Postpartum Depression in the Intergenerational Transmission of Child Maltreatment:

Longitudinal Evidence from Global Settings

by

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Melissa H. Watt

Dissertation submitted in partial fulfillment of
the requirements for the degree of Doctor of Philosophy
in the Department of Psychology & Neuroscience
in the Graduate School
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ABSTRACT

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Abstract

Childhood maltreatment is a potent and common form of early trauma that not only produces negative outcomes for individuals during their lifetime, but may also have consequences for the next generation. Mothers who have experienced childhood maltreatment are more likely to have children also exposed to maltreatment, a phenomenon known as the intergenerational transmission of maltreatment. The perinatal period, the earliest point of intersection between generations, may offer an opportunity to interrupt such transmission. This dissertation leveraged two longitudinal studies in diverse global settings to examine how childhood maltreatment influences maternal mental health during the postpartum period, in turn impacting children’s risk for maltreatment exposure and related outcomes. In Study 1, a UK-based longitudinal cohort of 1,116 mothers and their twin children (E-Risk) was used to: (1) explore maternal childhood maltreatment as a risk factor for postpartum depression; (2) test the bridging role of postpartum depression between maternal childhood maltreatment and long-term child outcomes, specifically child exposure to maltreatment, internalizing symptoms, and externalizing symptoms; and (3) examine the intergenerational effects of specific maltreatment subtypes. Structural equation modeling revealed that maternal childhood maltreatment predicted postpartum depression, which in turn predicted child maltreatment exposure between 5 and 12 years and subsequent child internalizing and
externalizing symptoms at 12 years. Indirect effects through postpartum depression were significant, robust across twin zygosity and child gender, and persisted after controlling for maternal covariates – though appeared to be carried by later maternal depression when included. In particular, emotional abuse emerged as a significant predictor of this pathway above and beyond other subtypes. In Study 2, similar aims were examined in a sample of 150 South African mothers followed through pregnancy and into the first postpartum year, with more proximal outcomes including maternal-infant bonding, infant development, and infant physical growth. Again, maternal childhood maltreatment predicted postpartum depression through 6 months, which then predicted child outcomes at 1 year. Indirect effects through postpartum depression were significant and persisted for maternal-infant bonding and infant growth after controlling for maternal and child covariates and accounting for antenatal distress. In particular, emotional neglect was a significant predictor of this pathway above and beyond other subtypes. Alterations in maternal emotion processing emerged as a potential explanatory mechanism. Together, findings from this dissertation underscore how postpartum depression may play a role in perpetuating negative outcomes across generations and in different global settings. Identifying and treating postpartum depression, as well as preventing its occurrence/recurrence, may help interrupt the intergenerational transmission of maltreatment and its sequelae.
Dedication

To the women around the globe, including my own mother,

who have helped me appreciate how families everywhere function,

and how mothers anywhere mother.
Contents

Abstract ........................................................................................................................................... iv

List of Tables ........................................................................................................................................ xi

List of Figures ........................................................................................................................................ xii

Acknowledgements .......................................................................................................................... xiii

1. Introduction ..................................................................................................................................... 1

  1.1 Scope and impact of childhood maltreatment ................................................................. 1

  1.2 Intergenerational transmission of maltreatment and psychopathology ..................... 2

  1.3 Potential role for postpartum depression in the intergenerational transmission pathway ................................................................. 4

  1.4 Global perspective .................................................................................................................. 6

  1.5 Specificity of maltreatment subtypes in intergenerational transmission ................. 7

  1.6 Specific aims .......................................................................................................................... 9

    1.6.1 Aims for Study 1 ........................................................................................................ 9

    1.6.2 Aims for Study 2 ....................................................................................................... 11

2. Study 1: UK ..................................................................................................................................... 13

  2.1 Methods ................................................................................................................................... 13

    2.1.1 Sample ...................................................................................................................... 13

    2.1.2 Procedures ............................................................................................................... 14

    2.1.3 Measures .................................................................................................................. 14

      2.1.3.1 Maternal childhood maltreatment ................................................................. 14

      2.1.3.2 Maternal postpartum depression ................................................................. 15
3. Study 2: South Africa

3.1 Methods

3.1.1 Sample

3.1.2 Procedures

3.1.3 Measures

3.1.3.1 Maternal childhood trauma

3.1.3.2 Maternal postpartum depression

3.1.3.3 Maternal-infant bonding

3.1.3.4 Child emotional/behavioral development

3.1.3.5 Child physical growth

3.1.3.6 Covariates

3.1.4 Analyses

3.1.4.1. Main analyses

3.1.4.2 Subtype analyses

3.1.5 Exploratory sub-study: Testing a potential mechanism

3.2 Results

3.2.1 Descriptive findings

3.2.2 Main findings

3.2.2.1 Influence of maternal childhood trauma on child outcomes

3.2.2.2 Mediating effect of postpartum depression

3.2.2.3 Subtype findings

3.2.4 Exploratory mechanism findings
List of Tables

Table 1: Maternal characteristics (Study 1) ................................................................. 24
Table 2: Child characteristics (Study 1) ................................................................. 24
Table 3. Descriptive findings on maternal predictors (Study 2) ........................................ 60
Table 4. Descriptive findings on child-related outcomes (Study 2) ................................. 60
Table 5. Direct path coefficients for main structural model for 12-year child outcomes (Study 1) ................................................................. 84
Table 6. Indirect path coefficients from maternal childhood maltreatment to child outcomes, adjusted (Study 1) ................................................................. 85
Table 7. Direct path coefficients for 12-year structural model including later maternal depression (Study 1) ................................................................. 86
Table 8. Indirect path coefficients from maternal childhood maltreatment through postpartum depression and later maternal depression to child outcomes (Study 1) .... 87
Table 9. Direct path coefficients for maltreatment subtypes model for 12-year outcomes (Study 1) ................................................................. 88
Table 10. Direct path coefficients for main structural model for 18-year child outcomes (Study 1) ................................................................. 93
Table 11. Direct path coefficients for 18-year structural model including later maternal depression (Study 1) ................................................................. 94
Table 12. Direct path coefficients for main structural model (Study 2) ............................ 95
Table 13. Direct path coefficients for models including antenatal distress (Study 2) ........ 96
Table 14. Path coefficients for trauma subtypes model (Study 2) ..................................... 97
Table 15. Exploratory Stroop models with masked fear stimuli (Study 2) ......................... 98
List of Figures

Figure 1. Main structural model with postpartum depression as mediator (Study 1) ..... 26
Figure 2. Adjusted structural model including later maternal depression (Study 1) ..... 28
Figure 3. Examining maltreatment subtypes in the full structural model (Study 1) ..... 32
Figure 4. Main structural model with postpartum depression (Study 2) ..... 61
Figure 5. Structural model including antenatal distress (Study 2) ..... 62
Figure 6. Relative influences of trauma subtypes on postpartum depression and subsequent outcomes (Study 2) ..... 64
Figure 7. Testing selective attention to fear as a potential mechanism linking maternal childhood trauma to postpartum depression (Study 2) ..... 65
Figure 8. Testing selective attention to masked fear stimuli in full model (Study 2) ..... 66
Figure 9. Main structural model with postpartum depression for child outcomes at 18 years (Study 1) ..... 91
Figure S10. Study selection flowchart in prior research synthesis ..... 103
Figure S11. Study assessment timeline in prior research synthesis ..... 109
Figure S12. Conceptual model in prior research synthesis ..... 126
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1. Introduction

1.1 Scope and impact of childhood maltreatment

Childhood maltreatment is a potent and common form of early trauma (Stoltenborgh, Bakermans-Kranenburg, Alink, & van Ijzendoorn, 2015), affecting over one million children in the US each year (Sedlak et al., 2010) and over 40 million children worldwide (Butchart, Phinney Harvey, Mian, Furniss, & Kahane, 2006). Exposure to childhood maltreatment has been shown to compromise healthy development across multiple critical domains, with detrimental effects persisting well into adulthood (Norman et al., 2012; Silverman, Reinhertz, & Giaconia, 1996). Short-term consequences of childhood maltreatment include physical injury (Cairns, Mok, & Welbury, 2005), emotional disturbance (Kazdin, Moser, Colbus, & Bell, 1985), and developmental delays (Trickett & McBride-Chang, 1995), as well as disrupted academic functioning and behavioral difficulties such as aggression, self-isolation, and delinquency (Lansford et al., 2007; Shonk & Cicchetti, 2001). The long-term consequences of maltreatment often include heightened risk for psychopathology of all kinds (Scott, McLaughlin, Smith, & Ellis, 2012), self-harm behavior (Klonsky & Moyer, 2008; Yates, Carlson, & Egeland, 2008), substance use (Lansford, Dodge, Pettit, & Bates, 2010) and chronic health problems (Springer, Sheridan, Kuo, & Carnes, 2007), as well as revictimization and/or violence towards others (Maxfield & Widom, 1996; Messman-Moore & Long, 2000; Widom, Czaja, & Dutton, 2008).
Correspondingly, the economic costs associated with childhood maltreatment – ranging from proximate burdens placed on the welfare, justice and foster care systems, as well as more distal costs on health and mental health care and in the form of lost workforce productivity and increased criminal activity – is estimated to exceed billions of dollars (Fang, Brown, Florence, & Mercy, 2012; Gelles & Perlman, 2012). Adding to this concerning picture, research suggests that childhood maltreatment not only produces negative outcomes for individuals during their lifetime, but also has consequences extending to the next generation (Roberts, O’Connor, Dunn, & Golding, 2004), thus potentially compounding the societal costs of maltreatment with each subsequent generation.

1.2 Intergenerational transmission of maltreatment and psychopathology

The traditional child abuse and neglect literature has long observed that parents who have experienced childhood maltreatment are much more likely to have children likewise exposed to maltreatment (Egeland, Jacobvitz, & Sroufe, 1988; Pears & Capaldi, 2001; Thornberry & Henry, 2013), a phenomenon referred to as the intergenerational transmission of maltreatment (Berzenski, Yates, & Egeland, 2014; Thornberry, Knight, & Lovegrove, 2012). Although a wide range of transmission rates has been documented (between 7% and 70%; Oliver, 1993), likely due to varied study methodologies as well as populations studied (Ertem, Leventhal, & Dobbs, 2000), the cumulative literature suggests that roughly one third of individuals maltreated as children go on to
perpetuate maltreatment in the next generation, an estimate about six times higher than for the general population (Kaufman & Zigler, 1987). Parents maltreated as children have also been found to demonstrate increased maltreatment potential (DiLillo, Tremblay, & Peterson, 2000; Hall, Sachs, & Rayens, 1998; Rodriguez & Tucker, 2011) in addition to actual occurrences of maltreatment. Moreover, there is evidence that parents who have experienced childhood maltreatment are at risk for disrupted parenting behaviors more broadly (Bert, Guner, & Lanzi, 2009), which can also contribute towards maladjustment and psychopathology in the next generation – thereby transmitting to children the negative sequelae of maltreatment in addition to maltreatment itself.

Together, these observations raise an important question for child welfare: What contributes to the intergenerational transmission of maltreatment and its negative sequelae? A better understanding of what facilitates intergenerational transmission would inform approaches to interrupt such transmission and reduce risks in the next generation. After all, while intergenerational continuity is a robust phenomenon, it is by no means inevitable (Kaufman & Zigler, 1987; Zuravin, McMillen, DePanfilis, & Risley-Curtiss, 1996). A majority of parents who have experienced childhood maltreatment do not maltreat their own children, thereby “breaking out” of the cycle (Egeland et al., 1988).
1.3 Potential role for postpartum depression in the intergenerational transmission pathway

Ever since the phenomenon of intergenerational transmission of maltreatment has been proposed, various studies have sought to explore relevant mechanisms. Classic explanatory frameworks include (1) social learning theory (Bandura, 1973), through direct modeling of abusive behavior, and (2) attachment theory (Ainsworth, 1979; Bowlby, 1978), through disrupted working models of relationships which in turn influence subsequent relationships, including with one’s children (Zeanah & Zeanah, 1989). In this dissertation, it is proposed that maternal factors in the perinatal period, occurring at the earliest point of intersection between generations, may fundamentally contribute to perpetuating maltreatment and its sequelae in the next generation.

Emerging findings from a separate stream of literature – perinatal mental health – may be relevant for understanding this hypothesis.

Perinatal mental health research is concerned with the study of prevalence, risk factors, and consequences of mental disorders during the perinatal period, common of which is postpartum depression (Wisner, Parry, & Piontek, 2002). Of relevance, women who have experienced maltreatment as children have been found at greater risk for postpartum depression (Dennis & Vigod, 2013; Garabedian et al., 2011; Meltzer-Brody, Bledsoe-Mansori, et al., 2013; Muzik et al., 2013; Seng et al., 2013). While childhood maltreatment is a risk factor for psychopathology across the life course (Widom, DuMont, & Czaja, 2007), it may particularly kindle depression in the postpartum period,
a time fraught with hormonal shifts (Hendrick, Altshuler, & Suri, 1998), physical recovery from childbirth (Brown & Lumley, 2000), and heightened demands of caregiving (Campbell, Cohn, Flanagan, Popper, & Meyers, 1992) which may also re-activate memories of early caregiving received. In turn, postpartum depression is increasingly recognized for its short- and long-term impacts on child outcomes (Murray & Cooper, 1996), even beyond later maternal depression in the child’s life (Hay, Pawlby, Angold, Harold, & Sharp, 2003; Murray et al., 2010). This may be due to disruptions in maternal caregiving and attachment (Field, 2010; Moehler, Brunner, Wiebel, Reck, & Resch, 2006) during a particularly foundational time for child development (Bagner, Pettit, Lewinsohn, & Seeley, 2010), with potential for lasting changes at the molecular and functional level (Blaze, Asok, & Roth, 2015; Kundakovic & Champagne, 2015).

Thus, while it is generally acknowledged that there is no single factor responsible for intergenerational transmission of maltreatment (Dixon, Browne, & Hamilton-Giachritsis, 2005b), maternal postpartum depression can be expected to be an important conduit for transmitting negative outcomes to children, given the sensitive timing of its occurrence. In addition, postpartum depression may also serve as a useful early marker – that is, a clinical endophenotype – for intergenerational risk, one that is amenable to detection and intervention. For instance, treating maternal depression in early childhood is suggested to reduce later maltreating behavior (McCann, Voris, & Simon, 1992). However, to date, no studies have formally tested postpartum depression
as a target in the pathway between maternal childhood maltreatment and a range of long-term child outcomes, including child victimization, physical health, and psychopathology.

1.4 Global perspective

One limitation of existing literature is that the bulk of research and theory on the intergenerational transmission of maltreatment and its negative sequelae has been generated in high-income countries (HICs). Similarly, a recent review of literature on maternal childhood maltreatment and perinatal mood disorders (Choi & Sikkema, 2015) found a predominance of studies published from HICs, particularly Canada, Australia, and the US. Less is known about the consequences of maternal childhood maltreatment on mothers and children in low- and middle-income countries (LMICs), despite that childhood maltreatment is a common occurrence in many of these settings (Cyr, Michel, & Dumais, 2013; Skeen & Tomlinson, 2013). For example, South Africa is known to have high rates of interpersonal trauma (Gass, Stein, Williams, & Seedat, 2011; Jewkes & Abrahams, 2002), including childhood abuse – with over a third of South African women reporting a history of childhood sexual abuse (Seedat, Van Niekerk, Jewkes, Suffla, & Ratele, 2009).

Investigating the effects of childhood maltreatment, including on maternal postpartum depression and subsequent child outcomes, merits attention in LMICs. Maternal postpartum depression is suggested to have especially negative effects on
growth and survival in low-resource settings (Rahman, Patel, Maselko, & Kirkwood, 2008). Additionally, a birth cohort study in South Africa found prospective linkages between maternal postpartum depression and compromised child psychological development, even after adjusting for later maternal depression (Verkuijl et al., 2014). However, no known research in LMICs has brought together a full model encompassing maternal childhood maltreatment, postpartum depression, and subsequent child outcomes. Such a model could strengthen the case for addressing maternal depression and maltreatment history during the postpartum period in order to optimize intergenerational outcomes, providing insight for early interventions in global settings.

1.5 Specificity of maltreatment subtypes in intergenerational transmission

A second limitation of the existing intergenerational transmission literature is that it has tended to examine maltreatment exposure aggregated as a whole, rather than specific to maltreatment subtypes (Berzenski et al., 2014). Childhood maltreatment can occur in the form of direct abuse (acts of commission) and/or neglect (acts of omission) (Leeb, Paulogetti, Melanson, Simon, & Arias, 2008). Physical abuse and sexual abuse are the most studied forms of childhood maltreatment, while emotional abuse and neglect are only beginning to receive greater attention as equally if not more pernicious childhood exposures (Hibbard et al., 2012). Accordingly, there has been a recent call for research paradigms testing the intergenerational transmission pathway to shift from examining the role of maltreatment exposure aggregated as a whole, to specific
maltreatment subtypes (Berzenski et al., 2014). It is possible that certain forms of maltreatment and their consequences are more likely to be transmitted via postpartum depression to the next generation.

Based on a review of prior literature (Choi & Sikkema, 2015), childhood physical abuse has often shown associations with depression in the postpartum period (Dennis & Vigod, 2013; Garabedian et al., 2011; Plaza et al., 2012), when mothers are interacting physically with their infants and can be reminded of their own care experiences. To date, childhood emotional abuse has also been relatively unexplored as a maternal risk factor, though it is recognized as having ostensibly worse effects on development and psychopathology over the life course (Hibbard et al., 2012). Notably, childhood emotional abuse has also been found to be strongly associated with postpartum depression (Edwards, Galletly, Semmler-Booth, & Dekker, 2008b; Plaza et al., 2012), even relative to other subtypes (Minnes et al., 2008). Thus, exploring the intergenerational consequences of specific forms of maternal childhood maltreatment, particularly emotional abuse, is a prime area of research. Comparative analyses would improve our understanding of which forms of childhood maltreatment are most likely to be carried forward into the next generation and which maternal maltreatment histories would be most relevant for perinatal intervention.
1.6 Specific aims

To capitalize on existing gaps in literature, my dissertation aimed to address the following research question: How does childhood maltreatment – and its specific forms – impact maternal depression in the postpartum period, in turn impacting the next generation’s exposure to maltreatment and/or other negative sequelae? This was a question ideally addressed using large-scale longitudinal studies with data collected from mothers and their children. In this dissertation, I report findings from two studies in different global settings that capitalized on existing longitudinal cohorts to examine how forms of childhood maltreatment affect mothers’ mental health during the postpartum period, in turn impacting their children’s risk of exposure to maltreatment as well as health and mental health outcomes. This research built on a published systematic literature review (Choi & Sikkema, 2015) which found that women who have experienced maltreatment in their own childhood are at elevated risk of mental disorders during the perinatal period, but revealed gaps in knowledge about the long-term implications for the next generation and role of particular maltreatment subtypes. The two studies are introduced next with their specific aims and hypotheses.

1.6.1 Aims for Study 1

Using a British longitudinal cohort of 1,116 mothers and their same-sex twin children followed since childhood up to 18 years of age, Study 1 explored the impact of
maternal histories of childhood maltreatment on postpartum depression and subsequent child outcomes.

Specific Aim #1: Explore maternal childhood maltreatment (overall and specific subtypes) as a risk factor for postpartum depression in this sample.

Hypothesis #1: Maternal childhood maltreatment would significantly predict postpartum depression. In particular, childhood emotional abuse and physical abuse are expected to have strong associations with postpartum depression as an outcome.

Specific Aim #2: Test the bridging role of postpartum depression between maternal childhood maltreatment and various child outcomes, specifically child (1) exposure to maltreatment, (2) internalizing symptoms, and (3) externalizing symptoms.

Hypothesis #2: Postpartum depression would significantly mediate the relationship between maternal childhood maltreatment and child outcomes, specifically child (1) exposure to maltreatment, (2) internalizing symptoms, and (3) externalizing symptoms.

Specific Aim #3: Conduct analyses to examine the combined and specific intergenerational effects of maternal childhood maltreatment subtypes.

Hypothesis #3: Emotional abuse and physical abuse subtypes were expected to have large associations with child outcomes as mediated by postpartum depression.
1.6.2 Aims for Study 2

The aim of Study 2 was to explore similar hypotheses in a sample of 150 South African mothers followed through pregnancy and into the first postpartum year. Study 2 sought to fill a current gap in the literature by contributing to sparse data on the relationship between maternal childhood maltreatment and postpartum depression in low- and middle-income countries (LMICs), and extending a preliminary look at more proximal child outcomes, which may be similar or different in such settings.

**Specific Aim #1:** Explore maternal childhood maltreatment (overall and specific subtypes) as a risk factor for postpartum depression in this sample.

**Hypothesis #1:** Maternal childhood maltreatment would significantly predict postpartum depression. In particular, childhood physical abuse and emotional abuse are expected to have strong associations with postpartum depression as an outcome.

**Specific Aim #2:** Test the bridging role of postpartum depression between maternal childhood maltreatment and various child and parent-child outcomes in the first postpartum year, including maternal-infant bonding, infant emotional/behavioral development, and infant growth.

**Hypothesis #2:** Postpartum depression would mediate the relationship between maternal childhood maltreatment and child and parent-child outcomes, specifically poorer maternal-infant bonding, infant emotional and behavioral symptoms, and less favorable infant anthropometric indicators in the first year of life.
Specific Aim #3: Conduct analyses to examine the combined and specific intergenerational effects of maternal childhood maltreatment subtypes.

Hypothesis #3: Emotional abuse and physical abuse subtypes were expected to have large associations with child outcomes as mediated by postpartum depression.

If postpartum depression is indeed a mechanism by which forms of maltreatment and/or their sequelae persist across generations, then intervening in the perinatal period to address maternal depression would be a key opportunity to interrupt intergenerational transmission processes. Such empirical evidence would underscore the postpartum period as a prime window to focus efforts in preventing adverse child outcomes.
2. Study 1: UK

2.1 Methods

2.1.1 Sample

Participants were mothers and children involved in the Environmental Risk (E-Risk) Longitudinal Twin Study, which tracks the development of a birth cohort of 2,232 British children. This cohort sample was drawn from a larger birth register of twins born in England and Wales in 1994-1995. Full details about the sample are reported elsewhere (Moffitt, 2002). Briefly, the E-Risk sample was constructed in 1999-2000, when 1,116 families (93% of those eligible) with same-sex 5-year-old twins participated in home visit assessments. Families were recruited to represent the UK population of families with newborns in the 1990’s, based on (a) residential location throughout England and Wales and (b) maternal age, with over-selection of teenaged mothers and under-selection of older mothers having twins via assisted reproduction. As such, higher-risk households were deliberately oversampled to compensate for their selective loss from the register due to non-response and likely attrition over time. At follow-up, the resulting sample of households was representative of the full range of socioeconomic conditions in the UK, as captured by a neighborhood-level socioeconomic index (ACORN; A Classification of Residential Neighborhoods, developed by CACI Inc. for commercial use in the UK). ACORN utilizes census and other survey-based geodemographic data to classify neighborhoods across the UK into five categories ranging from “wealthy achievers”
(Category 1), “urban prosperity” (Category 2) and “comfortably off” (Category 3) to “moderate means” (Category 4) and “hard-pressed” neighborhoods (Category 5). The ACORN distribution of households participating in the E-Risk study closely matched the nationwide distribution across all categories (Odgers et al., 2012), though underrepresented the “urban prosperity” category because such households are likely to be childless (e.g., young professionals).

2.1.2 Procedures

Home visit assessments began in 1999-2000 when children were 5 years old, and follow-up assessments were conducted when children were 7 (98% participation), 10 (96% participation), 12 (96% participation), and 18 years old (93% participation). Informed consent was initially obtained from mothers and assent given by the children through their 12-year assessment. At all phases, procedures were approved by the Joint South London and Maudsley and the Institute of Psychiatry NHS Research Ethics Committee.

2.1.3 Measures

2.1.3.1 Maternal childhood maltreatment

Mothers were administered the Childhood Trauma Questionnaire (CTQ) as part of a structured face-to-face interview. The CTQ (Bernstein & Fink, 1998) is a widely used 28-item scale that retrospectively measures exposure to maltreatment before the age of 18, including physical abuse, sexual abuse, emotional abuse, emotional neglect, and
physical neglect. Respondents were asked to rate various maltreatment experiences before age 18 on a five-point Likert scale, ranging from 1 = never true to 5 = very often true. An overall maltreatment score was available as a continuous variable (with possible scores ranging between 25-125), as were continuous scores for each of the five maltreatment subtypes (with possible scores ranging between 5-25). For descriptive purposes, dichotomous variables were created to reflect moderate-to-severe exposure to each of the childhood maltreatment subtypes, as per CTQ manual cut-offs (Bernstein & Fink, 1998). Substantial exposure to any childhood maltreatment was determined by moderate-to-severe exposure to at least one or more maltreatment subtypes.

2.1.3.2 Maternal postpartum depression

At the first assessment, mothers were interviewed about their lifetime depressive symptoms up to when the twins were 5 years old, using the Diagnostic Interview Schedule based on DSM-IV criteria (American Psychiatric Association, 2000). Mothers who met criteria for lifetime major depressive disorder were then asked to refer to the Life History Calendar (LHC) in order to specify the timing of their depressive episodes. The LHC is a reliable visual method for recalling the occurrence, timing and duration of life events, including psychopathology (Caspi et al., 1996). Specifically, the reliability of recalling depressive episodes using the LHC method was separately evaluated using a one-month test-retest and determined to be high, at 93% (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005). Mothers were asked to indicate whether they had experienced
depression during various reference periods, including the first year following the twins’ birth (i.e., postpartum year). For the postpartum depression variable, maternal reports of depression experienced specifically during the postpartum year were extracted into a dichotomous variable.

2.1.3.3 Child exposure to maltreatment

Child exposure to physical and sexual maltreatment by an adult was assessed using a validated structured interview protocol (Dodge, Bates, & Pettit, 1990) administered to mothers at each early phase of assessment (5 years, 7 years, 10 years, 12 years of twins’ lives). In this protocol, standardized questions were designed to sensitively and validly elicit information about potential maltreatment (e.g., “Do you remember any time when [your child] was disciplined severely enough that he or she may have been hurt?”). At each phase, any positive reports were probed by the interviewer for further details about the incident and to rule out accidental harm or harm from peers. This narrative information was documented in a dossier along with maternal narratives and any referrals made. Each dossier was maintained over phases of assessment and then independently reviewed at 12 years by two clinical psychologists to reach consensus about the likelihood of maltreatment occurrence anytime between five to 12 years of age. Inter-rater agreement between coders exceeded 90% initially and discrepancies were resolved through consensus review. Child exposure to harm was indexed in a three-level categorical variable: none, probable, and definite. For this study,
a dichotomous variable was created to reflect likely maltreatment exposure (probable or definite) versus no such exposure, as in previous research (Jaffee, Caspi, Moffitt, & Taylor, 2004).

2.1.3.4 Child internalizing symptoms

Child internalizing symptoms were measured at 12 years using the Child Behavior Checklist (CBCL) (Achenbach, 1991b; Achenbach & Rescorla, 2001) for mothers and the Teacher Report Form (TRF) (Achenbach, 1991a) for schoolteachers. Mother and teacher ratings were summed to provide a comprehensive measure of child symptomatology across settings, as in previous research (Cairns et al., 2005), yielding continuous scores on withdrawn and anxious/depressed subscales that were combined to form the overall internalizing problems scale.

2.1.3.5 Child externalizing symptoms

Child externalizing symptoms were measured at 12 years also using the CBCL for mothers (Achenbach, 1991b; Achenbach & Rescorla, 2001) and TRF for teachers (Achenbach, 1991a). Mother and teacher ratings were summed to provide continuous scores on aggressive and delinquent behavior subscales that were combined to form the overall externalizing problems scale.

2.1.3.6 Covariates

Later maternal depression, a key study covariate, was assessed in study mothers using the Life History Calendar across subsequent assessments. A variable was created
to reflect the number of years in which the mother was depressed between 1 and 10 years of the twins’ lives. Maternal age in years was recorded when twins were five years old. For this study, a new variable was computed to reflect maternal age when twins were born approximately five years earlier. Finally, maternal socioeconomic status was indexed using a standardized composite of family income, education, and social class indicators measured at the first assessment. These correlated indicators were found to load significantly onto a single latent factor (Trzesniewski, Moffitt, Caspi, Taylor, & Maughan, 2006). Based on the population-wide distribution of scores on this latent factor, mothers were divided into three tiers reflecting overall socioeconomic standing. This three-level categorical variable was coded in this study to reflect increasing levels of socioeconomic disadvantage. Regarding child characteristics, categorical information on child sex (male versus female) and twin zygosity status (monozygotic versus dizygotic) was also obtained at the first assessment.

2.1.4 Data analysis

2.1.4.1 Main analyses

Descriptive analyses were initially conducted in SPSS (SPSS IBM v.23, Armonk, NY) to understand the patterns of childhood maltreatment and depression as experienced by study mothers, and to characterize outcomes experienced by study children. Then, structural equation modeling (SEM) was conducted using Mplus v.7.3 (Muthén & Muthén, 1998-2014) to further examine the interrelationships between
maternal childhood maltreatment, maternal postpartum depression, and child outcomes. SEM allows for associations to be simultaneously evaluated rather than estimating multiple independent regressions, and also permits estimation of both direct and indirect effects (Bollen, 1987). Data from both twins were included in the SEM analyses. To account for nesting within mothers, the dataset was structured at the family level so that each twin’s data were included as variables within the same family case, with elder versus younger twin variables distinguished accordingly. Twin outcomes on a same variable (e.g., externalizing symptoms) were mapped with equal factor loadings onto a latent outcome variable, as consistent with the common fate model for dyadic data (Ledermann & Kenny, 2012; Peugh, DiLillo, & Panuzio, 2013), with factor mean and variance set to zero and one, respectively. As a sensitivity analysis for this twin-based approach, a similar structural model was initially tested where only one child from each pair was randomly selected for a singleton analysis without latent twin variables.

To test continuity of maltreatment and related sequelae across generations, path analyses were initially conducted using maximum likelihood estimation to examine maternal childhood maltreatment scores as a predictor of individual latent child outcomes. Then, an overall structural model was used to test the extent to which postpartum depression mediates the relationship between maternal history of childhood maltreatment and child exposure to maltreatment and subsequent child internalizing and externalizing symptoms. Specifically, maternal childhood maltreatment was
predicted to influence postpartum depression; postpartum depression was predicted to influence child harm exposure between the ages of 5 and 12; and child harm exposure was predicted to subsequently influence child internalizing and externalizing symptoms at 12 years. Direct paths were also estimated between maternal childhood maltreatment and child outcomes, and between postpartum depression and child internalizing and externalizing symptoms, to explore any influences above and beyond the proposed pathway. Child internalizing and externalizing symptom residuals were allowed to covary. A chi-square difference test revealed no significant worsening of model fit when outcome means and variances were specified as equal across elder and younger twins, supporting interchangeability, so this specification was preserved throughout model testing. Given the inclusion of categorical variables in the full model (e.g., postpartum depression), weighted least squares means and variance adjusted (WLSMV) estimation was used. All possible indirect effects of maternal childhood maltreatment on child outcomes were queried. Similar analyses were conducted for child outcomes at 18 years, however methods and results are reported in Supplemental Materials (S1.2).

SEM results were evaluated sequentially. First, to assess overall model fit, comparative fit indices (CFI/TLI, with acceptable values >.90) and the root mean square error of approximation (RMSEA, with acceptable values <.08) were examined as statistical tests of the goodness of fit of the overall model (Schreiber, Nora, Stage, Barlow, & King, 2006). Second, the direct effects of maternal childhood maltreatment on
child outcomes were examined. Third, the mediating effect of postpartum depression was evaluated. After this initial evaluation, maternal age and socioeconomic disadvantage were entered as covariates for all endogenous variables in the model. Finally, paths were added structurally to test the extent to which later maternal depression might carry the mediating effect of postpartum depression on child outcomes. This final model was again evaluated for overall fit as well as its direct and indirect effects. All effects were interpreted using standardized coefficients depending on the scale (continuous or categorical) of the independent variable.

2.1.4.2 Moderation analyses

To probe for potential differences in the resulting model across monozygotic (MZ: identical) and dizygotic (DZ: fraternal) twins, multiple-group testing was performed with the final model to determine whether pathways differed across MZ and DZ twins. Chi-square difference tests using the DIFFTEST function were conducted to compare a model in which all structural paths leading to child outcomes were constrained across MZ and DZ groups, against a model in which these parameters were free to vary. In each model, pathways between maternal variables (e.g., maternal trauma to postpartum depression) were fixed as equal across groups, as they were not theoretically suspected to differ between twin types. All other parameters were free to vary. If the chi-square difference statistic was non-significant, the more restricted model was considered acceptable given no substantial worsening in fit; conversely, a
significant result would suggest that imposing parameter restrictions led to significantly worsened model fit, such that groups should be considered separately.

Then, to probe for potential child sex differences in susceptibility to maternal risk, multiple-group modeling with similar constraints was used to determine whether any structural paths to child outcomes differed across male versus female twins.

2.1.4.3 Subtype analyses

To examine which maltreatment subtype(s) most strongly predicted postpartum depression and subsequent child outcomes, a similar structural model was tested to account for maltreatment subtypes using five predictor variables decomposed from the overall maltreatment variable (physical abuse; sexual abuse; emotional abuse; emotional neglect; physical neglect), to evaluate whether specific forms of maternal childhood maltreatment influence different child outcomes, both directly and indirectly through postpartum depression. Inter-correlations between maltreatment subtypes were automatically factored into the model given their specification as exogenous variables.

2.2 Results

2.2.1 Missing data

Among the 1,116 twin pairs, a subset had CTQ data completed by an individual other than the mother (e.g., father, grandparent). These cases were excluded from the present study because the CTQ data would not reflect maternal trauma and did not consistently correspond to a particular type of individual for reliable comparison. 1,038
twin cases had CTQ data that was completed by the mother herself. Of these, 22 cases had missing maternal data (eight on postpartum depression and 14 on CTQ) and were excluded from analysis, resulting in a final sample of 1,016 twin cases and their mothers. The subsample with missing data did not differ significantly from the final analytic sample on socioeconomic disadvantage, child sex, twin zygosity, or maternal age.

### 2.2.2 Descriptive findings

Sample characteristics are summarized in Table 1 for mothers and Table 2 for children. Of note, 18% of mothers endorsed clinical depression in the postpartum year, and the majority of these women (83%) reported at least some depression in later years. Almost one in four mothers (24%, N=248) experienced at least one type of childhood maltreatment at the moderate/severe level. The most common maltreatment subtype was emotional neglect (16%), followed by emotional abuse and sexual abuse (both 11%); histories of physical abuse were reported by 8% of mothers and 6% reported physical neglect. Among the children, there was a relatively even balance in gender as well as monozygotic versus dizygotic twins. One in five children was determined to have likely experienced physical or sexual harm between 5 to 12 years of age.
Table 1: Maternal characteristics (Study 1)

<table>
<thead>
<tr>
<th>Maternal variables</th>
<th>N = 1,016</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age; mean (s.d.)</td>
<td>28.0 (5.9)</td>
</tr>
<tr>
<td>Socioeconomic risk</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>33% (330)</td>
</tr>
<tr>
<td>Moderate</td>
<td>33% (336)</td>
</tr>
<tr>
<td>Low</td>
<td>34% (350)</td>
</tr>
<tr>
<td>Childhood maltreatment</td>
<td></td>
</tr>
<tr>
<td>Overall; mean (s.d.)</td>
<td>34.0 (15.9)</td>
</tr>
<tr>
<td>Physical abuse</td>
<td>8% (79)</td>
</tr>
<tr>
<td>Sexual abuse</td>
<td>10% (105)</td>
</tr>
<tr>
<td>Emotional abuse</td>
<td>11% (107)</td>
</tr>
<tr>
<td>Physical neglect</td>
<td>6% (59)</td>
</tr>
<tr>
<td>Emotional neglect</td>
<td>16% (161)</td>
</tr>
<tr>
<td>Postpartum depression</td>
<td>18% (180)</td>
</tr>
<tr>
<td>Any later depression</td>
<td>39% (396)</td>
</tr>
</tbody>
</table>

Table 2: Child characteristics (Study 1)

<table>
<thead>
<tr>
<th>Child variables</th>
<th>N = 2,032</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>51% (1044)</td>
</tr>
<tr>
<td>Male</td>
<td>49% (988)</td>
</tr>
<tr>
<td>Twin zygosity</td>
<td></td>
</tr>
<tr>
<td>Identical (MZ)</td>
<td>54% (1102)</td>
</tr>
<tr>
<td>Fraternal (DZ)</td>
<td>46% (930)</td>
</tr>
<tr>
<td>Harm exposure between 5-12 years</td>
<td>21% (422)</td>
</tr>
<tr>
<td>Internalizing symptoms at 12 years;</td>
<td>4.7 (3.8)</td>
</tr>
<tr>
<td>mean (s.d.)</td>
<td></td>
</tr>
<tr>
<td>Externalizing symptoms at 12 years;</td>
<td>6.9 (6.9)</td>
</tr>
<tr>
<td>mean (s.d.)</td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms at 18 years;</td>
<td>1.8 (3.0)</td>
</tr>
<tr>
<td>mean (s.d.)</td>
<td></td>
</tr>
<tr>
<td>Conduct disorder symptoms at 18</td>
<td>2.1 (2.3)</td>
</tr>
<tr>
<td>years; mean</td>
<td></td>
</tr>
</tbody>
</table>

Among mothers who reported postpartum depression, 40% (N=72/180) had at least one twin who was later exposed to maltreatment, compared to 22% (N=188/836)
among mothers without postpartum depression. Even in cases of discordant maltreatment exposure where only one twin was exposed to maltreatment, independent sample t-tests indicated that the twin who was exposed to maltreatment had slightly higher – but not significantly different – internalizing or externalizing scores than the non-exposed twin, suggesting that a shared context where at least one child is being maltreated may have consequences for both twins.

### 2.2.3 Structural findings

#### 2.2.3.1 Continuity of maltreatment and related sequelae across generations

In individual path analyses, maternal childhood maltreatment significantly predicted child harm exposure ($B=.32$, $p<.001$), internalizing symptoms ($B=.33$, $p<.001$) and externalizing symptoms ($B=.35$, $p<.001$), supporting continuity of maltreatment and related sequelae in the next generation. These significant paths persisted even when adjusted for maternal age and socioeconomic disadvantage.
2.2.3.2 Mediating effect of postpartum depression on child outcomes

The main structural model with postpartum depression as mediator (Figure 1; significant paths shown, with standardized coefficients) fit the data well, $\chi^2(20)=60.06$, $p<.001$, RMSEA=.04, CFI/TLI=.99/.98, explaining 15% of variance in child harm exposure, 20% of variance in child internalizing symptoms, and 24% of variance in child externalizing symptoms (all $p<.001$). With regards to direct effects, maternal childhood maltreatment significantly predicted postpartum depression ($B=.22$, $p<.001$). In turn, postpartum depression significantly predicted child exposure to harm ($B=.22$, $p<.001$), which then predicted child internalizing ($B=.27$, $p<.001$) and externalizing symptoms ($B=.34$, $p<.001$). Maternal childhood maltreatment also had significant direct paths to each child outcome ($p<.001$, see Figure 1 for standardized coefficients).
In terms of mediating effects, there was a significant indirect effect of maternal childhood maltreatment on child harm exposure through postpartum depression (B=.05, p=.002). For child internalizing symptoms, total indirect effects of maternal childhood maltreatment through the hypothesized model were also significant (B=.11, p<.001). Decomposition of specific indirect effects showed significant mediating pathways through the combined pathway of postpartum depression → subsequent child harm (B=.01, p=.011) and through child harm exposure alone (B=.07, p<.001). The indirect pathway through postpartum depression without child harm exposure was non-significant. For child externalizing symptoms, total indirect effects were similarly significant (B=.12, p<.001) and decomposition of specific indirect effects again showed significant mediating pathways through postpartum depression → subsequent child harm (B=.02, p=.004) and through child harm exposure alone (B=.09, p<.001). The indirect pathway through postpartum depression without child harm exposure was non-significant. A sensitivity analysis using only outcomes from one randomly selected child from each twin pair (not shown) revealed the same pattern of significant direct and indirect effects as in this twin-based model, thus the more comprehensive twin-based approach was conserved in subsequent analyses.

The model retained good fit even when maternal age and socioeconomic disadvantage were added as covariates, $X^2(26)=64.76$, p<.001, RMSEA=.038, CFI/TLI=.99/.98, and the overall pattern of significant direct and indirect effects
remained the same. In addition, socioeconomic disadvantage significantly predicted each of the child outcomes (B=.14, p=.010 for harm exposure; B=.20, p=.002 for internalizing symptoms; B=.32, p<.001 for externalizing symptoms), while maternal age was a significant predictor of child harm exposure (B=-.12, p=.004) such that younger mothers had a higher risk of later child harm. This adjusted model explained 19% of variance in child harm exposure, 22% of variance in child internalizing symptoms, and 30% of variance in child externalizing symptoms (all p<.001).

2.2.3.3 Exploring the potential role of later maternal depression

![Figure 2. Adjusted structural model including later maternal depression (Study 1)](image)

When later maternal depression was included structurally to the adjusted model so that it followed postpartum depression and was also allowed to predict each of the child outcomes (see Figure 2; significant paths shown, with standardized coefficients),
the resulting model continued to fit the data well, $X^2(30)=96.35$, $p<.001$, RMSEA=.047, CFI/TLI=.98/.97. In this model, maternal childhood maltreatment significantly predicted postpartum depression ($B=.32$, $p<.001$). Postpartum depression then significantly predicted later maternal depression ($B=.66$, $p<.001$) which in turn predicted child harm exposure ($B=.18$, $p=.027$), internalizing symptoms ($B=.20$, $p=.016$) and externalizing symptoms ($B=.15$, $p=.033$); however, postpartum depression was no longer a significant direct predictor of child outcomes. Maternal childhood maltreatment continued to have significant direct paths to each of the child outcomes ($p<.001$, see Figure 2 for coefficients). Socioeconomic disadvantage predicted later maternal depression ($B=.13$, $p=.004$), though not postpartum depression.

In terms of mediating effects, there was a significant total indirect effect of maternal childhood maltreatment on child harm exposure ($B=.07$, $p<.001$), specifically through the pathway that included both postpartum depression and later maternal depression ($B=.04$, $p=.032$). Similarly, there was a significant total indirect effect of maternal childhood maltreatment on child internalizing symptoms ($B=.10$, $p<.001$), through the specific pathways of postpartum depression → later maternal depression ($B=.05$, $p=.021$) and through child harm exposure only ($B=.05$, $p=.004$), but not solely through postpartum depression. The indirect pathway through postpartum depression → later maternal depression → child harm exposure to internalizing symptoms was marginally insignificant ($B=.008$, $p=.080$) after adjusting for covariates. For child
externalizing symptoms, there was a significant total indirect effect of maternal
crudlna childhood maltreatment (B=.11, p<.001), with specific pathways through postpartum
depression → later maternal depression (B=.03, p=.039), postpartum depression → later
maternal depression → child harm exposure (B=.01, p=.046), and through child harm
exposure only (B=.06, p<.001), but not solely through postpartum depression. With the
inclusion of later maternal depression, the model explained 20% variance in child harm
exposure, 24% variance in child internalizing symptoms, and 34% of variance in child
externalizing symptoms (all p<.001).

2.2.3.4 Testing the moderating effects of child characteristics

For the moderating effect of twin zygosity, chi-square difference tests using the
last structural model revealed no significant worsening in model fit from a freed model
(in which all structural paths to child outcomes were allowed to vary across identical
and fraternal twins) to the more constrained model (in which these paths were set as
equal), $\chi^2 \Delta(17)=18.06, p=.385$, suggesting no substantial difference between monozygotic
and dizygotic twins on outcome susceptibility to predictors within this model. Means
and variances of child outcomes were allowed to vary across MZ and DZ groups as
constraining these parameters produced worse model fit; according to univariate higher-
order statistics, DZ twins had comparable means to MZ twins but greater variances.

When the moderating effect of child sex was tested, chi-square difference tests
revealed a significant worsening in model fit from the freed model (in which all
structural paths to child outcomes were allowed to vary across male and female twins) to the more constrained model (in which these paths were set as equal), $X^2\Delta(17)=29.41$, $p=.031$. However, follow-up difference tests that constrained paths either from (1) maternal childhood maltreatment to child outcomes (direct effects) or (2) postpartum or later maternal depression to child outcomes (mediator effects) were non-significant, suggesting that these paths were comparable across male and female twins, at least when adjusting for all paths considered. Significant worsening in fit was found only when (3) paths from maternal demographic covariates to child outcomes were constrained across male and female twins, $X^2\Delta(6)=18.43$, $p=.005$. Inspection of unconstrained path coefficients between groups revealed that in this model, maternal socioeconomic disadvantage predicted child harm exposure among male twins ($p=.005$) but not female twins ($p=.993$); internalizing symptoms for female twins ($p=.005$) but not male twins ($p=.282$); and externalizing symptoms for both twin groups ($p<.001$). Younger maternal age at birth significantly predicted child harm exposure only for female twins ($p<.001$). These findings suggest some differential susceptibility of male versus female children to contextual risks but not to the overall intergenerational transmission of maltreatment through maternal depression. Again, means and variances were allowed to vary across male and female twins; as expected, boys on average had higher externalizing symptoms than girls.
2.2.3.5 Examining relative contributions of maltreatment subtypes

![Diagram](image)

Figure 3. Examining maltreatment subtypes in the full structural model (Study 1)

When all maltreatment subtypes were entered instead of overall maltreatment in the final structural model with covariates, the resulting model (Figure 3; significant paths shown, with standardized coefficients) fit the data well, $X^2(46)=104.94$, $p<.001$, RMSEA=.036, CFI/TLI=.98/.96, and explained comparable variance in child outcomes as the overall maltreatment model. Among maltreatment subtypes, maternal histories of emotional abuse and sexual abuse were significant predictors of postpartum depression ($B=.24$, $p<.001$ for emotional abuse; $B=.12$, $p=.005$) above and beyond the other subtypes, though when considered individually without the influences of other subtypes, each of the five maltreatment subtypes significantly predicted postpartum depression.
Postpartum depression continued to predict later maternal depression (B=.63, p<.001), which in turn predicted child harm exposure (B=.18, p=.012) and subsequent internalizing symptoms (B=.20, p=.010) and externalizing symptoms (B=.16, p=.011). Additionally, maternal history of emotional neglect had significant direct paths to child harm exposure (B=.12, p=.017), child internalizing symptoms (B=.10, p=.042), and marginally externalizing symptoms (B=.07, p=.060). Maternal history of physical abuse had a significant direct relationship with child externalizing symptoms (B=.14, p<.001).

In terms of mediating effects, there were significant total indirect effects on child harm exposure from maternal histories of emotional abuse (B=.06, p=.005) and sexual abuse (B=.03, p=.022), but not other subtypes. Specific indirect pathways through postpartum depression → later maternal depression were significant for emotional abuse (B=.03, p=.035) and marginally significant for sexual abuse (B=.01, p=.060). Total indirect effects on child internalizing symptoms through the model were significant from maternal emotional abuse (B=.04, p=.042) and emotional neglect (B=.04, p=.030), but not other subtypes. From emotional abuse, specific indirect pathways were significant through postpartum depression → later maternal depression and marginally for the full pathway through postpartum depression → later maternal depression → child harm (B=.006, p=.081). From emotional neglect, specific indirect pathways were non-significant, though marginally significant through child harm exposure (B=.03, p=.053). Total indirect effects on child externalizing symptoms through the model were
significant from maternal emotional neglect (B=.04, p=.017) and marginally emotional abuse (B=.04, p=.063), but not other subtypes. Emotional neglect had specific indirect effects on externalizing symptoms through child harm exposure only (B=.03, p=.03). From emotional abuse, specific indirect pathways through postpartum depression → later maternal depression and through postpartum depression → later maternal depression → child harm were both significant (B=.02, p=.04; B=.007, p=.048, respectively), even though the combination of all indirect pathways was not.

2.3 Discussion

2.3.1 Summary and integration

This study examined the mediating role of postpartum depression in the intergenerational transmission of maltreatment and its psychological sequelae. It drew on a large birth cohort that was nationally representative of household conditions across the UK, in which mothers were not selected for their trauma histories or depression risk. The fact that almost one in five of mothers in the sample endorsed clinically significant postpartum depression and nearly one in four had been exposed to at least one substantial form of childhood abuse or neglect supports the prevalence of these exposures at a population level and a need to understand how such exposures might affect outcomes in the next generation.

As expected, maternal childhood maltreatment was associated with greater risk for postpartum depression. In turn, postpartum depression significantly mediated the
relationship between maternal childhood maltreatment and child harm exposure between 5 to 12 years, with subsequent influences on child internalizing and externalizing symptoms at 12 years. Additionally, this mediating effect persisted even when accounting for the potentially confounding contributions of socioeconomic disadvantage, suggesting that the structural links observed between maternal childhood maltreatment, postpartum depression, and child outcomes were not simply explained by underlying levels of environmental adversity.

These core results integrate findings from two streams of literature: (1) maternal childhood maltreatment and postpartum depression, and (2) postpartum depression and child outcomes, towards a more nuanced framework of the intergenerational transmission of maltreatment. In the first stream of literature, history of exposure to childhood maltreatment has been associated with increased risk for postpartum depression across a range of studies (Alvarez-Segura et al., 2014; Choi & Sikkema, 2015).

In the second stream of literature, postpartum depression has been associated with early disruptions in maternal care that could predict a range of long-term child psychological outcomes (Forman et al., 2007; Logsdon, Wisner, & Pinto-Foltz, 2006; Lupien et al., 2011; Murray et al., 2011). In this study, postpartum depression was linked to child psychological outcomes mainly through later risk of child harm, a pathway that deserves interpretation.
Results from this study suggested that the mediating effect of postpartum depression from maternal childhood maltreatment on child harm and subsequent outcomes was primarily carried by the recurrence of later maternal depression, as consistent with recent literature (Agnafors, Sydsjö, deKeyser, & Svedin, 2012; Sanger, Iles, Andrew, & Ramchandani, 2015). Some mothers who continue to be depressed may directly engage in abusive behavior, perhaps due to limited emotional resources in response to child misbehavior (Burke, 2003; Shay & Knutson, 2008); however, an alternative explanation is they may also be less able to actively monitor child safety or effectively protect the child from a violent partner or abusive acquaintance. The majority of women with postpartum depression experienced at least some depression in later years, highlighting the persistent nature of maternal depression beyond the postpartum period. Chronicity of later maternal depression was then directly associated with risk of child harm exposure as well as internalizing symptoms and externalizing symptoms. The stronger effects of later maternal depression relative to postpartum depression might be explained in various ways. First, its occurrence is more proximal to child symptoms being assessed; developmental plasticity may have allowed intervening events to continue shaping outcomes beyond the postpartum period (Champagne, 2010). Second, it could be that cumulative exposure, as indexed by later maternal depression following postpartum depression, is particularly harmful (Halligan, Murray, Martins, & Cooper, 2007; Hay, Pawlby, Waters, & Sharp, 2008). Third, an interaction effect may
exist where early exposure to postpartum depression may actually sensitize some children with genetic vulnerabilities to develop psychopathology but only when encountering later stressors such as continued maternal depression (Starr, Hammen, Conway, Raposa, & Brennan, 2014), representing a G x E x E interaction.

Moreover, findings suggested that the overall intergenerational transmission pathway was robust to sex differences. Exploratory moderator findings suggested that male and female children differed only on their susceptibility to contextual influences such as maternal socioeconomic disadvantage. A growing body of literature has suggested boys may be more sensitive to early environmental adversity (DiPietro & Voegtline, 2015; Rutter, Caspi, & Moffitt, 2003; Sandman, Glynn, & Davis, 2013), including maternal psychopathology in the perinatal period (Choe, Sameroff, & McDonough, 2013; Korhonen, Luoma, Salmelin, & Tamminen, 2012; McGinnis, Bocknek, Beeghly, Rosenblum, & Muzik, 2015). However, differential susceptibility by child sex to maternal depression or history of childhood maltreatment was not supported in the current data. Lack of findings could reflect timing of child outcomes assessed. Another British longitudinal cohort (Quarini et al., 2016) also revealed no sex differences related to maternal postpartum depression for child depression outcomes at 12 years; rather, differences only emerged at 18 years, with boys showing greater long-term vulnerability following maternal postpartum depression.
When different maternal childhood maltreatment subtypes were considered together, emotional abuse emerged above and beyond other subtypes as a significant predictor of postpartum depression. While this was consistent with prior literature (Edwards et al., 2008b; Minnes et al., 2008) and study hypotheses, the present study extended this finding by examining its implications for child outcomes. Given that maltreatment subtypes often co-occur rather than occur in isolation, it is possible that when other forms of maltreatment are held constant, a history of emotional abuse is an indicator that the early maltreatment environment was particularly noxious since it also contained a strong psychological component in addition to any external harm. Literature increasingly recognizes the uniquely harmful effects of early emotional maltreatment (Hibbard et al., 2012; Spinazzola et al., 2014; Teicher, Samson, Polcari, & McGreenery, 2006), even after controlling for comorbidities with other subtypes (Spinhoven et al., 2010; Turner et al., 2012). Study findings also suggested that maternal exposure to emotional neglect during childhood has a particularly strong influence on later risk for child harm exposure and child internalizing symptoms, while maternal history of physical abuse was especially predictive of child externalizing symptoms above and beyond other subtypes. For clinicians, inquiring about childhood features of emotional abuse and neglect, in addition to reports of past physical or sexual abuse, may yield more nuanced insights about mothers and children at greatest risk.
As suggested in this study, specific facets of maltreatment such as the presence of emotional abuse or neglect may have particular associations with later risks during the transition to parenthood. Traditionally, intergenerational transmission research has examined broadly whether or not maltreatment has occurred in one generation and whether it reoccurs in the next generation, accruing a substantial evidence base (Kaufman & Zigler, 1987; Pears & Capaldi, 2001; Thornberry & Henry, 2013). More nuanced assessment of maternal maltreatment histories – not only subtypes experienced (Berzenski et al., 2014) but also the timing, severity, duration, and multiplicity of these experiences – is an increasingly compelling direction for the intergenerational transmission literature. The recently developed 52-item Maltreatment and Abuse Chronology of Exposure (MACE) scale (Teicher & Parigger, 2015) may be a useful tool for gathering this future data. Understanding different dimensions of maltreatment exposure in one generation and how they uniquely perturb developmental systems that may later affect psychopathology and/or parenting behavior (Andersen et al., 2008; Cicchetti, 2016) could help explain for whom there is continuity versus discontinuity in the next generation.

It must be noted that maternal childhood maltreatment continued to directly predict child outcomes above and beyond the mediating pathway, suggesting only partial mediation by maternal depression. This confirms that the intergenerational transmission of maltreatment and its consequences is a multifactorial phenomenon.
Additional mediators of the intergenerational transmission of maltreatment could include other corollaries of early maltreatment, such as hostile or controlling parenting styles (Delker, Noll, Kim, & Fisher, 2014), disorganized caregiving behaviors (Jacobvitz, Leon, & Hazen, 2006), substance abuse (Lo & Cheng, 2007), and increased risk for domestic violence, conflict and/or divorce (Bensley, Van Eenwyk, & Wynkoop Simmons, 2003; Colman & Widom, 2004), which also impact child development and psychopathology (Harold, Elam, Lewis, Rice, & Thapar, 2012; Holt, Buckley, & Whelan, 2008; Park & Schepp, 2014) without necessarily involving maternal depression or child harm. While individual risk factors require attention, algorithms of combined risk may be a promising direction for comprehensive prevention efforts.

2.3.2 Study limitations

Several limitations of this study should be noted. First of all, study assessments did not index severity of postpartum depression, which would capture a range of symptomatology and potentially be more informative in predicting later outcomes (Fihrer, McMahon, & Taylor, 2009). Maternal childhood maltreatment was also measured retrospectively with the CTQ, which may be subject to recall biases including under-reporting (Brewin, Andrews, & Gotlib, 1993). However, the CTQ has been found to correspond well to other sources of maltreatment information (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997) and should be considered a reasonable index of actual exposure. Second, as the E-Risk study was conducted in the UK, results and policy
implications may not be generalizable to other settings. For example, maternal and child health care systems in the UK are nationally managed, such that British mothers on average may be more able than those in the US to access supports and services throughout the course of their children’s development, potentially altering intergenerational transmission pathways. Additionally, the use of a twin-based sample could also limit generalizability to singleton families, despite sensitivity analyses using only one randomly selected twin from each pair. For example, parents of twins have higher risk for mental health difficulties than parents of singletons (Wenze, Battle, & Tezanos, 2015). However, findings from this study resonate with recent results from the US-based Nurses’ Health Study II (Roberts et al., 2015) which also found intergenerational links between maternal childhood maltreatment, maternal mental health, and child psychological outcomes in non-twin families.

This study also did not explicitly rule out the possibility of genetic mediation of intergenerational transmission, in which genetic factors are shared between mothers and their children that predispose them to similarly elicit maltreatment from their environment and/or demonstrate overall psychological vulnerability. For example, research has found child temperament, a putatively genetically determined characteristic (Saudino, 2005), may elicit both negative parenting behavior and parental depression (Dix & Yan, 2014). One indirect approach to examining genetic versus environmental mediation in this study is that if substantial genetic mediation was
present, it could be expected that maternal pathways to shared twin risk would be significantly stronger for monozygotic twins who fully share their genetic material than for dizygotic twins who only share half of their genetic material. In this study, monozygotic twins did not differ significantly from dizygotic twins on their shared susceptibility to maternal childhood maltreatment or maternal depression in terms of risk for harm exposure, externalizing symptoms or internalizing symptoms, suggesting some degree of environmental mediation. However, this transmission question could be further clarified with adoptive mother-child cohorts, as literature has suggested that adoptive mothers experience comparable levels of depression during their child’s first year of life (Mott, Schiller, Richards, O’Hara, & Stuart, 2011; Senecky et al., 2009). One drawback with using adoptive cohorts is not accounting for exposures that occurred during pregnancy, which could be addressed by additionally measuring antenatal experiences of birth mothers (Laurent et al., 2013) or alternatively recruiting egg donor in vitro fertilization (IVF) samples where the mother carries the pregnancy without being genetically related to the child. Converging evidence across these types of studies would yield more definitive answers.

2.3.3 Implications for treatment and prevention

In terms of implications for treatment and prevention, this study has highlighted the predictive utility of postpartum depression in the intergenerational transmission of maltreatment and its mental health sequelae. Study findings support recent
recommendations by the US Preventive Services Task Force (Siu & the U.S. Preventive Services Task Force, 2016) to screen for postpartum depression when appropriate follow-up options are available. Identifying and treating maternal depression in the earliest years may be an important way to interrupt cycles of trauma and improve both maternal and child outcomes: an efficient strategy from a public health standpoint.

At the same time, this study presents mixed findings on the postpartum period as a critical window, mechanistically, for later child outcomes. Consistent with a recent systematic review, the effect of postpartum depression tends to be conditioned on the occurrence of depression at other time points (Sanger et al., 2015). If early treatment gains are not maintained through later years, resolving postpartum depression may not be sufficient to interrupt intergenerational transmission of negative outcomes. For example, one study in Pakistan (Maselko et al., 2015) followed children’s cognitive, physical and emotional outcomes several years after their mothers had received a cognitive-behavioral intervention for perinatal depression in a randomized controlled trial. While this intervention significantly reduced levels of postpartum depression for mothers at the time, these treatment effects did not translate into longer-term improvements for their children when compared to controls whose mothers had not been treated for their postpartum depression. This finding echoed a systematic review that also found mixed evidence for long-term benefits of treating postpartum depression with regards to child psychopathology (Gunlicks & Weissman, 2008).
Thus, one relevant question for prevention is: (1) Can treatment effects for maternal depression be maintained, to warrant early intervention? Psychotherapy is generally indicated for postpartum depression due to breastfeeding and other concerns related to psychotropic use (Goodman, 2009) and generally yields more self-sustaining effects over time compared to pharmacological treatment (DeRubeis, Siegle, & Hollon, 2008; Dobson et al., 2008). However, relapse after a phase of psychotherapy is still common (Beshai, Dobson, Bockting, & Quigley, 2011; Vittengl, Clark, Dunn, & Jarrett, 2007). MBCT was developed specifically to prevent depression relapse (Morgan, 2003) and has been suggested as especially effective for individuals who have experienced childhood trauma (Williams et al., 2014), potentially because of its focus on experiential processing. Studies also support the use of MBCT for preventing relapse where there is indication of a chronic depression trajectory (e.g., multiple prior episodes), whereas problem-solving approaches such as interpersonal therapy may be sufficient for individuals with newer-onset situational depression (Piet & Hougaard, 2011). MBCT was recently piloted for treating perinatal depression (MBCT-PD) in women with prior depressive episodes (Dimidjian et al., 2016) and showed promising effects of reduced relapse/recurrence in the postpartum period. Follow-up research should be conducted to examine how mothers with a history of trauma respond to this intervention and whether its effects can extend beyond the postpartum period into later years.
A second relevant question for prevention is: (2) How does postpartum depression becomes recurrent, and for whom? Research suggests that postpartum depression is not a homogeneous condition (Kettunen, Koistinen, & Hintikka, 2014; Vliegen, Casalin, & Luyten, 2014). For a subset of women, the presence of postpartum depression is a marker of more chronic vulnerability for depression across the life course. Notably, trauma history is associated with chronic depression (McMahon, Barnett, Kowalenko, & Tennant, 2005; Negele, Kaufhold, Kallenbach, & Leuzinger-Bohleber, 2015; Wiersma et al., 2009). Additionally, a history of prior depressive episode(s) is predictive of persistent depression in mothers after childbirth (van der Waerden, Galera, Saurel-Cubizolles, Sutter-Dallay, & Melchior, 2015). Mothers with chronic depression trajectories should be clinically distinguished from those who are experiencing a more “classic” state of postpartum depression associated with hormonal shifts and acute life transition. While both groups of women require support and intervention, there is a two-generation impetus to specifically identify and follow mothers with chronic depression trajectories.

If early treatment effects can be sustained and it is possible to determine who is at greatest risk for recurrent depression, then identifying and treating postpartum depression with the goal of interrupting depressive trajectories among women who have experienced childhood maltreatment may provide one way to interrupt the continuity of maltreatment and subsequent psychopathology. Screening for maternal depressive
symptoms has been recommended in the postpartum period (Earls, 2010; Siu & the U.S. Preventive Services Task Force, 2016) with ensuing referrals to psychological treatment for postpartum depression. A treatment course of evidence-based psychotherapy that focuses on full remission (Keller, 2003) with options for relapse prevention and maintenance among at-risk women would likely translate into optimal long-term child outcomes. Psychosocial interventions that support breastfeeding (Hahn-Holbrook, Haselton, Dunkel Schetter, & Glynn, 2013; Kendall-Tackett, 2007a), self-care in the midst of caregiving demands (Woolhouse, Small, Miller, & Brown, 2016), and management of immediate stressors (Swendsen & Mazure, 2000) may also help alleviate postpartum depression, and could be delivered by home visiting nurses (Dodge et al., 2013; Olds, 2006). Successful efforts have been made to integrate depression treatment into regular home visiting (Ammerman et al., 2013) but notably the treatment effects appear to be attenuated by maternal histories of childhood maltreatment (Ammerman, Peugh, Teeters, Putnam, & Van Ginkel, 2016), suggesting that depressed mothers with trauma histories may require specialized attention. Finally, involvement of fathers in the prevention and treatment of postpartum depression is increasingly recognized as a priority (Fisher et al., 2016) as paternal depression in the postpartum period is suggested to independently impact later child psychopathology (Ramchandani et al., 2008), potentially through improving the overall family environment (Gutierrez-Galve, Stein, Hanington, Heron, & Ramchandani, 2015).
Alleviating postpartum depression in any of these ways could be protective against risk transmission. Postpartum interventions may be a strategic investment insofar as mothers may be more open to treatment and support during this time (Leis, Mendelson, Tandon, & Perry, 2009). At the same time, the postpartum period may just be one entry point along the intergenerational bridge. Similar interventions could be targeted in the antenatal period, where women are similarly accessible to health providers and systems of care. If women with a history of childhood maltreatment can be prospectively identified during pregnancy (Luoma, Korhonen, Salmelin, Helminen, & Tamminen, 2015) or even the preconception period (Patton et al., 2015), they can receive preventive interventions for depression (Sockol, Epperson, & Barber, 2013) and be more closely monitored for depressive symptoms across the perinatal period. Managing depression during pregnancy may be particularly advantageous as antenatal depression is not only predictive of postpartum depression but evidence is mounting that antenatal depression may biologically program long-term child outcomes independently of later depression (Pearson et al., 2013), though this was not possible to examine in the E-Risk cohort since maternal depression during pregnancy was not documented. Future research should factor in the potential role of antenatal depression in the intergenerational transmission pathway and test relative contributions of antenatal, postpartum and later maternal depression to better guide timing of interventions.
Given the likelihood of recurrent depression, continued screening for maternal depression throughout child’s life is also warranted, and pediatric settings offer a prime opportunity to detect ongoing maternal depression that could affect child development (Earls, 2010). However, less than half of pediatricians in the US report screening for maternal depression (Kerker et al., 2016), despite formal recommendations by the American Academic of Pediatrics. Further evidence on child health outcomes associated with recurrent maternal depression such as obesity and asthma (Giallo et al., 2015; Lampard, Francke, & Davison, 2014) could increase relevance and urgency for pediatricians. In addition to training health providers on the effects of maternal depression and suitable screening tools, improved reimbursement mechanisms for screening at health visits and delineation of subsequent referral options may also help increase provider involvement in this critical activity (VanLandeghem, 2006).

2.3.4 Conclusion

In conclusion, this study found that postpartum depression, especially when followed by recurrent maternal depression, plays a mediating role in the intergenerational transmission of maltreatment. Mothers who have experienced childhood maltreatment are at an increased risk for postpartum depression and their postpartum depression may also be more persistent and difficult to resolve, with downstream consequences for children’s wellbeing. Notably, children’s risk for exposure to maltreatment and subsequent mental health problems can be predicted as
early as the perinatal period, offering a promising window of opportunity for prevention. Some gaps in knowledge remain, including whether and how interventions targeting early maternal depression can yield enduring effects for children, or require maintenance throughout childhood. Nonetheless, interventions that address both depression and trauma in the context of caregiving are critically needed for mothers in the perinatal period (Ammerman et al., 2016; Muzik et al., 2015) and may assist ongoing efforts to interrupt intergenerational cycles of maltreatment.
3. Study 2: South Africa

3.1 Methods

3.1.1 Sample

This study drew on a longitudinal cohort of 150 South African women followed during pregnancy and through the first postpartum year. Participants were recruited from pregnant women presenting for their first antenatal care visit at a midwife obstetric unit (MOU) or health clinic in Cape Town, South Africa. Women were eligible if they were ≥18 years old and had a gestational age of <20 weeks, as determined by a routine dating ultrasound. Women were excluded if they had evidence of a high-risk pregnancy (e.g., non-singleton, history of pregnancy loss, history of pregnancy complications, severe current medical condition such as cardiac or renal failure).

3.1.2 Procedures

Participants were assessed longitudinally, beginning in the first trimester of pregnancy (13-14 weeks) if presenting this early; second trimester (21-22 weeks); and the third trimester (32-33 weeks). They were then assessed 3-5 days post-delivery; at 6 weeks; at 6 months; and 1 year postpartum. Assessments were conducted in a private room adjacent to the clinic and all assessment items were verbally administered by a research assistant in either Afrikaans or English, depending on the woman’s preferred language of administration. Informed consent was obtained from all women prior to
participation, and procedures were approved by the ethics committee at the University of Stellenbosch.

### 3.1.3 Measures

#### 3.1.3.1 Maternal childhood trauma

Mothers reported their exposure to childhood abuse or neglect via the Childhood Trauma Questionnaire (CTQ), administered during the second trimester of pregnancy. The CTQ has been previously administered to South African women (Spies & Seedat, 2014), including in antenatal care settings (Choi, Sikkema, et al., 2015; Koen et al., 2014). As described in Study 1, this scale measures exposure to early maltreatment, including physical abuse, sexual abuse, emotional abuse, emotional neglect, and physical neglect. An overall maltreatment score is available as a continuous variable, as are continuous scores for each of the five maltreatment subtypes. Internal consistency of CTQ items was .73. For descriptive purposes, cut-offs for maltreatment subtypes at the moderate/severe level were applied per manual guidelines (Bernstein & Fink, 1998): ≥ 10 for physical abuse, ≥ 8 for sexual abuse, ≥ 13 for emotional abuse, ≥ 10 for physical neglect, and ≥ 15 for emotional neglect. In addition, exposure to one or more maltreatment subtypes was used to create a dichotomous variable reflecting any history of maternal childhood trauma.
3.1.3.2 Maternal postpartum depression

The Edinburgh Postnatal Depression Scale (EPDS) is a 10-item screening tool administered to mothers at each postpartum assessment (3-5 days post-delivery, 6 weeks, 6 months, and 1 year postpartum). The EPDS is widely administered across global research settings and has been validated for postpartum use in South Africa (De Bruin, Swartz, Tomlinson, Cooper, & Molteno, 2004; Lawrie, Hofmeyr, De Jager, & Berk, 1998). Items are scored on a four-point Likert scale ranging from 0 to 3, with reverse scoring on certain items. Scored items were summed to create a continuous total score ranging from 0 to 30, with higher scores reflecting greater depressive symptomology. Internal scale consistency in this sample ranged from .83 to .86 across time points. For descriptive purposes, a recommended cut-off for possible postpartum depression in South African women is a score of 12 or higher (De Bruin et al., 2004; Lawrie et al., 1998) and was used to create dichotomous variables reflecting the presence of postpartum depression at each time point. For this study, continuous EPDS scores at 3-5 days, 6 weeks and 6 months were selected as predictors of study outcomes in order to minimize conflation with reported outcomes at 1 year.

3.1.3.3 Maternal-infant bonding

Maternal-infant bonding at 1 year was measured using the Postpartum Bonding Questionnaire (Brockington et al., 2001). The PBQ is a validated screening tool (Brockington, Fraser, & Wilson, 2006) that measures potential disruptions in the mother-
infant relationship. The PBQ has been used with postpartum women in South Africa (Rotheram-Borus et al., 2014) and consists of 25 items that assess maternal feelings about her infant (e.g., “I feel distant from my baby,” “I regret having this baby,” “This baby doesn’t seem to be mine”). Items are rated on a 6-point Likert scale from “always” to “never.” Internal consistency of items was high (Cronbach’s alpha = .86) in the current sample. Following the coding scheme, scale items were summed to yield an overall continuous score, with higher scores reflecting greater disruptions in bonding. For descriptive purposes, a cut-off of 12 or higher on the bonding impairment subscale of the PBQ (Brockington et al., 2006) was used to estimate the proportion of mothers experiencing substantial bonding difficulties with their infant.

3.1.3.4 Child emotional/behavioral development

Symptoms of emotional and/or behavioral difficulties were broadly assessed using the Infant/Toddler Symptom Checklist (ITSCL) administered to mothers at 1 year. The ITSCL (DeGangi, Poisson, Sickel, & Wiener, 1995) is a validated screening tool that can be used with children aged 7 to 30 months, with score cut-offs specific for different age groups including 10 through 12 months as relevant for this study. The ITSCL assesses perceived child developmental difficulties in nine different domains, including self-regulation and socioemotional functioning, and has demonstrated predictive validity for later child problems (DeGangi, Breinbauer, Roosevelt, Porges, & Greenspan, 2000). The subscale for infant self-regulation consisted of nine items (e.g., “frequently
irritable and fussy,” “can’t change from one activity to another without distress,” “can’t calm self effectively”) and demonstrated acceptable internal consistency in the present sample (Cronbach’s alpha = .71). The subscale for infant socioemotional functioning consisted of 15 total items across all age groups, with eight items noted as appropriate for children at 12 months (e.g., “avoids eye contact,” “does not interact reciprocally,” “anxious/fearful”). These eight items demonstrated modest internal consistency (Cronbach’s alpha = .57) in the present sample. For descriptive purposes, a cut-off score of 4 was recommended for children at 12 months (DeGangi et al., 1995) to indicate potential problems in each domain.

3.1.3.5 Child physical growth

Infant anthropometric indicators (weight and length) were measured at one year. Exact physical measurements were conducted using conventional clinical practice, with most clothing removed before measurement. Infant weight was measured in kilograms using a standard electronic baby scale, while length was measured in centimeters using a standard measuring tape starting from the top of the head and following the body curve down the back towards the heel. For this study, standardized z-scores for infant weight-by-age and length-by-age were calculated based on World Health Organization age- and gender-normed growth standards using an SPSS macro (WHO Anthro, 2011).
3.1.3.6 Covariates

Antenatal distress was measured in the second trimester using the Kessler Psychological Distress Scale (K-10) (Kessler et al., 2002), a 10-item scale that assesses general symptoms of anxiety and depression. Items are rated on a 5-point Likert scale, ranging from “none of the time” to “all of the time,” with possible total scores ranging between 10-50. The K-10 has been validated in South Africa in pregnant populations (Spies et al., 2009), and showed high internal consistency in this sample (Cronbach’s alpha = .87). A cut-off of 25 was used for descriptive purposes to indicate clinically significant levels of distress at a moderate/severe level (Kessler et al., 2002). Finally, maternal age and education were self-reported during pregnancy and included as demographic covariates.

3.1.4 Analyses

3.1.4.1. Main analyses

Descriptives analyses were first conducted in SPSS (SPSS IBM v. 23, Armonk, NY) to characterize maternal exposures and child outcomes. Then, simple path analyses were conducted to examine the relationship between maternal childhood trauma and postpartum depression, as well as each child outcome. Next, structural equation modeling (SEM) was conducted in Mplus 7.3 (Muthén & Muthén, 1998-2014) using maximum likelihood (ML) estimation. A main structural model was tested with maternal childhood trauma predicting postpartum depression, and postpartum
depression predicting each of the three infant outcomes. Maternal depression scores across the first three assessments (through six months postpartum) were regressed onto a latent postpartum depression factor. Weight-for-age and height-for-age z-scores at one year were also regressed onto a latent infant growth factor, while subscale scores on infant self-regulation and socioemotional functioning were regressed onto an infant development factor. All factors had their means and variances standardized to zero and one, respectively. Infant growth at one year was also adjusted for birth weight to account for potential baseline differences. The variances of exogenous variables were explicitly estimated in the model estimation for distributional assumptions. Overall model fit was assessed using comparative fit indices (CFI/TLI, with acceptable values >.90) and the root mean square error of approximation (RMSEA, with acceptable values <.08) (Schreiber et al., 2006), followed by inspection of direct and indirect effects. All effects were interpreted using standardized coefficients depending on the scale (continuous or categorical) of the independent variable. Following initial evaluation, antenatal distress was included structurally so that it was regressed on maternal childhood trauma and predictive of postpartum depression. Finally, the resulting model was adjusted for maternal age and education.

3.1.4.2 Subtype analyses

To probe the relative influence of trauma subtypes on the pathway, the five subtypes (physical abuse, sexual abuse, emotional abuse, physical neglect, and
emotional neglect) were simultaneously entered in the final structural model with covariates, to examine direct effects on postpartum depression and indirect effects on child-related outcomes through postpartum depression. Inter-correlations between trauma subtypes were automatically factored into the model given their specification as exogenous variables.

3.1.5 Exploratory sub-study: Testing a potential mechanism

Selective attention to negative emotional stimuli in the postpartum period was investigated as a potential mechanism underlying postpartum depression and subsequent outcomes among women with a history of childhood trauma. Data on a computerized emotional Stroop task (van Honk et al., 2000) was available for a subset of participants (N=33) at 6 weeks postpartum. This subset did not differ significantly on sociodemographic characteristics (e.g., age, marital status, employment status) or risk variables (e.g., trauma history, depressive symptoms, antenatal distress) from the subset that did not complete the lab-based procedures, so the latter were considered to be missing at random in full-sample analyses.

In the emotional Stroop task, participants were presented with a set of emotional faces from the standardized Ekman and Friesen’s Pictures of Facial Affect (Ekman & Friesen, 1976). After a practice round, sixty colored faces were presented in total over the course of six trials, with two trials for each major emotion (e.g., fearful, happy, angry) – one unmasked (conscious) and one masked (unconscious) trial. Each trial included 10
faces (5 male, 5 females) and incorporated neutral facial expressions in order to calculate relative reaction times. In each trial, emotional and neutral faces were displayed randomly on a computer screen at eye-level at a distance of 60 centimeters from the participants' face. For each unmasked display, participants viewed a fixation cross for 750 milliseconds before being shown the target facial expression. During this time, participants were asked to name out loud the color of the face that appeared (i.e., red, green, or blue) as quickly as possible, and their responses were automatically registered by lab audio-equipment. For each masked display, participants were briefly shown the target facial expression for 25 milliseconds before it was replaced by a masking shape of the same color. Again, participants were asked to name the color of the shape that appeared as quickly as possible, and their responses were automatically registered by lab audio-equipment.

Selective attention scores were calculated based on participants’ response times for emotional faces minus their response times for neutral faces. Positive scores indicated increased response latency (longer response times) for emotional faces relative to neutral faces, suggesting more time spent paying attention to the emotional stimuli. On the other hand, negative scores indicated decreased response latency (shorter response times) for emotional faces relative to neutral faces. Outlier response times over 900ms were excluded from analysis, as in previous research (Putman, Hermans, & van Honk, 2004).
For this study, selective attention scores for fearful stimuli were examined. In separate models, selective attention scores for fear stimuli (masked and unmasked) were entered as mediating variables between maternal childhood trauma and the latent postpartum depression variable. As before, EPDS scores at the first two time points were allowed to covary.

3.2 Results

3.2.1 Descriptive findings

Nearly half of the mothers reported being single (48%) and just over half (53%) reported some form of employment. The average age was 25 (s.d. = 5.8) and only 41% had completed Grade 12. Descriptive data for key maternal predictors are summarized in Table 3. Rates of childhood trauma were elevated in this sample, with 57% of women reporting exposure to at least one substantial form of maltreatment. Physical neglect was the most common experience (35%), followed by emotional neglect (23%) and sexual abuse (23%). Over one in four women met the cut-off for postpartum depression at any given time point, with the highest proportion of women at 6 months (41%).
Table 3. Descriptive findings on maternal predictors (Study 2)

<table>
<thead>
<tr>
<th>Continuous score, mean (s.d.)</th>
<th>Met cut-off, % (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall maltreatment</td>
<td>43(14.7)</td>
</tr>
<tr>
<td>Physical abuse</td>
<td>6.9(3.3)</td>
</tr>
<tr>
<td>Sexual abuse</td>
<td>7.1(4.5)</td>
</tr>
<tr>
<td>Emotional abuse</td>
<td>9.4(4.5)</td>
</tr>
<tr>
<td>Physical neglect</td>
<td>8.8(3.4)</td>
</tr>
<tr>
<td>Emotional neglect</td>
<td>10.8(5.1)</td>
</tr>
<tr>
<td>Antenatal distress</td>
<td>22(7.5)</td>
</tr>
<tr>
<td>Postpartum depression</td>
<td></td>
</tr>
<tr>
<td>3-5 days (N=92)</td>
<td>9.0(6.1)</td>
</tr>
<tr>
<td>6 weeks (N=124)</td>
<td>10.8(6.4)</td>
</tr>
<tr>
<td>6 months (N=119)</td>
<td>9.3(5.8)</td>
</tr>
<tr>
<td>1 year (N=107)</td>
<td>9.7(5.8)</td>
</tr>
</tbody>
</table>

There was a fairly even balance of female and male children in the sample (55% versus 45%, according to available data at one year). Continuous child-related outcomes are summarized in Table 4. About 10% of mothers met the cut-off for bonding impairments with their infant. 24% of infants with available data met the cut-off for difficulties with emotional functioning, while 43% met the cut-off for difficulties with self-regulation.

Table 4. Descriptive findings on child-related outcomes (Study 2)

<table>
<thead>
<tr>
<th></th>
<th>Mean (s.d.)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bonding</td>
<td>9.0(11.8)</td>
<td>0-91</td>
</tr>
<tr>
<td>Development</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-regulation</td>
<td>3.4(3.6)</td>
<td>0-14</td>
</tr>
<tr>
<td>Emotional functioning</td>
<td>1.8(2.2)</td>
<td>0-14</td>
</tr>
<tr>
<td>Growth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>10.01(1.5)</td>
<td>5.6-13.9</td>
</tr>
<tr>
<td>Length (cm)</td>
<td>76.5(5.2)</td>
<td>64-88</td>
</tr>
</tbody>
</table>
3.2.2 Main findings

3.2.2.1 Influence of maternal childhood trauma on child outcomes

In individual path analyses, maternal childhood trauma predicted mother-infant bonding (B=.22, p=.016) and infant developmental difficulties (B=.22, p=.040) but did not directly predict infant growth. Infant growth, however, was predicted by postpartum depression, which was associated with maternal childhood trauma, so was included in subsequent models to explore potential indirect effects through postpartum depression.

3.2.2.2 Mediating effect of postpartum depression

![Figure 4. Main structural model with postpartum depression (Study 2)]

When postpartum depression was examined as a link between maternal childhood trauma and child outcomes, the model (Figure 4; significant paths shown, with standardized coefficients) fit the data adequately, $X^2(29)=40.44$, p=.077, RMSEA=.055, CFI/TLI=.95/.93. A single modification index suggested that postpartum
depression indicators at 3-5 days and 6 weeks should be allowed to covary, which made theoretical sense due to their proximity in time versus 6 months. The updated model did not alter the pattern of effects but fit the data more closely, $X^2(28)=29.24$, $p=.40$, RMSEA=.017, CFI/TLI=.995/.992, and was therefore retained. In this model, maternal childhood trauma significantly predicted postpartum depression ($B=.31$, $p=.001$), which in turn predicted bonding problems ($B=.59$, $p<.001$), developmental difficulties ($B=.29$, $p=.018$), and physical growth ($B=-.38$, $p<.001$) above and beyond infant birth weight. In terms of mediating effects, there were significant indirect effects from childhood trauma through postpartum depression to bonding problems ($B=.18$, $p=.003$) and physical growth ($B=-.12$, $p=.015$), but marginally not infant development ($B=.09$, $p=.061$).

Figure 5. Structural model including antenatal distress (Study 2)

When accounting structurally for antenatal distress (Figure 5; significant paths shown, with standardized coefficients), the model continued to fit well, $X^2(33)=39.72$,
p=.196, RMSEA=.037, CFI/TLI=.98/.96. Maternal childhood trauma predicted antenatal distress (B=.17, p=.027) and continued to predict postpartum depression (B=.22, p=.019) above and beyond antenatal distress, though as expected, antenatal distress also highly predicted postpartum depression (B=.47, p<.001). Accounting for antenatal distress, postpartum depression still predicted bonding problems (B=.74, p<.001) and physical growth (B=.35, p=.009), while antenatal distress actually predicted bonding problems in the reverse direction (B=-.28, p=.014) but not other outcomes. Postpartum depression no longer predicted infant developmental difficulties, but developmental difficulties covaried significantly with bonding problems (B=.26, p=.046) after bonding was predicted by both antenatal distress and postpartum depression. In terms of mediating effects, there were significant total indirect effects from maternal childhood trauma to bonding (B=.18, p=.015) and physical growth (B=-.12, p=.015). For bonding, there was a significant specific indirect path through postpartum depression only (B=.16, p=.025), whereas for physical growth, specific indirect paths were non-significant, suggesting a cumulative effect of all pathways in the model. Finally, these direct and indirect effects remained significant even after adjusting the model for maternal age and education level. Childhood maternal trauma continued to predict postpartum depression; and depression continued to predict bonding problems as well as infant growth. In addition, lower education levels significantly predicted developmental difficulties (B=-.28, p=.019) and antenatal distress (B=-.26, p=.001).
3.2.2.3 Subtype findings

When maternal trauma subtypes were simultaneously entered as predictors into the main structural model with postpartum depression, the model (Figure 6; significant paths shown, with standardized coefficients) continued to fit well, $\chi^2(56)=55.28$, $p=.502$, RMSEA=.000, CFI/TLI=1.0/1.0. Among trauma subtypes, history of emotional neglect was significantly predictive of postpartum depression ($B=.27, p=.015$) above and beyond the other subtypes. Postpartum depression continued to predict bonding ($B=.63, p<.001$), development ($B=.31, p=.015$), and growth ($B=-.39, p<.001$). Accounting for other paths in the model, maternal history of emotional neglect had a significant indirect effect on maternal-infant bonding ($B=.17, p=.026$) and physical growth ($B=-.10, p=.047$) through postpartum depression. No other trauma subtypes had significant direct or indirect

**Figure 6. Relative influences of trauma subtypes on postpartum depression and subsequent outcomes (Study 2)**
effects in the model after accounting for influences of the other subtypes. However, when considered individually without the influences of other subtypes, maternal childhood emotional abuse, physical neglect, and emotional neglect each predicted postpartum depression and had similar significant indirect effects on bonding and growth through postpartum depression.

3.2.4 Exploratory mechanism findings

![Diagram](image)

**Figure 7.** Testing selective attention to fear as a potential mechanism linking maternal childhood trauma to postpartum depression (Study 2)

For selective attention to masked fear stimuli, the model (Figure 7) fit well, $\chi^2(4) = 5.06, p = .28, \text{RMSEA} = .042, \text{CFI/TLI} = .99/.97$. Maternal childhood trauma inversely predicted selective attention to masked fear stimuli at six weeks ($B = -39, p = .002$), such that greater levels of trauma history were associated with decreased response latencies. In turn, selective attention to masked fear stimuli inversely predicted postpartum depression scores through six months ($B = -47, p = .001$), such that decreased response latencies predicted higher levels of depression. The indirect pathway from maternal childhood trauma to postpartum depression through selective attention was significant ($B = .19, p = .030$). On the other hand, the model for selective attention to unmasked fear stimuli fit inadequately, $\chi^2(4) = 10.06, p = .040, \text{RMSEA} = .100, \text{CFI/TLI} = .93/.83$. In this model
(not shown), maternal childhood trauma inversely predicted selective attention to unmasked fear stimuli ($B=-.38$, $p=.024$), but selective attention did not significantly predict postpartum depression, though the coefficient was in negative direction as before. Thus, the indirect pathway was also non-significant.

![Diagram of mediation model]

**Figure 8. Testing selective attention to masked fear stimuli in full model (Study 2)**

Next, selective attention to masked fear stimuli was entered into the basic mediating model tested earlier with child-related outcomes (Figure 8). The model fit closely $X^2(37)=36.41$, $p=.496$, RMSEA=.000, CFI/TLI=1.0/1.0. In addition to the direct paths tested previously, which remained significant, indirect paths were significant through the combined pathway of selective attention and subsequent postpartum depression for bonding ($B=.12$, $p=.025$) and infant growth ($B=-.08$, $p=.046$). Interestingly, when examining subtypes, selective attention to masked fear stimuli was most significantly
correlated with a history of physical neglect \((r=.42, p=.017)\) and also associated in the same direction with emotional neglect \((r=-.28, p=.128)\) though not significantly.

### 3.3 Discussion

#### 3.3.1 Summary and integration

Using a longitudinal cohort of South African women recruited in antenatal care and followed through the first postpartum year, this study presents preliminary evidence about the intergenerational impact of maternal childhood trauma on infant outcomes in a non-Western setting, with perinatal mental health as a key mediator. In this study, maternal childhood trauma predicted postpartum depression, which then predicted a range of child outcomes, particularly mother-infant bonding and infant growth. Together, findings suggested that postpartum depression might be one way in which trauma in one generation can indirectly impact the next generation.

Postpartum depression through the first six months strongly predicted mother-infant bonding problems at one year. It is plausible that negative affect associated with depression (Joormann & Siemer, 2011) influenced maternal interactive behavior as well as perceptions of bonding. This is noteworthy as maternal caregiving behavior and negative attitudes towards her baby can predict later child problems (Bor, Brennan, Williams, Najman, & O’Callaghan, 2003). Postpartum depression also initially predicted child emotional/behavioral development at one year, as consistent with larger studies in South Africa that have found stable associations between postpartum depression and
child long-term psychological development (Verkuijl et al., 2014); however, this association did not persist after adjusting for covariates. This may be due to the inclusion of maternal-infant bonding, which covaried significantly with infant development throughout subsequent models and may have accounted for the otherwise observed association.

Postpartum depression was also negatively associated with child growth as indexed by standardized height and weight indicators, even after controlling for initial birthweight. Adjusting for birthweight suggests that differences in infant growth were shaped by exposures after birth such as maternal depression. This finding is consistent with existing literature that suggests postpartum depression affects child health outcomes in resource-poor settings (Stewart, 2007; Surkan, Kennedy, Hurley, & Black, 2011) and adds to sparse evidence specifically from South Africa. One early study in Cape Town (Tomlinson, Cooper, Stein, Swartz, & Molteno, 2006) found no relationship between postpartum depression and infant growth after adjusting for birthweight; however, authors looked at maternal depression at 2 months as opposed to a longer timeframe across the postpartum year, and used infant weight as the growth outcome instead of both weight and length indicators. On the other hand, a more recent study (Avan, Richter, Ramchandani, Norris, & Stein, 2010) with a large birth cohort in Johannesburg reported a significant relationship between postpartum depression at six months and stunted child growth at two years, as captured by a set of similarly
standardized anthropometric indicators. Postpartum depression has been associated with early breastfeeding cessation (Dennis & McQueen, 2007) as well as disrupted care practices (Field, 2010), all of which may impact child nutrition and growth. Postpartum depression may also negatively affect immunological parameters (e.g., SlgA) transmitted directly through maternal breast milk (Kawano & Emori, 2015), increasing child risk for illness and infection (Breakey, Hinde, Valeggia, Sinofsky, & Ellison, 2015).

Additionally, the influences of maternal childhood trauma on postpartum depression and of postpartum depression on child outcomes persisted after adjusting for antenatal distress. In Western literature, maternal distress during pregnancy has been found to uniquely predict child emotional/behavioral adaptation, possibly through in-utero biological programming of reactivity to the environment (Waters, Hay, Simmonds, & Goozen, 2014). In this study, while antenatal distress was predictive of postpartum depression it did not independently relate to child outcomes except for inversely predicting bonding problems when adjusted for postpartum depression. Antenatal distress also did not predict child birthweight, contrary to an international meta-analysis (Grote et al., 2010) or a recent South African birth cohort (Brittain et al., 2015). Since only a general measure of antenatal distress with combined anxiety and depressive features was used in this study, it may not have thoroughly captured facets of maternal depression or anxiety that would have impacted gestational growth.
Early experiences of physical and emotional neglect were particularly common in this sample of South African mothers. This aligns with evidence from the Western Cape, which confirms high levels of material disadvantage in local townships in the post-apartheid era (Carter & May, 2001). In this wine-growing region where farmworkers were historically paid their wages in alcohol (London, 1999), alcohol misuse and frequent attendance at alcohol-serving venues are not uncommon among adults who drink (Martinez, Røislien, Naidoo, & Clausen, 2011; Schneider et al., 2007) and have been related to marked parental absence both physically and psychologically from homes and ensuing neglect of children’s material and emotional needs (Choi, Watt, Skinner, Kalichman, & Sikkema, 2015; Seedat et al., 2009). In this study, a history of childhood emotional neglect was particularly related to postpartum depression and subsequent outcomes above and beyond other subtypes, though emotional abuse and physical neglect also operated through similar pathways when considered individually. This points to the salience of psychological maltreatment histories for maternal and child wellbeing, even relative to more evident forms of childhood physical or sexual abuse (National Scientific Council on the Developing Child, 2012; Norman et al., 2012).

Of note, maternal childhood trauma and postpartum depression were linked to alterations in emotion processing at six weeks postpartum, specifically attention to fearful stimuli. On average, women with a childhood trauma history spent less time paying attention to subliminally presented fearful faces, suggesting automatic avoidance
of negative emotional stimuli (Putman et al., 2004). This avoidant presentation was then linked to greater symptoms of postpartum depression across the first six months. While preliminary, study findings are consistent with a recent systematic review on neural endophenotypes of postpartum psychopathology (Moses-Kolko, Horner, Phillips, Hipwell, & Swain, 2014), which summarizes a body of literature in which mothers with postpartum depression tend to show reduced amygdala activation and amygdala-prefrontal connectivity in response to negative infant-related stimuli as compared to non-depressed mothers (Barrett et al., 2012; Silverman et al., 2011; Silverman et al., 2007).

It is suggested by Moses-Kolko et al. (2013) and other sources (Laurent & Ablow, 2012) that blunted cortico-limbic responses among depressed mothers may reflect a tendency to disengage from distressing stimuli (Gollan, Hoxha, Getch, Sankin, & Michon, 2013; Webb & Ayers, 2015) that would otherwise be emotionally salient for non-depressed mothers in facilitating normative mother-infant attachment.

Altered emotional processing might be one explanation for how experiences of abuse and neglect in one generation may reoccur in the next generation. For example, one study (Kim, Fonagy, Allen, & Strathearn, 2014) has found that unresolved trauma in mothers similarly predicts blunted amygdala activation to own infant distress. In non-postpartum samples, trauma exposure has typically been linked to heightened amygdala responses to fearful stimuli, especially if PTSD is present (Bryant et al., 2008; Felmingham et al., 2010). However, this could reflect hypervigilance following acute
traumatic events whereas the effects of childhood trauma may differentially impact neural patterns over the life course, including weakening connectivity between areas related to automatic fear regulation in adulthood (Birn, Patriat, Phillips, Germain, & Herrringa, 2014), which then generalizes into parenting contexts (Moser et al., 2015). In the present study, among subtypes examined, a history of physical neglect was most strongly correlated with decreased maternal attention to fearful stimuli and may reflect a learned response to reflexively dampen emotional responses in the presence of chronic deprivation. More research needs to be conducted to distinguish neural and behavioral correlates of emotional processing following childhood trauma versus other trauma exposures, and also examine the interaction between childhood trauma, depression and emotional processing specifically in the postpartum period, as this window may be characterized by unique shifts in threat response and emotional salience within the emerging mother-infant attachment system (Kim, Strathearn, & Swain, 2016).

### 3.3.2 Study limitations

Limitations of this study included the modest sample size, which may have limited statistical power. However, the fact that significant relationships were detected between variables even with limited power suggests these relationships are robust in this sample, contributing important preliminary data to sparse literature on postpartum depression in this setting. Of course, care should be taken not to automatically generalize findings to other samples or settings until corroborated in larger studies.
Second, mother-infant bonding problems were measured by self-report only. Higher reported bonding problems could be an artifact of mothers’ emotional state instead of actual interactive disruptions, although we attempted to control for this by separating the timing of postpartum depression measures (through six months) from the bonding outcome (at one year). In addition, other studies using objective behavioral measures have found similar relationships between postpartum depression and actual parenting behavior among mothers (Martinez-Torteya et al., 2014), suggesting correspondence between self-report and observational measures of maternal-infant relationships. Third, this study did not fully rule out the possibility that maternal mental health and child physical growth were both predicted by a common third variable such as food insecurity (Tsai, Tomlinson, Comulada, & Rotheram-Borus, 2016). We attempted to adjust the model for maternal education level as a proxy for socioeconomic disadvantage but did not collect more detailed measures to interrogate this potential confound. Fourth, this study did not explicitly account for intimate partner violence during the postpartum period, though individuals who have experienced childhood trauma are more likely to be revictimized as adults (Desai, Arias, Thompson, & Basile, 2002a). At the same time, there is evidence that childhood trauma may have independent influences on postpartum depression regardless of later victimization (Malta, McDonald, Hegadoren, Weller, & Tough, 2012). Future research could aim to clarify the role of early and later maternal experiences in this intergenerational pathway.
Finally, this study did not examine potentially co-occurring postpartum PTSD in mothers, though comorbid depression and PTSD are especially linked to adverse maternal and child outcomes (Chemtob, Gudino, & Laraque, 2013). Future research should investigate whether maternal depression and PTSD influence maternal caregiving and child outcomes in additive, interactive, or contrasting ways.

### 3.3.3 Implications for treatment and prevention

Several implications emerged from this study. First of all, postpartum depression has tangible consequences for maternal and child outcomes in this setting, and can be prospectively predicted by factors assessed during pregnancy. Routine assessment of trauma history and mental health symptoms during initial antenatal visits may yield insight into which women are at greatest risk for postpartum depression. Particular attention should be paid to early experiences of psychological abuse and neglect, as these seemed most relevant for postpartum mental health. Initial assessment could allow health care providers to provide women basic psychoeducation about possible postpartum risks and then make referrals if desired.

While limited, there is a growing evidence base on interventions for perinatal depression that can be delivered by non-specialist health workers in LMICs (Chowdhary et al., 2014), including South Africa (Lund et al., 2014). Best practices for such interventions (Chowdhary et al., 2014) include integration within broader maternal and child health services; continuity of contact across pregnancy and postpartum; cultural
adaptations to the local context; and expansion beyond individual psychotherapeutic strategies to focus on child care, interaction and feeding. Of note, enhancing maternal sensitivity and responsiveness, as well as play and affection, has often been incorporated as an intervention target in these settings. Promoting attunement may interrupt automatic avoidance of infant’s distress cues among depressed mothers, as suggested in this study, and potentially improve mood and subsequent interactions more robustly than simply treating maternal depressive symptoms (Forman et al., 2007). Emerging evidence suggests it is also possible to use simple feedback techniques to retrain how mothers process infant distress (Carnegie et al., 2015). Resolving bonding disruptions may then result in secure mother-infant attachment, which has lifelong implications (Ranson & Urichuk, 2008).

Postpartum interventions with depressed South African mothers could also improve infant growth in the first year of life. Whether growth improvements can then be maintained throughout childhood deserves further investigation. There is some evidence for early critical windows for child growth (Victora, de Onis, Hallal, Blossner, & Shrimpton, 2010) but also that catch-up may be possible throughout childhood (Prentice et al., 2013), at least in some LMICs (Bennett, Schott, Krutikova, & Behrman, 2015). However, catch-up growth itself may have negative long-term health and cognitive implications (Jou, Lonnerdal, & Griffin, 2013; Martin, Connelly, Bland, & Reilly, 2016; Pérez-Escamilla, 2013), so ensuring a robust foundation of physical
development for children in their earliest years of life is likely a sound strategy. Increasing awareness of the links between maternal mental health and child physical growth among primary health care providers who conduct child growth monitoring through the first five years (Western Cape Government, 2016) could help to ensure timely intervention, whether maternal postpartum depression is a signal of food insecurity and other contextual risks for the growing infant or whether it directly disrupts feeding and other child care practices. In a recent qualitative study on perinatal depression in a South African township (Davies, Schneider, Nyatsanza, & Lund, 2016), mothers further conceptualized their depressive symptoms as directly grounded in contextual stressors such as unemployment, loss/abuse, unplanned pregnancy, limited social support, and for some HIV diagnosis, suggesting that interventions targeting perinatal depression should also focus on helping women problem-solve and/or cope emotionally with these stressors, while ultimately needing to shift larger structural barriers such as poverty, violence, and disease (Lund et al., 2011).

Interestingly, recent research in South African townships (Worthman, Tomlinson, & Rotheram-Borus, 2016) has revealed that mothers in this context view their influences on children as marginally important during the perinatal period; instead, they tend to prioritize early adolescence as the most critical time to prevent poor life outcomes. This may reflect a setting particularly conducive to adolescent risk, given high community rates of HIV, crime, and methamphetamine use (Seedat et al., 2009;
Watt et al., 2014) – but also suggests less familiarity with the idea of a “critical window” from pregnancy through the first two years of life, which has received popular support in Western settings (Center on the Developing Child at Harvard University, 2010; Leadsom et al., 2013). It is critical to respect and support local priorities while also sharing locally sourced evidence that may point to the need for an expanded focus on both adolescence and earlier windows such as the perinatal period (Worthman et al., 2016). If supported by emerging evidence, public outreach campaigns by radio or in local clinics could be employed to promote caregiver interest and engagement in early interventions.

Finally, a broader prevention issue raised in this study is a need to focus more attention on maltreatment, particularly on the emotional and physical neglect of children in this setting, as these exposures appear to affect mental health across the life course and into the next generation. While child neglect is typically more common than abuse it is often more difficult to detect (National Scientific Council on the Developing Child, 2012), including in South Africa where it is not well documented (Makoae, Roberts, & Ward, 2012). Increasing child protection in South Africa is a complex endeavor that this current study does not presume to inform; however, promising targets include reducing systemic contributors to child neglect such as poverty and substance abuse (Slack et al., 2004); increasing community awareness of current reporting responsibilities for various levels of child neglect (Children’s Institute, 2015);
defining policies and programs to respond to such reports; and in the short term, increasing existing accountability among community members to monitor child safety and wellbeing where parents are absent (Choi, Watt, et al., 2015).

### 3.3.4 Conclusion

In conclusion, this study provides preliminary evidence that corroborates existing literature from LMICs in which maternal mental health has been found to influence child health and development (Bennett et al., 2015; Surkan et al., 2011). In this study, maternal depression across the early postpartum period negatively influenced maternal-infant bonding and infant physical growth at one year, and was prospectively predicted by childhood trauma histories assessed during pregnancy. Findings thus also inform and extend sparse literature on the intergenerational consequences of early maltreatment in a non-Western setting. This is particularly needed in South Africa, a country with high levels of interpersonal trauma within disadvantaged township settings (Matthews, Benvenuti, & Mathews, 2014). Mothers who have experienced childhood abuse and/or neglect may be at increased risk for disrupted caregiving, with potential replication of maltreatment experiences in the next generation. Early interventions to improve maternal mental health and related outcomes may set a crucial foundation for violence prevention (Skeen, Tomlinson, Ward, Cluver, & Lachman, 2015), though continued research in South Africa is needed across the life course and across
multiple ecological levels to understand the extent of intergenerational transmission of maltreatment and related sequelae in this high-trauma setting and how to interrupt it.
4. Final Discussion

Findings from both studies in this dissertation have contributed to current understandings about the intergenerational transmission of maltreatment and its sequelae. Specifically, both studies demonstrate that postpartum depression plays a mediating role in linking maternal histories of childhood maltreatment to negative outcomes in the next generation.

While based in vastly different contexts, these dissertation studies provided complementary data. Study 1 (E-Risk) in the UK used a large sample of mothers and children followed over decades, and could account for later maternal depression. On the other hand, Study 2 in South Africa drew on a small sample with only proximal child-related outcomes at one year – but provided more detailed measures of postpartum depression across the first year, and also collected antenatal data (i.e., maternal distress) that could not be accounted for in Study 1. Study 2 also included a proximal objective measure of child physical health, an outcome not examined in Study 1, while Study 1 used reliable gold-standard measures for child mental health outcomes. Study 1 documented maltreatment exposures across both generations, explicitly confirming intergenerational transmission of maltreatment – while Study 2 demonstrated transmission of negative outcomes but will require longer-term research to confirm the extent to which children of trauma-exposed mothers are indeed more likely to be similarly exposed to abuse and neglect in South Africa. Finally, Study 2 contained a lab-
based component that allowed an exploratory investigation of underlying mechanisms for postpartum depression and subsequent outcomes following childhood trauma.

One question raised from this dissertation, particularly by Study 1 but also implicitly in Study 2, is the following: How critical is it to intervene specifically in the perinatal period? While helping depressed mothers after childbirth is a valuable goal in itself, studies have suggested that treating maternal depression in the perinatal period has long-term economic benefits related to improved child wellbeing (Bauer, Knapp, & Parsonage, 2016; Bauer et al., 2015). However, more evidence on the efficacy, sustainability, and long-term benefits of early maternal depression interventions for children may be necessary to prompt large-scale investments at a societal level at the cost of other interventions during children’s school-aged or adolescent years. Follow-up research with children of mothers who have participated in perinatal depression intervention trials is still needed in both Western and developing settings. Beyond examining main effects (i.e., whether children of treated mothers fare better years later), it would be useful to conduct moderator analyses: for whom are benefits of early interventions sustained, and why. This would highlight additional targets or intervention components that would ensure greatest long-term impact for the next generation – or confirm additional critical windows for intervention. Based on findings from Study 1, basic clinical research is also needed to better understand recurrent
depression as a phenomenon and how to improve treatment maintenance for depression following psychotherapy, including for postpartum depression.

Converging findings from this dissertation also support specific attention to the intergenerational influences of maltreatment subtypes, particularly psychological forms of maltreatment. Among subtypes, childhood emotional neglect was a relatively common experience among mothers in both samples, while physical neglect was particularly prevalent in the South African sample owing to the setting. Unless in cases of extreme deprivation, many situations of child neglect – where caregivers are regularly missing opportunities to respond to children’s emotional, intellectual, and/or physical needs – are not necessarily detected but appear to have enduring consequences nonetheless. Persistent caregiver unavailability and lack of responsiveness may be as physiologically and psychologically stressful as more overt forms of maltreatment (National Scientific Council on the Developing Child, 2012). However, establishing a multi-systemic agenda to address child neglect is challenging as the goal becomes to prevent the absence of normative care, which is difficult to quantify. Improved definitions of what constitutes child neglect and further research and surveillance on specific costs associated with neglect may help to guide prevention efforts.

Finally, while not the focus of this dissertation, it must be acknowledged that even in the presence of maternal risks, there was still substantial discontinuity in the transmission of maltreatment and related child outcomes. Mechanisms for resilience
have been proposed in existing literature to include safe and stable relationships with at least one adult during development (Jaffee et al., 2013). Of relevance to the postpartum period, resilient child outcomes may be more likely when depressed mothers with maltreatment histories are able to draw upon core memories from any positive interactions with caregivers in their early childhood as they interact with their own infants (Lieberman, Padrón, Van Horn, & Harris, 2005) and when they are able to continue displaying positive affect and delight towards their infants even if sometimes engaging in negative parenting behavior (Martinez-Torteya et al., 2014). These adaptive cognitive-behavioral capacities could be promoted at the same time that depressive symptoms are being managed.

Thus, by drawing on longitudinal studies in different global contexts, this dissertation confirmed existing literature on the association between childhood maltreatment and postpartum psychopathology, but also extended this literature by looking at the implications of this association for child outcomes. Of note, it raised compelling directions for future research in both Western and non-Western settings to examine how maternal trauma histories and depressive symptoms relate to emotion processing during the perinatal period and may then link to disrupted maternal-infant interactions and even later risk for maltreatment, potentially clarifying neural and behavioral endophenotypes that could be targeted in ongoing efforts to interrupt the intergenerational transmission of maltreatment – starting from the earliest years of life.
S1. Study 1 Supplemental Materials

S1.1 Coefficient tables for main study models

Table 5. Direct path coefficients for main structural model for 12-year child outcomes (Study 1)

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted B (SE) p-value</th>
<th>Adjusted B (SE) p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maternal predictors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postpartum depression ON</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal childhood maltreatment</td>
<td>.22(.04) &lt;.001***</td>
<td>.22(.04) &lt;.001***</td>
</tr>
<tr>
<td>Socioeconomic disadvantage</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Maternal age</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Child outcomes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Harm exposure ON</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal childhood maltreatment</td>
<td>.27(.04) &lt;.001***</td>
<td>.24(.04) &lt;.001***</td>
</tr>
<tr>
<td>Postpartum depression</td>
<td>.22(.06) &lt;.001***</td>
<td>.22(.06) &lt;.001***</td>
</tr>
<tr>
<td>Socioeconomic disadvantage</td>
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<tr>
<td>Maternal age</td>
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<td>.12(.04) .004**</td>
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<td></td>
</tr>
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<td>.20(.04) &lt;.001***</td>
</tr>
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<td>Postpartum depression</td>
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<td>.11(.07) .096</td>
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<td>.24(.07) &lt;.001***</td>
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<tr>
<td>Socioeconomic disadvantage</td>
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<td>.20(.06) .002**</td>
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<td>.19(.03) &lt;.001***</td>
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<td>.08(.05) .119</td>
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<td>.29(.05) &lt;.001***</td>
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<td>.32(.04) &lt;.001***</td>
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<td>-.008(.04) .835</td>
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<tr>
<td>X(df)</td>
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<td>64.79(26) -</td>
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<tr>
<td>RMSEA</td>
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<td>.038</td>
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<tr>
<td>CFI/TLI</td>
<td>.99/.98</td>
<td>.99/.98</td>
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*Note. Standardized path coefficients (STDYX) except where predictor was dichotomous/categorical, in which case only y-variable was standardized (STDY).*
<table>
<thead>
<tr>
<th>Pathway</th>
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<tr>
<td>Total</td>
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<tr>
<td>PPD</td>
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<td>.002**</td>
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<tr>
<td>Total</td>
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<td>&lt;.001***</td>
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<tr>
<td>PPD</td>
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<td>.11</td>
</tr>
<tr>
<td>Child harm</td>
<td>.06(.02)</td>
<td>&lt;.001***</td>
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<tr>
<td>PPD → child harm</td>
<td>.01(.005)</td>
<td>.017*</td>
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<tr>
<td><strong>Externalizing symptoms</strong></td>
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<td></td>
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<tr>
<td>Total</td>
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<tr>
<td>PPD</td>
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<td>.133</td>
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<td>Child harm</td>
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<td>PPD → child harm</td>
<td>.01(.005)</td>
<td>.005**</td>
</tr>
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</table>

Table 6. Indirect path coefficients from maternal childhood maltreatment to child outcomes, adjusted (Study 1)
Table 7. Direct path coefficients for 12-year structural model including later maternal depression (Study 1)

<table>
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<tr>
<th></th>
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<tr>
<td><strong>Maternal predictors</strong></td>
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<tr>
<td>Postpartum depression ON</td>
<td></td>
<td></td>
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<tr>
<td>Maternal childhood maltreatment</td>
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<td>Socioeconomic disadvantage</td>
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<td>Postpartum depression</td>
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<td>.004**</td>
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<td>.412</td>
</tr>
<tr>
<td><strong>Child outcomes</strong></td>
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<td>Harm exposure ON</td>
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<td>Maternal childhood maltreatment</td>
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<td>Postpartum depression</td>
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<td>Later maternal depression</td>
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<td>.882</td>
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<td>X-(df)</td>
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<td>-</td>
</tr>
<tr>
<td>RMSEA</td>
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<tr>
<td>CFI/TLI</td>
<td>.98/.97</td>
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86
Table 8. Indirect path coefficients from maternal childhood maltreatment through postpartum depression and later maternal depression to child outcomes (Study 1)

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Adjusted B (SE)</th>
<th>p-value</th>
<th>(Unadjusted) B (SE)</th>
<th>p-value</th>
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<tbody>
<tr>
<td><strong>Harm exposure</strong></td>
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<td></td>
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</tr>
<tr>
<td>Total indirect</td>
<td>.07(.02)</td>
<td>&lt;.001***</td>
<td>.08(.02)</td>
<td>&lt;.001***</td>
</tr>
<tr>
<td>PPD</td>
<td>.04(.03)</td>
<td>.23</td>
<td>.03(.03)</td>
<td>.369</td>
</tr>
<tr>
<td>PPD → LMD</td>
<td>.04(.02)</td>
<td>.032*</td>
<td>.05(.02)</td>
<td>.012*</td>
</tr>
<tr>
<td><strong>Internalizing symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total indirect</td>
<td>.10(.03)</td>
<td>&lt;.001***</td>
<td>.12(.03)</td>
<td>&lt;.001***</td>
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<td>.868</td>
<td>-.01(.03)</td>
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<td>.05(.02)</td>
<td>.04**</td>
<td>.06(.02)</td>
<td>.002**</td>
</tr>
<tr>
<td>PPD → LMD</td>
<td>.04(.02)</td>
<td>.021*</td>
<td>.05(.02)</td>
<td>.011*</td>
</tr>
<tr>
<td>PPD → LMD → child harm</td>
<td>.008(.005)</td>
<td>.080</td>
<td>.01(.006)</td>
<td>.042*</td>
</tr>
<tr>
<td><strong>Externalizing symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total indirect</td>
<td>.11(.02)</td>
<td>&lt;.001***</td>
<td>.13(.02)</td>
<td>&lt;.001***</td>
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<td>&lt;.001***</td>
<td>.08(.02)</td>
<td>&lt;.001***</td>
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<tr>
<td>PPD → LMD</td>
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<td>.039*</td>
<td>.04(.02)</td>
<td>.012*</td>
</tr>
<tr>
<td>PPD → LMD → child harm</td>
<td>.01(.005)</td>
<td>.046*</td>
<td>.02(.006)</td>
<td>.018*</td>
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Table 9. Direct path coefficients for maltreatment subtypes model for 12-year outcomes (Study 1)

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<tr>
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<tr>
<td><strong>Maternal predictors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postpartum depression ON</td>
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<td></td>
</tr>
<tr>
<td>Maternal physical abuse</td>
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<td>.596</td>
</tr>
<tr>
<td>Maternal sexual abuse</td>
<td>.12(.04)</td>
<td>.005**</td>
</tr>
<tr>
<td>Maternal emotional abuse</td>
<td>.24(.06)</td>
<td>&lt;.001***</td>
</tr>
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<td>Maternal emotional neglect</td>
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<td>.313</td>
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<td>.543</td>
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<td>.623</td>
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<td></td>
</tr>
<tr>
<td>Postpartum depression</td>
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<td>.003**</td>
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<td>.361</td>
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<td>.017*</td>
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<td>Postpartum depression</td>
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<td>.192</td>
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<td>Later maternal depression</td>
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<td>.012*</td>
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<td>Socioeconomic disadvantage</td>
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<td>.036*</td>
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<td>.007**</td>
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<td>.941</td>
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<td>Maternal emotional abuse</td>
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<td>.042*</td>
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<td>Postpartum depression</td>
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<td>.950</td>
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<td>Later maternal depression</td>
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<td>.001***</td>
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<td>.402</td>
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<td></td>
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<tr>
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<td>&lt;.001***</td>
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<tr>
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<td>Later maternal depression</td>
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<tr>
<td>Harm exposure</td>
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<td>Maternal age</td>
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*Model fit*

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<th>df</th>
<th>p-value</th>
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<tr>
<td>CFI/TLI</td>
<td>.98/.96</td>
<td></td>
<td></td>
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</table>
S1.2 Extension to 18-year outcomes

S1.2.1 Analyses

The main structural analyses conducted for child outcomes at 12 years were replicated with child depressive symptoms and conduct disorder symptoms at 18 years, to test whether pathways through postpartum depression and subsequent risk for maltreatment exposure could further predict internalizing and externalizing symptoms up to the point at which children are entering young adulthood. Child depressive symptoms and conduct disorder symptoms were self-reported by twins at 18 years during structured interviews using DSM-IV criteria.

S1.2.2 Results

Individual path analyses revealed that maternal childhood maltreatment predicted child depressive and conduct disorder symptoms at 18 years (B=.19, p<.001 for depressive symptoms; B=.15, p<.001 for conduct disorder symptoms), confirming intergenerational impact through to young adulthood. These associations remained significant even when adjusted for maternal age and socioeconomic disadvantage.
With postpartum depression included as a mediator between maternal childhood maltreatment and these later outcomes, a similar structural model (Figure 9; significant paths shown, with standardized coefficients) fit the data closely, $X^2(20)=30.28$, $p=.066$, RMSEA=.022, CFI/TLI=.996/.996. As before, postpartum depression was a significant predictor of child harm exposure ($B=.22$, $p<.001$), which in turn predicted child depressive symptoms ($B=.24$, $p=.003$) and conduct disorder symptoms ($B=.21$, $p<.001$) at 18 years. The pattern of significant indirect pathways from maternal childhood maltreatment to child depressive symptoms and conduct disorder symptoms remained the same as in the 12-year analysis, with significant effects through child harm exposure alone, and through the combined pathway of postpartum depression → subsequent child harm. Maternal childhood maltreatment no longer had significant direct paths to child depressive symptoms but continued to directly predict externalizing symptoms above and beyond the pathway. Furthermore, indirect effects remained the same even after adjusting for maternal age and early socioeconomic
disadvantage. Again, later maternal depression seemed to carry this pathway, particularly through its relationship to child harm exposure, when it was included structurally (not shown).
### S1.2.3 Coefficient table for 18-year models

Table 10. Direct path coefficients for main structural model for 18-year child outcomes (Study 1)

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<tr>
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<tr>
<td></td>
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<td>B (SE)</td>
</tr>
<tr>
<td><strong>Maternal predictors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postpartum depression ON</td>
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<td></td>
</tr>
<tr>
<td>Maternal childhood maltreatment</td>
<td>.22(.04)</td>
<td>&lt;.001***</td>
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</tr>
<tr>
<td>Maternal age</td>
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<td></td>
</tr>
<tr>
<td><strong>Child outcomes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Harm exposure ON</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal childhood maltreatment</td>
<td>.27(.04)</td>
<td>&lt;.001***</td>
</tr>
<tr>
<td>Postpartum depression</td>
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<td></td>
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<tr>
<td>Socioeconomic disadvantage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms ON</td>
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<tr>
<td>Maternal childhood maltreatment</td>
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<td>.167</td>
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<td>Postpartum depression</td>
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<td></td>
</tr>
<tr>
<td>Harm exposure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Socioeconomic disadvantage</td>
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<td></td>
</tr>
<tr>
<td>Maternal age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conduct disorder symptoms ON</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal childhood maltreatment</td>
<td>.08(.04)</td>
<td>.041*</td>
</tr>
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<td>Postpartum depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Harm exposure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Socioeconomic disadvantage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal age</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Model fit</strong></td>
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<tr>
<td>$X^2 (df)$</td>
<td>30.28(20)</td>
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<td>.022</td>
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<td>CFI/TLI</td>
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<td>.996/.994</td>
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Table 11. Direct path coefficients for 18-year structural model including later maternal depression (Study 1)

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<tr>
<td><strong>Maternal predictors</strong></td>
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<td></td>
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</tr>
<tr>
<td>Maternal childhood maltreatment</td>
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<td>.004**</td>
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<td>.412</td>
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<tr>
<td>Maternal childhood maltreatment</td>
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<td>.227</td>
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<td>.027*</td>
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<tr>
<td>Socioeconomic disadvantage</td>
<td>.10(.04)</td>
<td>.033*</td>
</tr>
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<td>Maternal age</td>
<td>-.11(.04)</td>
<td>.006**</td>
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<td>.681</td>
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<td>.081</td>
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<tr>
<td>X(df)</td>
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S2. Study 2 Supplemental Materials

S2.1 Coefficient tables for main study models

Table 12. Direct path coefficients for main structural model (Study 2)

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<td>Bonding ON</td>
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<td>&lt;.001***</td>
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Table 13. Direct path coefficients for models including antenatal distress (Study 2)

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<td>B (SE)</td>
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<td>ON Maternal childhood</td>
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</tr>
<tr>
<td>maltreatment</td>
<td>.22(.09)</td>
<td>.019*</td>
<td>.21(.09)</td>
<td>.022*</td>
</tr>
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<td>&lt;.001***</td>
<td>.42(.09)</td>
<td>&lt;.001***</td>
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<td>-</td>
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<td>.061</td>
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<tr>
<td>Maternal childhood</td>
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<td>.033*</td>
<td>.15(.08)</td>
<td>.059</td>
</tr>
<tr>
<td>maltreatment</td>
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<td>-</td>
<td>-26(.08)</td>
<td>.001**</td>
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<td><strong>Child outcomes</strong></td>
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<tr>
<td>Bonding ON</td>
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<tr>
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<td>.74(.11)</td>
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<td>.69(.11)</td>
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<tr>
<td>Antenatal distress</td>
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<td>.014*</td>
<td>-.30(.10)</td>
<td>.004**</td>
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<td>.691</td>
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<td>.019*</td>
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<td></td>
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<td>47.30(41)</td>
<td>-</td>
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<td>-</td>
<td>.032</td>
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<td>.98/96</td>
<td>-</td>
</tr>
<tr>
<td><strong>Maternal predictors</strong></td>
<td>B (SE)</td>
<td>p-value</td>
<td></td>
<td></td>
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<tr>
<td>Postpartum depression ON</td>
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<td></td>
</tr>
<tr>
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<td>.63(.08)</td>
<td>&lt;.001***</td>
</tr>
<tr>
<td>Development ON</td>
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<td></td>
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<td>.015*</td>
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<td>Birth weight</td>
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<td>Postpartum depression</td>
<td>-.39(.11)</td>
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### S2.2 Coefficient table for exploratory study models

#### Table 15. Exploratory Stroop models with masked fear stimuli (Study 2)

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<td>B (SE)</td>
<td>p-value</td>
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<tr>
<td>Masked fear processing</td>
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<td>Maternal childhood maltreatment</td>
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<td>.002**</td>
<td>-.39(.13)</td>
<td>.002**</td>
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<tr>
<td>Masked fear processing</td>
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<td>.001***</td>
<td>-.52(.13)</td>
<td>&lt;.001***</td>
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<tr>
<td><strong>Child outcomes</strong></td>
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<tr>
<td>Bonding ON</td>
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</tr>
<tr>
<td>Postpartum depression</td>
<td>-</td>
<td>-</td>
<td>.61(.08)</td>
<td>&lt;.001***</td>
</tr>
<tr>
<td>Development ON</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Postpartum depression</td>
<td>-</td>
<td>-</td>
<td>.30(.13)</td>
<td>.019*</td>
</tr>
<tr>
<td>Growth ON</td>
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</tr>
<tr>
<td>Birth weight</td>
<td>-</td>
<td>-</td>
<td>-.68(.08)</td>
<td>&lt;.001***</td>
</tr>
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<td>Postpartum depression</td>
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<td>-</td>
<td>-.41(.11)</td>
<td>&lt;.001***</td>
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<td>X2(df)</td>
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<td>36.41(37)</td>
<td>-</td>
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<td>-</td>
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<td>CFI/TLI</td>
<td>.98/.97</td>
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S3. Prior Synthesis of Literature

The following text has been reproduced with permission in accordance to journal and publisher policies and is the accepted version of a review manuscript published in *Trauma, Violence, & Abuse* (DOI:10.1177/1524838015584369), entitled "Childhood Maltreatment and Perinatal Mood and Anxiety Disorders: A Systematic Review." This review set the foundation for the studies undertaken in this dissertation.

**S3.1 Introduction**

Perinatal mood and anxiety disorders (PMADs) afflict a substantial proportion of childbearing women (Bennett, Einarson, Taddio, Koren, & Einarson, 2004; Melville, Gavin, Guo, Fan, & Katon, 2010; O’Hara & Swain, 1996) and represent a significant threat to maternal and child wellbeing. Evidence suggests that mood and anxiety disorders occurring during pregnancy predict negative obstetric and delivery outcomes (Chung, Lau, Yip, Chiu, & Lee, 2001; Kurki, Hiilesmaa, Raitasalo, Mattila, & Ylikorkala, 2000; Li, Liu, & Odouli, 2009) as well as poor infant and child outcomes (Hay, Pawlby, Waters, Perra, & Sharp, 2010; Misri et al., 2004; O’Connor, Heron, & Glover, 2002; Talge et al., 2007; van Batenburg-Eddes et al., 2009), while postpartum mood and anxiety disorders may compromise mother-child bonding and interaction (Field, 2010; Moehler et al., 2006) as well as child growth (Patel, DeSouza, & Rodrigues, 2003) and long-term development (Grace, Evindar, & Stewart, 2003; Kaplow & Widom, 2007; Kurstjens & Wolke, 2001). A growing appreciation of the wide-ranging and enduring consequences
of PMADs has raised recent public and scientific interest in risk factors to inform prevention and intervention.

One emerging predictor of PMADs is women’s exposure to violence (Kendall-Tackett, 2007b), with extant literature focused on concurrent partner victimization (Beydoun, Beydoun, Kaufman, Lo, & Zonderman, 2012; Howard, Oram, Galley, Trevillion, & Feder, 2013). Experiences of interpersonal trauma can be especially salient during pregnancy and postpartum, a time when important relationships and identities are being reorganized (Huth-Bocks, Krause, Ahlfs-Dunn, Gallagher, & Scott, 2013). Less well understood, however, is the relationship between PMADs and earlier experiences such as childhood maltreatment, a potent and common source of trauma (Carlson, Furby, Armstrong, & Shlaes, 1997; Gilbert et al., 2009; Roller, 2011).

It is established that childhood maltreatment increases risk for various affective disorders in adulthood (Kaplow & Widom, 2007), including depression (Widom et al., 2007), PTSD (Brewin, Andrews, & Valentine, 2000), and anxiety (Pynoos, Steinberg, & Piacentini, 1999). However, childhood maltreatment may influence a woman’s mental health especially during the perinatal period, for various reasons. First, the perinatal period is marked with unique stressors – rapid physical and emotional changes during pregnancy, strenuous challenges of labor and delivery, heightened caregiving demands in the postpartum – that could exacerbate or trigger onset of psychopathology in the presence of existing vulnerability, as consistent with a diathesis-stress model (Monroe &
Simons, 1991; Schumm, Stines, Hobfoll, & Jackson, 2005). Second, pregnancy and motherhood involve role transitions where early traumatic memories may likely resurface. Since maltreatment is most commonly perpetrated by the child’s own parents (US Department of Health and Human Services, 2012), becoming a parent could activate cognitions, emotions or biological responses related to childhood experiences of being parented, including abuse and neglect (Stöckl & Gardner, 2013; van der Kolk, 1994).

Despite a growing evidence base, there has been no comprehensive review to date on whether and how childhood maltreatment influences mood and anxiety disorders occurring in the perinatal period – and for which maltreatment exposures, populations, and disorder outcomes. This review synthesizes empirical literature on the relationship between maternal histories of childhood maltreatment and a range of mood and anxiety disorders during pregnancy and postpartum. Findings from this review are expected to inform the prevention and treatment of PMADs, and advance the research agenda to optimize maternal and child outcomes in the context of lifetime trauma.

**S3.2 Methods**

**Review procedure**

The study selection procedures are summarized in Figure S11. We searched MEDLINE, PsychINFO, Embase and CINAHL databases for articles on the relationship between childhood maltreatment (search terms: child* abuse OR child* maltreatment OR child* neglect) and mental disorders (search terms: depress* OR anxiety OR trauma*...
OR disorder OR mood disorder OR psychological disorder OR mental health) specifically in the perinatal period (search terms: pregnan* OR postpartum OR postnatal OR prenatal OR antenatal). Citations available up to December 1, 2013, were included for abstract review.

Abstracts were flagged for full-text review if they were topically relevant and consisted of English-language empirical studies with human subjects. Additional citations were identified from hand searches of key reference lists. We evaluated full articles on whether they satisfied the following inclusion criteria: (1) definition of early experiences consistent with maltreatment (i.e., physical abuse, sexual abuse, emotional abuse, emotional or physical neglect), reported to have occurred during childhood and/or before the age of 18; (2) clinical diagnoses or symptoms corresponding to DSM-V mood and/or anxiety disorder(s), occurring during pregnancy or up to one year postpartum, assessed using diagnostic or screening instruments; (3) a statistical analysis of the association between (1) and (2); and (4) findings published in a peer-reviewed journal.

When multiple reports originated from the same study, we reviewed individual reports for their outcome of interest. If reports examined distinct outcomes, or same outcomes at different time points (e.g., during pregnancy versus postpartum) as part of a longitudinal study, they were both kept in the review. If similar outcomes were
Figure S10. Study selection flowchart in prior research synthesis
examined across reports, the citation with the largest sample size was selected for review. There were four such studies excluded due to redundant outcomes (Dennis & Ross, 2006; Lev-Wiesel & Daphna-Tekoa, 2007; Seng, Low, Sperlich, Ronis, & Liben, 2009; Seng, Sperlich, & Low, 2008). For clarity, the term “antenatal” will be used throughout the review to refer to the period during pregnancy, and the term “postpartum” will be used throughout to refer to the period following delivery.

**Data extraction and analysis**

For each article that met the inclusion criteria, we first extracted relevant details on study design, sample characteristics, measured variables, and key findings into a standardized data extraction form. We then summarized studies by year, country, sample size, and research design, and also calculated the proportion of studies focused on (1) disadvantaged populations, and (2) adolescents. We then tabulated assessment instruments for childhood maltreatment and PMADs in terms of type and frequency, with relevant cut-offs noted. We also characterized the prevalence of childhood maltreatment exposures and PMAD outcomes using weighted means and ranges, using reported data where available.

We categorized study results by type of disorder (e.g., depression, anxiety, PTSD), and further divided these by antenatal versus postpartum outcomes. For each PMAD, we calculated the proportion of studies demonstrating a positive, null, or negative association between childhood maltreatment and that outcome. We also
conducted a descriptive analysis of studies examining different maltreatment subtypes. Due to the heterogeneity of studies and measurements, quantitative pooled analysis of effect sizes was not conducted. Finally, we examined the subset of studies that tested the impact of childhood maltreatment in multivariate models, to extract covariates and relevant results.

**S3.3 Results**

**Overview of studies included in the review**

Of the 876 unique citations retrieved, a total of 35 papers based on 30 distinct studies were included in the review (not shown in dissertation). Three papers came from the same longitudinal study in the US (Kulkarni, Graham-Bermann, Rauch, & Seng, 2011; Muzik et al., 2013; Seng et al., 2013), three from a longitudinal study in Israel (Lev-Wiesel & Daphna-Tekoah, 2010; Lev-Wiesel, Daphna-Tekoah, & Hallak, 2009; Yampolsky, Lev-Wiesel, & Ben-Zion, 2010), and two from another longitudinal study in Australia (Edwards, Galletly, Semmler-Booth, & Dekker, 2008a; Edwards et al., 2008b). Individual papers will subsequently be referred to as “studies” to denote their investigation of independent questions.

Research on this topic has increased exponentially over the past 15 years, with eight studies published in 2013. Studies were conducted in nine different countries, including France (k=1), Spain (k=1), Vietnam (k=1), Netherlands (k=1), UK (k=1), Israel (k=3), Canada (k=4), Australia (k=6), and USA (k=17). With one exception in Vietnam
(Fisher et al., 2013), research to date has been conducted in high-income countries (HICs). Within these high-income settings, about a third of studies (35%) focused predominantly on low-SES and/or minority populations.

Studies were mostly cross-sectional (23%) or longitudinal (60%) in their overall design, using currently pregnant and/or postpartum samples. Three (10%) presented data drawn from an intervention study (Lesser et al., 2000; Blalock et al., 2011; Grote et al., 2012), and two (7%) were retrospective, asking women to recall prior pregnancies (Garabedian et al., 2011; Meltzer-Brody, Boschloo, Jones, Sullivan, & Penninx, 2013). Participants were recruited mostly from clinic and/or community settings. Sample sizes ranged from 44 to 5380, with a mean of 750 participants (median=300, SD=1141).

Five studies (Gilson & Lancaster, 2008; Lesser & Koniak-Griffin, 2000; Meltzer-Brody, Bledsoe-Mansori, et al., 2013; Stevens-Simon & McAnarney, 1994; Tzilos, Zlotnick, Raker, Kuo, & Phipps, 2012) focused specifically on pregnant “adolescents” as defined in the scope of their study. Seven further studies included participants under the age of 18. Of these, two studies (Christl et al., 2013; Leigh & Milgrom, 2008; Plant, Barker, Waters, Pawlby, & Pariante, 2013) included 17 year-olds among their adult participants, possibly reflecting different cut-offs for adulthood outside the US. Other studies included individuals as young as 14 (Chung, Mathew, Elo, Coyne, & Culhane, 2008), 15 (Hayes, Campbell, Buckby, Geia, & Egan, 2010), and 16 (Blalock et al., 2011) years of age.
Most studies examined PMAD(s) as a primary outcome of interest, and all presented statistical findings regarding the relationship between childhood maltreatment and PMAD(s). The vast majority focused on depression, but anxiety and PTSD were also examined. Sixteen studies only examined antenatal outcomes, 12 only considered postpartum outcomes, and seven looked at both antenatal and postpartum outcomes. Of the latter, six investigated antenatal and postpartum as separate outcomes, while one study (Meltzer-Brody, Boschloo, et al., 2013) retrospectively analyzed "perinatal" (both antenatal and postpartum) as a combined outcome.

Data collection instruments

Instruments used to measure childhood maltreatment

In assessing childhood maltreatment, the mode of administration ranged from self-report questionnaires (49%), interview (43%), or either format. Many studies utilized single items, as stand-alone measures (e.g., Buist et al., 2011; Dayan et al., 2010; Chung et al., 2008; Gilson et al., 2008; Cohen et al., 2002) or embedded within risk assessment forms such as the Antenatal Psychosocial Questionnaire (Edwards et al., 2008a) or Antenatal Psychosocial Health Assessment (Dennis & Vigod, 2013). Definitions of maltreatment in these items ranged from broad questions such as “As a child were you hurt or abused in any way (physically, emotionally, sexually)?)” (Edwards et al., 2008a, 2008b) to more specific probes about maltreatment types, such as “When you were a child, did any parent, step-parent, or guardian or any other person make you have sex
any sex act, not just intercourse) by using force or threatening to harm you or someone close to you?” (Garabedian et al., 2011).

The most common multiple-item scale for assessing childhood maltreatment was the Childhood Trauma Questionnaire (CTQ). Other scales included the Child Sexual Assaults Scale (CSA) used in Israel, and the abuse subscales of the Early Trauma Inventory Self-Report (ETI-SR) used in Spain. A set of structured interview questions was also used in earlier studies (e.g., Benedict et al., 1999) to assess for childhood trauma. Assessments of broader life experiences including childhood maltreatment consisted of the Life Stressor Checklist (LSC) and the Personal Safety Questionnaire (PSQ), which is an adaptation of the Conflicts Tactics Scale.

**Instruments used to assess perinatal mood and anxiety disorders**

For perinatal depression, the most common assessment instrument was the Edinburgh Postnatal Depression Scale (EPDS). The second most common instrument was the Center for Epidemiologic Studies Depression Scale (CES-D). Other instruments included the Beck Depression Inventory-II (BDI-II); the Postpartum Depression Screening Scale (PDSS); the Brief Symptom Severity questionnaire (BSI); the Clinical Interview Schedule (CIS); and the Children’s Depression Rating Scale-Revised (CDRS-R) for an pregnant adolescent sample (Tzilos et al., 2012).

For assessing anxiety, instruments included the Spielberger State-Trait Anxiety Inventory (STAI), the Beck Anxiety Inventory (BAI), the anxiety subscale of the Hospital
Anxiety and Depression Scale (HADS), and the adjusted Generalized Anxiety Disorder (GAD) module of the World Health World Mental Health Composite International Diagnostic Interview (WMH-CIDI). For PTSD, instruments included the Post-Traumatic Stress Disorder Checklist – Civilian Version (PCL-C), the Post-Traumatic Stress Disorder Symptom Scale (PSS-I), and the PTSD module from the National Women’s Study.

Figure S11. Study assessment timeline in prior research synthesis
The timing of disorder assessment varied greatly across studies, as schematized in Figure S11. Pregnant participants were assessed as early as the first trimester (Buist, Gotman, & Yonkers, 2011; Meltzer-Brody, Bledsoe-Mansori, et al., 2013), into the second trimester (Dayan et al., 2010; Lev-Wiesel & Daphna-Tekoah, 2010; Rich-Edwards et al., 2011; Tzilos et al., 2012), and with numerous studies focusing on the third trimester (Fisher et al., 2013; Gilson & Lancaster, 2008; Huth-Bocks et al., 2013; Leigh & Milgrom, 2008; Plant et al., 2013). While some studies only assessed women of a certain gestational age, others included women at different stages of pregnancy (e.g., 7 to 32 weeks gestation in Lang et al., 2006). In the postpartum period, assessments were made as early as the immediate postpartum (e.g., 12-48 hours after delivery; Plaza et al., 2012) but more commonly at 6 weeks (Edwards et al., 2008b; Gilson & Lancaster, 2008; Meltzer-Brody, Bledsoe-Mansori, et al., 2013; Seng et al., 2013), 6 months (Lesser & Koniak-Griffin, 2000; Minnes et al., 2008; Muzik et al., 2013), and up to one year (Christl et al., 2013; Lang, Rodgers, & Lebeck, 2006; Lesser & Koniak-Griffin, 2000; Minnes et al., 2008).

**Prevalence rates**

**Childhood maltreatment and subtypes**

Studies varied in whether and how they reported prevalence of childhood maltreatment. Rates of any childhood maltreatment – defined by reporting studies (k=19) in varied ways but consistently encompassing more than one subtype of maltreatment (e.g., physical and sexual abuse), or unspecified – ranged from 16% (Malta
et al., 2012; Meltzer-Brody, Boschloo, et al., 2013) to 66% (Buist, 1998), with an overall weighted average of 30% across studies. Rates of childhood physical abuse in reporting studies (k=12) ranged widely from 7% (Cohen et al., 2002) to 58% (Huth-Bocks et al., 2013). For childhood sexual abuse, studies (k=17) also reported a wide range between 7% (Dayan et al., 2010) and 50% (Buist, 1998). Rates of childhood emotional abuse in reporting studies (k=8) ranged from 4% (Cohen et al., 2002) to 68% (Huth-Bocks et al., 2013). Rates of childhood emotional neglect in reporting studies (k=4) ranged from 34% (Blalock et al., 2011) to 75% (Huth-Bocks et al., 2013), and rates of childhood physical neglect in reporting studies (k=3) ranged from 25% (Blalock et al., 2011) to 49% (Huth-Bocks et al., 2013).

**Perinatal mood and anxiety disorders**

Rates of antenatal depression ranged from 11% (Rich-Edwards et al., 2011) to 50% (Fisher et al., 2013), with a weighted average of 24% across reporting studies (k=13). Rates of postpartum depression ranged from 11% (Cohen et al., 2002) to 23% (Edwards et al., 2008b), with a weighted average of 18% across reporting studies (k=13). When studies were divided by whether or not they focused on low-SES and/or minority populations, the average antenatal depression rate was found to be almost three times higher in studies focused on low-SES and/or minority populations compared to those not focused on such populations – 32% versus 13%, respectively. On the other hand, the average rate for postpartum depression was slightly higher in studies not focused on
low-SES and/or minority populations – 17% versus 14%. Rates of lifetime perinatal depression were estimated to be 41% in a Netherlands sample (Meltzer-Brody, Boschloo, et al., 2013).

Rates for antenatal anxiety varied across reporting studies (k=2), including 4% for generalized anxiety disorder averaged across pregnancy (Blalock et al., 2011) and 63% for clinically elevated anxiety symptoms in the last trimester (Gilson & Lancaster, 2008). Postpartum anxiety rates in reporting studies (k=2) included 16% (Malta et al., 2012) and 44% (Gilson & Lancaster, 2008). Antenatal PTSD rates were reported as 8% by one study (Kulkarni et al., 2011), while postpartum PTSD rates were 6% (Seng et al., 2013) and 23% (Muzik et al., 2013), though all reports of PTSD rates (k=3) originated from the same longitudinal study and were not available from other studies (Huth-Bocks et al., 2013; Lev-Wiesel & Daphna-Tekoah, 2010; Lev-Wiesel et al., 2009; Yampolsky et al., 2010).

Effects of childhood maltreatment on perinatal mood and anxiety disorders

Depression

Antenatal. Fourteen of 18 studies (78%) reported significant positive associations between childhood maltreatment and antenatal depression (as defined by odds ratio with 95% confidence interval or \( p < 0.05 \)), most often in the second and third trimesters of pregnancy (Benedict, Paine, Paine, Brandt, & Stallings, 1999; Blalock et al., 2011; Chung et al., 2008; Grote et al., 2012; Lang et al., 2006; Leigh & Milgrom, 2008; Lesser & Koniak-Griffin, 2000; Meltzer-Brody, Bledsoe-Mansori, et al., 2013; Plant et al., 2013;
Rich-Edwards et al., 2011; Stevens-Simon & McAnarney, 1994; Tzilos et al., 2012; Yampolsky et al., 2010). Of these, one study (Fisher et al., 2013) found that childhood maltreatment did not significantly predict antenatal depression at specific time points (early or late in gestation), but had a significant positive association with depression persisting across early and late pregnancy. Four studies found null relationships between childhood maltreatment and antenatal depression (Dayan et al., 2010; Edwards et al., 2008a; Gilson & Lancaster, 2008; Shea et al., 2007). These four studies were all clinic-based and conducted outside of the US, and with one exception (Edwards et al., 2008a) used the EPDS with a higher cut-off (≥13) to assess antenatal depression.

**Postpartum.** Thirteen of 16 studies (75%) reported significant positive associations between childhood maltreatment and depression at some point in the postpartum period (Buist, 1998; Dennis & Vigod, 2013; Edwards et al., 2008a; Garabedian et al., 2011; Gilson & Lancaster, 2008; Hayes et al., 2010; Lesser & Koniak-Griffin, 2000; Malta et al., 2012; Meltzer-Brody, Bledsoe-Mansori, et al., 2013; Minnes et al., 2008; Muzik et al., 2013; Plaza et al., 2012; Seng et al., 2013). Of these, two found that childhood maltreatment significantly predicted depression only at some points during the postpartum year; however, their findings were somewhat contradictory in that Lesser and Koniak-Griffin (2000) found childhood maltreatment to be predictive of early (four to six weeks) but not later (six or 12 months) depression, while Minnes et al. (2008) found that childhood maltreatment predicted later depression (six or 12 months...
postpartum) but not in the immediate postpartum. Another two studies found a significant bivariate relationship between childhood maltreatment and postpartum depression but not when adjusted for other variables (Hayes et al., 2010; Seng et al., 2013). Three studies found no associations at all (Cohen et al., 2002; Lang et al., 2006; Leigh & Milgrom, 2008). There were limited consistencies across these studies in terms of how and when depression was measured and the setting in which the studies were conducted.

**Anxiety**

*Antenatal.* One of four studies (25%) found a significant positive association between childhood maltreatment and anxiety symptoms during pregnancy (Lang et al., 2006). Two studies found no relationship with anxiety in the third trimester (Gilson & Lancaster, 2008) and across pregnancy more broadly (Grote et al., 2012). The other (Buist et al., 2011) reported a null finding for childhood maltreatment predicting anxiety disorder occurring only in pregnancy, but found a significant positive association with GAD that persisted from before pregnancy into pregnancy.

*Postpartum.* Two of four studies (50%) found that childhood maltreatment predicted anxiety in the postpartum period (Gilson & Lancaster, 2008; Lang et al., 2006). In these studies, different subtypes of childhood maltreatment were examined, and there were both positive and null associations found depending on the maltreatment subtype.
(see section on maltreatment subtypes). Two studies found no associations between childhood maltreatment and postpartum anxiety (Buist, 1998; Malta et al., 2012).

**PTSD**

*Antenatal.* All three studies (100%) described significant positive associations between childhood maltreatment and antenatal PTSD, with one study looking at PTSD before the third trimester (Kulkarni et al., 2011) and the other two focused on PTSD in the third trimester (Huth-Bocks et al., 2013; Yampolsky et al., 2010).

*Postpartum.* Two of two studies (100%) described significant positive associations between childhood maltreatment and postpartum PTSD at six months postpartum (Lev-Wiesel et al., 2009; Muzik et al., 2013). One of these (Lev-Wiesel et al., 2009) found that childhood maltreatment also predicted PTSD earlier at two months postpartum, and that the effect size was larger at two months than later at six months.

**Examining effects by maltreatment subtypes**

Almost half (k=17) of the 35 studies examined exposure to childhood maltreatment in terms of “any” early maltreatment, without discriminating between different subtypes. Three studies only considered a single subtype of maltreatment, specifically sexual abuse (Benedict et al., 1999; Lev-Wiesel & Daphna-Tekoah, 2010; Lev-Wiesel et al., 2009). Fourteen studies analyzed the contributions of different childhood maltreatment subtypes, including sexual abuse, physical abuse, emotional abuse, emotional neglect, and physical neglect. The combinations of subtypes examined were
inconsistent across studies, with some studies looking only at sexual versus physical abuse, and others parsing between all possible subtypes.

Childhood sexual and physical abuse were often each found to predict antenatal depression (Chung et al., 2008) and postpartum depression (Buist, 1998; Dennis & Vigod, 2013; Garabedian et al., 2011; Plaza et al., 2012) at a univariate level. However, multiple studies found that physical abuse— but not sexual abuse— predicted postpartum depression at the multivariate level (Dennis & Vigod, 2013; Garabedian et al., 2011; Plaza et al., 2012), suggesting physical victimization has a more robust independent effect than sexual abuse in the postpartum period. Related to this, physical abuse was observed to predict depression and anxiety at both six weeks and six months postpartum, while sexual abuse only predicted postpartum depression and anxiety at the later time point (Buist, 1998; Gilson & Lancaster, 2008). On the other hand, some studies observed sexual abuse to be a stronger predictor than physical abuse when considering depression and anxiety in the antenatal period (Lang et al., 2006; Chung et al., 2008).

In addition, several studies suggested that childhood emotional abuse and neglect had a particularly important impact on PMADs relative to all other subtypes. For instance, emotional abuse and/or neglect was found predict postpartum depression (Edwards et al., 2008b; Minnes et al., 2008) and anxiety (Lang et al., 2006) even when physical or sexual abuse did not. In addition, studies found that emotional abuse and/or neglect had the largest independent association with antenatal depression (Blalock et al.,
2011) and PTSD (Huth-Bocks et al., 2013) as well as lifetime perinatal depression 
(Meltzer-Brody, Boschloo, et al., 2013), even when other maltreatment subtypes were 
also associated.

**Do observed associations persist when adjusted for covariates?**

**Sociodemographic**

The associations observed between childhood maltreatment and PMADs often 
remained significant even when covariates were included. Childhood maltreatment was 
a significant predictor of PMADs even when adjusting for sociodemographic 
characteristics such as age (Buist et al., 2011; Chung et al., 2008; Fisher et al., 2013; 
Garabedian et al., 2011; Huth-Bocks et al., 2013; Leigh & Milgrom, 2008; Meltzer-Brody, 
Boschloo, et al., 2013; Rich-Edwards et al., 2011), race (Blalock et al., 2011; Buist et al., 
2011; Chung et al., 2008; Rich-Edwards et al., 2011), education (Chung et al., 2008; Leigh 
& Milgrom, 2008; Lev-Wiesel et al., 2009; Meltzer-Brody, Boschloo, et al., 2013), marital 
status (Buist et al., 2011; Chung et al., 2008; Garabedian et al., 2011; Meltzer-Brody, 
Boschloo, et al., 2013), and income or socioeconomic status (Benedict et al., 1999; Chung 
et al., 2008; Dennis & Vigod, 2013; Fisher et al., 2013; Huth-Bocks et al., 2013; Leigh & 
Milgrom, 2008; Malta et al., 2012; Plaza et al., 2012).

**Psychiatric**

With few exceptions (Dayan et al., 2010; Seng et al., 2013), most studies found 
that childhood maltreatment remained a significant predictor of PMADs when adjusting
for maternal psychiatric history (Malta et al., 2012; Plant et al., 2013) such as prior depression (Dennis & Vigod, 2013; Edwards et al., 2008b; Leigh & Milgrom, 2008; Meltzer-Brody, Boschloo, et al., 2013; Plaza et al., 2012; Tzilos et al., 2012), suggesting that the observed relationship between childhood maltreatment and PMADs is not simply due to childhood maltreatment increasing the risk of later psychopathology in general. For example, Plant et al. (2013) found that adjusting for psychiatric history did not explain antenatal depression beyond that already accounted for by childhood maltreatment. Similar findings were observed when adjusting for maternal history of substance use (Edwards et al., 2008b; Garabedian et al., 2011; Tzilos et al., 2012).

Antenatal psychopathology seemed to be an important covariate of childhood maltreatment for postpartum outcomes. One study (Leigh & Milgrom, 2008) found that when antenatal depression was included in the model, it nullified the otherwise significant association between childhood maltreatment and postpartum depression, suggesting that antenatal depression mediates the relationship between childhood abuse and postpartum depression. Meltzer-Brody, Bledsoe-Mansori, et al. (2013) too found that antenatal depression was the largest predictor of postpartum depression – though observed that childhood maltreatment remained significantly associated with postpartum depression even when adjusted for antenatal depression. Similarly, Lev-Wiesel and Daphna-Tekoah (2010) found that childhood sexual abuse predicted
postpartum PTSD when adjusting for antenatal PTSD, though antenatal PTSD was also a large significant predictor.

Psychological attributes were also tested as covariates, including negative cognitive style (Leigh & Milgrom, 2008), personality traits (e.g., neuroticism; Meltzer-Brody et al., 2013), low self-esteem (Leigh & Milgrom, 2008), and optimism or self-confidence (Edwards et al., 2008b; Malta et al., 2012). Most negative psychological factors were found to be predictive of PMADs along with childhood maltreatment, while the positive ones showed inconsistencies: optimism (Malta et al., 2012) did not significantly predict postpartum depression while childhood maltreatment did, but self-confidence (Edwards et al., 2008b) significantly predicted antenatal depression while childhood maltreatment did not.

**Perinatal & reproductive**

Childhood maltreatment remained a significant predictor of PMADs when adjusted for birth parity (Christl et al., 2013; Fisher et al., 2013), history of miscarriage, stillbirth, or abortion (Leigh & Milgrom, 2008; Plant et al., 2013), history of premenstrual syndrome (PMS; Plaza et al., 2012), preference for the sex of the baby (Fisher et al., 2013), and how positively the pregnancy was viewed (Meltzer-Brody, Bledsoe-Mansori, et al., 2013). These factors, when considered alongside childhood maltreatment and other variables, tended not to be associated with the mental health outcome, except history of
PMS (Plaza et al., 2012) and how positively the pregnancy was viewed (Meltzer-Brody, Bledsoe-Mansori, et al., 2013).

Various perinatal factors were also included as covariates to predict mood and anxiety disorders specifically in the postpartum period. These ranged from complicated pregnancy (Garabedian et al., 2011) and low infant birth weight (Hayes et al., 2010) to breastfeeding (Garabedian et al., 2011), parenting stress (Leigh & Milgrom, 2008), and postpartum energy levels (Malta et al., 2012). Low infant birth weight (Hayes et al., 2010) and parenting stress (Leigh & Milgrom, 2008) each emerged as significant predictors of postpartum depression while childhood maltreatment did not, though evidence of the latter's effect was likely reduced due to predictors such as antenatal depression or current violence already in the multivariate model.

**Life events**

The impact of childhood maltreatment on PMADs was further examined in light of negative life events, early and recent. Among childhood adversities considered by Chung et al. (2008) such as homelessness, parental incarceration, and witnessing gun violence, only childhood maltreatment (specifically, sexual abuse) significantly predicted antenatal depression. In terms of more recent adversities, several studies found in multivariate analyses that while childhood maltreatment remained a significant predictor, recent negative life events often emerged as a more statistically
powerful predictor of PMADs (Benedict et al., 1999; Fisher et al., 2013; Leigh & Milgrom, 2008).

**Relational**

*Early caregiving experiences.* One study (Chung et al., 2008) factored in early caregiving experiences such as having positive parental relationships or receiving hugs and emotional affirmation as a child. Maltreatment history showed an interaction with these early experiences, particularly positive maternal relationships, in that childhood sexual abuse only predicted antenatal depressive symptoms when women did not report positive early relationships with their mother.

*Social support.* Several studies found that childhood maltreatment significantly predicted antenatal depression even when adjusted for current social support (Leigh & Milgrom, 2008; Meltzer-Brody, Bledsoe-Mansori, et al., 2013). In these studies, social support consistently showed a protective inverse association with antenatal depression. Similarly, childhood maltreatment remained a significant predictor of postpartum depression when social support was included in the model, though social support had a significant protective effect in some cases (Meltzer-Brody, Bledsoe-Mansori, et al., 2013) and no association in others (Edwards et al., 2008b; Malta et al., 2012). Childhood sexual abuse also predicted postpartum PTSD when adjusting for social support, though social support itself was not a significant predictor (Lev-Wiesel et al., 2009).
**Partner variables.** Childhood maltreatment remained a significant predictor of PMADs when various partner-related variables were examined as covariates, including partner substance use (Dennis & Vigod, 2013) and partner relationship quality during pregnancy (Plant et al., 2013). One exception was observed in Dayan et al. (2010), where partner conflict was related to antenatal depression in multivariate analyses, while childhood maltreatment was not.

**Adulthood interpersonal trauma**

Fourteen studies considered the contributions of adulthood IPV in comparison or tandem with childhood maltreatment. Although one study found that neither form of violence exposure (childhood or adulthood) was predictive of antenatal depression (Dayan et al., 2010), others found that both exposures were similarly predictive of depression in the perinatal period, when considered in parallel analyses (Dennis & Vigod, 2013) or adjusting for each other in the same model (Meltzer-Brody, Boschloo, et al., 2013). Most commonly, studies either supported the conclusion that childhood maltreatment had a more powerful contribution than adulthood IPV, or vice versa. However, findings were mixed, as discussed next.

Six studies suggested that violence experienced as an adult was more predictive of PMADs than childhood maltreatment. Garabedian et al. (2011) reported that when adulthood exposure to violence was included in the model, childhood abuse was no longer a significant predictor of postpartum depression. Hayes et al. (2010) too found
that when both childhood and adulthood trauma were included in the same model, adulthood violence exposure predicted postpartum depression while childhood abuse did not. Similarly, Cohen et al. (2002) observed in parallel analyses that adulthood abuse – but not childhood abuse – was a significant predictor of postpartum depression. Although Rich-Edwards et al. (2011) found that childhood and adulthood exposures to abuse were both predictive of antenatal depression, even when considered together, the effects were greater for recent exposures in a dose-response fashion, with abuse during pregnancy having the largest effect size. Additionally, Fisher et al. (2013) found that adulthood exposure to violence was predictive of depression at more time points during pregnancy than childhood maltreatment.

On the other hand, five studies observed that childhood exposure to abuse was more predictive of PMADs than recent trauma. In parallel analyses, Malta et al. (2012) found that childhood maltreatment predicted postpartum depression, while adulthood IPV did not. Edwards et al. (2008b) reported that when both exposures were entered with other risk factors in a stepwise selection model, childhood abuse was included as a significant predictor of postpartum depression, but not past or current IPV. Benedict et al. (1999) found that childhood sexual abuse but not current violence during pregnancy was associated with antenatal depression. In addition, two studies found that, when adjusted for the influence of the other, childhood abuse and adulthood IPV both predicted antenatal PTSD (Huth-Bocks et al., 2013; Kulkarni et al., 2011) – however, in
both cases, childhood abuse had a larger effect on the mental health outcome than adulthood IPV. For instance, Huth-Bocks et al. (2013) found that when adulthood IPV was added to the model with childhood maltreatment already in it, it only contributed 5% of explained variance for PTSD, whereas childhood maltreatment accounted for 19%.

**S3.4 Discussion**

This systematic review examined the relationship between childhood maltreatment and perinatal mood and anxiety disorders (PMADs). It synthesized findings from 35 studies with a total of 26,239 participants. Across studies, an average of 30% of female participants reported a history of childhood maltreatment. Reported rates of PMADs, while varying, were largely consistent with existing literature (Gavin et al., 2005; Ross, McLean, & Psych, 2006) if not higher due to high-risk samples included among reviewed studies. The elevated levels of childhood maltreatment and PMADs among women emphasize the need for continued research on the psychological impact of lifetime trauma during the perinatal period.

This review documented an overall trend of association between childhood maltreatment and PMADs, though there were variations by type of disorder. Robust trends were found for perinatal depression, with over 75% of studies reporting a positive association, as well as for PTSD, with 100% of studies reporting a positive association. Findings regarding the relationship between childhood maltreatment and perinatal anxiety were less consistent, with 50% or less of studies reporting a positive
association. It could be that anxiety symptoms are relatively common during pregnancy (Ross et al., 2006) and not as strongly influenced by trauma history.

The overall finding that childhood maltreatment was linked to PMADs should be contextualized by studies that examined maltreatment subtypes. Definitive comparisons were challenging to establish due to varied combinations of subtypes explored in each study. However, studies suggested that a history of sexual abuse may have a stronger impact on mental health during pregnancy, while physical abuse may exert greater influence in the postpartum period, but evidence remains limited. Additionally, emotional abuse and neglect seemed to have an important role relative to all other subtypes in predicting PMADs. This deserves greater attention as emotional maltreatment occurs more frequently during childhood than physical or sexual forms of abuse (Hibbard et al., 2012).

**Synthesis: A proposed conceptual model**

Reviewed studies suggested that the association between childhood maltreatment and PMADs is fairly robust, occurring above and beyond possible confounding factors – most notably population characteristics (e.g., age, race, and SES), prior psychopathology, and other childhood adversities. The association between childhood maltreatment and PMADs also persisted when adjusting for the presence of current social support and negative life events, though these variables showed independent influences on PMADs as well. Given the overall associations observed
between childhood maltreatment and PMADs, and accounting for the covariates reported to attenuate or exacerbate these associations, a conceptual model with potential mediators and moderators is proposed in Figure S12, highlighting pathways and influences that might become especially salient in the perinatal period, as discussed next.

![Conceptual Model](image)

**Figure S12. Conceptual model in prior research synthesis**

**Mediating pathways**

*Psychiatric.* Childhood maltreatment may increase general psychopathology in adulthood (Kaplow & Widom, 2007; Springer et al., 2007; Twai et al. & Rodriguez-Srednicki, 2004; Widom et al., 2007), raising the question of whether PMADs are simply extensions...
of preexisting psychological conditions. Yet this review found that psychiatric history did not fully explain the relationship between early trauma and PMADs (Dennis & Vigod, 2013; Edwards et al., 2008b; Leigh & Milgrom, 2008; Malta et al., 2012; Meltzer-Brod, Boschloo, et al., 2013; Plant et al., 2013; Plaza et al., 2012; Tzilos et al., 2012), which suggests that the perinatal period could instead be a time of novel or heightened psychiatric risk for those with a trauma history. Pregnancy usually entails procedures and experiences that can feel stressful, invasive, and beyond the woman’s control (Roller, 2011), which may trigger otherwise latent trauma. Additional studies that examine the novel onset of psychopathology during pregnancy and distinguish it from preexisting conditions could help clarify this hypothesis.

**Social.** Childhood maltreatment is known to increase the likelihood of abusive partner relationships later in life (Chu, 1992; Desai, Arias, Thompson, & Basile, 2002b; Messman-Moore & Long, 2000), which may then contribute more proximally to perinatal psychopathology (Howard et al., 2013). Several reviewed studies found that women who had experienced childhood maltreatment also tended to experience abuse during pregnancy (Benedict et al., 1999; Huth-Bocks et al., 2013). Later victimization may be especially relevant for perinatal depression, since several – though not all – reviewed studies found that adjusting for adulthood IPV nullified the otherwise significant effect of childhood maltreatment on depression (Garabedian et al., 2011; Hayes et al., 2010). On the other hand, for PTSD, reviewed studies tended to find childhood maltreatment more
influential than adulthood IPV (Huth-Bocks et al., 2013; Kulkarni et al., 2011), suggesting its impact may operate relatively independently of later victimization. Childhood maltreatment may set into motion vulnerabilities for PTSD (Mehta et al., 2013) that unfold regardless of later social experiences. However, as described previously, the literature remains inconsistent about how much adulthood trauma accounts for the relationship between childhood maltreatment and PMADs, even depression. This causal pathway, though supported by some evidence, is likely incomplete. Relative contributions of childhood versus adulthood trauma need to be further untangled in studies that systematically measure and compare both.

**Cognitive.** While not specifically examined by reviewed studies, existing literature provides support for mechanisms unfolding at the cognitive level. Attachment and object relations theories, including the Dynamic-Maturational Model of attachment and adaptation (Crittenden, 2006), posit that individuals form cognitive schemas, or mental representations, of the self and others based on early interactive experiences with caregivers (Ainsworth, 1979). Experiencing maltreatment during childhood is thought to profoundly impact attachment and related representations (Chu, 1992; Crittenden & Ainsworth, 1989), increasing cognitive vulnerabilities for later depression (Hammen, 1992; Morley & Moran, 2011). Cognitive vulnerabilities related to childhood maltreatment may become especially salient during pregnancy, when attachment
representations are being activated and reorganized with regard to the unborn child and self as a caregiver (Malone, Levendosky, Dayton, & Bogat, 2010).

*Psychobiological.* A subset of reviewed studies suggested that childhood maltreatment might operate through psychobiological pathways leading to mood and anxiety disorders in the perinatal period. For example, history of childhood maltreatment was associated with HPA axis dysregulation during pregnancy (Shea et al., 2007) and thyroid disturbance in the postpartum (Plaza et al., 2012), both of which can underlie depression (Pariante & Lightman, 2008; Hage & Azar, 2012) and other mental disorders (Shea et al., 2005). Another psychobiological mechanism, not explored in reviewed studies but also plausible, is inflammation. Childhood maltreatment is linked to inflammation during adulthood (Danese, Pariante, Caspi, Taylor, & Poulton, 2007), and inflammatory markers have been found to predict depressive symptoms in adulthood (Dinan, 2009) and specifically during pregnancy (Christian et al., 2009). Women with a maltreatment history may be particularly vulnerable to mood and anxiety disorders during the perinatal period as it is a time marked with dramatic hormonal fluctuations (Kammerer, Vivette, & Glover, 2006) and increased inflammation states (Sacks, Studena, Sargent, & Redman, 1998). These psychobiological pathways require investigation, including how disruptions might be mediated by changes at the cellular and epigenetic level.
Moderator influences

In addition to potential pathways from childhood maltreatment to PMADs, this review uncovered risk and protective factors that may alter the strength of the overall association.

Risk factors. Reviewed studies found that recent adversities were often stronger predictors of antenatal depression when considered alongside childhood maltreatment (Benedict et al., 1999; Fisher et al., 2013; Leigh & Milgrom, 2008), though each variable tended to retain an independent influence. The impact may be synergistic, wherein PMADs are most likely for a woman who has experienced childhood trauma and is also experiencing current life stressors. However, this interaction has yet to be examined. Psychological traits such as neuroticism (Meltzer-Brody, Boschloo, et al., 2013) and genetic alleles linked to greater susceptibility to environmental influences (Dinan, 2009) may also interact with childhood maltreatment to amplify its later effects.

Protective factors. In reviewed studies, social support consistently showed a protective effect against PMADs, especially antenatal depression (Leigh & Milgrom, 2008; Meltzer-Brody, Bledsoe-Mansori, et al., 2013), but did not appear to completely mitigate the impact of childhood maltreatment in that maltreatment often remained a predictor of PMADs even when adjusted for social support. Earlier sources of support also emerged as a key protective factor. Positive early caregiver relationships buffered maltreated individuals from antenatal depression (Chung et al., 2008), consistent with
other recent work confirming that safe, stable, nurturing relationships are crucial for interrupting the intergenerational consequences of maltreatment (Schofield, Lee & Merrick, 2013; Jaffee, Bowes, Ouellet-Morin, et al., 2013). Experiencing such relationships may help repair cognitive schemas following early abuse and thereby reduce psychological vulnerability during women’s own transition to motherhood. Finally, individual factors such as adaptive coping and psychological flexibility could also moderate the impact of childhood maltreatment during the perinatal period, but require further investigation.

**Intergenerational consequences**

While not the main focus of this review, some studies examined the implications of maternal childhood maltreatment and related psychopathology for child and parenting outcomes, as included in Figure S12. These consequences ranged from reduced parenting morale (Malta et al., 2012), impaired parent-child bonding (Muzik et al., 2013), and lower quality of maternal-infant interaction (Lesser et al., 2000), all previously described as risk factors for poor child outcomes such as psychopathology (Springer et al., 2007) and victimization (Pianta, Egeland, & Erickson, 1989). In addition, one study found that maternal childhood maltreatment and antenatal depression actually predicted offspring maltreatment (Plant et al., 2012). These findings suggest that the relationship between maternal childhood maltreatment and PMADs may be
embedded in a larger pathway, by which traumatic childhood experiences in one
generation can profoundly influence outcomes in the next generation.

**Opportunities for clinical intervention**

Mapping onto the proposed conceptual model, there are several prime points
across the life course for clinical intervention. In addition to broader societal efforts to
prevent childhood maltreatment, efforts to protect the developmental trajectory of
maltreated individuals must be undertaken in childhood, through adolescence and into
childbearing years, to interrupt proposed pathways leading to PMADs. These could
include treating emergent psychopathology, preventing revictimization, reorganizing
cognitive schemas, and remediating biological consequences of early trauma. Early
interventions promoting high-quality social support and strong relationships with a
caregiver (Cicchetti, Rogosch, & Toth, 2006; Dozier et al., 2006; Lieberman & Van Horn,
2013; Perry, 2009; Timmer et al., 2006) may address some of these areas and have been
shown to buffer long-term mental health outcomes of maltreated children (Kaufman et
al., 2004), with protective effects observed downstream in the perinatal period (Chung et
al., 2008).

More proximal approaches to optimizing perinatal mental health in the context
of childhood maltreatment should include (a) trauma screening in the context of
preconception care (Sacks et al., 1998) among women of childbearing age, followed by
referrals for preventive trauma counseling and support, and (b) trauma-oriented
treatment of PMADs among pregnant and postpartum women with childhood maltreatment histories. The preconception and perinatal periods may be prime opportunities to address early trauma and its mental health sequelae, since women with previously undetected experiences of maltreatment may engage with the health care system during this time, and be amenable to treatment and support. Trauma-focused psychotherapy (Coussons-Read, Okun, & Nettles, 2007; Lieberman & Van Horn, 2011) during this time may help women with childhood maltreatment histories adapt to the role of motherhood and specifically explore the potential influences of early trauma – and resulting cognitive schemas and biological stress responses – on their mood and anxiety. Mindfulness-based treatments have also shown recent promise in improving cognitive-emotional and attachment processes among women with childhood trauma (Hay et al., 2010). Based on this review, such psychotherapeutic approaches would ideally be complemented by behavioral interventions and/or social services to increase social support, promote adaptive coping with negative life events, and address the presence and impact of any current IPV. In addition, since preliminary evidence suggests that childhood maltreatment subtypes contribute to differential mental health risk across the perinatal period, there may be a need to tailor the timing and delivery of interventions based on specific childhood trauma experiences. For example, women who have experienced sexual abuse may benefit from more intensive treatment in the antenatal period, a time where bodily changes and invasive procedures may incur
distress, while women with a history of physical abuse may require particular support in the postpartum period, when they are interacting with their infant and may be reminded of their own care experiences.

**Limitations of the review and studies reviewed**

Several methodological issues about this review should be noted. First, the review drew only on peer-reviewed journal sources and did not include gray literature such as non-refereed journal articles, book chapters, and dissertations. Study inclusion was also limited to maltreatment during childhood. Given the aim of this review was to explore the role of early trauma in predicting PMADs, several studies were excluded because they considered lifetime trauma but not specific to childhood (Kim, Harrison, Godecker, & Muzyka, 2013; Manzolli et al., 2010; Meltzer-Brody et al., 2011; Mezey, Bacchus, Bewley, & White, 2005; Milgrom et al., 2008; Records & Rice, 2009; Silverman & Loudon, 2010), suggesting a larger base of literature not covered in this paper. Second, there was substantial heterogeneity across reviewed studies, limiting the extent to which findings could be aggregated. Different instruments were used for measuring PMADs, and differential cut-offs were applied for the same instrument, even gold-standard tools such as the EPDS. Timing of assessment also ranged widely across the perinatal timeline (Figure 2). Maternal stress, depression and anxiety are known to vary across this timeline (Heron, O’Connor, Evans, Golding, & Glover, 2004) and can have differential impact on the child depending on the stage of pregnancy (Davis & Sandman, 2010) and
during the postpartum (Gunnar & Fisher, 2006). Sensitive developmental windows in the perinatal period should be used across studies to guide consistent timing of clinical assessments. Third, reviewed studies universally relied on the mother’s retrospective self-report, which may be confounded with her current mental health or biased due to reluctance to disclose sensitive information (Hardt & Rutter, 2004). Future work should capitalize on longitudinal cohorts followed across the life course, so that maltreatment experiences measured prospectively during childhood can be linked to mental health during the transition to parenthood. In addition, objective measures of childhood maltreatment (e.g., child protective services records) may help circumvent reporting biases, though they may miss cases of emotional or sexual maltreatment not detected by formal systems.

Despite these limitations, this study has multiple strengths and contributions. To our knowledge, this systematic review is the first to summarize childhood maltreatment’s specific association with a range of PMADs. This review also explored relative contributions of different maltreatment subtypes, where possible, with emotional maltreatment emerging as a particularly salient risk factor for PMADs. In addition, this review proposed potential mediators and moderators, shifting the inquiry from whether an association between childhood maltreatment and PMADs exists, to how and why that association might occur. In doing so, it has also begun to evaluate the degree to which childhood trauma has unique influence as compared to other
Intervening factors such as later trauma. Finally, this review highlights the perinatal period as a distinct time of psychological risk for women with childhood maltreatment histories.

Implications for future research

Scientific advances rest upon several key research efforts. As mentioned before, due to limitations of retrospectively assessing childhood maltreatment, prospective life-course designs are needed. While potential causal pathways have now been suggested, mediation analyses have not been conducted to date. Future research should strive to account for the multilevel pathways by which this overall association is proposed to occur, using formal structural modeling methods to test causality. Concurrently, moderator analyses must determine which intervening characteristics or experiences can most offset the impact of childhood maltreatment. Important to consider is that the observed associations are not deterministic – not all women who experience childhood maltreatment develop PMADs, and thus mixed-methods approaches could be employed to better understand processes of resilience.

Populations of emphasis for future research include adolescents and cross-cultural populations. Females exposed to childhood maltreatment are more likely to experience teen childbirth (Noll & Shenk, 2013), and pregnant teens tend to report higher rates of mental health difficulties (Koniak-Griffin, Walker, & de Traversay, 1996). Thus, studying trauma and PMADs in childbearing adolescents continues to be an
important area for inquiry (Meltzer-Brody, Bledsoe-Mansori, et al., 2013). In addition, though notable socioeconomic disparities exist in observed rates of antenatal depression, there has been limited investigation of the relationship between childhood maltreatment and PMADs in low- and middle-income countries (LMICs). This paucity of research stands in contrast to the emerging appreciation that perinatal mental disorders may be particularly problematic in LMIC settings where maternal care makes a critical difference for child survival and growth (Rahman, Patel, Maselko, & Kirkwood, 2008). Correspondingly, there is increasing recognition of the global prevalence and burden of childhood maltreatment (Cyr et al., 2013) especially in LMICs (Skeen & Tomlinson, 2013). Incorporation of research from LMICs will help to illuminate universal versus culturally bound pathways between childhood maltreatment and PMADs, to better inform intervention in these settings.

Future work should also investigate a broader spectrum of mental disorders. The evidence base on perinatal anxiety remains limited and methods of assessment varied across reviewed studies, so further work using standard instruments could help determine whether inconsistent findings stem from methodological or conceptualization issues. Other perinatal psychopathologies such as OCD and more severe disorders such as psychosis also require investigation. Finally, future work should explore the intergenerational consequences of childhood maltreatment. A wealth of research has found that parents who are maltreated as children are more likely to have children.
exposed to maltreatment (Collishaw, Dunn, O'Connor, & Golding, 2007; Dixon, Browne, & Hamilton-Giachritsis, 2005a; Dixon et al., 2005b; Sidebotham & Heron, 2006) and at risk for psychopathology (Roberts et al., 2004), a phenomenon referred to as intergenerational transmission of trauma. PMADs may play a role in this transmission pathway, occurring at the earliest point of intersection between two generations; however, further studies testing this hypothesis are needed. If so, the perinatal period may offer a key opportunity to interrupt intergenerational trauma processes.

**Conclusion**

Childhood maltreatment is a potent and common source of trauma (Carlson et al., 1997; Gilbert et al., 2009; Roller, 2011). Existing literature shows that maternal experiences of childhood maltreatment may uniquely predict perinatal mood and anxiety disorders (PMADs), especially depression and PTSD. Intervening with maltreated women across the life course may help mitigate affective psychopathology during the perinatal period and its far-reaching consequences for both mother and child.
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Biography

Karmel Choi graduated summa cum laude from Duke University with a bachelor’s degree in Psychology and certificate in Child Policy Research. She then received a Hart Research Fellowship from the Duke Sanford School for Public Policy to conduct community-based research with a maternal and child health NGO (Action Research and Training for Health) in India. Karmel began her PhD in clinical psychology at Duke in 2011. During her PhD training, she published in numerous scientific journals including Social Science & Medicine and Trauma, Violence, & Abuse, and received a Sulzberger-Levitan Social Policy Graduate Research Fellowship from the Duke Center for Child & Family Policy, a Doctoral Scholars Award from the Duke Global Health Institute, and a Myra and William Waldo Boone Fellowship from the Duke Graduate School. In recognition of her dissertation research, she was also awarded a prestigious Doris Duke Fellowship for the Promotion of Child Well-Being from Chapin Hall in Chicago. She is a member of the International Society for Traumatic Stress Studies (ISTSS) and the Society for Behavioral Medicine (SBM). She will complete her clinical internship in Behavioral Medicine at Harvard Medical School/Massachusetts General Hospital and pursue a career in psychological research.