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Maternal Depression in the Intergenerational Transmission of Childhood Maltreatment and Psychological Sequelae: Testing Postpartum Effects in a Longitudinal Birth Cohort

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1 Abstract (200 words)

2 Mothers who have experienced childhood maltreatment are more likely to have children also
3 exposed to maltreatment, a phenomenon known as intergenerational transmission. Factors in
4 the perinatal period may contribute uniquely to this transmission but timing effects have not
5 been ascertained. Using structural equation modeling with 1,016 mothers and their 2,032
6 children in the Environmental Risk (E-Risk) Longitudinal Twin Study, we tested the mediating
7 role of postpartum depression between maternal childhood maltreatment and a cascade of
8 negative child outcomes, specifically child exposure to maltreatment, internalizing symptoms,
9 and externalizing symptoms: (1) adjusting for later maternal depression, (2) comparing across
10 sex differences, and (3) examining the relative role of maltreatment subtypes. Mothers who had
11 been maltreated as children were at increased risk for postpartum depression, especially those
12 who had experienced emotional or sexual abuse. In turn, postpartum depression predicted
13 children's exposure to maltreatment, followed by emotional and behavioral problems. Indirect
14 effects from maternal childhood maltreatment to child outcomes were robust across child sex
15 and supported significant mediation through postpartum depression; however, this appeared to
16 be carried by mothers' depression beyond the postpartum period. Identifying and treating
17 postpartum depression—and preventing its recurrence—may help interrupt the
18 intergenerational transmission of maltreatment and its sequelae.

19
20 Keywords: Intergenerational transmission, child maltreatment, postpartum depression, perinatal
21 mental health, childhood trauma

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Introduction

Childhood maltreatment is a potent and common form of early trauma (Cicchetti & Toth, 2005; Stoltenborgh et al., 2015). Exposure to childhood maltreatment has been shown to compromise healthy development across critical domains (Cicchetti, 2016), with detrimental effects persisting into adulthood (Norman et al., 2012). Adding to this concerning picture, research suggests that childhood maltreatment not only produces negative outcomes for individuals during their own lifetime, but also has consequences extending to the next generation (Roberts et al., 2004). Studies have long observed that parents who have experienced childhood maltreatment are more likely to have children likewise exposed to maltreatment (Egeland et al., 1988; Pears & Capaldi, 2001), a phenomenon referred to as intergenerational transmission of maltreatment (Thornberry et al., 2012).

Although a wide range of transmission rates has been documented (Oliver, 1993), 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 heightened maltreatment potential (DiLillo et al., 2000; Rodriguez & Tucker, 2011) as well as disrupted parenting behaviors more broadly (Bert et al., 2009), which can contribute to maladjustment and psychopathology in the next generation—thereby transmitting to children negative sequelae of maltreatment in addition to maltreatment itself. Together, a better understanding of what facilitates intergenerational transmission of maltreatment and its sequelae is needed to inform critical efforts to interrupt such transmission.

Since the phenomenon of intergenerational transmission was proposed, studies have sought to explore relevant mechanisms. Classic explanatory frameworks have included (1)

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1 social learning theory (Bandura, 1973), through direct modeling of abusive behavior, and (2)
2 attachment theory (Ainsworth, 1979; Bowlby, 1978), through disrupted working models of
3 relationships that influence subsequent relationships, including with one's children (Zeanah &
4 Zeanah, 1989). Caregiver mental health may represent another key pathway. Maternal
5 depression has been associated with maltreating behaviors in early childhood (Windham et al.,
6 2004), potentially by reducing emotional resources to respond to caregiving demands.
7 Furthermore, maternal depression is relatively easy to detect by self-report or clinical interview
8 and also amenable to intervention, making it a tractable risk factor from a public health
9 perspective. In this study, it is proposed that maternal depression in the perinatal period,
10 occurring at the earliest intersection between generations, may fundamentally contribute to
11 maltreatment and its sequelae in the next generation. Emerging findings from a separate stream
12 of literature—perinatal mental health—may be relevant for this hypothesis.

13 Perinatal mental health research is concerned with the study of prevalence, risk factors,
14 and consequences of mental disorders during the perinatal period, common of which is
15 postpartum depression (Wisner et al., 2002). Of relevance, women who have experienced
16 maltreatment as children are found at greater risk for postpartum depression (Alvarez-Segura et
17 al., 2014; Choi & Sikkema, 2016). While maltreatment history is a risk factor for
18 psychopathology across the life course (Widom et al., 2007), it may particularly kindle
19 depression in the postpartum period, a time fraught with hormonal shifts (Hendrick et al.,
20 1998), physical recovery from childbirth (Brown & Lumley, 2000), and heightened demands of
21 caregiving (Campbell et al., 1992) which may also reactivate own early memories of
22 caregiving. In turn, postpartum depression is recognized for its short- and long-term impacts on
23 child outcomes (Choi et al., 2017; Murray & Cooper, 1996), even beyond later maternal

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1 depression in the child's life (Hay et al., 2003; Murray et al., 2010). This may be due to
2 disturbances in maternal caregiving behavior and exposure to depressed maternal affect during
3 a critical time for child development (Field, 2010), which could set a long-term foundation for
4 negative interactions and increased vulnerability to emotional/behavioral problems. Thus, while
5 there is likely no single factor underlying intergenerational transmission of maltreatment
6 (Dixon et al., 2005), maternal depression is expected to be an important conduit for
7 transmitting negative outcomes to children, particularly during sensitive developmental
8 windows such as the postpartum period.

9 Prior work with longitudinal cohorts has examined how the timing of maternal
10 depression affects child outcomes (Barker, 2013). To date, research has not disentangled timing
11 effects of maternal depression in relation to intergenerational transmission of maltreatment—
12 for instance, whether postpartum depression contributes critically to this transmission pathway,
13 or whether this is better explained by maternal depression across development broadly. A better
14 understanding of timing effects would inform when interventions should best be targeted.
15 According to a sensitive window hypothesis, maternal depression during the first year of life is
16 expected to be uniquely impactful above and beyond later exposure(s) (Bagner et al., 2010;
17 Bureau et al., 2009). This may be why treating maternal depression in early childhood has been
18 suggested to reduce later maltreatment (McCann et al., 1992). However, no studies have
19 formally examined postpartum and ensuing depression in the pathway between maternal
20 childhood maltreatment and long-term child outcomes, including child victimization and later
21 psychopathology. Alternatively, since early depressive episodes are known to kindle or
22 increase risk for further episodes (Monroe & Harkness, 2005), it could be that cumulative
23 exposure to maternal depression is most noxious.

1 Participants were mothers and children involved in the Environmental Risk (E-Risk)
2 Longitudinal Twin Study, which tracks the development of a birth cohort of British children.
3 This cohort sample was drawn from a larger birth register of twins born in England and Wales
4 in 1994-1995. Full details about the sample are reported elsewhere (Moffitt, 2002). Briefly, the
5 E-Risk sample was constructed in 1999-2000, when 1,116 families (93% of those eligible) with
6 same-sex 5-year-old twins participated in home visit assessments. Families were recruited to
7 represent the UK population of families with newborns in the 1990's, based on (a) residential
8 location throughout England and Wales and (b) maternal age, with over-selection of teenaged
9 mothers and under-selection of older mothers having twins via assisted reproduction. Higher-
10 risk households were deliberately oversampled to compensate for their selective loss from the
11 register due to non-response and likely attrition over time.

12 At follow-up, the resulting sample of households represented the full range of
13 socioeconomic conditions in the UK, as captured by a neighborhood-level index (ACORN; A
14 Classification of Residential Neighborhoods, developed by CACI Inc. for commercial use in
15 the UK). ACORN utilizes census and other survey-based geodemographic data to classify
16 neighborhoods across the UK into five categories ranging from “wealthy achievers” (Category
17 1; 26% E-risk families vs. 26% UK), “urban prosperity” (Category 2; 5% vs 12%) and
18 “comfortably off” (Category 3; 30% vs 27%) to “moderate means” (Category 4; 13% vs. 14%)
19 and “hard-pressed” neighborhoods (Category 5; 26% vs. 21%). The ACORN distribution of
20 households participating in the E-Risk study closely matched the nationwide distribution across
21 all categories (Odgers et al., 2012), though underrepresented the “urban prosperity” category
22 (e.g., young professionals) because such households are likely to be childless.

23 **Procedures**

1 Home visit assessments began in 1999-2000 when children were 5 years old, and
2 follow-up assessments reported in this article were conducted when children were 7 (98%
3 participation), 10 (96% participation), and 12 (96% participation). An overview of procedures
4 at each assessment is publicly available (Medical Research Council, 2017). Informed consent
5 was initially obtained from mothers and assent given by the children through their 12-year
6 assessment. At all phases, procedures were approved by the Joint South London and Maudsley
7 and the Institute of Psychiatry NHS Research Ethics Committee.

8 **Measures**

9 **Maternal childhood maltreatment.** Mothers were administered the Childhood Trauma
10 Questionnaire (CTQ) as part of a structured face-to-face interview. The CTQ (Bernstein &
11 Fink, 1998) is a widely used 28-item scale that retrospectively measures exposure to
12 maltreatment before the age of 18, including physical abuse, sexual abuse, emotional abuse,
13 emotional neglect, and physical neglect. An overall maltreatment score was available as a
14 continuous variable (with possible scores between 25-125), as were continuous scores for each
15 of the five maltreatment subtypes (with possible scores between 5-25). Continuous scores were
16 used for all main analyses. As in previous E-Risk research (Jaffee et al., 2013) and for
17 descriptive purposes, dichotomous variables were created to reflect moderate-to-severe
18 exposure to each of the childhood maltreatment subtypes, as per CTQ manual cut-offs
19 (Bernstein & Fink, 1998), and substantial exposure to any childhood maltreatment was
20 determined by moderate-to-severe exposure to at least one or more maltreatment subtypes.

21 **Maternal postpartum depression.** At the first assessment, mothers were interviewed
22 by a trained clinician regarding their lifetime depressive symptoms up to when the twins were 5
23 years old, using the standardized Diagnostic Interview Schedule based on DSM-IV criteria

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1 (American Psychiatric Association, 2000). Mothers who met criteria for lifetime major
2 depressive disorder were then asked to refer to the Life History Calendar (LHC) in order to
3 specify the timing of their depressive episodes. If mothers did not meet criteria for lifetime
4 major depressive disorder, their score on all reference periods was entered as zero. The LHC is
5 a reliable visual method for recalling the occurrence, timing, and duration of life events,
6 including psychopathology (Caspi et al., 1996). Specifically, the reliability of recalling
7 depressive episodes using the LHC method was separately evaluated using a one-month test-
8 retest and determined to be high at 93% (Kim-Cohen et al., 2005). Mothers were asked to
9 indicate whether they had experienced depression during various reference periods, including
10 the first year following the twins' birth (i.e., postpartum year). For postpartum depression,
11 maternal reports of depression specifically during the postpartum year were extracted into a
12 dichotomous variable.

13 **Later maternal depression.** Later maternal depression was assessed in study mothers
14 using the Diagnostic Interview Schedule and Life History Calendar together across subsequent
15 assessments. A variable was created to reflect the cumulative number of periods in which the
16 mother was depressed between 1 and 10 years of the twins' lives (1 to 4 years, 5, 6, 7, 8, 9, 10
17 years), *with cumulative periods modeled categorically (ordinally) in all main analyses*. This
18 variable did not include maternal depression at 12 years to minimize conflation with reported
19 child outcomes at 12 years. *For descriptive purposes*, a dichotomous variable reflecting any
20 later maternal depression between 1 to 10 years was *also* derived.

21 **Child exposure to maltreatment.** Child exposure to physical and sexual maltreatment
22 by an adult was assessed using a validated structured interview protocol (Dodge et al., 1990)
23 administered to mothers at each phase of assessment (5 years, 7 years, 10 years, 12 years of

1 twins' lives). Details of this measure have been previously reported (Arseneault et al., 2011;
2 Jaffee et al., 2013; Polanczyk et al., 2009). Briefly, standardized questions were designed to
3 sensitively and validly elicit information about potential maltreatment (e.g., "Do you remember
4 any time when your child was disciplined severely enough that he or she may have been hurt?"
5 or "Next, I want to ask specifically about harm to your child of a sexual nature."). At each
6 phase, any positive reports were probed by the interviewer for further details about the incident
7 and to rule out accidental harm or harm from peers. This narrative information was documented
8 in a dossier along with maternal narratives and any referrals made. Each dossier was
9 maintained over phases of assessment and then independently reviewed at 12 years by two
10 clinical psychologists to reach consensus about likelihood of maltreatment occurrence between
11 birth to 12 years of age. Initial inter-rater agreement between coders exceeded 90% and
12 discrepancies were resolved through consensus review. Child exposure to harm was indexed in
13 a three-level categorical variable: none, probable, and definite. Examples of probable harm
14 included instances in which a mother reported spanking her child and leaving marks or bruises,
15 where sexually inappropriate behavior from an adult was suspected and resulted in preventive
16 action, or where social services had been contacted due to concerns about child maltreatment.
17 Examples of definite harm included instances where children sustained serious injuries from
18 neglectful or abusive care, were discovered to be subject to inappropriate sexual contact from
19 an adult, or were already registered on a child protective registry. A dichotomous variable was
20 created to reflect likely maltreatment exposure (probable or definite) versus no such exposure,
21 as in previous research (Jaffee et al., 2004).

22 **Child internalizing and externalizing symptoms.** Child internalizing and
23 externalizing symptoms were measured at 12 years using the Child Behavior Checklist (CBCL)

1 (Achenbach, 1991b; Achenbach & Rescorla, 2001) for mothers and the Teacher Report Form
2 (TRF) (Achenbach, 1991a) for schoolteachers. Mother and teacher ratings were summed to
3 provide an overall measure of child symptomatology across settings (Cairns et al., 2005) and in
4 order to incorporate multiple sources of variance, since children may behave or express
5 emotions differently across settings. Continuous scores on withdrawn and anxious/depressed
6 subscales were combined to form the overall internalizing problems scale, **while continuous**
7 **scores on aggressive and delinquent behavior subscales were combined to form the overall**
8 **externalizing problems scale. Given combined mother-teacher ratings and use of a UK-based**
9 **sample, overall raw scores were used and reported for all analyses.**

10 **Covariates.** Maternal age in years reflected the mother's age when twins were born.
11 Maternal socioeconomic status was indexed using a standardized composite of family income,
12 education, and social class indicators measured at the first assessment. These correlated
13 indicators were found to load significantly onto a single latent factor (Trzesniewski et al.,
14 2006). Based on cohort-wide distribution of scores on this latent factor, mothers were divided
15 into three tiers reflecting overall socioeconomic standing. This three-level categorical variable
16 was coded in this study to reflect increasing levels of socioeconomic disadvantage. Regarding
17 descriptive child characteristics, categorical information on child sex (male versus female) and
18 twin zygosity status (monozygotic versus dizygotic) was also obtained at the first assessment.

19 **Analytic Strategy**

20 **Main analyses.** Descriptive analyses were initially conducted in SPSS (SPSS IBM
21 v.23, Armonk, NY) to understand patterns of childhood maltreatment and depression as
22 experienced by study mothers, and to characterize outcomes experienced by study children.
23 Then, structural equation modeling (SEM) was conducted using Mplus v.7.3 (Muthén &

1 Muthén, 1998-2014) to test the hypothesized interrelationships between maternal childhood
 2 maltreatment, maternal postpartum depression, and child outcomes. SEM allows for
 3 associations to be simultaneously evaluated rather than estimating multiple independent
 4 regressions, and also permits estimation of both direct and indirect effects (Bollen, 1987). Data
 5 from both twins were included in the SEM analyses. To account for nesting within mothers, the
 6 dataset was structured at the family level so that each twin's data were included as variables
 7 within the same family case, with elder versus younger twin variables distinguished
 8 accordingly. Twin outcomes on a same variable (e.g., externalizing symptoms) were mapped as
 9 indicators with equal factor loadings onto one latent outcome variable, as consistent with the
 10 common fate model for dyadic data (Ledermann & Kenny, 2012; Peugh et al., 2013), with
 11 factor mean and variance set to zero and one, respectively. As a sensitivity analysis for this
 12 twin-based approach, a similar structural model was initially tested where only one child from
 13 each pair was randomly selected for a singleton analysis without latent twin variables.

14 To test continuity of maltreatment and related sequelae across generations, preliminary
 15 path analyses were conducted using maximum likelihood estimation to examine maternal
 16 childhood maltreatment scores as a predictor of each individual latent child outcome. Next, an
 17 overall structural model was used to test the extent to which postpartum depression mediates
 18 the association between maternal history of childhood maltreatment and child exposure to
 19 maltreatment and subsequent child internalizing and externalizing symptoms. Given the
 20 inclusion of categorical variables (e.g., postpartum depression) in the overall model, all
 21 subsequent analyses used WLSMV (weighted least squares means and variance adjusted)
 22 estimation. Specifically, maternal childhood maltreatment was predicted to influence
 23 postpartum depression; postpartum depression was predicted to influence child harm exposure

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1 between birth and 12 years; and child harm exposure was predicted to subsequently influence
2 child internalizing and externalizing symptoms at 12 years. Direct paths were also estimated
3 between maternal childhood maltreatment and child outcomes, and between postpartum
4 depression and child internalizing and externalizing symptoms, to explore any influences above
5 and beyond the hypothesized pathway. Child internalizing and externalizing symptom residuals
6 were allowed to covary. Within each twin pair, children were expected to be interchangeable—
7 i.e., meaningfully different on outcome variables based on order of birth (elder versus
8 younger). This assumption was explicitly tested using a chi-square difference test, which
9 revealed no significant worsening of model fit for a nested model when outcome means and
10 variances were specified as equal across elder and younger twins, so this specification was
11 preserved throughout model testing. All possible indirect effects of maternal childhood
12 maltreatment on child outcomes were queried.

13 SEM results were evaluated sequentially. First, to assess overall model fit, comparative
14 fit indices (CFI/TLI, with acceptable values $>.90$) and the root mean square error of
15 approximation (RMSEA, with acceptable values $<.08$ and associated 90% CIs) were examined
16 as statistical tests of the goodness of fit of the overall model (Schreiber et al., 2006). Second,
17 the direct effects of maternal childhood maltreatment on child outcomes were examined. Third,
18 the mediating effect of postpartum depression was evaluated. After this initial evaluation,
19 maternal age and socioeconomic disadvantage were entered as covariates for all endogenous
20 variables in the model. Finally, paths were added structurally to test the extent to which later
21 maternal depression might carry the mediating effect of postpartum depression on child
22 outcomes. This final model was again evaluated for overall fit as well as its direct and indirect

1 effects. All effects were interpreted using standardized coefficients depending on the scale
2 (continuous or categorical) of the independent variable.

3 **Testing for moderation by child sex.** To probe for potential differences in
4 susceptibility to maternal risk across child sex, multiple-group testing was performed with the
5 final model to determine whether structural pathways differed across male and female children.
6 Chi-square difference tests were conducted to compare a model in which all structural paths
7 leading to child outcomes were constrained across male and female groups, against a model in
8 which these parameters were free to vary. In each model, pathways between maternal variables
9 (e.g., maternal trauma to postpartum depression) were fixed as equal across groups, as they
10 were not theoretically suspected to differ across male and female children. All other parameters
11 were free to vary. If the chi-square difference statistic was non-significant, the more restricted
12 sex-neutral model was considered acceptable given no substantial worsening in fit; conversely,
13 a significant result would suggest that imposing parameter restrictions led to significantly
14 worsened model fit, such that the sexes should be considered separately.

15 **Examining role of maltreatment subtypes.** To evaluate the relative impact of specific
16 forms of maternal childhood maltreatment on child outcomes, both directly and indirectly
17 through postpartum depression, a structural model based on the adjusted final model was tested
18 to account for maltreatment subtypes using five predictor variables decomposed from the
19 overall maltreatment variable (physical abuse; sexual abuse; emotional abuse; emotional
20 neglect; physical neglect). Inter-correlations between maltreatment subtypes were
21 automatically factored into the model given their specification as exogenous variables.

22 **Results**

23 **Missing Data**

1 Among the 1,116 E-Risk families, a small subset had CTQ data completed by an
2 individual other than the mother (e.g., father, grandparent). These cases were excluded from the
3 present study because the CTQ data would not reflect maternal trauma, leaving 1,038 families
4 with CTQ data completed by the mother herself. Of these, 22 cases had missing maternal data
5 (eight on postpartum depression and 14 on CTQ) and were excluded from analysis as **WLSMV**
6 **would handle this data using pairwise deletion**, resulting in a final sample of 1,016 mothers and
7 their children. The subsample with missing data did not differ significantly from the final
8 analytic sample on socioeconomic categories, child sex, twin zygosity, or maternal age.

9 **Descriptive Findings**

10 Sample characteristics are summarized in Table 1 for mothers and Table 2 for children.
11 Of note, 18% ($N=180$) of mothers endorsed clinical depression in the postpartum year, and the
12 majority of these women (83%, $N=150$) reported at least some depression in later years, while
13 17% of mothers ($N=30$) reported depression only in the postpartum year. Almost one in four
14 mothers (24%, $N=248$) experienced at least one type of childhood maltreatment at the
15 moderate/severe level. The most common maltreatment subtype was emotional neglect (16%),
16 followed by emotional abuse and sexual abuse (both 11%); physical abuse was reported by 8%
17 of mothers and 6% reported physical neglect. Among the children, there was a relatively even
18 balance across gender as well as across identical versus fraternal twins. One in five children
19 was determined to have experienced likely harm before 12 years of age.

20 Among mothers who reported postpartum depression, 40% ($N=72/180$) had at least one
21 child who was later exposed to maltreatment, compared to 22% ($N=188/836$) among mothers
22 without postpartum depression. Even in cases of discordant maltreatment exposure where only
23 one twin was exposed to maltreatment, independent sample t-tests indicated the twin who was

1 exposed to maltreatment had slightly higher but not significantly different internalizing or
 2 externalizing scores than the non-exposed twin, suggesting that a shared context where at least
 3 one child is being maltreated has consequences for both twins.

4 **Structural Model Findings**

5 **Continuity of maltreatment and related sequelae across generations.** In individual
 6 path analyses, maternal childhood maltreatment significantly predicted children's harm
 7 exposure ($B=.32, p<.001$), internalizing symptoms ($B=.33, p<.001$) and externalizing symptoms
 8 ($B=.35, p<.001$), supporting continuity of maltreatment and related sequelae in the next
 9 generation. These significant paths persisted even when adjusted for maternal age and
 10 socioeconomic disadvantage, which would be later added to the full structural model.

11 **Mediating effect of postpartum depression on child outcomes.** The main structural
 12 model with postpartum depression (Figure 1; significant paths shown, with standardized
 13 coefficients) fit the data well, $\chi^2(20)=60.06, p<.001$, RMSEA=.04 [.032-.058],
 14 CFI/TLI=.99/.98, explaining 15% of variance in child harm exposure, 20% of variance in child
 15 internalizing symptoms, and 24% of variance in child externalizing symptoms (all $p<.001$).
 16 With regards to direct effects, maternal childhood maltreatment significantly predicted
 17 postpartum depression ($B=.22, p<.001$). In turn, postpartum depression significantly predicted
 18 child exposure to harm ($B=.22, p<.001$), which in turn predicted child internalizing ($B=.27,$
 19 $p<.001$) and externalizing symptoms ($B=.34, p<.001$). Maternal childhood maltreatment also
 20 had significant direct paths to each child outcome ($p<.001$, see Figure 1 for standardized
 21 coefficients).

22 In terms of mediating effects, there was a significant indirect effect of maternal
 23 childhood maltreatment on child harm exposure through postpartum depression ($B= .05,$

1 $p=.002$). For child internalizing symptoms, total indirect effects of maternal childhood
2 maltreatment through the hypothesized model were also significant ($B=.11, p<.001$).
3 Decomposition of specific indirect effects showed significant mediating pathways through the
4 combined pathway of postpartum depression → subsequent child harm ($B=.01, p=.011$) and
5 through child harm exposure alone ($B=.07, p<.001$). The indirect pathway through postpartum
6 depression without child harm exposure was non-significant. For child externalizing symptoms,
7 total indirect effects were similarly significant ($B=.12, p<.001$) and decomposition of specific
8 indirect effects again showed significant mediating pathways through postpartum depression →
9 subsequent child harm ($B=.02, p=.004$) and through child harm exposure alone ($B=.09,$
10 $p<.001$). The indirect pathway through postpartum depression without child harm exposure was
11 non-significant.

12 **Sensitivity analyses.** First, an analysis using only one randomly selected child from
13 each twin pair revealed the same pattern of significant direct and indirect effects (not shown) as
14 in the twin-based model, thus the more comprehensive twin-based approach was conserved in
15 subsequent analyses. Second, the comprehensive model was examined separately based on
16 mother- versus teacher-only reports of child symptoms, as opposed to combined reports. Each
17 model continued to fit well, though minor pathway differences were found by informant (as
18 shown in Figure 2). Specifically, when mothers reported on child symptoms, all model
19 pathways were significant as before, but postpartum depression also predicted child
20 internalizing symptoms directly, and thus significantly mediated between maternal childhood
21 maltreatment and child internalizing symptoms above and beyond child harm exposure. When
22 teachers reported on child symptoms, the pathway from child harm exposure to internalizing
23 symptoms was not significant; however, postpartum depression predicted child externalizing

1 symptoms directly, and thus significantly mediated between maternal childhood maltreatment
2 and child externalizing symptoms above and beyond child harm exposure. While these results
3 were used to contextualize main findings (see Discussion), combined reports across mothers
4 and teachers were used in subsequent analyses since a comprehensive measure of child
5 symptoms across settings was desired and different reporters are able to provide
6 complementary evidence about children's emotional/behavioral problems (Cairns et al., 2005).

7 **Adjusting for sociodemographic factors.** The model retained good fit even when
8 maternal age and socioeconomic disadvantage were added as covariates, $X^2(26)=64.76, p<.001$,
9 RMSEA=.038 [.027-.050], CFI/TLI=.99/.98, and the overall pattern of significant direct and
10 indirect effects remained the same. In addition, socioeconomic disadvantage significantly
11 predicted each of the child outcomes ($B=.11, p=.010$ for harm exposure; $B=.16, p=.002$ for
12 internalizing symptoms; $B=.26, p<.001$ for externalizing symptoms), while maternal age was a
13 significant predictor of child harm exposure ($B=-.12, p=.004$) such that younger mothers had a
14 higher risk of later child harm. This adjusted model explained 19% of variance in child harm
15 exposure, 22% of variance in child internalizing symptoms, and 30% of variance in child
16 externalizing symptoms (all $p<.001$).

17 **Exploring the potential role of later maternal depression.** When later maternal
18 depression was included structurally in the adjusted model so it followed postpartum
19 depression and was also allowed to predict each of the child outcomes (see Figure 3; significant
20 paths shown, with standardized coefficients), the resulting model continued to fit the data well,
21 $X^2(30)=96.35, p<.001$, RMSEA=.047 [.036-.057], CFI/TLI=.98/.97. In this model, maternal
22 childhood maltreatment significantly predicted postpartum depression ($B=.32, p<.001$).
23 Postpartum depression then significantly predicted later maternal depression ($B=.66, p<.001$)

1 which in turn predicted child harm exposure ($B=.18, p=.027$), internalizing symptoms ($B=.20,$
2 $p=.016$) and externalizing symptoms ($B=.15, p=.033$); however, postpartum depression was no
3 longer a significant direct predictor of child outcomes. Maternal childhood maltreatment
4 continued to have significant direct paths to each of the child outcomes ($p<.001$, see Figure 3
5 for coefficients). Socioeconomic disadvantage predicted later maternal depression ($B=.13,$
6 $p=.004$), though not postpartum depression.

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

1 **Testing the moderating effects of child sex.** Means and variances were allowed to
 2 vary across male and female children; as expected, boys on average had higher externalizing
 3 symptoms than girls. When the moderating effect of child sex on structural paths was tested,
 4 chi-square difference tests revealed a significant worsening in model fit from the freed model
 5 (in which all structural paths to child outcomes were allowed to vary across males and females)
 6 to the more constrained model (in which these paths were set as equal), $\chi^2\Delta(17)=29.41, p=.031$.
 7 However, follow-up difference tests that constrained paths either from (1) maternal childhood
 8 maltreatment to child outcomes (direct effects) or (2) postpartum or later maternal depression
 9 to child outcomes (mediator effects) were non-significant, suggesting these paths were
 10 comparable across male and female children, at least when adjusting for all paths considered.
 11 Significant worsening in fit was found only when (3) paths from maternal demographic
 12 covariates to child outcomes were constrained across male and female children, $\chi^2\Delta(6)=18.43$,
 13 $p=.005$. Inspection of unconstrained path coefficients between groups revealed that in this
 14 model, maternal socioeconomic disadvantage predicted child harm exposure among males
 15 ($p=.005$) but not females ($p=.993$); internalizing symptoms for females ($p=.005$) but not males
 16 ($p=.282$); and externalizing symptoms for both sexes ($p<.001$). Younger maternal age at birth
 17 significantly predicted child harm exposure only for females ($p<.001$). These findings suggest
 18 some differential susceptibility of male versus female children to contextual risks but not to the
 19 overall intergenerational transmission of maltreatment through maternal depression.

20 **Examining maltreatment subtypes.** When all maltreatment subtypes were entered
 21 instead of overall maltreatment in the final structural model with covariates (Figure 4), the
 22 resulting model fit the data well, $\chi^2(46)=104.94, p<.001$, RMSEA=.036 [.027-.045],
 23 CFI/TLI=.98/.96, and explained comparable variance in child outcomes as the overall

1 maltreatment model, with similar pathways. Among subtypes, maternal histories of emotional
2 abuse and sexual abuse were significant predictors of postpartum depression ($B=.24, p<.001$ for
3 emotional abuse; $B=.12, p=.005$ for sexual abuse) above and beyond the other subtypes.
4 Additionally, maternal history of emotional neglect had significant direct paths to child harm
5 exposure ($B=.12, p=.017$), child internalizing symptoms ($B=.10, p=.042$), and marginally
6 externalizing symptoms ($B=.07, p=.060$). Maternal history of physical abuse had a significant
7 direct relationship with child externalizing symptoms ($B=.14, p<.001$).

8 **Discussion**

9 **Summary and Integration**

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

18 As expected, maternal childhood maltreatment was associated with greater risk for
19 postpartum depression. Mothers who reported postpartum depression had nearly twice the
20 prevalence (40% vs. 22%) of having a child exposed to later maltreatment. In SEM analyses,
21 postpartum depression significantly mediated the relationship between maternal childhood
22 maltreatment and child harm exposure, with subsequent influences on child internalizing and
23 externalizing symptoms at 12 years. This mediating effect persisted even when accounting for
24 socioeconomic disadvantage, suggesting that structural links observed between maternal

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1 childhood maltreatment, postpartum depression, and child outcomes were not simply explained
2 by underlying levels of environmental adversity. These core results integrate findings from two
3 separate streams of literature towards a more nuanced framework of intergenerational
4 transmission, where (1) history of childhood maltreatment has been associated with increased
5 risk for postpartum depression (Alvarez-Segura et al., 2014; Choi & Sikkema, 2016), and (2)
6 postpartum depression has been associated with a range of long-term child psychological
7 outcomes (Hay et al., 2003; Murray et al., 2010). In this study, postpartum depression was
8 linked to child outcomes mainly through risk of child harm, a cascade consistent with literature
9 on maternal depression in the perinatal period (Pawlby et al., 2011; Plant et al., 2017).

10 Contrary to the sensitive window hypothesis, results indicated the mediating effect of
11 postpartum depression from maternal childhood maltreatment on child harm and subsequent
12 outcomes was carried by later maternal depression, consistent with recent literature (Agnafors
13 et al., 2012; Sanger et al., 2015). Most women with postpartum depression experienced at least
14 some depression in later years, highlighting the persistent nature of maternal depression beyond
15 the postpartum period. Some mothers who continue to be depressed may directly engage in
16 abusive behavior, perhaps due to limited emotional resources in response to child misbehavior
17 (Shay & Knutson, 2008); however, an alternative is they may be less able to actively monitor
18 child safety or effectively protect the child from a violent partner or abusive acquaintance. The
19 stronger effects of later maternal depression relative to postpartum depression might be
20 explained in various ways. First, its occurrence is more proximal to child symptoms being
21 assessed; developmental plasticity may have allowed intervening events to continue shaping
22 outcomes beyond the postpartum period (Champagne, 2010). Second, it could be that
23 cumulative exposure, as indexed by later maternal depression following postpartum depression,

1 is particularly harmful (Halligan et al., 2007; Hay et al., 2008). Third, an interaction effect may
2 exist where early exposure to postpartum depression may sensitize children with genetic
3 vulnerabilities to develop psychopathology but only when encountering later stressors such as
4 continued maternal depression (Starr et al., 2014), representing a G x E x E interaction.

5 Moreover, findings suggested the overall intergenerational transmission pathway was
6 robust to sex differences. Differential susceptibility of boys to maternal depression or history of
7 childhood maltreatment was not supported in the current data, despite prior evidence of
8 potential sex differences (Choe et al., 2013; McGinnis et al., 2015). Lack of findings could
9 reflect timing of child outcomes assessed. Another British longitudinal cohort (Quarini et al.,
10 2016) also revealed no sex differences related to maternal postpartum depression for child
11 depression outcomes at 12 years; rather, potentially latent differences emerged at 18 years, with
12 male children showing greater vulnerability following maternal postpartum depression.

13 When different maternal childhood maltreatment subtypes were considered together,
14 emotional abuse emerged above and beyond other subtypes as a significant predictor of
15 postpartum depression. While consistent with prior literature (Minnes et al., 2008), the present
16 study extended this finding by examining its implications for child outcomes. Literature
17 increasingly recognizes the uniquely harmful effects of emotional maltreatment (Hibbard et al.,
18 2012; Spinazzola et al., 2014). Given that maltreatment subtypes often co-occur rather than
19 occur in isolation, a history of emotional abuse may also indicate the early maltreating
20 environment was particularly noxious, containing a strong psychological component in addition
21 to external harm. For clinicians, inquiring about childhood emotional abuse and neglect in
22 addition to reports of past physical or sexual abuse may yield nuanced insights about mothers
23 and children at greatest risk. Studying additional dimensions of maltreatment exposure—

1 including the timing, severity, duration, and multiplicity of these experiences (Teicher &
2 Parigger, 2015)—and how they uniquely perturb neurodevelopmental systems that may affect
3 later psychopathology and/or parenting may illuminate further opportunities for prevention.

4 It must be noted that maternal childhood maltreatment continued to predict child
5 outcomes above and beyond the maternal depression pathway, suggesting only partial
6 mediation. This confirms how intergenerational transmission is a multifactorial phenomenon.
7 Other mediators of this transmission could include hostile or controlling parenting styles,
8 maternal substance abuse, or mental health disorders such as PTSD. **Broader contextual factors**
9 **such as domestic violence, household food insecurity, and neighborhood environments**
10 **previously investigated in the E-Risk cohort (Belsky et al., 2010; Jaffee et al., 2007; Jaffee et**
11 **al., 2002) may also be relevant, though our goal was to specifically ascertain timing effects of**
12 **maternal depression.** Algorithms of combined risk developed using computational learning
13 methods (Mair et al., 2000) may be a promising direction for comprehensive prevention efforts,
14 in which maternal depression **and other contextual factors** should be included as key indicators.

15 Furthermore, while not the focus of this study, it must be acknowledged that even in the
16 presence of maternal risks, there was still substantial discontinuity in the transmission of
17 maltreatment and related child outcomes. Mechanisms for resilience have been previously
18 described in the literature (Cicchetti, 2013). Of relevance to the postpartum period, resilient
19 child outcomes may be more likely when depressed mothers with maltreatment histories draw
20 upon memories from early positive interactions with caregivers as they interact with their
21 infants (Lieberman, Padrón, Van Horn, & Harris, 2005) and when they continue displaying
22 positive affect towards their infants despite some negative parenting behavior (Martinez-

1 Torteya et al., 2014). Understanding how to promote these relational capacities in the presence
2 of depressive symptoms may yield novel directions for intervening with mothers at risk.

3 **Limitations and Future Directions**

4 Several limitations of this study should be noted. First of all, study assessments did not
5 index severity of postpartum depression beyond threshold for diagnosis, which would capture a
6 range of symptomatology and potentially be more informative in predicting later outcomes
7 (Fihrer et al., 2009). Given the study's initial focus on specific maternal risk factors such as
8 depression, comprehensive psychiatric evaluation was not undertaken; thus, other mental health
9 morbidities such as PTSD or anxiety that could also influence risk transmission were not
10 measured from the outset. Maternal childhood maltreatment was also measured retrospectively
11 with the CTQ, which may be subject to underreporting (Brewin et al., 1993) though has been
12 found to correspond well to other sources of maltreatment information (Bernstein et al., 1997)
13 and should be considered a reasonable index of actual exposure. Similarly, mothers reported on
14 postpartum depressive episodes as part of a retrospective clinical interview. Though this
15 validated measure has been shown to increase accuracy of recall (Caspi et al., 1996),
16 recollection of prior depressive episodes may be biased by current mood and functioning at
17 time of assessment. Findings would benefit from replication and extension using cohort studies
18 with prospective data on maternal risk factors starting before birth. Indeed, our results converge
19 with a recent study examining antenatal and postpartum depression in the transmission of
20 negative outcomes in a prospective cohort (Plant et al., 2017).

21 Second, as the E-Risk study was conducted in the UK, results and policy implications
22 may not be generalizable to other settings. For example, maternal and child health care systems
23 in the UK are nationally managed, such that average British mothers may have greater access

1 than mothers in the US to supports and services throughout the course of their children's
2 development, potentially mitigating intergenerational risks. Additionally, the use of a twin-
3 based sample could also limit generalizability to singleton families, despite sensitivity analyses
4 using only one randomly selected twin from each pair. For instance, mothers of twins may
5 experience higher levels of depression (Thorpe et al., 1991). However, findings from this study
6 resonate with others that have observed intergenerational links between maternal childhood
7 maltreatment, maternal mental health, and child psychological outcomes in non-twin families
8 (Plant et al., 2017; Roberts et al., 2015).

9 Third, there was potential for informant bias since mothers reported on their own
10 maltreatment history and depression as well as children's outcomes, albeit at different time
11 points. We attempted to mitigate this by separately testing the model using only mother or
12 teacher reports of child symptoms. Results suggested that the proposed intergenerational
13 pathway to child externalizing symptoms was robust across informants and particularly salient
14 when teachers reported these symptoms, while the pathway to child internalizing symptoms
15 was only significant when mothers reported these symptoms. Differential pathway results may
16 reflect how externalizing symptoms are more obvious across settings, while mothers may be
17 more likely than teachers to observe subtle internalizing symptoms within home contexts.
18 Drawing on multiple informants and/or objective child assessments in future studies will clarify
19 whether current findings for child internalizing symptoms are better explained by informant
20 bias or actual contextual variation. In addition, child harm exposure over time was documented
21 carefully by trained clinicians but did rely on caregiver self-report, which may result in biased
22 estimates, though formal child registries may also underestimate actual cases compared to
23 sensitively elicited personal reports (Gilbert et al., 2009).

1 This study did not explicitly rule out genetic mediation of intergenerational
2 transmission, in which genetic factors shared between mothers and their children predispose
3 them similarly to elicit maltreatment from their environment and/or demonstrate overall
4 psychological vulnerability. For example, difficult temperament—a putatively heritable
5 characteristic—may elicit both negative parenting behavior and parental depression (Dix &
6 Yan, 2014). To examine genetic versus environmental mediation, studies could draw upon
7 adoptive mother-child cohorts (McAdams et al., 2015) or recruit egg donor in vitro fertilization
8 (IVF) samples where the mother carries the pregnancy without being genetically related to the
9 child (Thapar et al., 2009), to examine whether evidence converges across these types of
10 studies. Finally, while postpartum depression appeared to partially link maternal trauma and
11 child outcomes, it could reflect causal effects of maternal depression prior to birth (e.g., during
12 pregnancy), though we were not able to explicitly test this in our study. Future research should
13 factor in the previously observed role of antenatal depression in intergenerational transmission
14 (Plant et al., 2013; Plant et al., 2017) and test relative contributions of antenatal, postpartum,
15 and later maternal depression to refine timing of interventions, as well as potentially synergistic
16 effects of combined maternal depression and maltreatment histories.

17 **Implications**

18 For treatment and prevention, this study has highlighted the predictive utility of
19 postpartum depression for the intergenerational transmission of maltreatment and its sequelae.
20 Elevated maternal depressive symptoms at any point after birth appear to increase risk of
21 intergenerational transmission, and these symptoms may appear as early as the postpartum
22 period. Postpartum depression could serve as a useful early marker—that is, part of a clinical
23 endophenotype—for intergenerational risk, one amenable to detection and intervention. This

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1 aligns with national recommendations (Siu & the U.S. Preventive Services Task Force, 2016)
2 to screen for postpartum depression when follow-up options are available. Postpartum
3 interventions may be strategic since mothers may be open to treatment and support during this
4 time (Leis et al., 2009). For example, efforts have been made to incorporate depression
5 treatment into home visiting programs (Ammerman et al., 2013) but notably program effects
6 appear to be attenuated by maternal histories of maltreatment (Ammerman et al., 2016),
7 suggesting that depressed new mothers with trauma histories may require specialized attention
8 and integrated interventions that simultaneously address trauma and mental health. **Among**
9 **psychotherapeutic options, one example is** mindfulness-based cognitive therapy (MBCT),
10 which was developed to prevent depression relapse (Morgan, 2003) and may be especially
11 effective for individuals with childhood trauma (Williams et al., 2014). MBCT has
12 demonstrated promise for preventing perinatal relapse in women with prior depressive episodes
13 (Dimidjian et al., 2016). Mothers with trauma histories could benefit from such intervention but
14 it is unknown whether effects would extend beyond the postpartum period into later years.

15 Identifying and treating maternal depression in the earliest years may be an important
16 way to interrupt cycles of trauma and improve maternal and child outcomes: an efficient
17 strategy from a public health standpoint. **However, postpartum depression may be most**
18 **predictive of poor child outcomes within a course of maternal depression that is ongoing or**
19 **recurrent, often including depressive episodes that onset before and/or during pregnancy.**

20 Research increasingly suggests that postpartum depression is not a homogeneous condition
21 (Kettunen et al., 2014; Vliegen et al., 2014). For a subset of women—such as those with
22 maltreatment histories (Nanni et al., 2012)—postpartum depression may reflect more chronic
23 lifelong vulnerability for depression. These mothers should be clinically distinguished from

1 those experiencing a more “classic” state of postpartum depression associated with hormonal
2 shifts and acute life transition. While both groups require support and intervention, this study
3 suggests there is a two-generation impetus for targeting mothers with chronic trajectories.
4 Given the likelihood of recurrent depression, mothers with maltreatment histories should be
5 monitored for depressive symptoms beginning in pregnancy and at later points in their child’s
6 life, and treated accordingly when symptoms elevate. Ob/gyn and pediatric settings offer a
7 prime opportunity to detect ongoing maternal depression and related risk factors that could
8 affect child development (Earls, 2010).

9 **Conclusion**

10 This study found that postpartum depression, especially when followed by recurrent
11 maternal depression, plays a mediating role in the intergenerational transmission of
12 maltreatment. Mothers who have experienced childhood maltreatment are at increased risk for
13 postpartum depression and their postpartum depression may also be more persistent and
14 difficult to resolve, with downstream consequences for children’s wellbeing. Notably, this
15 study contributes to a growing body of evidence that children’s risk for exposure to
16 maltreatment and subsequent mental health problems can be predicted as early as the perinatal
17 period, offering a promising window of opportunity for prevention. Some gaps in knowledge
18 remain, including whether and how targeting early maternal depression can yield enduring
19 effects for children, or require maintenance throughout childhood. Nonetheless, interventions
20 that address both depression and trauma in the context of caregiving are critically needed for
21 mothers in the perinatal period (Ammerman et al., 2016; Muzik et al., 2015) and may assist
22 ongoing efforts to interrupt intergenerational cycles of maltreatment.

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Figure Captions

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Figure 1. Main structural model with postpartum depression as mediator. *Rectangles* represent observed variables; *ovals* represent latent variables on which younger and elder twin scores have been regressed (paths not shown to improve readability). *Solid lines* represent paths with coefficients significant at $p<.05^*$ or $p<.001^{***}$, with only significant paths shown in the model.

Figure 2. Sensitivity analyses of mother- versus teacher-only informant reports. *Rectangles* represent observed variables; *ovals* represent latent variables on which younger and elder twin scores have been regressed (paths not shown to improve readability). *Solid lines* represent significant paths. Of note, *bolded lines* represent significant paths that were different across child outcomes as measured by mother- versus teacher-only reports.

Figure 3. Adjusted structural model including later maternal depression. *Rectangles* represent observed variables; *ovals* represent latent variables on which younger and elder twin scores have been regressed (paths not shown to improve readability). *Solid lines* represent paths with coefficients significant at $p<.05^*$ or $p<.001^{***}$, with only significant paths shown in the model.

Figure 4. Examining the relative contribution of maltreatment subtypes. *Rectangles* represent observed variables; *ovals* represent latent variables on which younger and elder twin scores have been regressed (paths not shown to improve readability). *Solid lines* represent paths with coefficients significant at $p<.05^*$ or $p<.001^{***}$, with only significant paths shown in the model.