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The Long-Term Effects of Maternal Depression: Early Childhood Physical Health as a Pathway to Offspring Depression

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Abstract

Purpose—Cross sectional and retrospective studies have highlighted the long-term negative effects of maternal depression on offspring physical, social, and emotional development, but longitudinal research is needed to clarify the pathways by which maternal depression during pregnancy and early childhood affects offspring outcomes. The current study tested one developmental pathway by which maternal depression during pregnancy might negatively impact offspring mental health in young adulthood, via poor physical health in early childhood.

Methods—The sample consisted of 815 Australian youth and their mothers who were followed for 20 years. Mothers reported on their own depressive symptoms during pregnancy and offspring early childhood. Youth completed interviews about health-related stress and social functioning at age 20, and completed a questionnaire about their own depressive symptoms two to five years later.

Results—Path analysis indicated that prenatal maternal depressive symptoms predicted worse physical health during early childhood for offspring, and this effect was partially explained by ongoing maternal depression in early childhood. Offspring poor physical health during childhood predicted increased health-related stress and poor social functioning at age 20. Finally, increased health-related stress and poor social functioning predicted increased levels of depressive symptoms later in young adulthood. Maternal depression had a significant total indirect effect on youth depression via early childhood health and its psychosocial consequences.

Conclusions—Poor physical health in early childhood and its effects on young adults’ social functioning and levels of health related stress are one important pathway by which maternal depression has long-term consequences for offspring mental health.
Keywords
Depression; child and adolescent health; social adjustment; community sample

Maternal depression during and directly after pregnancy has profound effects on offspring physical health early in life, and has been linked to problems such as low birth weight, growth retardation, diarrheal episodes, and feeding problems (1, 2, 3, 4, 5, 6). Additionally, maternal depression represents a significant public health cost, with children of depressed mothers showing lower rates of receiving appropriate immunizations (7), higher rates of hospitalization (8), and higher health expenditures (9). Thus, current evidence points to a strong association between maternal depression and offspring physical health throughout infancy and childhood.

Maternal depression during pregnancy likely compromises offspring physical health through a number of different mechanisms. First, depression leads to negative health behaviors, such as substance use and poor nutrition (3), which can affect the developing fetus. Importantly, prenatal depression also predisposes mothers to higher rates of depression later in life, which could continue to impact offspring development throughout early childhood (10, 11, 12). Second, depression has been linked to dysregulation in the hypothalamic-pituitary-adrenal (HPA) axis (13) and elevated inflammation (14, 15), which could affect developing biological systems in the fetus. In support of this hypothesis, several studies have shown that mothers’ prenatal stress or depression leads to alterations in the development of the HPA axis and immune system in offspring (16, 17, 18, 19). Such physiological alterations in turn predispose offspring to health problems such as lower birthweight (18, 20) and chronic illness (18, 21).

Poor physical health during childhood in turn can have social and emotional consequences for the offspring of depressed women. A variety of early physical health problems, such as low birth weight or chronic illness, predict increased rates of psychiatric disorders during adolescence and adulthood (22, 23, 24, 25), with depression an especially common diagnosis (23, 26). Thus, maternal depression puts some children at risk for health problems during early childhood, and these difficulties with physical health might in turn lead to increased rates of depression in offspring. Yet, research on long-term mental health outcomes in offspring is typically cross-sectional, with retrospective reports of the variables of interest, rather than longitudinal designs. Furthermore, few studies have acknowledged the interplay between offspring physical health and mental health outcomes by examining childhood physical health as one potential pathway from maternal depression to offspring depression.

It is also crucial for research on the interplay between physical and mental health to examine mechanisms by which poor physical health in childhood might predict increased depression later in life. Two potential pathways linking poor childhood health and increased depression involve ongoing stress and disability related to poor health (27) and poor social functioning (28). A range of early health problems, including pre-term birth, low birth weight, and chronic illness predict difficulties such as academic underachievement, school absence, and reduced productivity at school (27, 29). This ongoing health-related stress and impairment is one pathway by which early childhood physical illness might lead to later depression. In addition, both frequent, minor illnesses and severe chronic illness have been linked to problems with social adjustment, including fewer interactions with friends and distress in social relationships (28, 30). Thus, social difficulties are another pathway by which early childhood physical health problems might lead to later depression. Nevertheless, these
specific mechanisms of the effects of poor childhood health on later mental health outcomes have not been examined in longitudinal studies.

The current project addresses these issues by examining the longitudinal relationships among maternal depression, offspring physical health, and offspring depression, and by exploring two potential mechanisms of the effects of offspring physical health on later depression: ongoing health-related stress and problems with social functioning. We hypothesize that maternal depressive symptoms during pregnancy will predict risk for increased health problems for offspring in early childhood, possibly through ongoing maternal depression in early childhood. In addition, we hypothesize that childhood health problems will predict higher youth depressive symptoms in young adulthood. Finally, we also predict that health-related stress and poor social functioning will serve as two pathways by which early childhood health affects later youth depression. We will use a large community sample to examine whether this model is true for both children with and without chronic illness. This is an important question, given the potentially different effects of frequent, minor health problems and more serious chronic illness on psychosocial outcomes.

Method

Participants

Participants were 815 youth drawn from a larger sample of 7,775 children born between 1981 and 1984 studied as a birth cohort for the Mater Misericordiae Mothers’ Hospital-University of Queensland Study of Pregnancy (MUSP; 31). At youth age 15, 815 mother-child pairs were selected for follow-up study of youth risk for depression based on mothers’ patterns of elevation of scores on the Delusions Symptoms States Inventory (DSSI; 32), a 7-item self-report measure of depression and anxiety. Families at age 15 were selected to represent a range of symptom presence, chronicity, and severity of maternal depression, and were over-selected for maternal depression relative to the general population. From the original sample, 991 families were targeted for inclusion in the follow-up, and 815 consented and were included. Children in the high-risk subsample (n = 815) were not significantly different from the original birth cohort in terms of gender, \( \chi^2(1, N = 7,775) = .53, p = .48 \); income, \( t(7147) = .81, p = .42 \); or mother’s education, \( t(7612) = 1.70, p = .09 \).

The adolescent sample at age 15 was 50.6% male and 49.4% female. The families were largely lower middle class and predominantly Caucasian (91.4% Caucasian; 3.6% Asian; 5% other or not reported). The majority of mothers worked outside the home (66%), were married to the father of the target youth (66%; 21% married to someone else; 13% no partner).

At youth age 20, 705 youth from the original 815 participants completed follow-up questionnaires and interviews. Finally, when youth were ages 22–25, participants were contacted for a final follow-up to provide biological specimens and complete additional questionnaires. Of the 705 youth who participated in the age 20 assessment, 475 (57% female) participated in the age 22–25 questionnaires. Youth who participated in the final assessment had a similar income range (M = 3.05, SD = 2.1) and ethnic distribution (\( \chi^2 = 2.62, p = .62 \)) to the original sample. In addition, youth who participated in the age 22–25 follow-up did not differ from those who did not participate in terms of prenatal maternal depression (\( t(814) = -.73, p = .46 \)), maternal depression during early childhood (\( t(814) = 1.10, p = .27 \)), early childhood physical health (\( t(506) = .45, p = .66 \)), health-related stress at age 20 (\( t(703) = -1.25, p = .21 \)), social functioning at age 20 (\( t(703) = .70, p = .49 \)), or youth depressive symptoms at age 20 (\( t(631) = 1.30, p = .19 \)). However, males had a higher dropout rate than females (\( \chi^2 = 26.34, p < .001 \).

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Procedure

Mothers completed questionnaires during pregnancy, 3–4 days after the child’s birth, 6 months after birth, and at youth ages 5, 15, and 20. Children completed interviews and questionnaires at ages 15 and 20, and questionnaires once between ages 22 and 25. Interviews and questionnaires asked about mother and child psychopathology, youth stress, youth physical health, and demographic information. Interviews were conducted by trained postgraduate students in the participants’ homes or other convenient locations. Participants all gave informed consent (assent), and the institutional review/ethics panels of the University of Queensland, Emory University, and the University of California, Los Angeles approved the research protocols.

Measures

Maternal depressive symptoms—Maternal depressive symptoms during pregnancy were measured using the depression subscale of the DSSI, a 7-item self-report measure of depression and anxiety. This scale has been validated against the clinical judgments of experienced clinicians and psychiatrists, and shows good predictive and concurrent validity (32, 33). Maternal depressive symptoms during early childhood were measured using an average of mothers’ DSSI scores at three additional time points after offspring birth: 3–4 days, 6 months, and 5 years after birth (all three time points were significantly correlated: 3–4 days with 6 months ($r = .39, p < .001$); 3–4 days with 5 years ($r = .15, p < .001$); 6 months with 5 years ($r = .13, p < .001$)). Coefficient alphas for DSSI scores at the four time points ranged from .79 to .84.

Childhood physical health—A composite of offspring physical health during infancy and early childhood was created from six different indicators of health collected from mothers 3–4 days after offspring birth, 6 months after birth, and 5 years after birth.

Postnatal health problems. Mothers indicated whether offspring had suffered from any medical problems during or immediately after birth. Youth were coded as having postnatal medical difficulties if mothers indicated that children had experienced “moderate” or “major” medical problems.

Health problems in infancy. Six months after birth, mothers reported whether offspring had experienced any of a number of difficulties with their physical health (e.g., colic, diarrhea, feeding problems, colds). Youth were coded as having physical health difficulties in infancy if they were in the highest third of the sample in terms of the number of physical difficulties endorsed by mothers at least several times per month.

Healthcare utilization in infancy. Youth were coded as high healthcare utilizers if they were in the highest third of the sample in terms of the number of times their mothers reported seeking medical attention for their children in the first 6 months after birth.

Hospitalization by age 5. At age 5, mothers reported whether offspring had experienced multiple hospitalizations before the age of 5 or not.

Physical limitations at age 5. At age 5, mothers reported on whether offspring had physical health problems that limited their daily activities or not.

Chronic illness by age 5. Finally, mothers reported whether offspring suffered from at least one chronic illness (e.g., asthma, diabetes) before the age of 5 or not.

A second composite was also created to explore whether the effects of early childhood physical health problems would persist when chronic illness was not included. This
composite was identical to the first, except that it eliminated the chronic illness by age 5 variable (range 0–5).

**Health-related stress**—Ongoing stress and disability associated with physical health at youth age 20 was measured using the UCLA Life Stress Interview (34). This semi-structured interview has been used and validated in a number of other studies using adolescent and young adult samples (35, 36). Interviewers probed individuals’ experiences with chronic stress in various domains over the past 6 months and assigned objective severity ratings for health-related stress on a 5-point scale (with 1 indicating excellent health and 5 indicating extreme physical health problems). This measure of health-related stress has been validated against other indices of poor health (35), and ratings of health-related stress showed high interrater reliability in the current study (.77).

**Social functioning**—The UCLA Life Stress Interview also probes chronic stress across three domains of social functioning with peers: romantic relationship, relationship with a best friend, and social life. The interviewer assigned an objective severity rating for the level of chronic stress in each interpersonal domain on a 5-point scale (with 1 indicating excellent social functioning and 5 indicating extremely poor social functioning). Objective severity ratings for all 3 interpersonal domains were then summed to create an overall measure of social functioning at youth age 20. The interrater reliabilities for social domains ranged from .72 to .84. Validity data for these scales have been reported in Hammen et al. (2008) (36).

**Youth depressive symptoms**—Depressive symptoms were assessed once between ages 22 and 25 using the Beck Depression Inventory (BDI; 37). The BDI is a 21-item self-report measure with well-established psychometric properties, including high internal reliability (coefficient alpha = .94 in the current sample), as well as convergent and discriminant validity (37).

**Covariates**—Youth gender was controlled for in all paths containing youth variables, given the strong association between gender and depressive symptoms (38), and the higher rate of dropout among males in the current sample. A second model was run controlling for two potential confounding variables at youth age 15. Youth depressive symptoms at age 15, assessed with the BDI, were included as a covariate, given the fact that depressive symptoms are associated with both social functioning at age 20 and youth depressive symptoms at ages 22–25 (see Table 1). Family income at age 15, measured by maternal report using a 7-point scale, was also included as a covariate, given that low family income could be related to both decreased access to healthcare and increased depressive symptoms.

**Analyses**

Path models were tested within a structural equation modeling (SEM) framework, using robust maximum likelihood procedures to estimate standard errors. All analyses were carried out in Mplus v5 (39) using full information maximum likelihood methods to accommodate missing data. The first two models examined the main hypotheses controlling for the covariates described above. In addition, a third model was run to explore whether observed effects were attributable mainly to children with chronic illness. This model was identical to the first, but did not include childhood chronic illness in the childhood physical health composite. Moreover, the model was run in the subset of children whose mothers reported no offspring chronic illness by age 5 (n = 623).
Results

Descriptive statistics and Pearson correlations among the variables are presented in Table 1. First, a model that measured the indirect effects of maternal depression during pregnancy on offspring mental health outcomes in young adulthood, via the pathway of childhood physical illness and the impact of physical illness on ongoing disability and social adjustment at age 20, was tested (Figure 1). Standardized beta values are reported.

Fit indices revealed that this model was a good fit to the data: \( \chi^2 \) (df= 8, N=815) = 23.79, \( p = .003 \); CFI = .95; RMSEA = .05 (90% CI .03, .07); SRMR = .04. Prenatal maternal depressive symptoms had a significant indirect effect on offspring depressive symptoms in young adulthood (\( \beta = .01, p < .01 \)). In addition, parameter estimates for the hypothesized direct effects in the path model were all significant, controlling for the effects of youth gender. Maternal depressive symptoms during pregnancy were directly related to increased health problems for offspring before the age of 5, but also had a significant indirect effect on offspring health problems via ongoing maternal depression during early childhood (\( \beta = .06, p < .01 \)). Offspring health problems in turn predicted greater health stress and poorer social adjustment for offspring at age 20. Greater health-related stress at age 20 also predicted concurrent difficulties with social functioning. Finally, offspring health-related stress and poor social functioning predicted higher youth depressive symptoms at ages 22–25.

Importantly, when youth depressive symptoms and family income at age 15 were controlled for in paths predicting the psychosocial consequences of childhood physical illness, the model fit and patterns of significance remained largely unchanged (\( \chi^2 \) (df= 10, N=672) = 24.15, \( p = .007 \); CFI = .96; RMSEA = .05 (90% CI .02, .07); SRMR = .03).

Next, the same model was tested only in individuals who had no reports of chronic illness by age 5, using a composite of childhood physical illness that excluded chronic illness. Fit indices revealed that this model also provided adequate fit to the data: \( \chi^2 \) (df= 8, N=623) = 20.25, \( p = .009 \); CFI = .95; RMSEA = .05 (90% CI .02, .08); SRMR = .04. In addition, individual paths in the model showed the same pattern and significance as in the model that included chronic illness, except that childhood physical health by age 5 was no longer a direct predictor of difficulties with social functioning at age 20 (\( \beta = .03, p = .45 \)). Childhood physical health did continue to predict health-related stress and disability at age 20, which in turn predicted poorer social functioning.

Discussion

The present study used a longitudinal, community-based sample to show that poor childhood physical health is one pathway by which maternal depression affects youth depression in young adulthood. Moreover, findings revealed that ongoing stress related to physical health and poor social functioning in young adulthood are two mechanisms by which childhood physical health affects later youth depression.

Findings are consistent with previous evidence indicating that maternal depression is associated with a number of offspring health problems during early childhood (1, 5). Importantly, the current study provided support for the effects of maternal depression on childhood health using a longitudinal dataset, allowing us to draw more firm directional conclusions. In addition, this relationship between maternal depression and childhood physical health was observed even in children with no history of early childhood chronic illness, indicating that maternal depression can lead to more general health problems early in life.
Results also highlight the long-term effects of poor childhood physical health on psychosocial functioning. Previous work has indicated that a range of childhood physical health problems predict increased risk for later emotional distress (22, 23), but has rarely proposed and tested potential mechanisms of these effects. Current findings highlight ongoing health stress and poor social functioning as two pathways by which poor childhood physical health affects later offspring depression. Interestingly, secondary analyses exploring the long-term effects of general physical health in children with no history of chronic illness showed largely similar results. However, for children with no chronic illness, childhood health problems did not directly compromise later social functioning, and only affected social functioning indirectly through health-related stress. Thus, even frequent struggles with minor illness during childhood can confer risk for later health-related stress and social functioning, but chronic illness might place children at particular risk for later social difficulties. It is possible that chronic illness leads to more school absences (27), thereby preventing children from forming strong social bonds with peers and creating risk for increased social isolation (28). It might also be more difficult for children with chronic illness to participate in extracurricular activities and sports teams that encourage friendships with peers.

Several limitations of the current study should be acknowledged. The physical health composite does not capture the full range of severity for certain physical health problems, given that several continuous variables were dichotomized. In addition, this composite was based largely on maternal reports of offspring health, which might be negatively biased by maternal depression (40). Future research should therefore utilize multiple informants and/or biological markers of disease to more precisely measure offspring physical health.

The current sample was also over-selected for maternal depression. Although this sample allows us to gain insight into problems that might be especially relevant to offspring of depressed mothers, results in the present sample might not be representative of a random community sample. In particular, mothers with depression likely have higher rates of physical health problems. Findings should be interpreted in light of this fact and replicated in samples that are more representative of the general population. In addition, the rate of dropout in the current study by the final assessment was relatively high, given the longitudinal design. Although the final sample did not differ from the original sample on a number of key variables, there was a higher rate of dropout among males. Although gender was controlled for in all analyses, results should be interpreted in light of this fact.

Finally, the relationship between physical health and depression among family members is doubtless bidirectional. Children’s physical health problems can also influence mothers’ emotional distress. Moreover, because offspring physical health was only measured in early childhood, the present study was unable to determine whether physical health problems show continuity over the course of development. As a result, future research should use repeated measures to examine fluctuations in physical illness and depression as they unfold across development. Such research will help to clarify the roles of early childhood illness, as well as other physical problems that emerge across adolescence (e.g., acne, obesity), in predicting later social and emotional distress.

Despite these limitations, these findings reveal an important pathway by which maternal depression affects offspring depression in young adulthood, via early childhood health. Poor offspring health is only one of many variables involved in the intergenerational transmission of depression. Nevertheless, findings have important implications for clinical interventions in children of depressed mothers. Prevention efforts with depressed mothers during pregnancy and early childhood might improve the developmental trajectories of at-risk children. Moreover, interventions involving stress management and social skills training for
children with poor physical health might have important implications for their psychosocial outcomes later in life.

Future studies in this area should explore dysregulation in biological systems (e.g., HPA axis, immune system) that might be underlying the negative effects of maternal depression on offspring physical and mental health. Future research should also test potential moderators of the effects of maternal depression on early childhood health. For example, are the effects of maternal depression on early childhood physical health buffered by the presence of a non-depressed partner? Clarifying these mediating and moderating factors might in turn lead to more targeted interventions for the prevention of the adverse effects of maternal depression on childhood physical and mental health.

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References


Implications and Contribution: This study is unique in highlighting offspring physical health and its psychosocial consequences in young adulthood as a pathway of the long-term negative effects of maternal depression on offspring mental health. Findings can help to inform interventions designed to prevent the negative consequences of maternal depression on offspring physical and mental health.
Figure 1.
Results of structural equation model testing the indirect effects of maternal depressive symptoms during pregnancy on offspring depressive symptoms in young adulthood, via offspring physical health during childhood, and offspring health-related stress and social functioning at age 20. Gender was controlled for in all paths. All paths were significant, and standardized coefficients are shown.
* $p < .05$, ** $p < .01$
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<td>1.2</td>
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<td>3. Child physical health (age 5)</td>
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<td>.21**</td>
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Note.
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* p ≤ .05,
** p ≤ .01