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# Testing Bidirectional Effects between Maternal and Child Depression During Middle Childhood

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# Testing Bidirectional Effects between Maternal and Child Depression During Middle Childhood

by

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**Abstract**

**Background:** To date, the understanding of depression within families has primarily focused on a single direction, from parents to children. Extensive research has focused on this perspective, leading to the development of various hypotheses, such as the *spillover hypothesis* and the *intergenerational transmission of depression*. These unidirectional hypotheses suggest that parent depression influences the development of child depression. More recently, a new hypothesis has emerged – *child evocative effects*. This hypothesis proposes that there are more bidirectional and dynamic interactions within the family unit, wherein children can also influence parents' depression. **Objectives**: Using a prospective pregnancy cohort, we tested both potentially co-occurring phenomena. First, we tested the potential bidirectional effects of mother and child depressive symptoms across four waves of data during the middle childhood period. Second, we tested whether child sex and family socioeconomic status moderated associations. **Methods:** This study was based on data from 1801 mothers and children from the All Our Families cohort from Calgary, Alberta. Maternal and child depression and demographic information was assessed through validated self-report measures of depressive symptoms at four timepoints (Time 1: Spring 2020, child age 9.66 years; Time 2: Spring 2021, child age 10.40 years; Time 3: Fall-Winter 2021-2022, child age 11.08 years, and Time 4: Winter 2023, child age 12.82 years). Child sex and family socioeconomic status was reported by mothers at Time 1. **Results:** Results of a random-intercept cross-lagged panel analysis revealed that child depression at Time 1 predicted higher maternal depression at Time 2 (β = .12; 95% CI .02, .22). Additionally, child depression at Time 2 predicted higher maternal depression at Time 3 (β = .17; 95% CI .07, .26). The obverse association was not supported. Child sex and family socioeconomic status did not moderate associations. **Conclusions:** Contrary to conventional theorizing, we found evidence for child-evocative effects but not maternal spillover effects of depressive symptoms. Our study sheds light on the nuances of how depression

potentially develops within families and challenges conventional theorizing of a unidirectional spillover from caregiver to child depression. It establishes a framework for future research to incorporate bidirectional and potentially transactional relationships when considering depression transmission within families. Furthermore, it emphasizes the need to incorporate the complex dynamics of family interactions into prevention and intervention efforts.

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## Introduction

Depression can be a debilitating experience for children with suboptimal outcomes on development, social functioning, and life course psychological health (Rao & Chen, 2009). As a result, concerted empirical attention has been devoted to understanding its etiology. Decades ago, researchers advanced the notion of a unidirectional "*spillover hypothesis*" – that is, that parent functioning and depression[1] "spilled over" onto children's functioning and depression (Erel & Burman, 1995; Goodman et al., 2020; Madigan et al., 2018; Nelson et al., 2009). Although there is support for the spillover hypothesis, a different hypothesis has recently emerged, known as child evocative effects. This hypothesis focuses more on the potential for children's depression to "evoke" depression in parents (Lowthian et al., 2022; Wilkinson et al., 2021). While both hypotheses are compelling, the majority of studies to date, which are largely cross-sectional, only examine the unidirectional spillover hypothesis. It is critical to advance understanding of the potential bidirectional and temporal associations between parent and child depression to appropriately pinpoint targets for prevention and intervention.

Recently, advanced methodologies have been created to adequately test the "chicken or the egg" question, i.e., what comes first, maternal or child depression? Specifically, the random-intercept cross-lagged panel model (RI-CLPM) is a state-of-the art structural equation-based statistical model that allows for enhanced understanding of simultaneous, continuous, and bidirectional longitudinal associations (Hamaker et al., 2015). RI-CLPMs can elucidate the directionality of associations between co-occurring phenomena in a manner that closely

[1] The use of the term depression in this paper refers to depressive symptoms, not to a formal diagnosis of a depressive disorder. Any references to a diagnosis of depression (e.g., Major Depressive Disorder) will be explicit.

approximates causality, by controlling for trait-like individual differences that endure over time (Hamaker et al., 2015). Since the cross-lagged parameters in RI-CLPM represent the proportion of uniquely explained variance in an outcome not shared with any other predictor in the model (Schuurman et al., 2016), the objective of my master's thesis is to test how parent and child depression is linked over the course of middle childhood, to in turn shed light on the bidirectional longitudinal associations between caregiver and child mental health.

**Child Depression: Symptoms, Prevalence, and Course**

Depression in children encompasses a broad variety of behaviours and attitudes including depressed or irritable mood, anhedonia, fluctuations in weight, sleep or psychomotor changes, concentration difficulties, feelings or thoughts of worthlessness, guilt, or suicidality (American Psychiatric Association, 2022). Child depression is thought to differ somewhat in presentation and onset from the adult criteria. Specifically, in childhood depression, irritability is included as a possible diagnostic criterion and the temporal precedence of symptoms may deviate from adults, with anhedonia developing earlier than other symptoms (Watson et al., 2020).

Depression is one of the most prevalent psychiatric disorders in childhood (Mullen, 2018). The prevalence for meeting Major Depressive Disorder (MDD) diagnostic criteria in Canadian youth is as high as 10% (Georgiades et al., 2019), and the lifetime prevalence is estimated to be 20% (Lewinsohn et al., 1994). Moreover, compelling evidence has emerged illustrating an increase in the prevalence of childhood depressive symptoms both prior to 2019 (National Healthcare Quality and Disparities Report, 2022) and during the COVID-19 pandemic (Madigan, Racine, et al., 2023), with greater increases for females and children from lower

socioeconomic families. Pandemic-related increases in depression are commensurate with evidence showing that precipitating factors for child depression have also increased, including isolation and social distancing (Brooks et al., 2020), screen time (Madigan et al., 2022), school, learning and extracurricular activity disruptions (Tsujimoto et al., 2023), loneliness (Farrell et al., 2023), and reduced physical activity (Neville et al., 2022). While the pandemic-related increase in depressive symptoms has been well documented, more effort, attention, and resources are required to advance understanding of the etiology of depression, especially for supporting those in the post-pandemic period.

The importance of childhood psychological health cannot be overstated considering that depression in childhood predicts an increased risk for a multitude of highly aversive long-term outcomes, such as personality pathology (Beauchaine et al., 2009), substance abuse (Groenman et al., 2017), suicide (Vander Stoep et al., 2011), decreased adult family income, increased economic costs (Smith & Smith, 2010), and increased demand on emergency mental health services (Madigan, Korczak, et al., 2023). Thus, identifying malleable factors relevant to the onset and course of depression in childhood allows for more targeted interventions, better treatment responsiveness, and reduced likelihood of recurrence and longer-term health difficulties. Considering the time-sensitive nature of childhood depression, the immediate individual suffering, and the short and long-term costs for the child, their family and across society, the identification of risk factors that may perpetuate, or exacerbate child mental health problems should be considered a priority.

**Maternal Depression: Symptoms, Prevalence, and Course**

Adult depression has several similarities in diagnostic criteria and symptomatology as child depression, such as anhedonia, low mood and changes in sleep or weight (American Psychiatric Association, 2022). Adult depression is highly prevalent, with an estimated 8% of adults over 20 showing clinically significant levels of depression (Brody et al., 2013). Research has consistently demonstrated greater prevalence in women than men (Brody et al., 2013), and this increased prevalence rate is maintained in motherhood (Ertel et al., 2011). Prior to the pandemic, the prevalence of depression in mothers was approximately 10% annually (Ertel et al., 2011). Throughout the COVID-19 pandemic, the prevalence of maternal depression increased. One longitudinal study showed that rates of clinically significant rates of maternal depressive symptoms increased to 35% during the pandemic, from 19% before the pandemic (Racine et al., 2021). This increase demands greater attention and exploration to better understand and thus treat this rapid deterioration in maternal mental health.

Mothers were uniquely positioned to experience distress and changes throughout the COVID-19 pandemic (Racine et al., 2022). Specifically, mothers reported experiencing increases in risk factors related to depression, such as perceived stress in response to changes in social support (MacMillan et al., 2021; Racine et al., 2022). Additionally, mothers purportedly bore the brunt of additional childcare responsibilities during the pandemic (Zoch et al., 2021), likely contributing to increased maternal stress levels (Penna et al., 2023). Furthermore, mothers are emotionally impacted by financial strain (Williams & Cheadle, 2016), which also increased during the pandemic (Gayatri & Puspitasari, 2022). These experiences, individually or collectively, can exacerbate maternal depression, which in turn can lead to less attention and focus directed at children and their developmental, mental health, and relational health needs

(Goodman, 2020).

While the increase in pandemic-related maternal depression has been well-established, the implications of the increase on child depression are not well understood. Moreover, 50% of recurrent episodes of depression occur within the first two years of the initial depressive episode, and women are 1.5-3 times more likely to experience depression (Goodman, 2007). This highlights the need for further exploration of the increase in maternal depression and its potential implication for both mothers and children.

**Maternal and Child Depression: Theoretical Models and Hypotheses**

*Bioecological Model.* This model of development posits that human development and well-being is a process involving a series of reciprocal interactions between an individual and their immediate environment (Bronfenbrenner, 1979; Bronfenbrenner & Ceci, 1994). In other words, people become more effective at navigating interactions with their immediate environment the more often the interactions occur. Specifically, the model posits that human development is the result of natural dispositions, the availability of bioecological resources one harnesses, the knowledge and skill required to adapt to their environment, and the demand characteristics of the microsystem which can enhance or discourage adaptations. Moreover, the bioecological model of development understands people as nested within different levels of the ecology. Specifically, interactions with individuals in the microsystem (e.g., a family), as well as the influences of the larger systems in which the microsystem is nested (e.g., how the community can shape family behaviors and interactions), can influence an individual's outcomes.

*Socio-ecological Model*. This model also includes a component that addresses social interactions within the levels of systems, particularly the close individuals one has direct contact with within a microsystem (e.g., a child interacting with their parents; Bronfenbrenner, 1979). These interactions can directly impact development, and these immediate or direct processes are known as proximal influences. Alternatively, larger systems or influences that impact microsystems (e.g., economic recession or global pandemic) are known as distal influences (Bronfenbrenner & Ceci, 1994). Proximal influences can enhance the development and functioning of individuals by making them more attuned to the immediate microsystem, whereas distal influences have an indirect effect on the microsystem. Not all microsystem events or interactions cause an adaptation, but the more consistent and longstanding an interaction, the more likely it is to cause an adaptation (Bronfenbrenner & Morris, 2006). Furthermore, for a proximal factor to be influential, the effect must be bidirectional. In other words, reciprocity is essential for the proximal influence to have an effect (Bronfenbrenner & Morris, 2006). For proximal processes to continually evoke adaptations, they require increasingly sophisticated, reciprocal interactions.

The bioecological and socioecological models view development as ongoing, with individuals constantly responding to proximal and distal influences. In understanding maternal and child depression, both mothers and children are within one microsystem, depression in either the mother or the child has the capacity to alter the overall microsystem. As both are continually adapting to that system, they are thus influenced by the other person. Overall, the bioecological and socioecological models require reciprocity. For one member of a microsystem to be able to influence it and other members within the system, there must be an exchange.

Taken together, these models reiterate the necessity of bidirectionality, suggesting that while maternal depression could spillover onto child depression, so too could child depression instigate maternal depression.

*Spillover Hypothesis.* In addition to the impact of maternal depression on an individual, there are also broader effects on the family environment. Parental depression can be associated with less nurturing and engaging parenting (Goodman, 2020; Kiernan & Huerta, 2008), as well as reduced ability to understand and attend to a child's emotional needs (Field, 2010). This can, in turn increase children's depression (Goodman, 2020), effectively spilling over onto children's mood and behavior. In the current study, we focus on the spillover of *maternal* depression given that it is two times more common in women compared to men (Bromet et al., 2011), and that rates of maternal depression during the COVID-19 pandemic have notably increased in Albertan mothers (Racine et al., 2022).

*Child Evocative Effects.* As noted, one limitation of the spillover hypothesis in explaining the transmission of depression from parents to children is that it fails to consider reciprocal or child evocative effects. Specifically, parents' mental health problems may be "activated" by their children's mental health issues. This hypothesis stems from theoretical work on parenting such as the Coercive Process Model (Patterson, 2004). For example, a coercive cycle may begin with a child misbehaving followed by a reprimand from the parent, which in turn results in the child's increased misbehavior to gain additional attention. In this scenario, the child evokes the behavior of the parent.

Empirical examinations of this hypothesis have shown that child aggression and disruptive behavior has been found to predict greater levels of parental negativity (Plamondon

et al., 2018) and depression (e.g., Gross et al., 2008) at later time points. While child evocative effects have been well researched in the study of aggression and conduct problems, limited research has examined how children can evoke later emotional problems and/or depressive symptoms in parents (e.g., Gross et al., 2008; Wilkinson et al., 2021). Research examining child emotional disorders suggests that parents of children with emotional disorders are more likely to have later subsequent mental health problems compared to parents of children without emotional disorders (Wilkinson et al., 2021). Theoretically, child depression may contribute to feelings of failure or inadequacy in child-rearing (Elliott & Bowen, 2018), which in turn may impact the well-being of parents.

**Bidirectional evidence of Parent-Child Mental Health**

Evidence on the potential for bidirectional associations of parent to child emotional difficulties has only recently emerged. For example, one study with a sample of 1,911 mother-child dyads following children at ages 0, 3, 4, 7, 11, 14, and 17 from the United Kingdom, found evidence of bidirectional associations between mother's mental health problems and parent-reported child emotional problems across time using a RI-CLPM analysis (Lowthian et al., 2023). The cross-lag paths suggested that maternal mental health problems at child age 11 were positively associated with child emotional problems when children were age 14, a finding consistent with the spillover hypothesis. However, child emotional problems at age 7 were also positively associated with maternal mental health problems at age 11, which is consistent with child evocative effects. The authors also found evidence for moderated effects based on socioeconomic status, with higher socioeconomic status protecting against both maternal and child emotional problems.

An additional study supplied further evidence of bidirectional associations between parental psychological distress and child internalizing symptoms, which includes depression. In a sample of 10,746 parent-child dyads from the United Kingdom, parents reported on themselves and their child at the ages of 3, 5, 7, 11, 14 and 17. The direction of associations between maternal psychological distress and children's internalizing problems was assessed (Speyer et al., 2022). The results illustrate bidirectional associations between parents and children, including different associations based on sex. Specifically, the data showed mother to child effects, supporting the spillover hypothesis, whereby maternal psychological distress consistently predicted later internalizing problems for both boys and girls. Furthermore, the child to mother effects were also revealed, wherein boys' internalizing problems were associated with increased maternal psychological distress at 11 and 14 years of age. While these findings of bidirectional evidence were not consistent throughout the age range of the children, it does provide evidence for their existence and may signal that the sex of the child and some periods of child development may pose disproportionate risk for parents.

**Limitations of Previous Research**

Initial testing of bidirectional effects of maternal and child mental health has revealed both spillover and child evocative effects; however, for several reasons, re-examination is required. First, the findings require replication and extension during a time of heightened stress for many families, such as a global pandemic, when symptoms of both maternal and child depression increased (Madigan et al., 2023; Racine et al., 2021). Second, existing research has only included measures of parent-reported child emotional problems. Single informant designs can increase the risk of reporter bias. Moreover, parent reported child problems may be less

accurate depictions of a child's symptoms of mental health and true internal state, especially for internalizing problems like depression (De Los Reyes et al., 2015). Third, in previous research, waves of data collection intervals were 3 to 4 years, which could lead to potential oversight of crucial nuances pertaining to the strength or pattern of bidirectional effects during rapid periods of child development and mental health (Lowthian et al. 2023).

**Methods for Examining Bidirectional Effects**

There are two primary methods that have been established for detecting the bidirectional influence of two variables on one another across time. First, the Cross-lagged Panel Model (CLPM) where "cross" refers to the bidirectional estimate from one variable to the other (Duncan, 1969), and lagged refers to the different timepoints that these estimates are being made across. CLPM control for correlations within timepoints, as well as construct stability, to enable the crossed estimates to predict a true effect rather than within-construct variance. While considered to be a novel and noteworthy methodological advance, the CLPM has been critiqued for not adequately accounting for some of the more stable or trait-like associations in both the predictor and outcome variables. Specifically, since no trait-like or individual differences can be considered, the model may incorrectly assume causal relationships (Hamaker et al., 2015).

To address this limitation, the RI-CLPM was developed to account for the stable, trait-like associations that exist between datapoints as a means of improving accuracy of cross-lagged predictions (e.g., association between parent and child depression across time). Thus, RI-CLPM consider both between and within individual effects. Specifically, random intercept estimates are calculated for all constructs being assessed and estimates are thought to

represent the stable between person components. Paths are estimated between timepoints across and within constructs. For within construct paths (e.g., child depression Time 1, child depression Time 2), the coefficient represents the within-person effect, whereas the cross-lagged paths (e.g., child depression Time 1, maternal depression Time 2) represent the between-construct effects. The RI-CLPM's use of random intercept (i.e., constraining all the loadings on a single factor to 1) is thought to improve the prediction of within-construct estimation by partitioning out the between person variance. Considering the relatively recent development of this statistical method, as well as the requirement for high quality, longitudinal data from maternal-child dyads, research using this analytical tool to approximate causality and determine directionality in mother child depression is extremely limited.

**The Current Study**

Given the individual, familial, and societal toll of depression, it is essential to advance understanding of its development and impact on families and family dynamics. The COVID-19 pandemic has highlighted increased severity of depression in both mothers (Racine et al., 2022) and children (Madigan, Racine, et al., 2023), which may impact transmission processes. To advance understanding of the directionality and temporality of maternal and child depression during a period of heightened stress for most families, the first aim of this study is to assess whether symptoms of maternal depression impact childhood depression and/or vice versa across four time points during middle childhood (child ages 9, 10, 11 and 12). This will be tested with advanced methods for examining bidirectional effects, namely RI-CLPM. Based on existing research, I hypothesized that bidirectional effects would be observed.

The second objective of this study was to examine whether child sex and family

socioeconomic status moderate maternal-child depression associations within the RI-CLPM. Several pandemic-related studies have found evidence that mothers and girls experienced greater increases in depression during the pandemic (Madigan, Racine, et al., 2023; Racine et al., 2022) and larger increases in depression have been observed during the pandemic in children with higher socioeconomic status (Madigan, Racine, et al., 2023). Thus, my exploratory hypotheses are that bidirectional effects may be strongest between mothers and girls, and for children with higher socioeconomic status. Identifying the transmission of depression in the parent-child relationship will help identify individuals at risk, better inform family dynamics, and identify targets for prevention and interventions that can help to mitigate risk of depression onset and exacerbation.

## Method

### Participants

This sample is drawn from All Our Families (AOF), a pregnancy cohort from Calgary, Alberta. Recruitment took place in primary health care offices and public health laboratory services and inclusion criteria required pregnant mothers to be over 18, fluent in English, have a gestational age greater than 24 weeks, and to be receiving community prenatal care. Additional details regarding study recruitment have been published elsewhere (McDonald et al., 2013). Consenting mothers (N = 3387) received comprehensive questionnaires at recruitment, and approximately 2400 families have been followed consistently over time at 4 months postpartum and when their children were 1, 2, 3, 5, and 8 years old. Furthermore, consenting mothers and assenting children completed separate questionnaires three times throughout the COVID-19 pandemic, first between May and July of 2020 (Time 1; T1), second between March

and April of 2021 (Time 2; T2), and third between November 2021 and January 2022 (Time 3; T3). Data collection for mothers and children also occurred once following the end of the pandemic, between January and March 2023 (Time 4; T4). Data from the 3 COVID-19 waves and the post COVID-19 wave of data, which include both maternal and child reports of depression, were used in the current study. Participant compensation consisted of a gift card for a nominal amount at each timepoint. All procedures were approved by the University of Calgary Research Ethics Board.

This study was informed by data from 1801 dyads that provided usable data for at least one data wave. Mothers were an average of 41.56 years old (*SD* = 4.35) at T1. Most mothers in this study had graduated post-secondary education (65.7%), had an average household income greater than $125,000, were married or in a common-law relationship (95.92%) and were White/Caucasian (76.6%). Children were an average of 9.66 years old (*SD* = 0.81) at T1 and were mostly male (52.2%). Dyads that did not participate or provide complete data by the end of the pandemic tended to have older mothers $t(925) = 2.51$, $p = .001$, and older aged children $t(773) = 9.59$, $p = .001$. Additionally, responders and non-responders did not differ on pre-pandemic depression $t(932) = 1.53$, $p = .127$; however, at the beginning of AOF data collection ($N = 3387$), non-responders had a significantly lower average income range than continued responders, $\chi^2(10)= 56.27$; $p < .001$, as well as less education, $\chi^2(5)= 46.36$; $p < .001$, and were less likely to be married, $\chi^2(1)= 13.27$; $p < .001$. All sample demographic information for study completers can be found in Table 1.

**Measures**

***Child Depression***

Youth self-reported on symptoms of depression using the Behaviour Assessment System for Children (BASC-3; Reynolds & Kamphaus, 2015). Summed scores are converted to standardized T-scores which range from 0-100, with scores exceeding 70 indicating clinically significant impairment. BASC-3 T-scores are based on comparisons to samples of same age peers. The BASC-3 has demonstrated excellent validity and reliability in detecting mental health difficulties in children (Reynolds & Kamphaus, 2015). Child depression was measured using the BASC-3 Depression Subscale which demonstrated good internal consistency in this sample across timepoints ($\alpha$ = .83 - $\alpha$ = .88).

***Maternal Depression***

Maternal depression was determined by mothers self-reporting using the *Center for Epidemiologic Studies Depression Scale-10* (Andresen et al., 1994). The CESD-10 measures the presence and severity of depressive symptoms experienced, and responses are summed with higher scores indicating greater severity of depressive symptoms. The CESD-10 has a recommended cutoff of 10 to indicate clinically significant impairment (Andresen et al., 1994). The CESD-10 demonstrated good internal consistency and validity in adults (Irwin et al., 1999), and demonstrated good internal consistency in this sample across all timepoints ($\alpha$ = .85 - $\alpha$ = .87).

***Moderators***

Two moderators were assessed as past research has provided evidence for their influence on maternal and child depression throughout the COVID-19 pandemic, allowing for the improved identification of those at disproportionate risk. First, we assessed whether child sex (maternal report; male vs. female) moderates the associations between maternal and child

depression. Additionally, we assessed whether family reported total household income at the first data wave of the pandemic (measured in $10,000 increments from 0 to $100,000, between $100,000 to $124,999, $125,000 to 149,999, or $150,000 or more) moderates the maternal-child depression association. For this study, family annual income was used as a proxy for socioeconomic status, as socioeconomic status was found to be relevant to our variables of interest in past research (e.g., Lowthian et al., 2023). For the moderation analyses, we created a dichotomized variable, placing families into either high (> $80,000) or low (< $80,000) income categories before testing whether family socioeconomic status moderated the association between maternal-child depression. This cutoff was chosen due to its proximity to the Canadian median income and as it maximizes our sample size in the low-income group (*N* = 193) compared to the high group (*N* = 1083).

**Statistical Analysis**

All statistical analyses were conducted in R 4.3.0 (R Core Team, 2023) and MPlus Version 8.1 (Muthen & Muthen, 2017) to test the proposed research questions. We calculated Cronbach's alpha to assess the internal consistency of the included measures and mean scores of all dependent and independent variables across the four timepoints. Data were initially screened for assumptions, including normal distribution, outliers, impossible values, and for homogeneity of variance. Correlations were calculated for all variables of interest and the results can be observed in Table 2.

A RI-CLPM was used to assess the relationship between mother and child depression. As RI-CLPM controls for within-person and across-time effects, results are expected to approximate the causal effects of parent-child depression across timepoints. Model fit was

determined based on the criteria outlined by Hu and Bentler (1999). Hu and Bentler discuss a cutoff value for the Standardized Root Mean Residual (SRMR) of .08, Root Mean Square Error of Approximation (RMSEA) of .06, a Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) of >.95.

To evaluate whether child sex and family socioeconomic status moderated the associations between maternal-child depression, a multiple group model comparison was conducted. In testing whether the moderators impacted the strength of associations, we followed the protocol outlined by Mulder & Hamaker (2021). For this analysis, categorical variables (e.g., child sex) function as a grouping variable that allows for the means, lagged regression coefficients, variances and covariances to vary between groups. Differences between groups in the lagged coefficients represent moderation effects, and these effects can be tested by comparing the chi squared difference of the multi-group RI-CLPM. Specifically, in the null condition, the lagged coefficients are constrained (e.g., both groups are considered equal) and then compared to the alternative condition, where the lagged coefficients are not constrained (e.g., groups are free to vary), to determine whether the two models significantly differ.

To test missing data, Little's MCAR test was conducted and was non-significant, suggesting that missing data was at random $\chi^2$ (784) = 794.18, $p$ =.392. To account for missing data, models were run with full information maximum likelihood estimation, consistent with other longitudinal cohort studies using RI-CLPM (McArthur et al., 2021; Neville et al., 2021). Standardized coefficients were computed for each of the within-variable, between variable, and the cross-lagged effects to better allow for interpretation. For these standardized coefficients,

the magnitude of effects was interpreted as slight (≤.09), small (≥.10 to ≤.29), moderate (≥.30 to ≤.49), or large (≥.50; Cohen, 1992).

## Results

### Descriptive Statistics and Correlations

As illustrated in Table 2, significant positive associations were found between maternal and child depression at all timepoints. Family socioeconomic status was significantly correlated with maternal depression at all timepoints. Child sex was only significantly correlated with child depression at the final timepoint and was not significantly associated with maternal depression at any timepoint.

### Longitudinal Bidirectional Associations Between Maternal and Child Depression

In assessing the standard RI-CLPM (See Figure 1), the model was first tested to determine model fit to the observed data. In comparing the proposed model to the fit indices, the model demonstrated a good fit to the data ($\chi^2$= 23.70; *p* = .005; RMSEA = 0.03; SRMR = .019; CFI = .996; TLI = .986.

Statistically significant autocorrelations were observed for the first three timepoints of maternal depression; however, the final timepoint failed to reach statistical significance. This indicates substantial within-construct stability in maternal depression over most time points. Autocorrelations for child depression at all timepoints were significantly and positively associated with one another. That is, on average, children's depression symptoms were stable across adjacent time points. In addition, the stable association between the maternal and child random intercepts showed a significant and positive autocorrelation (β= .27 [95% CI .17 to

.27]), suggesting that maternal and child depression demonstrated temporally stable or trait like associations across time.

In assessing the coefficients between mother and child depression at each time point, only the third timepoint demonstrated a significant, small effect (β= .14 [95% CI .05 to .23]). Alternatively, the first, second, and fourth timepoints did not meet the threshold for significance (See Figure 1 and Table 3), which suggests some variability in the magnitude and significance of the association between maternal and child depression over time. Specifically, this suggests that at the third timepoint, maternal and child depression covaried in a stable, trait-like manner, and that greater levels in one variable were associated with greater levels in the other variable. Alternatively, the lack of association at other timepoints may be due to several factors, such as differences in reactions to the COVID-19 pandemic and associated stressors, or disparate developmental trends.

Regarding the cross-lags within the RI-CLPM, child depression at Time 1 predicted elevations in maternal depression at Time 2, demonstrating a small but significant effect (β= .12 [95% CI .02 to .22]). Additionally, child depression at Time 2 predicted elevations in maternal depression at Time 3, again demonstrating a small but significant effect (β= .17 [95% CI .07 to .26]). Altogether, this suggests that, after accounting for consistent within-construct stability across maternal and child depression, child depression predicted elevated levels of later maternal depression during the COVID-19 pandemic; however, following the end of pandemic-related restrictions (T4), this observed effect failed to reach significance (See Table 3 and Figure 1). Moreover, we failed to observe any significant obverse cross-lagged effects that would support maternal depression spilling over to child depression at any timepoints (See Table 3

and Figure 1 for all values). Finally, we compared cross-lagged effects with their obverse effect (e.g., the cross lagged effect of Child T1 Depression onto Maternal T2 Depression against the cross lagged effect of Maternal T1 Depression onto Child T2 Depression). The magnitude of the cross lagged effects between T1 and T2 did not significantly differ from one another, $t(1766) = -1.68$, $p = .093$, nor did T2 and T3 , $t(1766) = -1.43$, $p = .153$, or T3 and T4, $t(1766) = -0.23$, $p = .818$.

**Moderation Analyses**

The comparison between the constrained model ($\chi^2(30) = 41.95$) and the unconstrained model ($\chi^2(18) = 31.12$) testing if child sex was a moderator resulted in a non-significant difference, $\Delta\chi^2(12) = 10.83$, $p = .543$, suggesting no significant difference in the lagged associations based on child sex. Moreover, we assessed whether the RI-CLPM was moderated by family socioeconomic status using the same method. The constrained model ($\chi^2(30) = 50.02$) and the unconstrained model ($\chi^2(18) = 38.90$) resulted in a non-significant difference, $\Delta\chi^2(12) = 11.12$, $p = .519$, illustrating that we observed no significant difference in the lagged associations based on family socioeconomic status. This suggests that neither child sex nor family socioeconomic status significantly changed the estimates of bidirectional effects of maternal and child depression.

## Discussion

Most research to date has focused on unidirectional associations linking parent to child depression, with little consideration of the potential bidirectional nature of this association. The current study used a RI-CLPM to estimate the directionality and temporality between maternal and middle childhood depression across four waves of data during the pandemic, a period

marked by heightened family stress. Contrary to conventional theorizing, our analyses revealed evidence for child-evocative effects only. Specifically, child depression early in the pandemic (Time 1 and 2), predicted heightened maternal depression later in the pandemic (Time 2 and 3), respectively. The obverse, maternal depression to later child depression, was not observed. Thus, our findings provide evidence for child evocative effects, but not the spillover hypothesis, suggesting that elevations in child depression alter the family microsystem, likely activating or exacerbating maternal depression.

The observed unidirectional cross-lagged results partially replicate recent research showing child evocative effects (Lowthian et al., 2023; Speyer et al., 2022); however, these studies also demonstrated parent to child spillover effects within their RI-CLPM models (Lowthian et al., 2023; Speyer et al., 2022), which were not supported in our analyses. The disparity between our findings and the bidirectional effects observed in previous research warrants further exploration. One plausible explanation is that our data are based on multi-informant reporting, while the previous research has relied on a single informant design (maternal report only). Single informant reports carry inherent validity risks, such as the potential for overreporting of child depression symptoms by mothers who are themselves struggling with depression (Najman et al., 2001). This overreporting could increase with time, given the stable and reoccurring nature of depression. Moreover, depression can contribute to negative interpretations and cognitive biases (e.g., Nieto et al., 2020). For these reasons, our use of child-reported depression symptoms may be a more accurate reflection of the child's internal experiences (De Los Reyes et al., 2015), particularly if the parent is experiencing

depression and depression-related cognitive difficulties (e.g., negative cognitive biases, cognitive distortions).

While there are methodological explanations for our evidence of child evocative effects, several possible pandemic-related co-occurring factors could also explain the timing and nature of the evocative associations. First, child screen time use increased rapidly at the outset of the pandemic (Madigan et al., 2022), and this increase was sustained over time in the All Our Families cohort (Plamondon et al., 2023). Screen time in middle childhood has been linked to children's internalizing problems (Eirich et al., 2022). Thus, increases in screen time might explain the initial rise in child depression. Second, due to pandemic-restrictions, such as stay or work from home orders, the frequency of maternal-child interactions, and family interactions more broadly, increased. This increased exposure may have provided an opportunity for mothers to both recognize and potentially be impacted by their child's mood and behaviors. Ultimately, with the abrupt change in child depression, and the changes in relational dynamics within the microsystem between mothers and children, the notable increase in child depressive symptoms could have taken a disproportionate toll on mothers' mental health and contributed to their own depression.

Ultimately, our findings provide evidence that child depression contributes to elevations in mothers' depression and serves as a starting point for future research to explore these effects. Moreover, future studies should explore the mechanisms that could explain how depression is transmitted or influences parents and children within families. While our research questions primarily focused on a unique period of family life, our results may be generalizable outside of this specific global event. For example, historical research during times of natural

disasters were informative for policy and practice developments during the COVID-19 pandemic (Sakurai & Chughtai, 2020). Nonetheless, the results of this study require replication in non-pandemic periods.

While evidence emerged for child evocative effects, we did not find evidence for the spillover hypothesis. There are several possible explanations for this finding that are worth considering. First, during the pandemic and post-pandemic period, children in our study were emerging into adolescence, a period marked by increases in emotionally intimate, trusting, and supportive peer relations (Berndt, 2004). As emerging adolescents spend more time with their peers, both in-person and through social media and gaming platforms, their social networks and the quality of these relationships are developing, possibly functioning as a support network should they need it. The development of larger, more emotionally connected relationships for children could possibly mitigate any spillover effects. Moreover, positive childhood friendships may help to protect against the spillover of maternal depression to children, considering that friendships during this period can predict long-term healthy psychological functioning (e.g., van Harmelen et al., 2017). Second, differences in the trajectories of maternal and child depression might also explain our findings. According to the bioecological model of development, changes within a microsystem might not immediately manifest as psychopathology. Instead, they can lead to short-term adaptations that become detrimental over the long-term. These findings support calls for additional long-term research into the effects of the pandemic on children (Wade, Prime & Browne, 2020; 2023), as pandemic influences may impact mental health for years to come.

Additionally, we tested whether the magnitude of the cross-lagged effects differed for mother to child and child to mother depression. Consistent with past research testing differences in cross-lagged effects (Hassan et al., 2024), we also found no significant differences. However, this finding does not substantively change our interpretation of the significant cross-lagged effects observed from child to parent depression. First, from a conceptual standpoint, the relationship between parent to child depression likely stems from both shared environmental factors, as well as genetics (e.g., Goodman et al., 2020). When accounting for this shared heritability, only child depression was prospectively associated with deviations in mothers later depression scores. Second, from a statistical standpoint, the cross-lagged effects for child to mother depression did not include zero, providing good evidence of a small effect of child depression on later maternal depression. In contrast, the mother to child estimates did include zero and failed to reach statistical significance, revealing poor evidence that deviations in maternal depression predicted heightened child depression at later time points.

In addition to testing bidirectional associations, we also tested whether child sex or family socioeconomic status moderated associations between maternal and child depression. Model fit indices revealed that neither of these variables were significant moderators, suggesting that child sex or family socioeconomic status did not necessarily protect against or predicted disproportionate risk to the within-construct, between-constructs, or cross-lagged effects in our model. Regarding child sex, one explanation for the lack of significance could be the age of children during our study. Specifically, children were on average 9 years old at the start of the study which is earlier than the average onset of puberty in Canada (Al-Sahab et al.,

2010). As such, it may be that the well-established differences in depressive symptoms between boys and girls that emerge in the adolescence years (e.g., Hankin et al., 1998), were not observed in this study. In support of this explanation, our correlation analyses suggest that child sex was only significantly associated with child depression at our final timepoint, when children were on average 12 years old. Moreover, it is important to note that we examined child sex, not gender as a moderator. Child gender should be explored as a moderator in future research.

With regards to family socioeconomic status, while unexpected, some evidence has emerged that the COVID-19 pandemic may have elicited an atypical response, with children from higher socioeconomic status families reporting greater difficulties during the pandemic than their lower socioeconomic status peers (e.g., Madigan et al., 2023). In addition, our sample is comprised of predominantly higher socioeconomic families, with most parents having graduated post-secondary educational institutions and family annual incomes exceeding $125,000. Taken together, it is possible that we may have been insufficiently powered to detect differences between socioeconomic levels.

Within-construct trends of maternal depression showed consistent, stable within-construct associations early to late-pandemic; however, maternal depression at the third timepoint (January 2022) failed to predict maternal depression at the final timepoint (January 2023). The lack of stability in the association between the third and the final timepoint may represent a “return to normal” for mothers. For example, all remaining public health restrictions were terminated in June 2022, and following that, families in Alberta had wide availability to COVID-19 vaccinations and largely returned to consistent routines, such as

regular school attendance for children, extracurricular activities, and social engagements. Accordingly, mothers reported levels of depression at the post-pandemic timepoint in our sample appear fairly consistent with past studies including in community samples caregiving research (e.g., González et al., 2017). The early pandemic was rife with uncertainty and a loss of control which has been linked with elevated depression (Andrews et al., 2023; Eren et al., 2023); however, following restrictions lifting, vaccines becoming available, and life "returning to normal," pandemic related concerns that contributed to the stability of maternal depression lapsed, explaining the reduction in overall depression.

In contrast, child depression was stable both within and post-pandemic. Specifically, child depression demonstrated strong within-construct stability, with large positive effect sizes. This suggests that child depression increased throughout the pandemic and that this elevation was sustained into the post-pandemic period. This finding is consistent with longitudinal (Park et al., 2024), meta-analytic evidence (Madigan et al., 2023; Wang et al., 2022), and scoping review findings (Wolf & Schmitz, 2024) suggesting pandemic increases in child depression, over and above expected typical increases in depression (McLaughlin & King, 2015). There are numerous proximal factors relevant to pandemic-related increases in child depression, including increased child loneliness (Thakur et al., 2022), elevations in screen time exposure (Madigan et al., 2022; Plamondon et al., 2023), reductions in physical activity (Neville et al., 2022), and elevations in parental mental health problems or stress (Racine et al., 2021). Thus, while post-pandemic life was intended to "returned to normal," the effects of the pandemic on youth mental health compared to maternal mental health, may have been more cumulative and lasting in nature (Wade et al., 2020).

## Clinical Implications

Our findings that child depression is a highly stable construct draws attention to the need for allocation of resources and mental health supports. Depression in childhood predicts several pernicious outcomes including decreased academic performance, relational functioning, employment opportunities, physical health, and elevated risk for later problems with psychopathology in adulthood (Thapar et al., 2022). Thus, there is a need for interventions that mitigate the effects of the pandemic on youth before mental and physical health problems take root. Additionally, our evidence of child evocative effects provides new understanding to family and dyadic interventions. Specifically, our findings suggest that treatments conceptualize psychopathology and therapeutic interventions in a more relational lens, with the mental health of those within the family as possible protective or risk factors. Finally, our results suggest that interventions targeted at treating families and improving their relationships have the potential to improve the shared microsystem, relations between individuals in the microsystem, and ultimately improve the mental health of all members of a microsystem (e.g., parents and children). Moreover, interventions targeting family relations may be particularly relevant to implement broadly during times of crisis, particularly if there are restrictions that increase the amount of time and interactions that family members have. For example, the Connect Attachment Program (Moretti, Pasalich & O'Donnell, 2018) is a manualized form of therapy that helps to improve sensitive parenting skills and attachment security in adolescents (Bao & Moretti, 2023), and has been shown to improve both parent (Moretti & Obsuth, 2009) and child mental health (Moretti et al., 2015), as well as the overall relationship quality (Kristen et al., 2023; Moretti et al., 2012).

## Limitations

Several study limitations should be noted. First, families that participated in the COVID-19 period data waves had a higher average socioeconomic status, younger moms and children, and were less likely to be married than those in earlier data waves. This may limit the generalizability of our sample to lower socioeconomic families. Second, our measures of depression were questionnaire-based, and our study design would be improved with the use of clinical diagnostic measures in future research. Third, our study does not test biological factors (e.g., genetics) which are highly relevant to the development and maintenance of depression across generations (Goodman, 2020), and could help explain some of our observed effects. Fourth, given the lack of evidence for the spillover hypothesis, incorporating different methods and timeframes (e.g., pulse models, looking at longer term predictions of depression) might reveal a different pattern of associations and add advanced understanding of these effects. Finally, our study only assessed maternal depression and our results should be replicated with paternal mental health variables.

## Conclusion

Overall, the current study provides high-quality evidence of the directional nature between maternal-child depression, which is opposite to conventional theorizing. Specifically, we found that child depression predicted later heightened maternal depression, but not vice versa. To our knowledge, this is the only multi-informant RI-CLPM on maternal and child depression. Moreover, our results provide insights into pandemic related influences that persist into the post-pandemic period. Specifically, that depression in one family member (i.e., children) exacerbated the depression of other family members (i.e., mothers). Additionally, this

study highlights how maternal and child depression were stable across the pandemic period; however, this stability persisted for children into the post-pandemic period whereas a "rebound" was observed for mothers during the post-pandemic period. This continuation of child distress is over and above that expected with typical development (Park et al., 2024), suggesting that pandemic effects on children may continue to have reverberations if resources and interventions are not made available to them, and their families.

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***Table 1: Demographic and Mental Health Characteristics***

| Variable | Statistic |
|---|---|
| Maternal age at T1, *M* (*SD*) | 41.56 (4.35) |
| Child age at T1, *M* (SD) | 9.66 (0.81) |
| Boys | 9.66 (0.85) |
| Girls | 9.64 (0.78) |
| Child Sex, *n* (%) | |
| Male | 537 (52.2) |
| Female | 491 (47.8) |
| Maternal Education, *n* (%) | |
| Some Elementary of High School | 11 (1.3%) |
| Graduated High School | 36 (4.3%) |
| Some college/university or trade courses | 109 (12.9) |
| Graduated college, trades or university | 555 (65.7) |
| Some graduate school | 20 (2.4) |
| Completed graduate school | 112 (13.3) |
| Ethnicity, *n* (%) | |
| Black/African American | 18 (1.4%) |
| East Asian | 71 (5.5%) |
| Indigenous | 18 (1.4%) |
| Latin American | 19 (1.5%) |
| Middle Eastern | 21 (1.6%) |
| South Asian | 42 (3.3%) |
| Southeast Asian | 30 (2.3%) |
| White/Caucasian | 984 (76.6%) |
| Mixed/Other | 81 (6.3%) |
| Family Annual Income pre-pandemic *n* (%) | |
| $175,000 or more | 411 (32.2%) |

| | |
|---|---|
| $125,000 - $174,999 | 292 (22.9%) |
| $100,000 - $124,999 | 251 (19.7%) |
| $80,000 - $99,999 | 129 (10.13%) |
| $60,000 - $79,999 | 87 (6.8%) |
| $40,000 - $59,999 | 56 (4.4%) |
| $30,000 - $39,999 | 25 (2.0%) |
| Less than $29,999 | 25 (2.0%) |
| Maternal Mental Health Characteristics *M* (*SD*) | |
| T1 CES-D | 8.06 (5.75) |
| % with clinically significant depression | 33.57% |
| T2 CES-D | 7.77 (5.74) |
| % with clinically significant depression | 32.09% |
| T3 CES-D | 7.94 (5.67) |
| % with clinically significant depression | 33.33% |
| T4 CES-D | 7.45 (5.15) |
| % with clinically significant depression | 29.92% |
| Child Mental Health Characteristics *M* (SD) | |
| T1 Depression | 49.59 (9.63) |
| Boys | 50.23 (10.65) |
| Girls | 49.14 (9.18) |
| % with clinically significant depression | 5.20% |
| T2 Depression | 49.66 (11.08) |
| Boys | 49.05 (10.85) |
| Girls | 50.32 (11.30) |
| % with clinically significant depression | 7.17% |
| T3 Depression | 51.54 (13.18) |
| Boys | 50.71 (12.70) |
| Girls | 52.56 (13.63) |

| | |
|---|---|
| % with clinically significant depression | 9.88% |
| T4 Depression | 54.36 (15.35) |
| Boys | 51.17 (13.35) |
| Girls | 57.26 (16.78) |
| % with clinically significant depression | 14.77% |

*Note CES-D, Center for Epidemiological Studies Depression Scale and the scores exceeding 10 met the threshold for clinical significance, BASC, Behaviour Assessment System for Children measured Child Depression. Time 1 Spring 2020, child aged 9.66; Time 2 Spring 2021, child aged 10.40; Time 3 Fall-Winter 2021-2022, child aged 11.08, and Time 4 Winter 2023, child aged 12.82. Child scores meeting the threshold for clinical significance exceeded a T-score of 70 on the BASC-3 Depression Subscale.*

***Table 2. Bivariate Correlations of Maternal – Child Depression, Child Sex and Family Socioeconomic Status***

| | T1 CES-D | T2 CES-D | T3 CES-D | T4 CES-D | T1 BASC | T2 BASC | T3 BASC | T4 BASC | Child Sex | Family SES |
|---|---|---|---|---|---|---|---|---|---|---|
| T1 CES-D | 1 | | | | | | | | | |
| T2 CES-D | .68*** | 1 | | | | | | | | |
| T3 CES-D | .60*** | .65*** | 1 | | | | | | | |
| T4 CES-D | .55*** | .61*** | .64*** | 1 | | | | | | |
| T1 BASC | .13*** | .17*** | .13*** | .13*** | 1 | | | | | |
| T2 BASC | .12*** | .19*** | .23*** | .16*** | .57*** | 1 | | | | |
| T3 BASC | .18*** | .19*** | .22*** | .18*** | .42*** | .61*** | 1 | | | |
| T4 BASC | .11*** | .14*** | .20*** | .13*** | .31*** | .44*** | .60*** | 1 | | |
| Child Sex | .04 | .01 | -.03 | -.02 | .07 | -.04 | -.03 | -.18*** | 1 | |
| Family SES | -.09** | -.10** | -.09** | -.07* | -.04 | -.04 | -.06 | -.03 | -.01 | 1 |
| *M* | 8.06 | 7.77 | 7.94 | 7.45 | 49.59 | 49.66 | 51.54 | 54.36 | | |
| *SD* | 5.75 | 5.74 | 5.67 | 5.15 | 9.63 | 11.08 | 13.18 | 15.35 | | |

*Note CES-D, Center for Epidemiological Studies Depression Scale, BASC, Behaviour Assessment System for Children. SES, Socioeconomic status*

*$p < .05$, **$p < .01$, ***$p < .001$

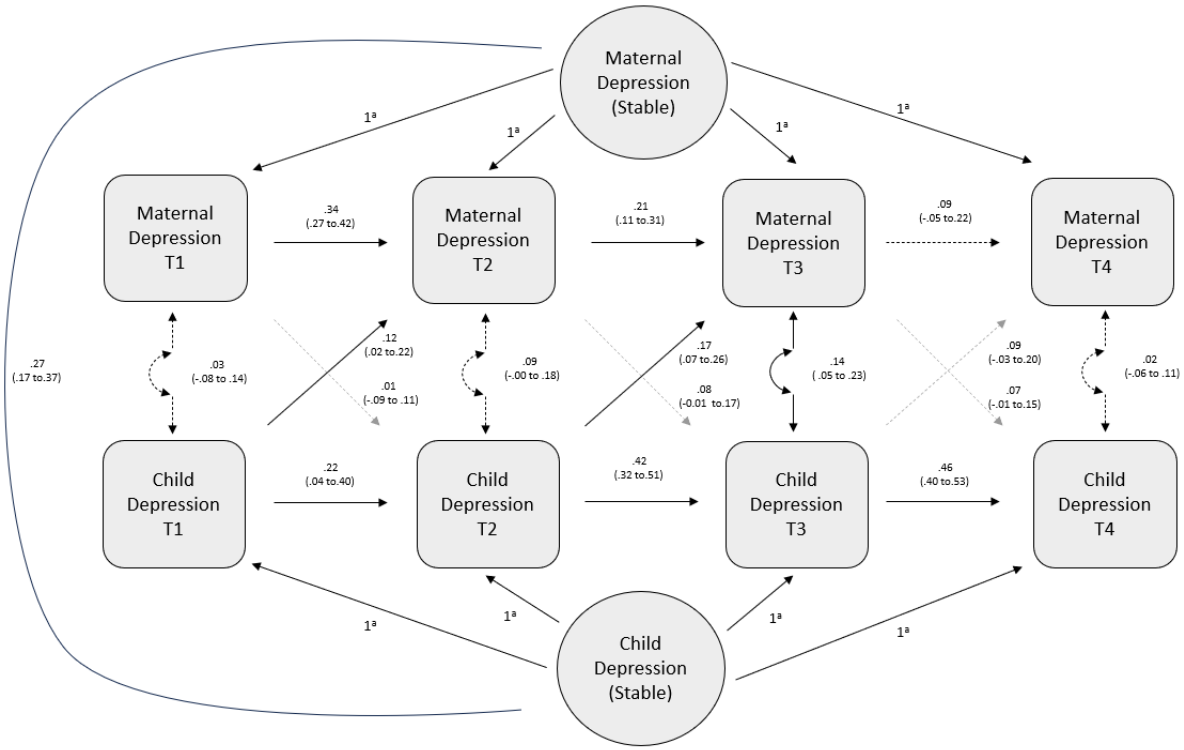
***Table 3. Basic RI-CLPM Standardized and Unstandardized Autoregressive and Cross-Lagged Coefficients***

| Paths | β (95% CI) | B (95% CI) |
|---|---|---|
| Autoregression Parameters | | |
| Maternal Depression | | |
| T1-T2 | .34 (.27 to .42) | .33 (.25 to .41) |
| T2-T3 | .21 (.11 to .31) | .20 (.10 to .30) |
| T3-T4 | .09 (-.05 to .22) | .07 (-.04 to .18) |
| Child Depression | | |
| T1-T2 | .22 (.04 to .40) | .27 (.05 to .49) |
| T2-T3 | .42 (.32 to .51) | .55 (.44 to .66) |
| T3-T4 | .46 (.40 to .53) | .56 (.48 to .64) |
| Cross-Lagged Coefficients | | |
| Mother to Child | | |
| M1-C2 | .01 (-.09 to .11) | .02 (-.20 to .23) |
| M2-C3 | .08 (-.01 to .17) | .23 (-.03 to .48) |
| M3-C4 | .07 (-.01 to .15) | .27 (-.03 to .56) |
| Child to Mother | | |
| C1-M2 | .12 (.02 to .22) | .07 (.01 to .12) |
| C2-M3 | .17 (.07 to .26) | .07 (.03 to .11) |
| C3-M4 | .09 (-.03 to .20) | .02 (-.01 to .05) |
| Time Invariant Coefficients | | |
| M1-C1 | .03 (-.08 to .14) | .77 (-2.23 to 3.83) |
| M2-C2 | .09 (-.00 to .18) | 2.67 (-.03 to 5.38) |
| M3-C3 | .14 (.05 to .23) | 4.94 (1.63 to 8.24) |
| M4-C4 | .02 (-.06 to .11) | .87 (-2.19 to 3.93) |

*Note M1 to M4 represent Maternal scores on the Center for Epidemiological Studies Depression Scale. C1 to C4 represent Child depression scores on the BASC, Behaviour Assessment System for Children*

***Figure 1. Basic RI-CLPM Model***

*Random intercept cross-lagged panel model testing bidirectional effects of maternal and child depression across 4 timepoints and controlling for stable between-person differences.*




*Note.* Solid lines represent statistically significant associations while dotted lines failed to reach significance. [a] pathways were constrained to 1 in order to isolate time-invariant, between dyad, differences in maternal and child depression.

***Figure 2. Timeline of COVID Pandemic and Restrictions in Alberta***

*Timeline of the COVID-19 pandemic related restrictions that were implemented across Alberta Canada.*