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The Role of Perinatal Depression in the Development of Co-occurring Aggression and Anxiety/Depression Problems

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Abstract

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Maternal depression is a well-established risk factor for a range of negative developmental outcomes in offspring, including internalizing and externalizing problems. However, the role of maternal depression in the development of co-occurring internalizing and externalizing problems is not well understood. Given the chronicity and severity of co-occurring child behavior problems, it is important to understand the contributing risk factors and mechanisms for these outcomes. Using a group-based trajectory modeling technique, the current study identified developmental trajectory groups that exhibited chronic co-occurring aggression and anxiety/depression problems, only chronic aggression problems, and only chronic anxiety/depression problems. Exposure to perinatal maternal depression (both prenatal and postnatal) was examined in relation to these distinct behavioral problem groups. Mediation and moderation paths were also tested to understand the mechanisms through which maternal depression may have its impact and to identify subgroups that might be at higher risk. Results revealed differential patterns in the risk and mechanisms of risk transmission from prenatal/postnatal depression to chronic co-occurring, chronic aggression, and chronic anxiety/depression problems. In particular, our results revealed direct associations between prenatal and postnatal maternal depression and co-occurring problems, and prenatal maternal depression and chronic aggression. In addition, we found support for the mediating roles of stressful family environments and negative maternal parenting. Implications of these findings on psychopathology research and prevention/intervention strategies were discussed.

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Introduction

Overview

Maternal depression is a well-established risk factor for a wide-range of negative child outcomes including various types of child psychopathology. However, relatively little is understood about the impact of maternal depression on the development of co-occurring internalizing and externalizing behavioral problems, despite the fact that rates of co-occurrence are high. The current study examined the effects of maternal depression on the development of co-occurring anxiety/depression and aggression across development in a longitudinal sample. The current study had two primary aims. The first aim was to identify developmental trajectory classes for offspring anxious/depressed and aggression problems separately, and dual trajectory classes for co-occurring anxious/depressed and aggression problems. The second aim was to examine whether maternal depression was associated with the trajectory classes for co-occurring anxiety/depression and aggression. The third aim was to further test theory-driven moderators and mediators in this risk process. The rationale and hypotheses underlying the second and third aims are based on the theoretical framework proposed by Goodman and Gotlib (1999) concerning the transmission of risk from depressed mothers to their offspring. The current study makes unique contributions to our understanding of the mechanisms through which perinatal maternal depression impacts co-occurring internalizing and externalizing problems versus either internalizing or externalizing problems considered separately.

Maternal Depression and Risk for Child Psychopathology

Depression in women is an important public health issue given its high lifetime prevalence of 6% to 17% in the general population (Kessler, 2006). Women are particularly at risk for depression during their childbearing ages (Meltzer-Brody, 2011). Depression in mothers is a well-documented risk factor for a range of adverse outcomes across child development. In child psychopathology, conditions marked by dysregulated mood or emotion, such as depression and anxiety, are often classified as internalizing disorders/problems, whereas conditions characterized by dysregulated behavior including hyperactivity, impulsivity, oppositionality, defiance, disruptiveness, aggression, and antisocial features are often referred to as externalizing disorders/problems (Achenbach & Edelbrock, 1978, 1984). Children of depressed mothers have been shown to be at increased risk for internalizing and externalizing behavior problems, when examined separately. For example, previous research shows that children with depressed mothers experience higher rates and more severe forms (e.g., earlier onset, greater functional impairments) of depression compared to children without depressed mothers and other groups (e.g., Hammen, 1991). In addition, the effects of maternal depression have been extended to other internalizing problems such as anxiety disorders, externalizing problems such as aggression, and a range of problems in affective, cognitive, interpersonal, and neurobiological functioning (Goodman & Tully, 2006).

In a recent meta-analysis conducted by Goodman et al. (2011) compared the strengths of the associations between maternal depression and several adverse child outcomes, including three psychopathology groups: internalizing behavioral problems, externalizing behavioral problems, and general psychopathology. A total of 399 independent effect sizes from193 studies between 1982 and 2009 were summarized in

this meta-analysis. The meta-analytic results suggested similar pooled effect sizes for internalizing and general psychopathology with weighted mean *r*s equal to .23 (95% CI: [.22, .24]) and .24 (95% CI: [.22, .26]) respectively, and a slightly smaller weighted mean *r* of .21 (95% CI: [.20, .22]) for externalizing behavioral problems. The effect sizes for maternal depression are considered small to medium as it only explains between 4.4% and 5.8% of the variance in behavioral problems, though significant between-study heterogeneities were reported for these effect sizes (Goodman et al., 2011).

It seems then that maternal depression presents as a risk for several different types of child psychopathology, including conditions that are theorized to be distinct from each other (e.g., internalizing versus externalizing). It is also important to note that the effect sizes for associations with maternal depression do not vary drastically by the type of behavioral problems, although tremendous heterogeneities are apparent across studies. These findings are consistent with the concept of multifinality in developmental psychopathology (Cicchetti and Rogosch, 1996). Nonetheless, an important question that remains unanswered is whether maternal depression confers risk through similar or different mechanisms and developmental pathways to child internalizing problems compared to externalizing problems or the co-occurrence of both internalizing and externalizing problems.

The developmental psychopathology approach calls for understanding the developmental processes by examining the mechanisms for risk transmission. For maternal depression specifically, Goodman and Gotlib (1999) proposed the Integrative Model for the Transmission of Risk to Children of Depressed Mothers, which suggests that the risk of maternal depression is transmitted to the offspring through several inter-

correlated mechanisms. The following section provides a brief review of the model, particularly the parts of the model that are closely relevant to the current study.

The Models of Risk

Goodman and Gotlib (1999) summarized the large body of literature on the risk of maternal depression for psychopathology and proposed a model primarily focused on the mechanisms for the transmission of risk from depressed mothers to their children. Mechanisms in this model are conceptualized as intervening or causal variables that mediate the effects of maternal depression on negative outcomes in children (Goodman and Gotlib, 1999; Goodman 2007). Four mechanisms were proposed and empirical support for each was evaluated: 1) "heritability of depression," 2) "innate dysfunctional neuroregulatory mechanisms," 3) "exposure to mother's negative and/or maladaptive cognition, behaviors, and affect," and 4) "exposure to a stressful environment" (Goodman & Gotlib, 1999, pp. 461). The first two mechanisms are genetic and biological processes or vulnerabilities to depression that children may inherit from their depressed mothers or acquire before birth. Mechanisms 3 and 4 are most pertinent to the current investigation, as our study was focused primarily on social and familial risk factors associated with maternal depression.

Mechanism 3 postulates that depressed mothers may expose their children to negative cognitions, behavior, and affect, which may contribute to problematic parenting and influence the children's social and cognitive development; children may acquire and display cognition, behavior, and affect exhibited in their mothers through modeling or social learning. Indeed, there is sufficient evidence showing that children are exposed to their mothers' negative cognitive styles and self-perceptions (e.g., attend to negative

events, internal, stable, and global attribution style for negative events), depressive behaviors (e.g., more negative content and behavior in interaction with others), and affect (e.g., sad and irritable affect). In the current study, we examined whether low levels of maternal positive attitude towards the infant and low levels of maternal warmth and affection mediate the risk of maternal depression for the development of co-occurring anxiety/depression and aggression.

Mechanism 4 entails the family context for children with depressed mothers, particularly the stress that is associated with the mother's depression. The research on stress generation theory has shown that depressed adults may experience more stressful life events, particularly dependent stress (e.g., interpersonal stressors), as a result of their depression (Hammen, 1991). Therefore, it was proposed that children's exposure to the family environment, which may contain high numbers of stressors (e.g., marital discord) that are associated with mother's depression, might mediate the negative impact of maternal depression on children's behavioral outcomes. As reviewed in Goodman and Gotlib (1999), there is strong evidence showing that marital discord is associated with depression. Marital interactions for depressed people are characterized by negativity, hostility, conflict, and tension, and marital discord is associated with children's adverse behavioral outcomes. Poverty is another factor that has been associated with maternal depression and increased stress exposure in children at risk (Goodman & Gotlib, 1999). In the current study, marital discord and poverty, indicated by low maternal dyadic satisfaction and low family income respectively, were tested as potential mediators of the association between maternal depression and offspring anxiety/depression and aggression.

Goodman and Gotlib (1999) also proposed several moderators, including fathers' mental health and availability, timing and course of maternal depression, as well as child characteristics such as gender, intellectual/social/cognitive skills, and temperament. In this study we examined both prenatal and early postnatal maternal depression to test potential differential effects of timing. We also examined several possible moderators of the associations between maternal depression and various child behavioral outcomes. Identifying moderators of these relationships allows us to understand which groups of children are more or less at risk, and what factors influence the strength of the association in the direction of either increasing or decreasing the negative impact of maternal depression. In the current study, we have information on father's psychopathology, child gender, and child's verbal ability, and we tested whether these proposed variables moderate the of maternal depression on co-occurring or pure anxious/depressed and aggressive behavioral problems.

Goodman and Gotlib's model (1999) examined associations between maternal depression and "childhood or adolescent depression" or "other child disorders." The specificity of the hypothesized mechanisms in terms of the prediction of particular child outcomes is largely unknown. The extant literature on the topic of maternal depression rarely differentiates co-occurring versus single behavior problem groups. Specifically, when internalizing problems are studied as a negative outcome of maternal depression, it is unclear whether these children also exhibit externalizing problems and vise versa. The primary goal of the present study was to test whether the Goodman and Gotlib (1999) model predicts co-occurring anxiety/depression and aggression in offspring, and in particular whether the hypothesized mediators and moderators are more relevant in the

case of these co-occurring behavior problems or in the case of pure problem outcomes. This particular theoretical model was chosen because preliminary evidence suggests that mother depression predicts to a trajectory of co-occurring internalizing and externalizing problems across early development, as well as to the development of pure problem outcomes (Fanti and Henrich, 2010). However, a study of the mechanisms for these risks and the potential moderators for these detrimental outcomes is lacking. If the mediators and moderators suggested by Goodman and Gotlib (1999) are relevant to specific types of outcomes, they might suggest potential points of prevention and early intervention for high-risk children.

Co-occurrence between Internalizing and Externalizing Problems

The internalizing-externalizing behavior distinction has received extensive empirical support by early factor analytic studies conducted using child behavior checklists (Achenbach & Edelbrock, 1978; Achenbach, 1966) and more recent studies examining the latent structure of common DSM disorders in childhood (Cosgrove et al., 2011; Hewitt et al., 1997; Lahey et al., 2004). Although internalizing and externalizing problems are conceptualized and empirically supported as distinct dimensions, moderate correlations between the two latent dimensions have been consistently reported in both the adult (e.g., $r = .51$) and child (e.g., $r = .66$) psychopathology literature (Krueger, Caspi, Moffitt, & Silva, 1998; Krueger, 1999; Lahey et al., 2004). In fact, at the disorder level, this phenomenon is often referred to as the problem of *comorbidity* (i.e., the overlap across mental disorders defined by the current diagnostic systems), and specifically in this case *heterotypic comorbidity,* the comorbidity between disorders from different diagnostic groupings such as depression and conduct disorder (Angold,

Costello, & Erkanli, 1999). In the present study, we focused on the co-occurrence between internalizing and externalizing behavior problems (i.e., anxiety/depression and aggression). The term *co-occurrence* is used here to refer to the simultaneous presence of both internalizing and externalizing symptomatology in one individual (Kaplan, Crawford, Cantell, Kooistra, & Dewey, 2006). The term comorbidity is generally avoided because of the criticisms and controversies in the extant literature around what the term entails (Kaplan et al., 2006; Lilienfeld, 2003).

The co-occurrence between internalizing and externalizing disorders or problems is highly prevalent across the life-course and is found in both clinical and community samples (Gilliom & Shaw, 2004; Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). The co-occurrence between internalizing and externalizing problems may also be present early in childhood and as such, it can place children at risk for developing school and peer problems, negative parent-child interactions, subsequent internalizing and/or externalizing psychopathology, and negative developmental outcomes (Gilliom & Shaw, 2004; Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003; Kovacs & Devlin, 1998; Moffitt, 1993). Some evidence has indeed suggested that children with co-occurring problems tend to have more severe and chronic symptomatology (Kovacs & Devlin, 1998; Newman, Moffitt, Caspi, & Silva, 1998; Nottelmann & Jensen, 1995; Youngstrom, Findling, & Calabrese, 2003) and generally worse developmental outcomes than those with pure problems (see review in Nottelmann & Jensen, 1995). However, other studies suggest that these combinations of co-occurring disorders may lead to less severe outcomes. For example, low levels of aggression may alleviate certain adverse outcomes associated with depression, and lead to lowered risk for major depression, and relatively

better academic adjustment outcomes (Harrington, Rutter, & Fombonne, 1996). Similarly, some studies have shown that children with co-occurring anxiety and conduct problems exhibited less deviant behaviors than those with "pure" conduct problems (Walker et al., 1991). In one study, scores on the CBCL anxiety/depression scale were found to moderate treatment outcomes among school-age children with externalizing problems (e.g., conduct problems, oppositional defiant problems), such that having higher levels of anxiety/depression predicted a greater reduction in the levels of externalizing behaviors (Beauchaine, Webster-Stratton, & Reid, 2005). Given these contradictory findings concerning long-term outcomes for co-occurring behavior problems, it is also important to take into account the chronicity of behavior problems across development. In the current study, we did so by examining growth patterns of behavior problems across development from age 5 to 20 years.

Anxiety and depression often co-occur with externalizing problems in children (Angold & Costello, 1993). In a review of general population studies, 7.2 - 83.3% of depressed youth and 5.9 - 69.2% of youths with anxiety also had Conduct Disorder (CD) or oppositional defiant disorder (ODD); among youths with CD/ODD, 2.2 - 45.9% and 7.1 - 55.3% had depression and anxiety, respectively within one-year time frame (see Table 3 in Angold et al., 1999). Not only are anxiety and depression both highly likely to co-occur with externalizing problems, the co-occurrence represents a complex condition that is not well understood. As mentioned above, it is unclear whether co-occurring anxiety or depression and externalizing problems are associated with worse or better long term outcomes, as evidence exists for both. Further, it has also been suggested that gender may be an important factor in developing co-occurring anxiety or depression

among children with conduct problems (Loeber & Farrington, 2000). Specifically, the risks for developing anxiety or depression are much higher among girls with conduct problems than those without (Zoccolillo, 1992). In general, the nature of the heterotypic co-occurrence between anxiety/depression and conduct problems and the role of gender in this co-occurrence need further study.

Indeed, the lack of understanding does not just pertain to anxiety/depression and conduct problems; to a larger extent, it is not well understood how and why internalizing and externalizing disorders or problems co-occur despite the well-documented prevalence. Several authors have delineated the causes for the co-occurrence of disorders across and within diagnostic groupings, and among the proposed causes are methodological artifacts (e.g., shared diagnostic criteria), flaws in the conceptualization of psychopathology, and substantive reasons such as shared etiology (Angold et al., 1999; Lilienfeld, 2003). At this point, there is a general consensus that the co-occurrence of disorders is a "real" phenomenon that requires substantive explanations and further study. Some researchers argue that co-occurring disorders might represent meaningful syndromes (e.g., Angold & Costello, 1992; Lilienfeld, 2003; Kovacs, Paulauskas, Gatsonis, & Richards, 1988; Seligman & Ollendick, 1998). Others have looked into common underlying causal factors such as shared genetic influences or higher-order personality/temperament dimensions (e.g., negative emotionality, behavioral inhibition; see review in Lilienfeld, 2003). Indeed, a better understanding of the true meaning of "comorbidity" and its causes, development, and consequences would add an important piece to our understanding of the development of psychopathology. The majority of the extant research on psychopathology, however, has focused exclusively on single

disorders, by either excluding individuals with co-occurring disorders or overlooking the co-occurring symptoms that are present in the study (Jensen, 2003; discussed in Oland & Shaw, 2005; Seligman & Ollendick, 1998). As a result, there is a paucity of research on the etiology of co-occurring disorders. The distinction between "pure" versus co-occurring psychopathology remains unclear, as do the reasons why some children only display "pure" disorders but others develop co-occurring problems. Clinically, little is written on the guidelines for treating "comorbid" disorders, despite the fact that "comorbidity" is acknowledged as a common clinical reality rather than exception. Therefore, more research is needed to examine the numerous unresolved questions about co-occurring disorders.

Modeling Developmental Trajectories

Modeling developmental trajectories and identifying trajectory groups using group-based approaches have been appealing to clinical researchers, and such applications are increasingly popular (Nagin, 2010). A developmental trajectory describes the change over time of a behavioral outcome of interest. Trajectory groups refer to clusters of individuals that follow similar developmental trajectories of a behavioral outcome over time or age. Two group-based methods are often applied to address clinical research questions about developmental trajectories. One is Latent Class Growth Analysis (LCGA), alternatively called Group Based Trajectory Modeling (GBTM), which was developed by Nagin and colleagues (Nagin, 2001, 2005). The other is Growth Mixture Modeling (GMM), developed by Muthen and colleagues (2001). LCGA uses a finite number of trajectory groups to approximate the unknown continuous distribution of trajectories across individuals. Groups in LCGA are conceptualized as

clusters of individuals who follow approximately the same developmental trajectory and are technically specified without within-group variability. In GMM, however, groups are conceptualized as subpopulations of individuals that literally follow different growth curves, with random-effects or within-group variability included in the model. Despite these important distinctions (see detailed comparison in Nagin 2005, 2010; Muthen 2008, 2010), both group-based methods share the same ultimate goal of identifying individuals that follow distinct developmental pathways. Nonetheless, it has been argued that LCGA may be more suitable for clinical research questions, as developmental trajectory groups of behavioral outcomes are unlikely to reflect fundamentally discrete groups from distinct subpopulations (Nagin, 2010). For the purpose of substantive review, the following section focuses on studies that have adopted either LCGA or GMM methods to study the development of behavioral problems over time. However, the current study adopted LCGA in identifying developmental trajectory groups of anxiety/depression and aggression across childhood and into early adulthood.

A few recent studies have examined the developmental trajectories of "pure" and co-occurring internalizing and externalizing problems, applying group-based methods in longitudinal samples. Methodologically, these recent studies on dual trajectories of childhood disorders have moved the field beyond documenting the prevalence of co-occurring disorders, by examining the cross-sectional correlates of co-occurring disorders, and/or documenting the growth curve of single disorder or symptom dimensions. Substantively, more intriguing research questions can be asked with group-based methods, for example, how many trajectories classes underlie the co-occurrence of internalizing and externalizing problems across development, what are the shapes and

levels of trajectory classes, what are the antecedents or risk factors as well as outcomes for co-occurring problems versus pure problems, and whether co-occurring problems are meaningful syndromes as some authors (Angold et al., 1999; Angold & Costello, 1992) have suggested. These questions have important implications for the conceptualization and classification of psychopathology as well as for prevention and intervention (e.g., early identification of high risk children, guidelines for dealing with children with more than one diagnosis).

Several researchers have examined the heterotypic co-occurrence of depression and conduct problems (CP) or delinquency during adolescence (Chen & Simons-Morton, 2009; Diamantopoulou, Verhulst, & van der Ende, 2011; Reinke, Eddy, Dishion, & Reid, 2012). For example, Chen and Simons-Morton (2009) identified small proportions of the samples (8.8% of the boys and 3.7% girls) self-reporting high depression and CP over time in a school-based, non-clinical, longitudinal sample of 2,453 adolescents followed from 6 to 9th grade. A gender difference was reported for co-occurring CP among depressed children such that nearly half (42.9%) of the boys who were in the high depression trajectory were also in the high CP trajectory, compared to only 10.2% for girls. The prevalence rates for high depression among children of high CP trajectories were comparable across genders (~6%). Compared with the other groups, the co-occurring depression and CP group had significantly more problem peers, higher family discord, less parent knowledge, and poorer social competence. The co-occurrence group also had significantly poorer academic outcomes than the low problem group at the endpoint of the study, but did not significantly differ from the pure problem groups on these outcomes (Chen & Simons-Morton, 2009).

In contrast, in a similar investigation of gender differences in the co-occurrence of depressive symptoms and delinquency, Diamantopoulou et al. (2011) found that girls were more likely than boys to follow trajectories of self-reported high-level, co-occurring depression and delinquency across adolescence (age ranges 11-18 years) in a combined sample of multiple birth cohorts (total *n* = 1,423). Two co-occurring problem groups were identified, namely increasing-depression-high-delinquency (8% of the boys and 29% of the girls) and decreasing-depression-high-delinquency (24% of the boys and 9% of girls) groups. The increasing-high group membership was predicted by childhood aggression in boys and childhood depression in girls; the decreasing-high group membership was predicted by childhood depression and delinquency in boys and childhood depression in girls. These co-occurrence groups were generally associated with worse adult outcomes in terms of continued behavioral problems and low self-esteem (Diamantopoulou et al., 2011).

Reinke et al. (2012) investigated the co-occurrence between disruptive behavioral problems and depressive symptoms in a school-based sample (*n* = 361) of adolescents from neighborhoods with elevated rates of delinquency followed from grades 5 to10. Consistent with Chen and Simons-Morton's (2009) findings, this study identified only a small co-occurrence of high-level problems that lasted across development (1%), but found that depression increased the risk for disruptive behavior problems and vice versa. Of note, Reinke et al. (2012) reported that gender did not significantly predict class membership, which is contrary to the two previous studies (Chen & Simons-Morton, 2009; Diamantopoulou et al., 2011). This inconsistent finding may have been due to a small sample size and lack of statistical power. This study also reported more negative

adulthood outcomes for the co-occurring problem classes compared to low problem classes in terms of police arrest, substance use, mental health diagnoses, and subsequent depression and disruptive behaviors (pure problem classes were not yielded or examined; Reinke et al., 2012).

Only one study to date has examined the co-occurrence of the Child Behavior Checklist (CBCL; Achenbach, 1991) internalizing and externalizing problems in childhood using a group-based modeling approach. In this study, Fanti and Henrich (2010) used LCGA to examine the co-occurrence between total CBCL internalizing and externalizing problems in a community sample (n = 1,232) followed from 2 to 11 years of age. This study identified several trajectories for internalizing (i.e., low, moderate, high) and externalizing problems (i.e., low, moderate desister, moderate, high desister, chronic) as well as co-occurring problems (e.g., chronic co-occurring, pure internalizing, pure chronic externalizing, low risk, and 7 more classes). It also compared co-occurring, pure, and low problem classes in terms of early childhood risk factors such as temperament, early cognitive abilities, early home environment (e.g., enriched and positive home environment), and maternal postpartum depression. Overall, this study found that co-occurring and pure problem classes were not differentiated by the individual and contextual risk factors tested, suggesting shared etiological factors and multifinality (i.e., the same etiology leading to different developmental outcomes). Co-occurring and pure problem classes were also associated with similarly high levels of negative outcomes such as delinquency and risky behaviors, providing evidence for equifinality (i.e., the same outcome resulting from multiple predictors or developmental paths).

The few studies that examined the pattern and etiology of co-occurring internalizing/depression and externalizing/aggression thus far have focused mostly on the adolescent phase of development. No prior studies have examined the developmental trajectories for co-occurring problems across long periods of development, i.e., from childhood to adolescence to early adulthood. The current study has the advantage of conducting group-based modeling of change across development in a birth cohort with data collected at multiple waves across development. The outcome variables of interest (i.e., different types of behavioral problems) were collected from mother reports on widely used rating scales (i.e., CBCL) at age 5, 15, and 20, spanning from early childhood, to mid-adolescence, to early adulthood. Data on a rich set of early risk factors were collected at multiple time points during pregnancy, at birth, and during infancy. These variables include well-established risk factors for psychopathology such as maternal depression, early biological risk factors such as birth weight, pregnancy complications, and prenatal exposure, and family contextual variables such as stressors, social economic status, and parental marital satisfaction. The extensive data collection efforts at multiple waves spanning from pregnancy to early adulthood enable us to temporally separate early risk factors during prenatal and perinatal periods from subsequent behavioral problems during childhood, adolescence, and adulthood.

The studies reviewed above largely applied a multiple risk factor approach when examining the predictors for developmental trajectory classes. The risk factors identified in these studies for (but not necessarily specific to) the co-occurring internalizing and externalizing problems included problem peers, low levels of child social competence, parent-child conflict, previous behavioral problems, early difficult temperament, early

poor cognitive skills, early negative home environments, and maternal postpartum depression. Most relevant to the current study, maternal postpartum depression was a significant risk factor for co-occurring internalizing and externalizing problems as well as pure internalizing or externalizing problems across early childhood (Fanti & Henrich, 2010). Though these studies documented whether certain precursor factors or events were associated with membership in the co-occurring problem class, they did not examine the mechanisms for these associations, or how these risk factors acted together to place children on developmental pathways to negative outcomes. To build upon previous research findings and address the gaps in the literature, we examined the mechanisms for the association between maternal depression and child co-occurring aggression and anxiety/depression within a developmental framework suggested by Goodman and Gotlib's (1999) model. We tested specific hypotheses about the mechanisms of associations that were derived from the model as outlined in detail below.

The Present Study

The current study aimed to examine the impact of maternal depression on the development of co-occurring versus pure anxiety/depression and aggression from early childhood, to mid-adolescence, to adulthood in a longitudinal sample. Group-based methods were used to identify developmental trajectory classes for co-occurring or pure behavioral problems. Not only did the current investigation aim to identify underlying trajectory classes (specific aim 1), the primary goal for the current investigation was to understand the role of maternal depression in the development of co-occurring problems, especially *why* and *how* children are at risk for developing co-occurring behavioral problems versus one single type of behavioral problem during the developmental course

from childhood to young adulthood (specific aims 2 and 3). Unlike previous studies that tested multiple risk factors for developmental trajectory classes of behavior problems without a theoretical model, our study focused primarily on maternal depression as a risk factor, and applied the developmental risk model of Goodman and Gotlib (1999) to examine mechanisms that might explain the transmission of risk from depressed mothers to their children. The strengths of the current study include the use of a longitudinal sample with repeated measures, the adoption of a person-oriented, group-based approach to examine clinically interesting questions about development of psychopathology, and the focus on predictors of trajectory classes within a theoretical framework. The specific aims and hypotheses for the current study are as follows:

Specific Aim 1. Determine the developmental trajectory classes of anxiety/depression and aggression related behavioral problems, both individually and together, from childhood, to adolescence, to early adulthood.

Hypothesis 1. Based on previous studies of the developmental trajectories of behavioral problems in children and adolescents (e.g., Fanti & Henrich, 2010; Reinke et al., 2012), we hypothesize that we will identify: 1) a small group of individuals exhibiting high-levels of both internalizing and externalizing behavioral problems over time (i.e., a group with chronic co-occurring problems); 2) two small groups with individuals exhibiting only one type of behavioral problems over time (i.e., chronic internalizing or externalizing only group); and 3) a large group, containing the majority of the sample, exhibiting low levels of both types of behavioral problems over time (i.e., low problem group).

Specific Aim 2. Examine whether the trajectory class memberships (e.g., co-occurring vs. pure vs. low problem groups) identified in Aim 1 are differentially predicted by maternal depression in early childhood.

Hypothesis 2. Based on the well-established association between maternal depression and negative behavioral outcomes, we predicted significant main effects of maternal depression on trajectory class memberships. Specifically, because prior evidence shows that maternal depression may be more strongly associated with general psychopathology and internalizing problems than with externalizing problems, we expected to find that maternal depression would be more strongly associated with the co-occurring problem class than the pure externalizing problem class.

Specific Aim 3. Our third aim was to test whether family factors (e.g., mother's marital satisfaction, poverty) and characteristics of the early mother-child relationship *mediate* the relationship between maternal depression and trajectory class memberships as well as whether child gender, child cognitive ability, and father psychopathology *moderate* this relationship.

Hypothesis 3. Based on Goodman and Gotlib's (1999) model, we hypothesized that maternal negative attitude, low maternal warmth, marital discord, and poverty would mediate the risk of maternal depression for the development of co-occurring anxiety/depression and aggression, and that father's psychopathology, child gender, and child's verbal ability would moderate the association between maternal depression and behavioral trajectory class memberships. We predicted that children of depressed mothers would be more likely to be in the co-occurring problem group if paternal psychopathology was also present, and if the child had lower verbal abilities. Since the

findings for gender differences were in opposite directions in the previous studies (Chen & Simons-Morton, 2009; Diamantopoulou et al., 2011), the specific direction of gender moderating effects was not hypothesized.

Methods

Participants and Procedures

Participants in the current study were drawn from the Mater University Study of Pregnancy (MUSP), a birth cohort study of 7,223 mothers and their offspring born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital in Brisbane, Queensland, Australia (Keeping et al., 1989). The original MUSP aimed to examine psychosocial factors and children's health and development. 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; refers to as the "initial cohort" from here forward) 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).

Fifteen years after birth, a subset of the mother-offspring pairs from the initial cohort were recruited, oversampled for maternal depression as assessed by the Delusions-Symptoms State Inventory (DSSI; Bedford & Foulds, 1977) collected at four time points between pregnancy and child's age 5 years as described above. These DSSI scores were used to identify the level and frequency of elevated depression using specific algorithms (see details in Hammen, Brennan, & Shih, 2004). Nine hundred ninety-one families met

inclusion criteria based on the mothers' DSSI scores. Among these 991 families, 815 families consented and were included (82%), 103 families did not give consent, 68 families could not be reached, and 4 families did not participate because of the child's disability or death (Hammen et al., 2004). Participants in the 15-year follow-up study included 815 mothers (mean age = 41 years, 76.8% were currently married or cohabiting), and their targeted adolescent child (412 males and 403 females, mean age = 15.2 years, *SD* = 0. 29). Fathers were included if available. 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).

The high risk sample of families (*n* = 815) were re-contacted at child age 20 years, at which time 705 families were located, consented, and participated in the follow-up study. Compared to the age 15 sample participants who were not followed up, youth participants in the 20-year follow-up were more likely to be female and from higher income families. Respondents and non-respondents were not significantly different in terms of maternal or youth depression status (reported in Hammen, Brennan, & Le Brocque, 2011).

The participants included in the current analyses are those who participated in the age 15 follow-up (*n* = 815). This sample has 49.5% female, 92.3% Caucasian-descendent, 4.0% Asian-descendent, 2.0% Maori/Islander, and 1.9% Aboriginal. 38.2% of the participants have one sibling, 33.7% have two siblings, and 28.1% have more than three siblings. The mean age for the mother at childbirth was 25.53 (*SD* = 5.09). The mean level of parent education was Grade 10 (equivalent to high school graduation in the

US). The median family income was in the range of 15,600-20,799 Australian dollars per year.

Measures

Behavioral problems (Repeated-Measured Outcome Variables)

Age 5 Years. Behavioral problems at age 5 years were measured using selected items from the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1981, 1983). Mothers rated each item on a 3-point scale (*often*, *sometimes*, *never*). The CBCL is used to assess behavioral problems in children between 4 and 18 years old. The modified version of the CBCL used in the current study contains 33 of the 113 items from the original CBCL. Items that were excluded were those that have low base rates at age 5. Factor analysis of the modified CBCL yielded three broad dimensions (i.e. internalizing, externalizing, thought problems) that were identified in the full CBCL (Achenbach, 1991). The three subscale scores achieved acceptable internal consistency (alphas = .76, .75, and .84, for internalizing problems, social/attentional/thought problems, and externalizing problems, respectively) in the initial cohort with 5,342 mother-child dyads at age 5 (see Bor et al., 1997; Williams et al., 1998). This modified version of CBCL was also validated against the full CBCL using mother report. The externalizing, internalizing, and thought problem subscale scores across the two CBCL versions were highly correlated with r = .94, .89, and .96 respectively. The sensitivity and specificity of the modified CBCL for identifying children with elevated ratings on the full CBCL were satisfactory (see details in Williams et al., 1998).

Age 15. Behavioral problems during adolescence were assessed at the 15-year follow-up using mother report of the full CBCL (Achenbach, 1991). Mothers rated each

CBCL item on a 3-point scale ranging from 0 (*not true*), 1 (*sometimes true*), to 2 (*often true*) in terms of how well the item described the child.

Age 20. Behavioral problems during early adulthood were assessed at the 20-year follow-up, during which the mothers completed the Adult Behavior Checklist (ABCL; Achenbach & Rescorla, 2003). The current study only used the age-15 CBCL and age-20 ABCL items that were selected in the modified CBCL used at 5-year follow-up to maintain consistency in measures across development.

The CBCL/ABCL items from the *anxious/depressed* scale included in the current study (available at age 5, 15, *and* 20) are: "cries a lot," "feels worthless or inferior," "feels too guilty," "nervous, high-strung, or tense," "too fearful or anxious," and "worries." The CBCL/ABCL items from the *aggression* scale are: "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 internal consistency coefficients for the scores of anxious/depressive and aggressive behaviors are satisfactory (reported in Table 1)

For the analysis of the three repeated measures of aggression, there were 596 cases with data at all three time points, 200 cases with data at two time points (72.5% were missing data at the last time point), and 19 cases with data at only one time point. For the analysis of the three repeated measures of anxious/depressed behaviors, there were 597 cases with data at all three time points, 194 cases with data at two time points (71.6% were missing data at the last time point), and 26 cases with data at only one time point.

Perinatal Maternal Depression (Primary Explanatory Variables)

The Delusions Symptoms States Inventory (DSSI; Bedford & Foulds, 1978) depression subscale was used to measure maternal depressive symptoms at each assessment phase of the study (i.e., during pregnancy, 3 to 4 days after childbirth, 6 months, 5 years). The DSSI was developed to identify individuals with clinically significant psychopathology in the community by detecting signs and symptoms of mental disorders that impair individuals' social functioning. The DSSI has been extensively validated and used in the literature (e.g., Bagshaw, 1977; Bedford & Deary, 1999). The depression subscale 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*). The internal consistency coefficients of the DSSI depression scores across the four time points range from .71 to .81 (reported in Brennan et al., 2000). In the current study, DSSI depression scores at birth and 6 months were averaged to create a postnatal depression score. The DSSI score obtained during pregnancy was measured as a separate indicator of prenatal maternal depression. It was obtained from the mothers at their first prenatal clinic visit (mean gestation = 18.14 weeks, *SD* = 5.85).

Mediators

Two groups of mediators were assessed in this study - family related factors and mother child interaction factors. Family-related factors included maternal dyadic satisfaction and low family income (poverty). These family-related factors generally fall under *mechanism 4* (exposure to stressful environment) in Goodman and Gotlib's

(Goodman & Gotlib, 1999) model. Mother-child interaction factors included maternal feelings toward her infant and maternal parenting and discipline style in early childhood. These factors fall under *mechanism 3* (exposure to mother's negative and/or maladaptive cognitions, behaviors, and affect) in Goodman and Gotlib's (Goodman & Gotlib, 1999) model.

Family-related factors

Family income (poverty). At each assessment phase, family income was reported on the following scale of Australian Dollars: 1 = $0-5,199; 2 = $5,200-$10,399; 3 = $10,400 - $15,599; 4 = $15,600-$20,799; 5 = $20,800-$25,999; and 6 = $26,000 or more. Family income reported by the mother when the child was six months and 5 years of age was used as a measure of family stress associated with poverty.

Maternal dyadic satisfaction. Maternal dyadic satisfaction and adjustment were assessed using the dyadic satisfaction items from the Spanier Dyadic Adjustment Scale (DAS; Spanier, 1976) at each of four assessment time points (i.e., during pregnancy, at birth, and 6 months and 5 years after birth). The DAS is a 32-item scale, and the current study employed the eight items that comprise the *dyadic satisfaction* subscale. Scores on this subscale have satisfactory internal consistency (alphas = .81 to .86 across the four time points) in the initial cohort (n = 5,342). Continuous dyadic satisfaction scores at 6 months and 5 years were tested as potential mediators.

Maternal positive attitude and warmth

Maternal positive attitude towards the infant. At the infant 6-month follow-up, maternal subjective feelings towards child care were assessed by asking the mothers to what degree they agreed with the following statements: "caring for my baby is very

satisfying," "I feel so angry that sometimes I could smack my baby," "my baby makes me too tired," "my baby is so good I hardly know he/she is there," "I sometimes feel like hitting my baby," "I feel fed up looking after my baby all day." Mothers rated these items on a 5-point scale, from *Strongly Agree*, *Agree*, *Neutral*, *Disagree*, to *Strongly Disagree*. Item 1 and 4 were reverse coded, and the items on this scale achieved acceptable internal reliability (*alpha* = .75).

Maternal warmth towards the child. At the 5-year follow-up, mothers were asked about their displays of affectionate behaviors (e.g., at times too busy to comfort the child and not wanting to cuddle the child) toward their 5-year-old child. Scores on these two items were reverse coded and then averaged for data analyses.

Moderators

We tested whether father's psychopathology, child gender, or child cognitive skills moderated the relationship between maternal depression and trajectory classes of co-occurring behavioral problems.

Father psychopathology. A subsample of the fathers (*n* = 522) provided self-report data on their history of Axis I disorders on the SCID at the age 15 follow up. In addition, all mothers were queried about father psychopathology at the age 15 follow up on the Family History Research Diagnostic Criteria (FHRDC) interview (Andreasen, Endicott, Spitzer, & Winokur, 1977) in the cases where fathers did not get directly assessed by the SCID. Fathers in this sample were classified as having a lifetime history of Axis I psychopathology (*yes* or *no*) if they were classified as having lifetime psychiatric diagnosis by either self-reported SCID or mother-reported FHRDC. In our

sample, 463 (56.8%) children had no history of paternal psychopathology by age 15, while the fathers of 352 (43.2%) children had lifetime history of psychopathology.

Child gender. The targeted child's gender is a dichotomous moderator, males versus females.

Child cognitive functioning. At the 5-year follow-up, a subset of children (*n*=3,767 out of 5,342) were assessed directly by research assistants using the Peabody Picture Vocabulary Test- Revised (PPVT-R; Dunn & Dunn, 1981), a standardized measure of receptive language skills. Though some children did not complete the PPVT purely due to logistic reasons (e.g., scheduling), children with and without PPVT test did differ significantly on important variables, including maternal age at birth and maternal depression (reported in Brennan et al., 2000).

Potential confounds

The following variables were tested as potential confounds: maternal age at child's birth; marital status (coded as married to the child's father, married/cohabiting with another person, or not currently married or cohabiting from birth to 5 years); maternal substance use during pregnancy, including smoking, alcohol, and drug use (average daily consumptions of cigarettes, alcohol, coffee, tea, and marijuana during pregnancy were assessed at child birth through maternal self-report of behaviors during the last trimester); pregnancy outcome variables, including birth weight in grams and gestational age in weeks, obtained from medical records; and father and mother's antisocial behavior, measured by asking the mother whether she or her partner had ever been arrested. After testing for significant confounds (see Table 5), gestational age and maternal smoking during the last trimester were controlled for in regression-based

analyses. Descriptive statistics for gestational age are reported in Table 1. For maternal smoking, 61% (n = 499) of the sample never smoked during pregnancy, 6.4% (n = 52) rarely smoked, 3.9% (n = 32) some of the time, and 28.3% (n = 230) smoked most of the time.

Data Analysis Plan

The data analyses are grouped into three sections. First, latent class growth analysis (LCGA) was conducted to identify latent trajectory classes for each type of behavioral problem (i.e., anxiety/depression, aggression) as well as the dual trajectories for co-occurring behavioral problems (i.e., anxiety/depression *and* aggression). Second, multinominal logistic regression was used to examine maternal depression as a predictor of latent trajectory class memberships obtained from the dual trajectory LCGA analyses. Third, mediation analyses and moderation analyses were conducted to better understand the link between maternal depression and latent trajectory class memberships. All LCGA analyses were conducted in Mplus 7. Full information maximum likelihood estimation was used to retain children with incomplete data. Models were estimated using 500 random permutations of starting values, and rerun with different sets of starting values, to avoid local solution and check the stability of solution. Regression-based analyses were conducted in Stata 12.

Latent class growth analysis (LCGA)

Following the recommendations in the literature (e.g., Jung & Wickrama, 2008; Bengt Muthén, 2006), the initial step prior to conduct LCGA was to run a single-class, univariate latent growth-curve model to inspect the growth curve for each type of problem behavior assuming no underlying heterogeneity of the growth trajectory. To

account for the unequal intervals of the repeated measurements, the first two loadings for the slope factor for observed variables measured at age five, 15, and 20 (i.e., mother ratings of anxious/depressed and aggression items on CBCL) were fixed at zero and two, and the third loading was set free to allow for non-linearity (i.e., latent basis model; Meredith & Tisak, 1990). This specification is the same for all the remaining growth models. The second step is to specify unconditional latent class models for each type of behavior problem.

Dual trajectory analyses. A dual trajectory LCGA model was specified (see *Figure 1*) to obtain the conditional probability of membership in each trajectory for one type of behavioral problem given the membership in each trajectory for another type of behavioral problem, as well as the joint probability of membership in trajectories across behavior problems.

Model selection procedure. Model comparison and selection followed the described procedures: 1) inspection of the graphs that show the shape and location of the different estimated class trajectories. Based on Nagin and Tremblay (2001), a more parsimonious model (i.e., with fewer distinct classes) is preferred when the exact number of classes is of low theoretical importance (e.g., no a prior hypothesis about the number of classes); 2) The average posterior probabilities of class membership and the entropy value were considered to check the precision of classification and the degree to which the classes were distinguishable. Based on Nagin (2005), average probabilities greater than or equal to .70 indicate satisfactory fit; based on Muthén (2000), an entropy value greater than .70 is satisfactory as it indicates clear classification and greater power to predict class membership; 3) Model fit statistics were evaluated using the Bayesian information

criterion (BIC) and the Lo, Mendel, Rubin (LMR) *p*-value. The Akaike information criterion (AIC) values were also reported. As recommended of Nylund et al. (2007, 33-34), the model with a low BIC value and a significant LMR *p*-value comparing the *k* and the *k*-1 class model should be chosen as the better fitting model. A non-significant *p*-value ($p > .05$) indicates that the *k*-1 class model is preferred over the *k* class model.

Multinomial logistic regression

After the categorical latent trajectory class variable was obtained from the dual trajectory LCGA analysis, multinominal logistical regression was used to examine whether maternal depression predicted the latent class variable. The regression coefficients indicate, for example, the expected change in the log-odds of being in the co-occurring problems class versus the low problem group for one-unit increase in the degree of maternal depression (indicated by the maternal self-reported DSSI scores). We also reported relative risk ratios (RRR), which was obtained by exponentiating the multinomial regression coefficient (logit) and is commonly interpreted as odds ratios (OR). RRR is interpreted as follows: for each one-unit change in the predictor, the relative risk ratio of the outcome relative to the referent group is expected to change by a factor of the parameter estimate, holding all other variables in the model constant (UCLA: Statistical Consulting Group, n.d.).

Mediation and moderation analyses. The conceptual model of the mediation and moderation analyses is illustrated in *Figure 2*. Multiple mediation analyses were conducted using Structural Equation Modeling (SEM) in Mplus 7. As recommended (MacKinnon, Fairchild, & Fritz, 2007), bootstrapping resampling procedures were used to obtain the standard errors and confidence intervals for the direct and indirect/mediated

effects. Bootstrap standard error and bias corrected bootstrapping confidence intervals along with *p*-values were reported for effects in mediation models.

For the evaluation of the SEM model fit, we reported the following goodness-of-fit indices: the χ^2 test statistic and its associated degrees-of-freedom (*df*) and *p* value, the Tucker-Lewis index (TLI), the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). Assessment of the adequacy of model fit was based on guidelines suggested in the literature, including: CFI and TLI greater than 0.95 for reasonably good fit (Hu & Bentler, 1999) and values between 0.90 - 0.95 for acceptable model fit (Bentler, 1990); RMSEA ≤ .08 for adequate fit and ≤ .05 for close fit (Browne & Cudeck, 1993). The fit of a single model was evaluated using the combination of CFI, TLI, and RMSEA, as each individual fit index has its limitations and no consensus has been reached regarding the use of a single fit index to evaluate the adequacy of model fit (Loehlin, 2004).

Results

Descriptive Statistics

Table 1 shows the descriptive statistics of continuous study variables. Table 2 shows the bivariate correlation matrix of the primary independent and dependent outcome variables.

Latent Growth Curve (LGC) model

Aggression.

Figure 3 shows that on average mother-rated aggression scores decreased across age 5, 15, and 20. The model fit statistics indicate excellent fit: $\chi^2 = 3.673$, $df = 2$, $p = .159$, $RMSEA = 0.032$ (90% CI: [0.000, 0.083]), $CFI = 0.993$, $TLI = 0.989$, and $SRMR =$

0.027. The unstandardized means for the Intercept and Slope latent factors were 5.184 (*SE* = 0.122, $p < .001$) and -1.438 (*SE* = 0.066, $p < .001$), respectively. The unstandardized variances for Intercept and Slope latent factors were 7.675 (*SE* = 0.709, $p < .001$) and 1.449 (*SE* = 0.221, $p < .001$), respectively. The covariance between Intercept and Slope was -2.083 (*SE* = 0.344, $p < .001$). These results suggest that there was significant inter-individual variability in terms of the initial status of aggression and the rate of change over time. The rate of change was negatively correlated with the initial levels of aggression, such that higher initial status was associated with lower rate of change.

Anxious/Depressed.

Figure 3 shows that on average there was a decrease across age 5, 15, and 20 for anxious/depressed behavior problems. The model fit statistics indicate acceptable fit: $\chi^2 = 9.175$, *df* = 2, $p = .010$, *RMSEA* = 0.066 (90% CI: [0.027, 0.112]), *CFI* = 0.975, *TLI* = 0.963, and *SRMR* = 0.035. The unstandardized means for the Intercept and Slope latent factors were 2.603 (*SE* = 0.076, $p < .001$) and -0.643 (*SE* = 0.038, $p < .001$), respectively. The unstandardized variances for Intercept and Slope latent factors were 1.568 (*SE* = 0.181, $p < .001$) and 0.079 (*SE* = 0.011, $p < .001$), respectively. The covariance estimate between Intercept and Slope was non-significant (*r* = -0.018, *SE* = 0.119, $p = .882$) and was thus fixed at 0 in the final model. Residual variance for age 15 anxious/depressed was also non-significant and thus fixed at 0 in the final model (as suggested by modification indices), while the residual variances for age 5 and 20 anxious/depressed were freely estimated. These results also suggest that there was significant inter-

individual variability in terms of the initial status of anxious/depressed behavioral problems and the rate of change over time.

Latent Class Growth Analysis

Latent class growth analysis (LCGA) was then conducted to explore the heterogeneity in the group developmental trajectories. The goals for LCGA were to identify different developmental trajectories, class probabilities, and the posterior probabilities of class memberships for all individuals.

Aggression.

One- to 5- class LCGA solutions were estimated for the single outcome aggression measured at age 5, 15, and 20. The choice of the number of classes was made based on a combination of indices including AIC, BIC, the Lo-Mendell-Rubin adjusted LRT test statistics, and entropy, as well as sample size considerations. Table 3 shows that as the number of classes increases the AIC and BIC, or Information Criteria (ICs), kept dropping while entropy values remain relatively the same. The entropy values were around 0.8, suggesting that the classes were well separated. The largest changes in ICs occur between 1- to 2-class models and between 2- to 3-class models, compared to the much smaller changes in ICs from 3- to 4- and 4- to 5-class models. The LMR adjusted LRT test had non-significant *p*-value for the 4-class model, suggesting that 3 classes are sufficient and that 4 classes were not necessary. Therefore, 2-class and 3-class solutions were compared based on the number of individuals in each class and visual inspection of the class trajectories. The 3-class solution identified a chronic problem group (3.2%, n = 26), a medium-maintaining problem group (20.1%, n = 164), and a low-decreasing normative group (76.7%, n = 625). The 2-class solution identified a chronic problem

group (12.1%, *n* = 99) and a low-decreasing normative group (87.9%, *n* = 716). The percentages and numbers are from final proportion and class counts based on individuals' most likely latent class patterns. Given that one of the primary goals for the current paper was to investigate co-occurring aggression and anxious/depressed problems, the number of individuals in the chronic problem group from the 3-class solution was too small (*n* = 26) to further break down in terms of the anxious/depressed problem classes. The 2-class solution was thus adopted for further analysis of dual trajectories of aggression and anxious/depressed problems.

Anxiety/Depression.

One- to 5-class LCGA solutions were also estimated for the anxious/depressed behavioral problems measured at age 5, 15, and 20. The ICs kept decreasing as the number of classes increased. The entropy values stayed around 0.9, suggesting that the classes were well separated. Similarly, the biggest increment of fit occurred between 1- to 2- and 2- to 3- class models. The LMR adjusted LRT test had significant p-value for the 4-class model, suggesting that 3 classes are sufficient. Two-class and 3-class solutions were then compared. The 3-class solution yielded a chronic problem group (2.9%, *n* = 24), a medium-increasing problem group (18.2%, *n* = 149), and a low-problem normative group (78.8%, *n* = 642). The 2-class solution yielded a chronic-increasing problem group (14.8%, *n* = 121) and a low-problem normative group (85.2%, *n* = 694). The 2-class solution was adopted for the dual trajectory analysis because of the small size of the chronic problem group (2.9%, *n* = 24) from 3-class solution.

Co-occurring Aggression and Anxious/Depressed

The co-occurrence of aggression and anxious/depressed problems was examined using parallel process LCGA. A dual trajectory model was fitted with all possible classes between 2-class aggression and 2-class anxious/depressed problems (2x2) estimated. This model identified a chronic co-occurring aggression and anxiety/depression group (10.6%, $n = 86$), a low anxious/depressed high aggression or pure aggression group (2.2%, $n = 18$), a high anxious/depressed low aggression or pure anxious/depressed group (5.6%, $n = 46$), and a low-problem normative group (81.6%, $n = 665$). Table 4 shows the class memberships for the four problem groups and the tabulation of the four groups by gender. *Figure 4* shows the plot of the four dual trajectory groups. The class assignment information based on each individual's most likely class membership was exported and used as the outcome variable for the remaining analyses.

Maternal Depression and Behavioral Problem Classes

The relations between maternal depression and the four behavioral problem classes were then tested using multinomial logistic regression. Separate multinomial regressions were conducted to predict class membership on the basis of mother's self-reported depression during pregnancy and the postpartum period (averaged between maternal DSSI depression score at birth and 6 months). Potential confounding variables were then tested in terms of their associations with the outcome variables using logistic regression and then tested for their associations with the predictors using correlation and simple linear regression (see Table 5). Based on these analyses, gestational age and maternal smoking were included as control variables in all analyses predicting to trajectory class outcomes.

Prenatal maternal depression.

Compared to the low-problem normative group, a one-unit increase in the score of prenatal maternal depression was associated with a 0.14 unit change in the log-odds of being in the chronic co-occurring problem group (*RRR* = 1.15, *p* = .049, 95% CI: [1.00, 1.33]), controlling for the effects of gestational age (*RRR* = 0.89, *p* = .090, 95% CI: [0.77, 1.02]) and maternal smoking during pregnancy (*RRR* = 1.17, *p* = .054, 95% CI: [0.98, 1.39]). One-unit increase in the score of prenatal maternal depression was also associated with a 0.34 unit change in the log-odds of being in the high aggression low anxious/depressed problem group (*RRR* = 1.41, *p* = .009, 95% CI: [1.09, 1.82]) relative to the low problem group, controlling for the effects of gestational age (*RRR* = 0.85, *p* = .222, 95% CI: [0.66, 1.10]) and maternal smoking during pregnancy (*RRR* = 1.30, *p* = .139, 95% CI: [0.92, 1.85]). Prenatal maternal depression was not associated with a higher chance of being in the high anxious/depressed low aggression problem group (*RRR* = 1.09, *p* = .363, 95% CI: [0.90, 1.32]) relative to the low problem normative group, controlling for gestational age and maternal smoking during pregnancy. Prenatal maternal depression also did not differentiate the high anxious/depressed low aggression group (*RRR* = 0.95, *p* = .648, 95% CI: [0.76, 1.19]) or the high aggression low anxious/depressed group (*RRR* = 1.22, *p* = .160, 95% CI: [0.92, 1.62]) from the chronic co-occurring group, controlling for gestational age and maternal smoking during pregnancy.

Postnatal depression.

Compared to the low-problem normative group, one-unit increase in the score of maternal *postnatal* depression was associated with a 0.18 unit change in the log-odds of being in the chronic co-occurring problem group (*RRR* = 1.20, *p* = .033, 95% CI: [1.01,

1.42]), controlling for the effects of gestational age (*RRR* = 0.90, *p* = .120, 95% CI: [0.78, 1.03]) and maternal smoking during pregnancy (*RRR* = 1.19, *p* = .054, 95% CI: [1.00, 1.41]). Postnatal depression did not differentiate the low aggression high anxious/depressed group (*RRR* = 1.14, *p* = .249, 95% CI: [0.91, 1.42]) or the high aggression low anxious/depressed group (*RRR* = 0.92, *p* = .683, 95% CI: [0.63, 1.36]) from the normative group, controlling for gestational age and maternal smoking during pregnancy. Postnatal depression also did not differentiate the low aggression high anxious/depressed group (*RRR* = 0.95, *p* = .705, 95% CI: [0.73, 1.24]) or the high aggression low anxious/depressed group (*RRR* = 0.77, *p* = .210, 95% CI: [0.51, 1.16]) from the chronic co-occurring group, controlling for gestational age and maternal smoking during pregnancy.

Mediation Analyses

Following the analyses of the main effects of perinatal depression, mediation analyses were conducted to test the mechanisms through which perinatal depression may lead to chronic co-occurring or pure problems. Recent advances in mediation analyses suggest that mediation should be tested both with and without the presence of main effects. Given this, we ran mediation analyses for the effects of prenatal and postnatal depression on all three outcomes: chronic co-occurring, pure aggression, and pure anxiety/depression.

Mediation analyses were conducted using structural equation modeling in Mplus. *Figure 5* shows the specification of the mediation model, which follows the serial multiple mediators model discussed in Hayes (2013, pp. 145). Three sets of mediators measured at child 6 months and 5 years were specified in this model, namely maternal

dyadic satisfaction, family income, and maternal positive attitude or warmth towards the child. Three indirect paths/effects were specified for each set of mediators: 1) a specific indirect effect through the mediator measured at 6 months, 2) a specific indirect effect through the mediator measured at 5 years, and 3) specific indirect effects through the mediators measured at both 6 months and 5 years. Prenatal and postnatal maternal depression were tested as the independent variables in separate models. In addition, each mediation model was tested using logistic regression for each binary outcome, namely chronic co-occurring problem, chronic pure aggression, and chronic pure anxiety/depression groups. The low problem group was the referent group in the logistic regression models for each chronic behavior problem outcome.

Chronic Co-occurring Problems

Prenatal depression. *Figure 6* shows the results from the mediation model for the effects of prenatal depression on the risk of being in the chronic co-occurring problems (n = 86) versus the low problems (n = 665) group. The model fit statistics suggested excellent fit: χ^2 = 25.471, df = 18, p = .113, *RMSEA* = 0.024 (90% CI: 0.000, 0.044), *CFI* = 0.987, and *TLI* = 0.969. The R^2 for this model was 16.1%. Path coefficient estimates are reported and significant mediators/paths/coefficients are marked in *Figure 6*, while the indirect effect estimates and bias correct bootstrap 95% CIs are reported here.

Maternal dyadic satisfaction *at 6 months* was a marginally significant mediator with a sum of indirect effects of 0.020 (*SE* = 0.011, 95% CI: [0.000, 0.043]). The specific indirect/mediated path through maternal dyadic satisfaction at 6 months, which is the indirect path in blue in *Figure 6*, was marginally significant with an estimate of 0.020 (*SE* = 0.012, 95% CI: [-0.001, 0.045]). The sum of indirect effects via maternal dyadic

satisfaction *at 5 years* was negligible with an estimate of 0.000 (*SE* = .007, 95% CI: [-0.016, 0.014]).

Family income *at 6 months* was a significant mediator with a sum of indirect effects of 0.022 (*SE* = 0.010, 95% CI: [0.007, 0.046]). The specific indirect path through family income at 6 months, which is the bolded indirect path in red in *Figure 6*, was significant with an estimate of 0.018 (*SE* = 0.010, 95% CI: [0.002, 0.042]). The sum of indirect effects via family income *at 5 years* was minimal with an estimate of 0.007 (*SE* = 0.006, 95% CI: [-0.001, 0.022]).

Maternal warmth at 5 years was a significant mediator with a sum of indirect effects of 0.015 (*SE* = 0.008, 95% CI: [0.003, 0.033]). The specific indirect path via *both* maternal negative attitude at 6 months *and* maternal warmth at 5 years, which is the bolded indirect path in green that goes through both mediators in *Figure 6*, was significant with an estimate of 0.004 (*SE* = 0.002, 95% CI: [0.001, 0.010]). The specific indirect effect via maternal warmth at 5 years, which is the bolded indirect path in green that only goes through maternal warmth in *Figure 6*, was significant with an estimate of 0.011 (*SE* = 0.006, 95% CI: [0.002, 0.027]). The sum of indirect effects via maternal attitude toward the infant at 6 months was minimal with an estimate of 0.001 (*SE* = 0.010, 95% CI: [-0.018, 0.020]).

Postnatal depression. *Figure 7* shows the results from the mediation model for the effects of postnatal depression on the risk of being in the chronic co-occurring problem groups as the outcome. This model yielded excellent fit: $\chi^2 = 25.252$, $df = 18$, $p = .118$, *RMSEA* = 0.024 (90% CI: 0.000, 0.044), *CFI* = 0.989, and *TLI* = 0.974. The R^2 estimate was 16.2%. Consistent with the mediation model for prenatal maternal

depression and the chronic co-occurring group, the effects of postnatal depression on chronic co-occurring problems were also mediated by maternal dyadic satisfaction and family income at 6 months, as well as low maternal positive attitude at 6 months and low maternal warmth at 5 years.

Maternal dyadic satisfaction at *6 months* was a marginally significant mediator with a sum of indirect effects of 0.039 (*SE* = 0.019, 95% CI: [0.000, 0.078]). The specific indirect path through maternal dyadic satisfaction at 6 months, which is the indirect path in blue in *Figure 7*, was marginally significant with an estimate of 0.039 (*SE* = 0.019, 95% CI: [0.000, 0.078]). The sum of indirect effects via maternal dyadic satisfaction at 5 years was negligible with an estimate of 0.000 (*SE* = 0.010, 95% CI: [-0.020, 0.019]).

Family income at 6 months was a significant mediator with a sum of indirect effects of 0.023 (*SE* = 0.011, 95% CI: [0.006, 0.049]). The specific indirect path through family income at 6 months, which is the bolded indirect path in red in *Figure 7*, was significant with an estimate of 0.019 (*SE* = 0.011, 95% CI: [0.002, 0.046]). The sum of indirect effects via family income *at 5 years* was minimal with an estimate of 0.007 (*SE* = 0.007, 95% CI: [-0.002, 0.025]).

Maternal warmth at 5 years was a significant mediator with a sum of indirect effects of 0.022 (*SE* = 0.011, 95% CI: [0.004, 0.045]). The specific indirect path via *both* maternal negative attitude at 6 months *and* maternal warmth at 5 years, which is the bolded indirect path in green that goes through both mediators in *Figure 7*, was significant with an estimate of 0.009 (*SE* = 0.004, 95% CI: [0.002, 0.019]). The specific indirect effect via maternal warmth at 5 years, which is the bolded indirect path in green that only goes through maternal warmth in *Figure 7*, was significant with an estimate of

0.013 (*SE* =0.008, 95% CI: [0.002, 0.032]). The sum of indirect effects via maternal attitude toward the infant at 6 months was minimal with an estimate of 0.000 (*SE* = 0.021, 95% CI: [-0.043, 0.039]).

Chronic Aggression

Prenatal depression. *Figure 8* shows the results from the mediation model for the effects of prenatal depression on the risk of being in the chronic aggression (*n* = 18) versus the low problems (*n* = 665) groups. The model fit statistics suggested excellent fit statistics: $\chi^2 = 25.506$, *df* = 18, *p* = .112, *RMSEA* = 0.024 (90% CI: 0.000, 0.044), *CFI* = 0.988, and *TLI* = 0.965. R^2 estimate was 19.7%. For this model, only the sum of indirect effects via maternal dyadic satisfaction at 5 years was significant (estimate = 0.032, *SE* = 0.017, 95% CI: [0.007, 0.074]), which was the sum of the specific indirect effect via maternal dyadic satisfaction at 6 months *and* at 5 years (estimate = 0.024, *SE* = 0.010, 95% CI: [0.007, 0.048]) and the specific indirect effect directly via maternal dyadic satisfaction at 5 years (estimate = 0.008, *SE* = 0.011, 95% CI: [-0.008, 0.036]). The direct/main effect of prenatal depression was still significant after accounting for the mediated paths.

Postnatal depression. *Figure 9* shows the results from the mediation model with the chronic aggression group as the outcome. The model fit statistics suggested excellent fit: $\chi^2 = 25.278$, *df* = 18, *p* = .117, *RMSEA* = 0.024 (90% CI: 0.000, 0.044), *CFI* = 0.988, and *TLI* = 0.972. R^2 estimate was 13.1%. Only the sum of indirect effects via dyadic satisfaction was significant (estimate = 0.038, *SE* = 0.020, 95% CI: [0.005, 0.091]), which included the specific indirect effect via maternal dyadic satisfaction at 6 months *and* 5 years (estimate = 0.041, *SE* = 0.019, 95% CI: [0.005, 0.082]) and the specific

indirect effect only via dyadic satisfaction at 5 years (estimate = -0.002, *SE* = 0.011, 95% CI: [-0.026, 0.017]). The sum of indirect effects was non-significant for family income at 6 months and 5 years as well as maternal negative attitude towards the baby at 6 months and maternal warmth at 5 years. It should be noted that there were no significant direct/main effects of postnatal depression on chronic aggression.

Chronic Anxiety/Depression

Prenatal depression. For the serial multiple mediator model with the chronic anxiety/depression group ($n = 46$), the model fit statistics also suggested excellent fit: $\chi^2 = 25.477$, $df = 18$, $p = .127$, $RMSEA = 0.024$ (90% CI: 0.000, 0.044), $CFI = 0.985$, and $TLI = 0.966$. R^2 estimate was 4.6%. None of the indirect effects were significant in this model.

Postnatal depression. For the serial multiple mediator model with high anxious/depressed low aggression problem group, the model fit statistics also suggested excellent fit: $\chi^2 = 25.247$, $df = 18$, $p = .118$, $RMSEA = 0.024$ (90% CI: 0.000, 0.044), $CFI = 0.988$, and $TLI = 0.974$. R^2 estimate was 5.2%. None of the indirect effects were significant in this model.

Moderation Analyses

The proposed moderators (i.e., child gender, father psychopathology, child cognitive skills) were tested using multinomial regression with interaction terms. Gestational age and maternal smoking during pregnancy were controlled for in the regression models with interaction terms entered for each moderator and independent variable combination.

Gender.

Child gender was not a significant predictor of being in the chronic co-occurring problem group (*RRR* = 1.09, *SE* = 0.25, *p* = .709, 95% CI: [0.70, 1.71]), pure aggression group (*RRR* = 0.83, *SE* = 0.40, *p* = .702, 95% CI: [0.32, 2.13]), or pure anxiety/depression group (*RRR* = 1.24, *SE* = 0.38, *p* = .485, 95% CI: [0.68, 2.26]).

The interaction term between *prenatal* maternal depression and gender was not significant for the chronic co-occurring problem group (*RRR* = 0.89, *SE* = 0.13, *p* = .435, 95% CI: [0.68, 1.18]), pure aggression group (*RRR* = 1.30, *SE* = 0.34, *p* = .313, 95% CI: [0.78, 2.16]), or pure anxiety/depression group (*RRR* = 0.87, *SE* = 0.17, *p* = .485, 95% CI: [0.60, 1.28]). The interaction term between *postnatal* depression and gender was not significant for the chronic co-occurring problem group (*RRR* = 0.91, *SE* = 0.16, *p* = .577, 95% CI: [0.65, 1.27]), pure aggression group (*RRR* = 1.48, *SE* = 0.59, *p* = .320, 95% CI: [0.68, 3.23]), or pure anxiety/depression group (*RRR* = 1.14, *SE* = 0.27, *p* = .563, 95% CI: [0.73, 1.80]).

Father psychopathology.

Having a father with any SCID axis I diagnosis by child age 15 was significantly associated with higher risk of being in the chronic co-occurring problem group (*RRR* = 1.68, *SE* = 0.39, *p* = .024, 95% CI: [1.07, 2.64]). Father having any SCID axis I diagnosis was also marginally associated with higher risk of being in the pure aggression group (*RRR* = 1.74, *SE* = 0.53, *p* = .070, 95% CI: [0.96, 3.17]) or the pure anxiety/depression group (*RRR* = 2.30, *SE* = 1.13, *p* = .089, 95% CI: [0.88, 6.01]).

The interaction term between *prenatal* maternal depression and father psychopathology was not significant for the chronic co-occurring problem group (*RRR* = 0.84, *SE* = 0.12, *p* = .225, 95% CI: [0.64, 1.11]), pure aggression group (*RRR* = 0.75, *SE*

= 0.20, *p* = .282, 95% CI: [0.45, 1.26]), or pure anxiety/depression group (*RRR* = 1.00, *SE* = 0.20, *p* = .981, 95% CI: [0.69, 1.47]). The interaction term between *postnatal* depression and father psychopathology was not significant for the chronic co-occurring problem group (*RRR* = 1.00, *SE* = 0.17, *p* = .993, 95% CI: [0.72, 1.40]), pure aggression group (*RRR* = 0.76, *SE* = 0.30, *p* = .483, 95% CI: [0.35, 1.63]), or pure anxiety/depression group (*RRR* = 1.02, *SE* = 0.23, *p* = .933, 95% CI: [0.65, 1.59]).

Child cognitive abilities.

Higher children's verbal ability (PPVT score at age 5) was associated with lower risk of being in the chronic co-occurring problem group (*RRR* = 0.98, *SE* = 0.01, *p* = .020, 95% CI: [0.96, 0.997]) but not for the pure aggression (*RRR* = 1.00, *SE* = 0.02, *p* = .907, 95% CI: [0.96, 1.04]) or anxiety/depression group (*RRR* = 0.99, *SE* = 0.01, *p* = .312, 95% CI: [0.96, 1.01]).

The interaction term between *prenatal* maternal depression and child cognitive ability was not significant for the chronic co-occurring problem group (*RRR* = 1.00, *SE* = 0.01, *p* = .951, 95% CI: [0.99, 1.01]), pure aggression group (*RRR* = 1.01, *SE* = 0.01, *p* = .508, 95% CI: [0.99, 1.03]), or pure anxiety/depression group (*RRR* = 1.01, *SE* = 0.01, *p* = .547, 95% CI: [0.99, 1.02]). The interaction term between *postnatal* depression and child cognitive ability was not significant for the chronic co-occurring problem group (*RRR* = 1.00, *SE* = 0.01, *p* = .972, 95% CI: [0.98, 1.02]), pure aggression group (*RRR* = 0.98, *SE* = 0.01, *p* = .270, 95% CI: [0.95, 1.01]), or pure anxiety/depression group (*RRR* = 1.01, *SE* = 0.01, *p* = .590, 95% CI: [0.99, 1.03]).

Discussion

The current study examined the risk of perinatal exposure to maternal depression on long-term behavioral outcomes in the offspring. Building upon the well-established link between maternal depression and negative child outcomes, the current study was motivated to fill several gaps in the literature: 1) the specificity of the mechanisms comparing the impact of maternal depression on distinct behavioral outcomes, such as co-occurring problems versus pure problems; 2) documenting the long-term effects of prenatal versus postnatal depression on child behavioral outcomes; and 3) using a longitudinal design to test for mediators through which maternal depression exerts its effects. In the current study, group-based methods were used to identify clusters of children following similar developmental trajectories of aggressive and anxious/depressed behavioral problems from early childhood to adolescence and young adulthood. Prenatal and postnatal maternal depression were then assessed for their associations with distinct trajectory groups, including chronic co-occurring problems, chronic aggression, and chronic anxious/depressed problem groups. Further, mediators and moderators of the associations between prenatal/postnatal maternal depression and trajectory groups of behavioral problems were tested. Results from each of these analyses are interpreted below. Strengths, limitations, implications, and future directions are then discussed.

Developmental Trajectory Groups

The average growth curve for aggression, characterized by a decline from age 5 to age 15 and 20, was consistent with previously reported developmental trajectories for the CBCL Aggression scale from early childhood to adolescence (Bongers, Koot, van der Ende, & Verhulst, 2003). The average growth curve for anxiety/depression showed a

drastic decline from age 5 to 15 followed by a surge from age 15 to 20, which was not consistent with the general increase across development for CBCL Anxious/Depressed scale previously reported (Bongers et al., 2003). It should be noted that the 6 anxiety/depression items used in the current study were a subset of the 14 items from CBCL Anxious/Depressed. The previous studies were also conducted in community samples whereas the current study used a high-risk sample with half of the child participants being exposure to maternal depression.

The developmental trajectories for aggression, anxiety/depression, and the co-occurrence of aggression and anxiety/depression were larger in their sizes compared with the trajectory classes identified by Fanti & Henrich (2010), though Fanti & Henrich (2010) examined the dual trajectory groups for CBCL internalizing and externalizing problems (from 2-11 years) rather than specifically the aggression and anxiety/depression scales. Both studies identified trajectory groups that exhibit chronic anxiety/depression or internalizing problems, chronic aggression or externalizing problems, and chronic co-occurrence of both problems. In both studies, the sizes of the chronic internalizing or externalizing problem groups were much smaller than the co-occurrence group, suggesting that co-occurrence is more common than pure problems. Specifically, 1.9% of Fanti & Henrich's (2010) sample was identified as "pure chronic externalizing" group compared to 2.6% as "chronic aggression" in our sample; 2.9% of their sample was identified as "pure chronic internalizing" group compared to 6.2% of our sample as "chronic anxiety/depression"; and 3.7% of their sample were identified as "chronic co-occurring" group respectively compared to 10.3% as "chronic co-occurring problems" group in our sample. It is worth noting that our sample was a high-risk sample in that

about half of the children were exposed to maternal depression before reaching age 15 whereas the Fanti & Henrich's (2010) sample was a community sample. Greater exposure to maternal depression may be an explanation for the larger sizes of behavioral problem trajectory classes in our study. Nonetheless, both studies suggest that the co-occurring/comorbid groups are present early in life and are sizeable. They are also larger than the pure problem groups. Along with the epidemiological studies on comorbidity, these results support the importance of studying the etiology, development, outcomes, and treatment of co-occurring problems. Currently individuals with comorbid disorders are often excluded in pharmaceutical and psychological treatment studies, despite that in reality comorbidity is the rule rather than the exception. Given the sizeable group of individuals who develop co-occurring problems, it would be important in future treatment research to include this group so that guidelines for treating comorbid disorders can be established.

Specificity of the Effects of Maternal Depression

Overall main effects of maternal depression

Consistent with the recent meta-analysis of the effects of maternal depression (Goodman et al., 2011), the effect sizes reported in the current study are considered small (relative risk ratios range from 1.15 to 1.41), regardless of the timing of maternal depression and the various outcomes examined. It should be noted that a depression rating scale was used to assess maternal depression, and therefore the effect sizes are interpreted as expected increase in the relative risk of having the outcome when depression score increase by 1, which is in fact a relatively small increment compared to, for instance, a binary variable of maternal depression diagnosis. Nonetheless, because a

large range of risk factors (e.g., genetic factors, hormonal factors, neighborhood and societal influences) have been reported to contribute to behavioral problems and disorders, it is not surprising that one variable (i.e., maternal depression) only explains a small amount of the total variance.

Main effects on chronic co-occurring problems

Findings from the current study suggested that both prenatal and postnatal depression were associated with chronic co-occurring problems of anxiety/depression and aggression. Furthermore, the effect size for postnatal depression (*RRR* = 1. 20) was slightly greater than the effect size for prenatal depression (*RRR* = 1.15) for the prediction of membership in the chronic co-occurring problem group, which is consistent with a previous finding that more recent timing of maternal depression predicted higher levels of behavioral problems (Brennan et al., 2000). It also appears that co-occurring problems may be driving the association between perinatal depression and child behavioral problem outcomes. When individuals with co-occurring problems were separated from the aggression and anxiety/depression groups, most of the associations between perinatal depression and either chronic aggression or anxiety/depression were non-significant, with the exception of the unique association between prenatal depression and chronic aggression problems.

Main effects on chronic aggression

We also found that prenatal depression predicted offspring having chronic aggressive problems but not chronic anxious/depressed problems. Although the lack of association between prenatal depression and chronic anxious/depressed problems was somewhat surprising, this finding is consistent with a previous longitudinal study of 349

mother-child dyads that also reported that prenatal depression (assessed during the last trimester) predicted externalizing problems at child 4-5 and 8-9 years old but not internalizing problems (Luoma et al., 2001, 2004). Our findings further suggested that this effect might be unique to prenatal depression, because only prenatal but not postnatal depression predicted chronic aggressive problems: the effect size dropped from a relative risk ratio (*RRR*) of 1.41 ($p = .009$) for prenatal depression to no effect for postnatal depression (*RRR* = 0.92, $p = .683$). This finding is also consistent with Luoma et al. (2001, 2004) who found that the effect on externalizing problems was specific to prenatal but not postnatal depressive symptoms (Luoma et al., 2001, 2004). Furthermore, the association between prenatal depression and chronic aggression was still present after accounting for a significant indirect effect through maternal dyadic satisfaction at child 6 months and 5 years, suggesting that additional mediating mechanisms are likely to be present and are yet to be identified. For example, it has been found that women with perinatal depression, similarly to individuals with nonpuerperal depression, show abnormalities of the HPA axis activity (Bloch et al., 2005) as well as abnormal response to reproductive hormonal changes (Bloch et al., 2000). Although abnormalities in HPA axis activity and reproductive hormonal levels are not unique to prenatal depression, we know little about their long-term effects on the developing fetus in utero. Therefore, it would be important to examine the effects of these abnormalities on the developing fetus and their potential mediating role in the relationship between prenatal depression and chronic aggressive behavior problems.

Main effects on chronic anxiety/depression

The current study did not identify any significant direct or indirect effects from perinatal maternal depression to chronic anxious/depressed behavioral problems. This finding is inconsistent with the extensive literature on the associations between maternal depression and child internalizing problems/disorders, including studies that showed such associations using the current sample (e.g., Spence, Najman, Bor, O'Callaghan, & Williams, 2002). This finding is unexpected particularly given that the current sample had a higher rate of maternal depression. One would expect that the child participants in our sample were at greater risk for developing internalizing problems/disorders (e.g., anxious/depressed problems) as they presumably had higher genetic predisposition *and* environmental exposure to depressed mothers and the associated risk factors. It is important to note, however, that this null finding is not entirely inconsistent with the well-documented link between maternal depression and child internalizing problems. Our results did show that perinatal maternal depression was associated with significantly higher risk of offspring developing both chronic anxious/depressive and aggressive problems (i.e., chronic co-occurring problems). Previous studies also rarely contrast the effects of maternal depression on pure internalizing versus co-occurring problems. Therefore, it might be that the effect size of maternal depression on pure internalizing problems is small and we would need a larger sample size to detect such small effect. It is also possible that maternal depression is more strongly associated with internalizing problems when in combination with externalizing problems. The lack of direct effect of perinatal maternal depression on chronic anxiety/depression problems needs replication and further investigation (e.g., other potential indirect effects should be examined).

Mediating role of stressful family environment

The current study examined two sets of indicators of stressful family environment, namely maternal dyadic satisfaction and family income measured at both child 6 months and 5 years, as potential mediators for the transmission of the risk of maternal depression. Findings from these analyses are supportive of Goodman and Gotlib's model (1999) and add important evidence to the specificity of the mechanisms for distinct child behavioral outcomes. In particular, two findings from these mediation analyses are worth highlighting. First, our results suggested that, for both prenatal and postnatal depression, maternal dyadic satisfaction (though marginally significant) and family income *at child 6 months* mediated the effects of maternal depression on co-occurring behavioral problems. Interestingly, maternal dyadic satisfaction and family income *at child 5 years* were *not* significant mediators of the effects for either prenatal or postnatal depression. These findings suggest that the first year of life may be a critical period for stressful family environment to play a mediating role in the association between maternal depression and chronic co-occurring behavior problems in children. Indeed, infants rely entirely on their family environment for physical and emotional security as well as social engagement and experiences and the first year of childhood is thought to be critical in the normal development of neuroregulatory systems (e.g., hypothalamic-pituitary-adrenocortical [HPA] functioning). For example, it has been suggested that sensitive and responsive caregiving as well as healthy family processes are critical for the developing HPA axis during infancy and early childhood (Repetti, Taylor, & Seeman, 2002; Tarullo & Gunnar, 2006). As such, problematic family processes, such as poverty, overt family conflict, and non-nurturing family relationships, not only may represent immediate adversity to infants (e.g., exposure to abuse and violence) but also

fail to support infants in developing effective self-regulation. Deficits in these biological regulatory systems, as a result of or aggravated by risky family processes, have long-lasting adverse effects on mental and physical outcomes (Repetti et al., 2002). Our findings fit with this literature in suggesting that the mediating role of risky family processes may be particularly relevant to infants and their depressed mothers.

Second, for both prenatal and postnatal depression, the serial indirect paths through maternal dyadic satisfaction at 6 months *and* 5 years significantly mediated the effects of maternal depression on chronic aggression problems. The main effect of prenatal depression was still significant after accounting for this indirect path of serial multiple mediators to chronic aggression problems. It is also important to note that this was the only significant mediated path between prenatal/postnatal depression and chronic aggression problems. Taken together, these findings suggest that the mechanisms for the risk of maternal depression might be different in the development of chronic co-occurring behavioral problems versus chronic aggression problems. It appears that *continued* maternal dyadic *dis*satisfaction, which presumably reflects marital conflict, plays an important role in the development of chronic aggression problems for children exposed to either prenatal or postnatal depression, or both.

It is possible that the maternal dyadic *dis*satisfaction reflects unmeasured mental health problems in the child's father, which in turn increases the risk for developing behavioral problems. Or alternatively the absence of father's mental health problems may decrease the risk for child behavioral problems, as the role of father was discussed as a potential resilience factor in Goodman and Gotlib's model (1999). In the current study, we reported that father's psychopathology measured at child age 15 was not a significant

moderator for the main effects of perinatal maternal depression on chronic co-occurring problems or chronic aggression. This null finding, however, does not preclude the possibility of moderated mediations by paternal psychopathology for the indirect paths from perinatal maternal depression to child negative behavioral outcomes through early stressful family environment (e.g., marital conflicts, family poverty). For example, it is possible that father's involvement in child rearing and his mental health status during early childhood might moderate the path from perinatal maternal depression and stressful family environment, such that for those children with depressed mothers, higher paternal involvement with childcare and absence of paternal psychopathology might lead to a less stressful family environment during early childhood. Similarly, father's presence and mental health during early childhood may also moderate the path from stressful family environment to chronic co-occurring problems or chronic aggression, such that a more active and mentally healthy father may alleviate the negative impact of early stressful family environment on the development of behavioral problems. A better measurement of paternal psychopathology during early childhood will enable the examination of the examination of such moderated mediation models.

Mediating role of low maternal positive attitude and warmth

Our mediation analyses yielded significant indirect effects of perinatal maternal depression on offspring *chronic co-occurring problems* through lower maternal positive attitude toward the infant measured at 6 months and lower maternal warmth towards the child measured at 5 years. This finding also provides strong support for Goodman and Gotlib's model (1999), which postulates that the exposure to mother's negative behaviors/affect/cognition mediates the effect of maternal depression. Negative parenting

practices have long been recognized in depressed mothers (see meta-analytic review in Lovejoy, Graczyk, O'Hare, & Neuman, 2000) and are believed to contribute to the transmission of cognitive vulnerability and depression to children (see reviews in Goodman & Gotlib, 1999; Goodman, 2007). Our findings fit with this literature and provide strong evidence for this mechanism.

We also found that low maternal positive attitude and warmth did *not* mediate the significant link between prenatal maternal depression and chronic aggression problems or the link between postnatal maternal depression and chronic aggression. This finding should be contrasted with the significant mediating effects of perinatal maternal depression and chronic aggression through continued maternal dyadic dissatisfaction, as harsh parenting is often associated with marital conflict, and both contribute to externalizing problems (e.g., Burke, 2003; Marchand, Hock, & Widaman, 2002). Our finding is consistent with an exposure hypothesis, which postulates that exposure to marital conflict elicits and promotes child aggression (Cummings, Goeke-Morey, & Papp, 2004). However, we did not examine the effects of having a child who would later develop chronic aggression on marital conflict and maternal depression. Recent work has suggested that certain temperament (e.g., fearless temperament) during early childhood prospectively predicted conduct problems and callous-unemotional traits (CP+CU) later in childhood and adolescence (Barker, Oliver, Viding, Salekin, & Maughan, 2011). Our chronic aggression group in the absence of anxiety/depression problems is likely to resemble the CP + CU group. Therefore, it would be important to also include child temperament characteristics to better elucidate the transactional process between

maternal depression and child chronic aggression in the context of a risky family (e.g., continued parental conflict).

Lack of support for any indirect/mediated effects from perinatal depression to chronic anxiety/depression was again somewhat unexpected. Exposure to harsh parenting and marital conflicts have been linked to maternal depression and shown to increase the risk of children developing internalizing problems (e.g., Davies, Cummings, & Winter, 2004; Shaw, Keenan, Vondra, Delliquadri, & Giovannelli, 1997). Yet maternal dyadic satisfaction and maternal positive attitude and warmth did not have mediated effects for chronic anxiety/depression problems in our high-risk sample. It is likely that the indirect effects for this association were not included in our model. It is also possible that the effects of maternal depression on pure internalizing problems are only specific to more chronic maternal depression with an early onset (indicating greater genetic loading). Nonetheless, if our null finding were replicated, it might also be possible that maternal depression, at least during the perinatal period, is not a specific risk factor for chronic internalizing problems in the offspring, but a general risk factor for chronic aggression and co-occurring problems. In that case, other risk factors and processes should be examined for the development of chronic anxiety/depression problems.

Potential moderators

There are several potential methodological reasons for the non-significant results regarding the moderators. For example, child PPVT scores were not available for the full sample. The father psychopathology measure was also suboptimal, as it was not obtained directly from fathers in many cases. These problems may have resulted in reduced sample size and/or statistical power to test for interaction. Further, gender was also not a

significant moderator for the main effects of perinatal maternal depression, but potential gender moderations of the mediated paths identified in our results should be conducted before concluding that gender does not play a moderating role.

Strengths

One of the major strengths of the current study is the use of a longitudinal, prospective study design, which allows for testing multiple mediators likely relevant to the causal pathways from maternal depression to child behavioral problems. The mother-child dyads were followed from pregnancy to 20 years after birth, which is among the longest follow-up periods in longitudinal studies of maternal risk factors and child outcomes. Our predictors, mediators, and outcomes were also generally collected in a temporal order, such that the maternal depression was measured during pregnancy and within 6 months after childbirth, mediators were measured at 6 months and 5 years, and outcomes were measured at age 5, 15, and 20. The prospective nature of the current study and the temporality of the study variables make our findings less prone to errors when making causal inferences compared to evidence obtained from cross-sectional studies. The current study was also grounded in a developmental psychopathology theory for the risk transmission of maternal depression, which guided the examination of mediators and moderators. The use of Latent Class Growth Analysis allowed for identifying clusters of individuals that developed chronic co-occurring problems versus chronic aggression or anxiety/depression problems. This method is considered more advantageous than using clinical cut-offs, cluster analysis, or correlations because latter statistical methods are incapable of accounting for growth or changes over time (Nagin, 2005).

Limitations

Several limitations should be noted when interpreting the results of the current study. First, the current study used summary scores in the group-based analysis identifying the trajectory groups. Although previous studies using growth curve and group-based modeling methods typically use the summary scores (i.e., the sum of all item ratings on a scale) of psychological measures, using measurement models (i.e., constructing a factor model using all item ratings) as the outcome variable at each time point would be more methodologically sound because measurement errors would be better accounted for. Longitudinal measurement invariance over time should be established to ensure that the items relate to the latent construct in the same way across time points (e.g., invariant factor loadings, thresholds, intercepts, residuals). Latent growth factors and latent classes can then be added as higher-order factors to the latent construct/outcome. A major difficulty in applying this method to the current analyses was that the CBCL item ratings were categorical, which greatly increases the complexity of the model and analyses. Second, only a subset of the CBCL items was available across all three time points (10 out of 20 aggression items; 6 out of 14 anxiety/depression items). The reason for our lack of complete CBCL items at age 5 was that the 5-year follow-up preceded the publication of the Preschool CBCL. Relatedly, we were not able to separately examine anxiety and depression, because the reliability coefficients for the scores of anxiety and depression items (4 items each according to DSM-oriented scale) were too low. Third, the current study relied on mother reports of maternal depression and child behavioral outcomes as well as measures of mediators. Future studies should replicate the current findings using multiple sources of information (e.g., behavioral observation, father reports, and teacher reports).

Fourth, our study was also limited by its relatively small sample size and few repeated measures of the outcome. As a result, we weren't able to examine more developmental trajectory classes, particularly ones that capture the non-linear growth that were previously reported in other studies. More trajectory groups with more repeated measures would also allow for examining multifinality and equifinality, such as what predicts individual with diverging and converging developmental pathways. Fifth, we did not examine bidirectional effects between the depression in mothers and the behavior problems in their children and thus do not provide a full picture of the transactional process of the risk transmission. As mentioned above, difficult temperament characteristics in children are important to include in the model, as these temperaments may increase harsh parenting, decrease parental warmth, and exacerbate family risk and parental psychopathology, which in turn might worsen the child's behavioral problems and lead to long-term negative outcomes. Relative to this limitation, we also did not examine additional measurements of maternal depression after child 6 months, which makes it difficult to examine the potential child effects on maintaining maternal depression.

Implications

Findings from the current study have implications for psychopathology research. Since our findings point to the specificity of mechanisms through which maternal depression has its negative impact on distinct behavioral outcomes, it may be important to distinguish those children presenting with both internalizing and externalizing problems from those presenting with primarily one type of behavioral problem. Our findings also support distinguishing prenatal from postnatal depression, as some

important differential associations were found in relation to child behavioral outcomes (e.g., unique impact of prenatal depression on chronic aggression).

Our findings of long-term effects of perinatal depression on child development suggest that prevention and early intervention for maternal depression during pregnancy and postpartum should be advocated. Currently in the U.S. routine screening of perinatal depression is "strongly encouraged but not mandated" (American College of Obstetricians and Gynecologists, 2010). One of the current controversies in medicine involves how to best advise pregnant woman and new mothers about the risk of untreated prenatal and postpartum depression versus the risk of pharmacological treatment during this time (Meltzer-Brody, 2011). Our study, along with a large body of psychological literature on the risk of maternal depression on fetus/child development, suggests that perinatal depression should be routinely screened and psychotherapy may also be an important recommendation for depressed mothers and their families in addition to pharmacological treatment. Currently cognitive behavioral therapy (CBT) and interpersonal psychotherapy (IPT) have both shown effectiveness in helping women with mild-to-moderate depression during pregnancy and the postpartum period (O'Hara, Stuart, Gorman, & Wenzel, 2000; Spinelli & Endicott, 2003; Yonkers et al., 2009). Our findings suggest that these early interventions for maternal depression may also have long-term effects in reducing child behavioral problems. Some psychologists have already been advocating for intensive perinatal intervention for mothers at risk (e.g., maternal depression, substance use) as a starting point of an experimental preventive intervention strategy for reducing disruptive behaviors in children (Tremblay, 2010). Given all the problematic family processes and parenting behaviors that are suggested to

mediate the risk of maternal depression on negative child outcomes, psychological treatment should be recommended in addition to pharmacological treatment with the goal to improve the health and mental health outcomes of the mothers, children, and their families.

Several intervention strategies and targets are supported by our mediation results. First, as our study suggests that infancy or early childhood may be a critical risk period, it may be particularly important for the psychotherapy treatment to take place as early as possible (during pregnancy and first year of the child's life). In addition, it would be important to require the partner of the depressed mother to also participate in the treatment so that any marital conflict and dissatisfaction could be addressed and the infant's potential exposure to family conflict may be minimized. For example, partner- and family-assisted therapies, which have shown to be efficacious as a treatment for perinatal depression (Baucom, Shoham, Mueser, Daiuto, & Stickle, 1998), might be particularly useful in reducing child behavioral problems and improving the developmental outcomes.

Our findings also confirmed the mediating role of negative parenting for the association between perinatal depression and child co-occurring problems. This finding highlights the need to target maternal attitude and warmth towards the child to reduce the child's risk of being exposed to such negative behaviors in the depressed mother and to minimize subsequent development of both internalizing and externalizing behavioral problems. Psychotherapy that specifically targets maternal negative attitude towards child rearing may be helpful. For example, a behavioral family intervention program known as Triple P – Positive Parenting Program targets parenting behaviors and has shown to result

in reduction in conduct problems in preschoolers (Sanders, Markie-Dadds, Tully, & Bor, 2000). Based on the current finding, intervention like Triple P could be extended to treat children with co-occurring anxiety/depression and aggression.

Future Directions

Future directions may include both methodological improvements and further substantive investigations. As noted above, replications for the current findings are needed, particularly using samples that have additional time points and measurements of key variables from other sources/methods. Establishing measurement invariance and using measurement models for the longitudinal outcomes are also important in increasing statistical power, as measurement errors will be better accounted for using such approaches. Substantively, given our findings of mediation paths, one important future step is to examine *for whom* these mediation processes would lead to the adverse behavioral outcomes by conducting moderated mediation analyses. Though the current results suggest that none of the theory-driven moderators significantly moderated the effects of maternal depression on negative child outcomes, it is still possible that the moderators impact one or several mediated paths. Future studies should also explore biological mechanisms (e.g., dysregulation of the HPA axis, hormonal changes during perinatal period) that might mediate the effects of prenatal depression and child aggressive behavioral problems. Continued research on the etiology and pathogenesis of perinatal maternal depression will evidently be crucial given the long-lasting negative effects on child behavioral outcomes. A better understanding of the underlying pathogenesis of perinatal depression will also help elucidate the mediating mechanisms that contribute to negative child developmental outcomes. Further, given the high co-

occurrence between depression and anxiety (e.g., Angold & Costello, 1993) and the evidence that the presence of additional psychopathology confers higher risk to the mother-child dyad (Carter, Garrity-Rokous, Chazan-Cohen, Little, & Briggs-Gowan, 2001), the co-occurrence of perinatal depression and anxiety in mothers should be taken into account as well as the effects of maternal comorbidity on various child behavioral outcomes. Finally, as maternal depression represents both increased genetic vulnerability and an environmental stressor for the children, future studies that use genetically informed samples (e.g., twin registry) or have molecular genetic information on the mother-child dyads will be able to better separate genetic versus environmental effects in the transmission of the risk of maternal depression to co-occurring versus pure behavioral outcomes.

Conclusions

The current study examined the main/direct and indirect effects of prenatal and postnatal maternal depression on the development of chronic co-occurring aggression and anxiety/depression problems, chronic aggression, and chronic anxiety/depression, relative to low behavior problems in offspring. The dual developmental trajectory groups for aggression and anxiety/depression problems from early childhood to adolescence to young adulthood were identified using group-based trajectory modeling methods. Prenatal and postnatal maternal depression were differentially associated with distinct behavioral problem outcomes. Specifically, although both prenatal and postnatal depression were associated with increased risk of offspring developing chronic co-occurring problems, only prenatal depression was associated with increased risk of offspring developing chronic aggression problems, and neither of the maternal depression

measures were associated with risk of offspring chronic anxiety/depression problems, relative to the risk of having low behavior problems throughout development. Stressful family environment and low maternal positive attitude and warmth during early childhood were found to mediate the effects of prenatal and postnatal maternal depression on developing co-occurring problems. Only continued marital conflict mediated the effects of prenatal and postnatal depression on developing chronic aggression problems. No indirect effects were identified for developing chronic anxiety/depression problems. These results suggest that co-occurring internalizing and externalizing problems as well as chronic internalizing or externalizing problems are present throughout child development into young adulthood. The current study makes a unique contribution by revealing the differential effects and mechanisms associated with perinatal maternal depression on different child outcomes, particularly focusing on co-occurring problems.

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Table 1.

Descriptive statistics for continuous study variables

Repeated measures	Obs.	Range	*Mean*	*SD*	*Median*	*Alpha*
Aggression scale						
Summary score at age 5	789	0-18	5.81	3.44	5	0.82
Summary score at age 15	765	0-17	2.87	2.98	2	0.84
Summary score at age 20	653	0-17	2.91	3.15	2	0.84
Anxious/Depressed scale						
Summary score at age 5	773	0-11	2.52	2.19	2	0.76
Summary score at age 15	776	0-10	1.32	1.80	1	0.77
Summary score at age 20	654	0-12	2.27	2.24	2	0.79
Primary predictors						
Prenatal DSSI score	705	0-7	1.56	1.62	1	
Postnatal DSSI score	705	0-7	1.37	1.34	1	
Mediators						
DAS score at 6 months	769	11-50	42.25	5.38	43	
DAS score at 5 years	762	10-50	40.17	6.39	42	
Family income at 6 months	776	1-7	4.07	1.09	4	
Family income at 5 years	788	1-7	4.47	1.52	4	
Maternal attitude at 6m	814	11-50	38.34	6.98	40	
Maternal warmth at 5y	815	2-5	4.30	0.61	4.5	
Moderators						
Child PPVT score	673	60-130	99.21	13.20	100	
Confounds						
Gestational age	814	26-43	39.34	1.73	40	

Note. Anxious/depressed scale has 6 items common across all time points. Aggression scale has 9 items common across all time points. Maternal attitude at 6m indicates maternal positive attitude towards the infant measured at 6 months with higher scores indicating greater positive attitude. For maternal warmth at 5 years, higher scores indicating higher levels of warmth toward the child. Descriptive statistics of categorical moderators and confounding variables are reported in text.

Table 2.

Bivariate correlations for primary predictor and outcome variables

	Variables	1	2	3	4	5	6	7	8
1	Anxious/Depressed age 5	1.00							
2	Anxious/Depressed age 15	.35**	1.00						
3	Anxious/Depressed age 20	.31**	.58**	1.00					
4	Aggression age 5	.50**	.26**	.20**	1.00				
5	Aggression age 15	.23**	.57**	.37**	.37**	1.00			
6	Aggression age 20	.20**	.35**	.55**	.29**	.56**	1.00		
7	Prenatal DSSI score	.17**	.10*	.12**	.09*	.10**	.13**	1.00	
8	Postnatal DSSI score	.16**	.13**	.11**	.14**	.10**	.05	.46**	1.00

Note. Summary scores for anxious/depressed and aggression scale at age 5, 15, and 20 were outcome variables used for the growth curve and latent growth class analyses. Prenatal and postnatal DSSI depression scores were primary predictors in the current study.

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

Table 3.

Model fit information for LCGMs of Aggression *and* Anxiety/Depression

Model	AIC	BIC	Entropy	LMR adjusted LRT test	*p*-value
Aggression					
1-class model	11395.141	11423.360			
2-class model	10927.983	10975.015	0.890	458.074	0.0001
3-class model	10743.199	10809.043	0.877	185.853	0.0007
4-class model	10661.869	10746.527	0.878	86.117	0.1382
5-class model	10601.361	10704.831	0.888	66.045	0.2919
Anxious/Depressed					
1-class model	9562.325	9585.841			
2-class model	8911.037	8958.069	0.897	517.285	0.0000
3-class model	8719.535	8785.379	0.910	192.329	0.0149
4-class model	8593.285	8677.943	0.922	129.422	0.1036
5-class model	8530.765	8634.235	0.904	67.985	0.0349

Note. AIC = Akaike Information Criterion. BIC = Bayesian Information Criterion. LMR adjusted LRT test= Lo, Mendel, Rubin (LMR) adjusted Log Likelihood Ratio Test.

Table 4.

Class memberships for the co-occurring and pure problems based on the most probable class probabilities

Group	Anxious/ Depressed	Aggression	Descriptors	*n*	%	Male	Female
1	High	Low	Chronic Anxiety/Depression	46	5.64	21	25
2	High	High	Chronic Co-occurring	86	10.55	42	44
3	Low	Low	Low Problems	665	81.60	339	326
4	Low	High	Chronic Aggression	18	2.21	10	8

Note. Pearson $\chi^2(3) = 0.7714$, $p = 0.856$ for gender by classes tabulation.

Table 5.

Test of potential confounds in relations to primary predictors and outcomes

	Coef	*SE*	*z*/*t*	*p*
Co-occurring Problems (n = 86) vs. Low Problems				
Gestational age	-0.17	0.08	-2.05	**0.040**
Maternal smoking				
Rarely	0.07	0.50	0.14	0.888
Some of the time	0.31	0.56	0.56	0.576
Most of time	0.51	0.25	2.04	**0.041**
Birth weight	-0.0004	0.0002	-1.93	0.054
Chronic Aggression (n = 18) vs. Low Problems				
Gestational age	-0.11	0.11	-0.98	0.325
Maternal smoking				
Rarely	0.32	1.08	0.30	0.766
Some of the time	-	-	-	-
Most of time	1.16	0.50	2.33	**0.020**
Birth weight	-0.0004	0.0002	-1.93	**0.054**
Chronic Anxiety/Depression (n = 46) vs. Low Problems				
Gestational age	-0.02	0.09	-0.23	0.816
Maternal smoking				
Rarely	-0.11	0.62	-0.18	0.856
Some of the time	-0.75	1.03	-0.73	0.466
Most of time	-0.41	0.37	-1.11	0.269
Birth weight	0.00005	0.0003	0.17	0.865
Prenatal maternal depression				
Gestational age	-0.02*	-	-	0.523
Maternal smoking				
Rarely	0.12	0.26	0.46	0.647
Some of the time	0.32	0.32	1.00	0.315
Most of time	0.69	0.14	5.00	**0.000**
Birth weight	-0.0002	0.0001	-1.26	0.207
Postnatal maternal depression				
Gestational age	-0.07*	-	-	0.073
Maternal smoking				
Rarely	0.26	0.21	1.24	0.215
Some of the time	-0.44	0.26	-1.69	0.092
Most of time	0.37	0.11	3.22	**0.001**
Birth weight	-0.0002	0.0001	-1.62	0.106

Note. For Low Problems group, $n = 665$. * indicates correlation coefficients. Only potential confounds that significantly predicted the primary outcome Co-occurring problems were reported here.

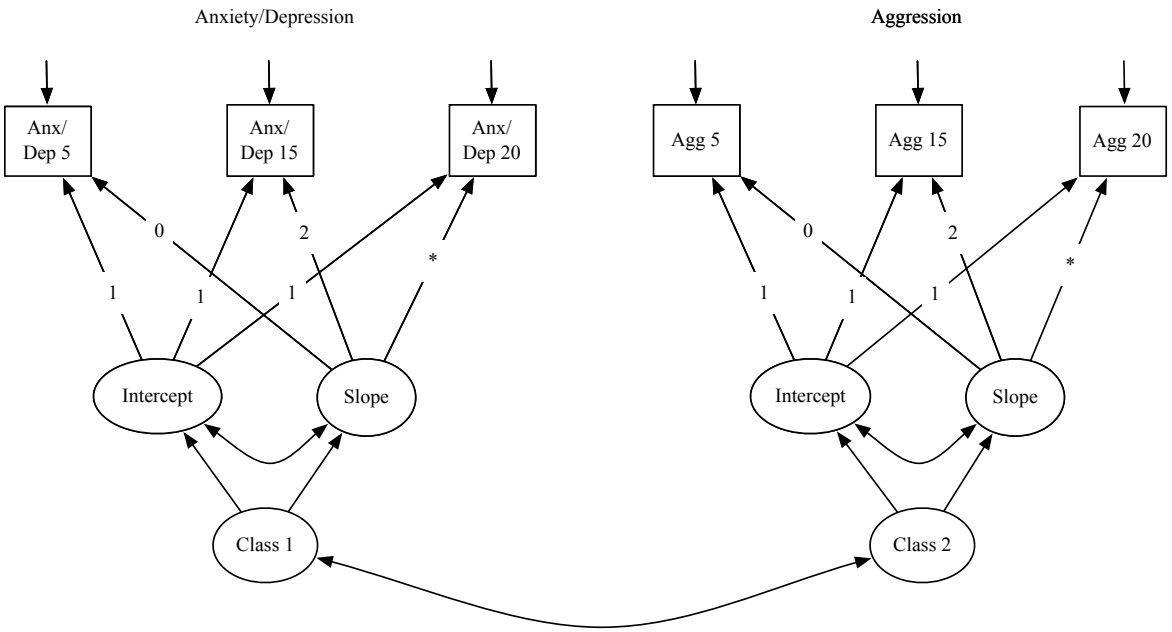

Figure 1. Path diagram showing the specification of the dual trajectory Latent Class Growth Analysis (LCGA). Anxiety/Depression and Aggression problems measured at age 5, 15, and 20 were manifest outcome variables. Intercept and Slope represent two latent growth factors. Loadings for the Intercept were fixed at 1; loadings for the Slope for age 5 and 15 follow-ups were fixed at 0 and 2, and were free for age 20. Class 1 and 2 represent latent class variables for Anxiety/Depression and Aggression problems, respectively.

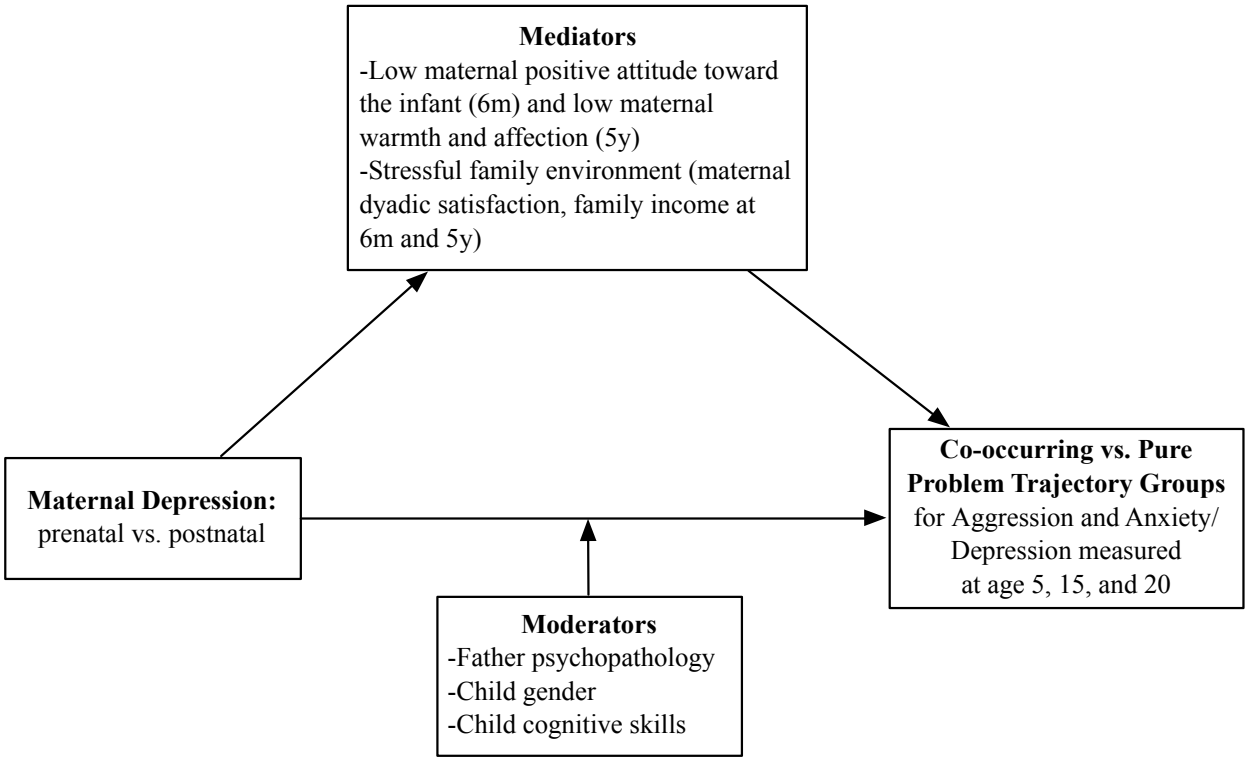

Figure 2. A generic path model showing the mediation and moderation analyses. 6m and 5y indicate that the specific mediator was measured at child 6 months or 5 years. Co-occurring vs. pure problem trajectory groups were the final class counts based on the most probable class memberships obtained from the dual trajectory Latent Class Growth Analyses (LCGA).

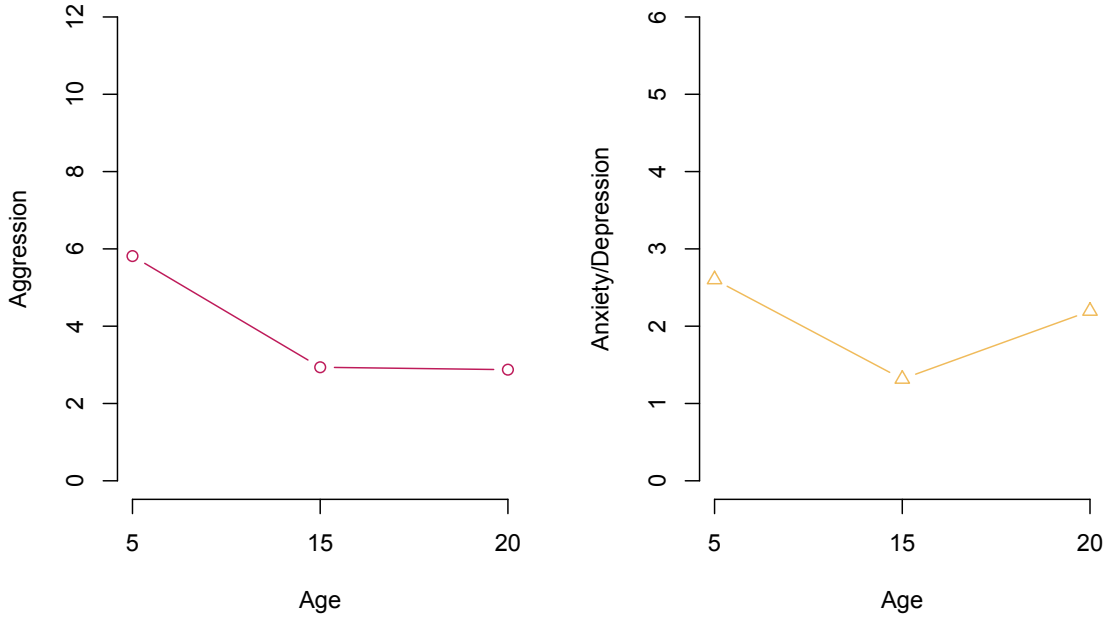

Figure 3. Average trajectories for *Aggression* and *Anxiety/Depression* obtained from Latent Growth Curve modeling.

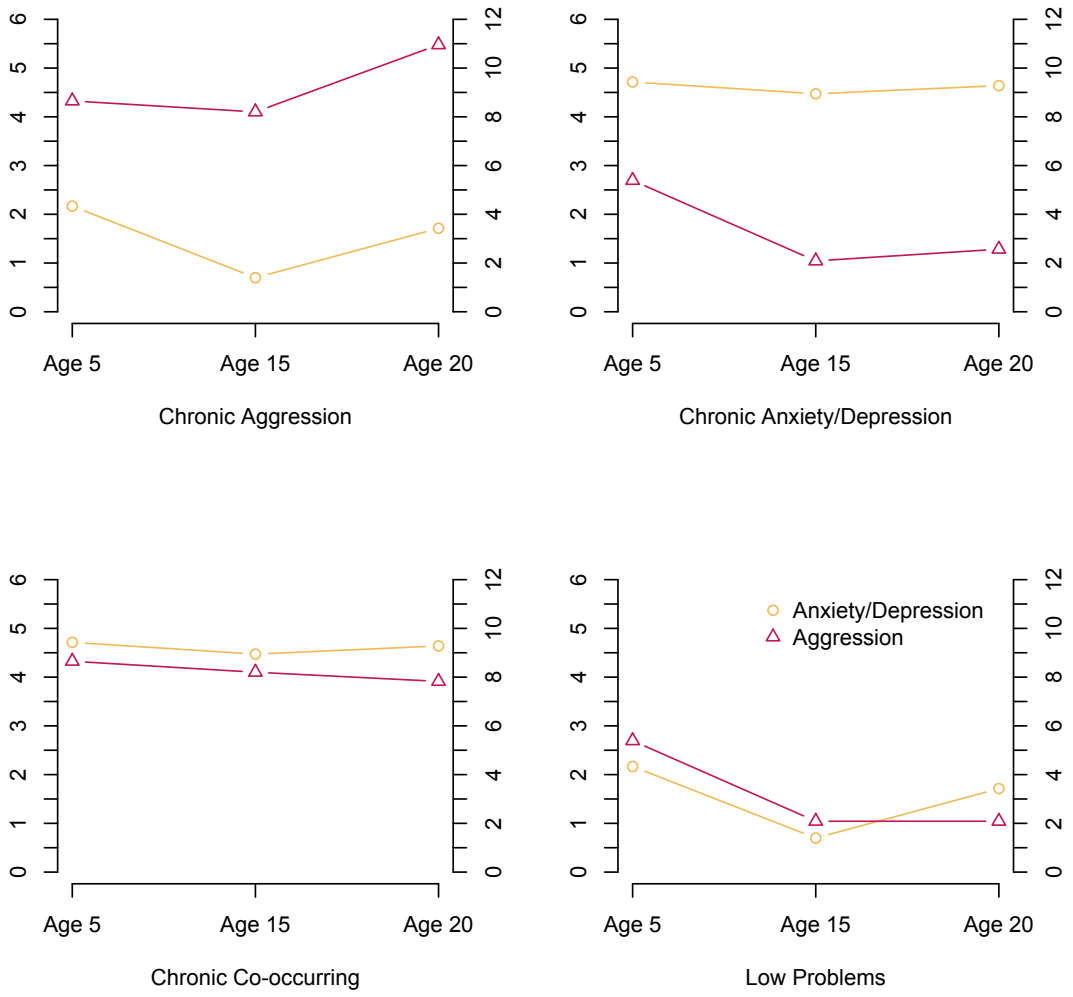

Figure 4. Dual trajectory groups for *Aggression* and *Anxiety/Depression* obtained from dual trajectory Latent Class Growth Analysis (LCGA). The y-axes on the left (range: 0-6) indicate Anxiety/Depression level and the ones on the right (range: 0-12) indicate Aggression level. Note that the y-axes levels are not equivalent to raw scores on the CBCL.

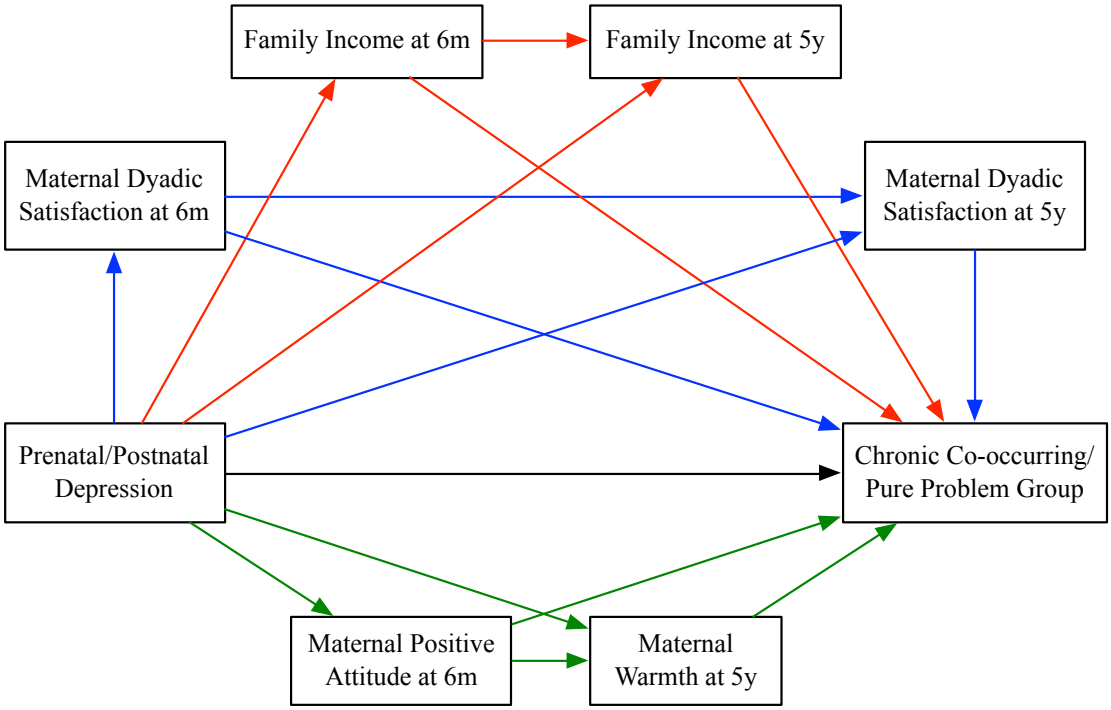

Figure 5. Illustration of the full serial multiple mediators model. Indirect paths going through maternal dyadic satisfaction were in blue, family income in red, and maternal positive attitude and warmth in green. 6m and 5y indicate that the specific mediator was measured at child 6 months or 5 years.

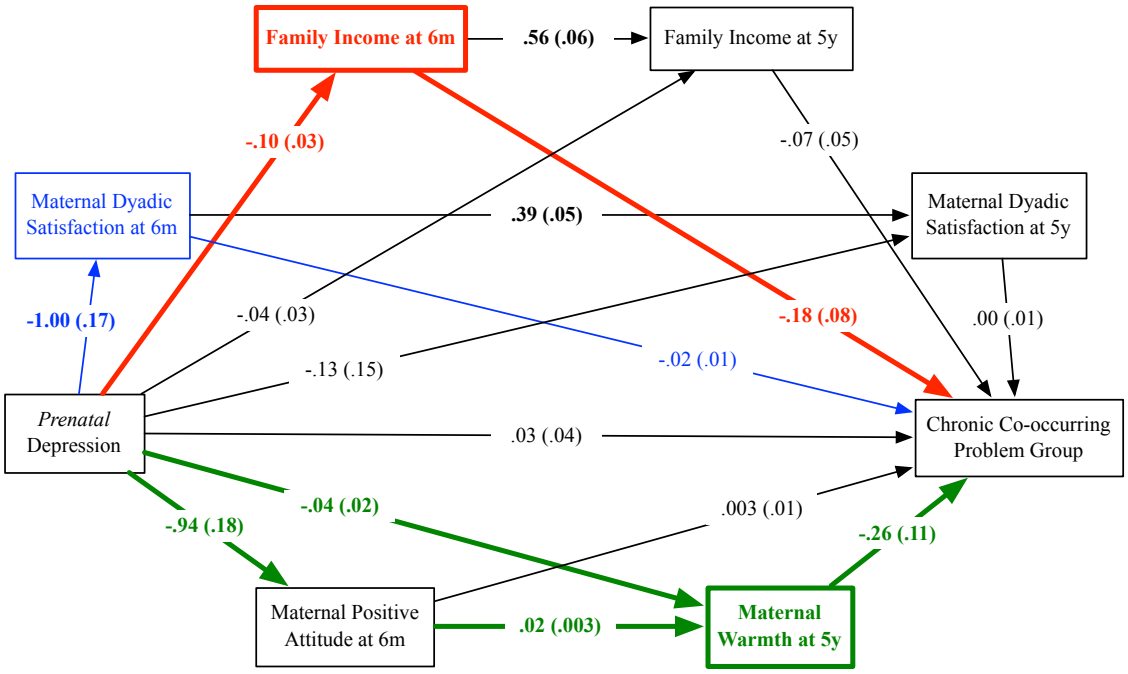

Figure 6. Mediated paths for the association between *prenatal* depression and chronic co-occurring problem group. Path coefficients are unstandardized with standard errors in parentheses (obtained from bootstrapping procedure). Bolded path coefficients are significant estimates. Bolded *and* colored indicate either mediators with significant sum of indirect effects or significant mediated paths. Colored but not bolded indicate either a mediator with marginally significant sum of indirect effects or marginally significant mediated paths.

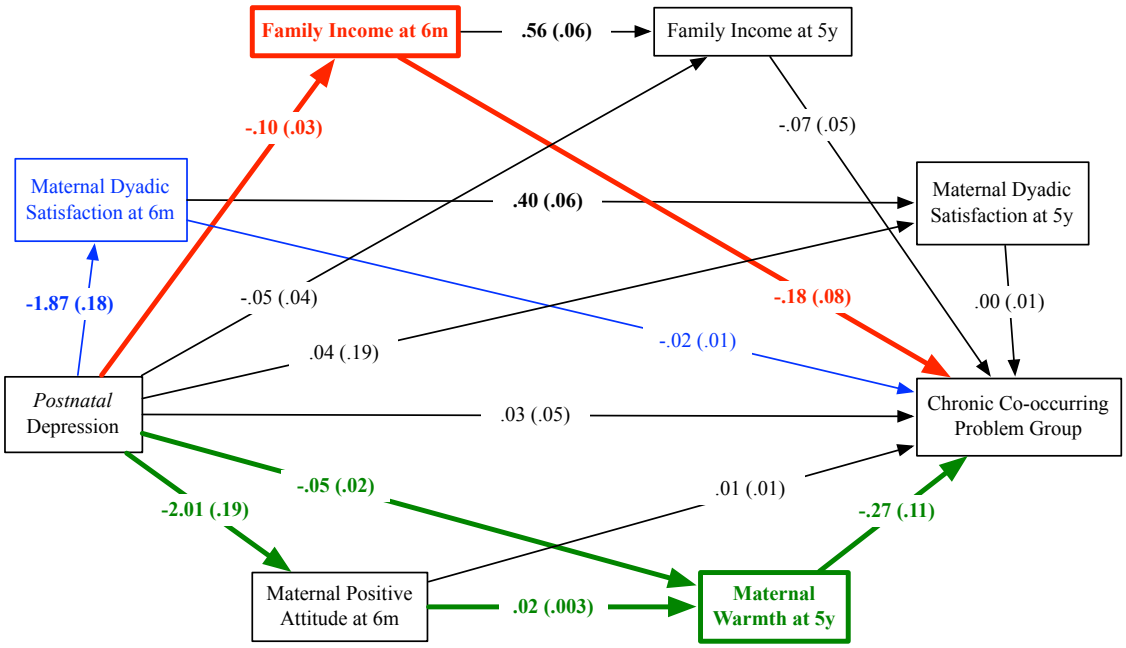

Figure 7. Mediated paths for the association between *postnatal* depression and chronic co-occurring problem group. Path coefficients are unstandardized with standard errors in parentheses (obtained from bootstrapping procedure). Bolded path coefficients are significant estimates. Bolded *and* colored indicate either mediators with significant sum of indirect effects or significant mediated paths. Colored but not bolded indicate either a mediator with marginally significant sum of indirect effects or marginally significant mediated paths.

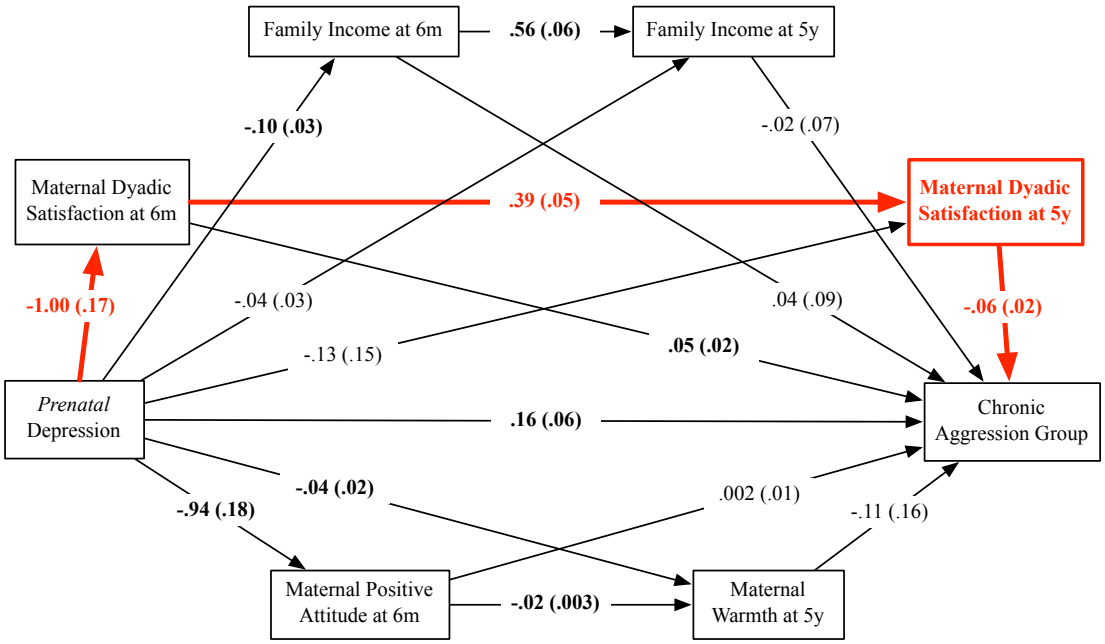

Figure 8. Mediated paths for the association between *prenatal* depression and chronic pure aggression group. Path coefficients are unstandardized with standard errors in parentheses (obtained from bootstrapping procedure). Bolded path coefficients are significant estimates. Bolded *and* colored indicate either mediators with significant sum of indirect effects or significant mediated paths.

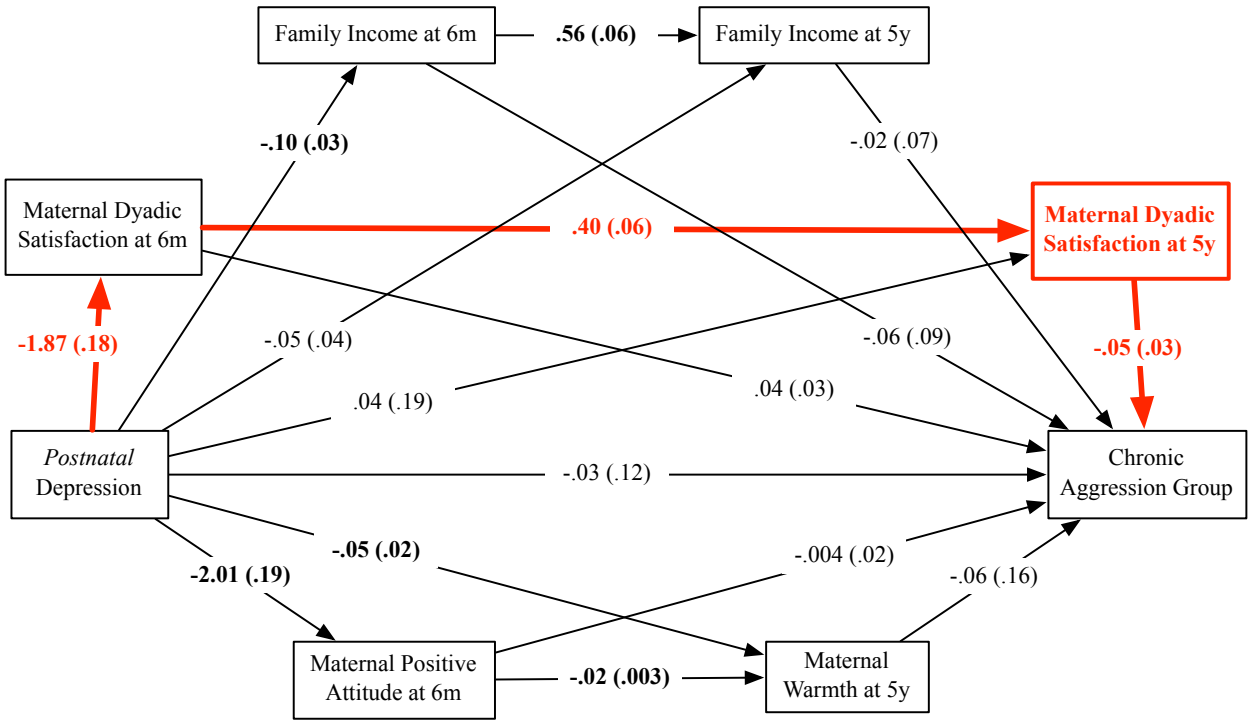

Figure 9. Mediated paths for the association between *postnatal* depression and chronic pure aggression group. Path coefficients are unstandardized with standard errors in parentheses (obtained from bootstrapping procedure). Bolded path coefficients are significant estimates. Bolded *and* colored indicate either mediators with significant sum of indirect effects or significant mediated paths.