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**Regular Article**

# A transactional mediation model of risk for the intergenerational transmission of depression: The role of maternal criticism

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**Abstract**

In this study, we sought to combine two lines of research to better understand risk for the intergenerational transmission of depression. The first focuses on the role of maternal criticism as a potential mechanism of risk for depression in youth while the second builds from interpersonal and stress generation models regarding the potential impact of youth depression on future escalations in maternal criticism. Specifically, we examined the role of maternal criticism within a transactional mediation model using data from a multi-wave study. Participants were 251 mother–offspring pairs consisting of mothers with ($n = 129$) and without ($n = 122$) a history of major depressive disorder (MDD) during their child's lifetime who completed assessments every 6 months for 2 years. We found support for the hypothesized transactional mediational model in which maternal expressed emotion-criticism (EE-Crit) mediated the link between maternal history of MDD and residual change in youth's depressive symptoms over the previous 6 months and, reciprocally, youth depressive symptoms mediated the relation between maternal MDD history and residual change in EE-Crit 6 months later. These results indicate that maternal criticism and offspring depressive symptoms may contribute to a vicious cycle of depression risk, which should be considered for interventions targeted toward youth at risk of developing MDD.

**Keywords:** depression risk; expressed emotion; maternal criticism

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There is clear evidence for the intergenerational transmission of major depressive disorder (MDD; for a review, see Goodman, 2020). Indeed, offspring of mothers with MDD are three to six times more likely to develop MDD themselves than offspring of never depressed mothers (Gotlib et al., 2020). Despite this high risk, relatively little is known about the specific mechanisms that mediate the intergenerational transmission of depression risk. This type of information is essential for the development of more targeted preventive interventions designed to break the cycle of the intergenerational transmission of depression.

One potential mechanism of risk is maternal criticism. Research has shown that mothers with depression exhibit higher levels of criticism and hostility toward their children than do nondepressed mothers (for a review, see Goodman, 2007). Although much of this research is based on children's reports of maternal criticism, which may be subject to response bias, similar results have been obtained from studies focused on observer-coded levels of criticism. Specifically, researchers have used the Five-Minute Speech Sample (FMSS) (Magaña et al., 1986) to objectively code levels of criticism (expressed emotion-criticism [EE-Crit]) based on speech samples of mothers talking about their children. These studies have shown that mothers with a history of MDD exhibit higher levels of EE-Crit than never depressed mothers (e.g., Frye & Garber, 2005; Tompson et al., 2010), and that a maternal history of depression predicts elevated levels of EE-Crit 2 years later (Frye & Garber, 2005).

There is strong evidence for a link between maternal criticism (measured using the FMSS and self-report) and youth depression, though most of the existing research has been cross-sectional. For example, studies have shown that youth with current depressive disorders, compared to never depressed youth, are more likely to have parents who express high levels of EE-Crit (Asarnow et al., 1994, 2001; Silk et al., 2009). Further, higher levels of self-reported maternal criticism (Anhalt & Morris, 2008; Jacquez et al., 2004) and maternal EE-Crit (Frye & Garber, 2005; Han & Shaffer, 2014) are associated with higher levels of internalizing symptoms in youth. In addition to these cross-sectional studies, there is also evidence from two prospective studies that maternal EE-Crit predicts prospective changes in youths' depressive symptoms and onset of depressive diagnoses (Burkhouse et al., 2012; Silk et al., 2009).

In combination, therefore, previous research has supported each of the individual links in a mediation model in which maternal criticism may serve as a mechanism of risk for the intergenerational transmission of depression. There is also evidence that negative parenting more generally mediates the link between depression in mothers and child outcomes, including child depressive symptoms (for a review, see Goodman et al., 2020). However, no studies have formally evaluated whether maternal criticism mediates the link between a maternal history of MDD and change in children's depressive symptoms over time. The first goal of this study, therefore, is to specifically examine the potential mediating role of maternal criticism. In so doing, we focused on levels of

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EE-Crit to avoid relying on mothers' or children's self-reported levels of maternal criticism, which may be confounded with mothers' or children's depression, potentially inflating relations with these variables.

Another limitation of previous research that the study sought to address was that existing studies have focused on the impact of maternal criticism on youth depressive symptoms even though theorists recognize that many, if not most, relations between parenting behavior and youth psychopathology is transactional rather than unidirectional. Indeed, research has supported bidirectional relations between children's symptoms of psychopathology and various parenting behaviors (Hipwell et al., 2008; Serbin et al., 2015). In the current study, therefore, in addition to maternal criticism predicting changes in youth depressive symptoms, we hypothesized that youth depressive symptoms would also predict changes in maternal criticism. This is consistent with interpersonal models of depression, which emphasize not only the impact of interpersonal relations on depression risk, but also the impact of depression on interpersonal relations, with depressed individuals hypothesized to elicit negative reactions from others (Coyne, 1976). Our hypothesis is also consistent with stress generation models in which individuals with depression are hypothesized to contribute to the generation of additional stress in their lives, particularly interpersonal stress (Hammen, 1991, 1999). This generation of stress is thought to be particularly important when considering ongoing risk for depression, as it can create persistently stressful interpersonal contexts (Hammen & Shih, 2014). Regarding criticism specifically, researchers have hypothesized that symptoms related to depression (e.g., hopelessness and rumination) may cause strain in close relationships and elicit criticism from others (Joiner et al., 2005; McLaughlin & Nolen-Hoeksema, 2012).

In youth, there is clear evidence that elevated depressive symptom levels predict future increases in interpersonal stress (Connolly et al., 2010; Gibb & Hanley, 2010; Morabito et al., 2022; Shapero et al., 2013). There is also preliminary evidence from one study that youth depressive symptoms predict future increases in levels of maternal criticism (Nelemans et al., 2014), though this study focused on self-reported levels of maternal criticism. The transactional relation between depression and factors that contribute to the disorder (e.g., interpersonal stress) has been conceptualized as a "vicious cycle of risk" in that they can contribute to the cyclical perpetuation of one another, exacerbating both depression and interpersonal stress (Hammen, 2009; Teasdale, 1983). To formally evaluate this transactional model, we assessed levels of maternal criticism (EE-Crit) and youth depressive symptoms across a multi-wave prospective study.

The goal of this study, therefore, was to test a transactional mediation model of risk for the intergenerational transmission of depression. Specifically, we assessed levels of EE-Crit and youth's depressive symptoms every 6 months for 2 years (five assessments total) among mother–offspring dyads with or without a maternal history of MDD during their child's lifetime. We predicted that levels of EE-Crit at each assessment would predict change in youth depressive symptoms during the follow-up. Consistent with interpersonal models of risk and the stress generation theory, we also hypothesized the reverse direction of influence in which youth depressive symptoms at each assessment would predict prospective increases in EE-Crit. We then tested the full transactional mediational model to determine whether EE-Crit mediates the relation between maternal MDD and youth depressive symptoms over time and whether youth depressive symptoms mediate the link between maternal MDD and levels of EE-Crit over time, consistent with a transactional or vicious cycle of risk. Finally, given that offspring of mothers with a history of MDD are at increased risk for anxiety as well as depression (for a review, see Goodman et al., 2011), we evaluated the specificity of the model to youths' risk for depression versus anxiety. Importantly, there is evidence that EE-Crit is associated with youth depression but not anxiety (Asarnow et al., 2001), suggesting that EE-Crit may represent a mechanism of risk specifically for depression in offspring of mothers with MDD.

## Method

### Participants

Participants in this study were 255 mother–offspring pairs recruited from the community through different forms of advertisements (e.g., flyers, newspapers). Of the mothers, 129 had a history of *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 2000) MDD during the child's life and the remaining 116 mothers did not have any lifetime history of any mood disorder. Of this latter group, four developed MDD during the follow-up period and were therefore excluded, leaving us with a final sample of 251 mother–offspring dyads. Exclusion criteria for both groups included symptoms of schizophrenia, alcohol or substance abuse within the last 6 months, or a history of bipolar disorder. The participating offspring had to be between the ages of 8–14 at the initial assessment and be the biological child of the participating mother. Exclusion criteria for the youth were that they could not have a developmental or learning disability, per the mother's report. Furthermore, only one child per family could participate in the study. If more than one child was eligible for participation, then one child was chosen at random.

In terms of demographic characteristics, the average age of the mothers in our sample was 40.45 years ($SD = 6.96$). Of the mothers in our sample, 89.2% were White, 4.6% were Black, 3.1% were multiracial, and the remaining 3.1% were from other racial/ethnic groups. The children in our sample were 51.3% female and the average age was 11.28 years ($SD = 1.90$) at baseline. In terms of race/ethnicity, 82.6% were White, 10.3% were multiracial, 5.1% were Black, and the remaining 2.0% were from other racial/ethnic groups. Descriptive statistics are presented in Table 1.

### Measures

The Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I; First et al., 1994) was used to assess for current and past DSM-IV Axis I disorders in mothers at the baseline assessment. As noted above, 129 mothers met for an MDD diagnosis during their child's lifetime of whom 21 met criteria for current MDD at the baseline assessment. To assess inter-rater reliability, a random selection of 20 SCID-I interviews were coded by a second interviewer and the reliability of MDD diagnoses was excellent ($\kappa = 1.00$).

The FMSS (Magaña et al., 1986) was used to assess mothers' levels of EE-Crit at each assessment. For the FMSS, the mother is asked to speak for 5 minutes about her child and how the two of them get along together. The FMSSs were coded by individuals trained to reliability standards and who were kept unaware of the other study variables (e.g., maternal diagnoses). Mothers are rated as high on EE-Crit if any of the following three criteria are met: their initial statement about the child is negative (e.g., "He is a very

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**Table 1.** Descriptive statistics for study variables

| | Mothers with a history of MDD | Mothers with no history of MDD |
|---|---|---|
| Child age (T1) | 11.37 (2.02) | 11.37 (1.83) |
| Child gender (% girls) | 48.84% | 54.10% |
| Child race (% white) | 72.09% | 90.98% |
| T1 EE-Crit | 0.79 (0.92) | 0 .56 (0.82) |
| T2 EE-Crit | 0.78 (0.81) | 0.57 (0.79) |
| T3 EE-Crit | 0.62 (0.81) | 0.50 (0.73) |
| T4 EE-Crit | 0.58 (0.75) | 0.52 (0.74) |
| T5 EE-Crit | 0.67 (0.78) | 0.39 (0.65) |
| T1 CDI | 7.44 (5.88) | 4.79 (5.23) |
| T2 CDI | 6.68 (6.62) | 3.63 (4.16) |
| T3 CDI | 5.87 (5.01) | 3.55 (3.98) |
| T4 CDI | 5.27 (5.06) | 2.91 (3.75) |
| T5 CDI | 5.79 (5.27) | 3.40 (4.73) |
| T1 MASC | 46.40 (17.48) | 39.01 (13.68) |
| T2 MASC | 40.17 (16.61) | 36.47 (13.29) |
| T3 MASC | 37.88 (14.27) | 35.32 (14.35) |
| T4 MASC | 36.10 (13.77) | 32.42 (11.50) |
| T5 MASC | 34.16 (16.91) | 32.85 (14.10) |

*Note.* Unless otherwise noted, values represent means (and standard deviations). MDD = major depressive disorder; EE-Crit = expressed emotion-criticism; CDI = Children's Depression Inventory; MASC = Multidimensional Anxiety Scale for Children.

difficult person"), they report a negative relationship (e.g., "We yell at each other all the time"), or they make one or more critical statements during the FMSS (e.g., "I can't stand when she . . . ") as defined by the FMSS coding system. A borderline rating of EE-Crit is assigned if mothers express dissatisfaction about their child that is not extreme enough to be rated as a criticism (e.g., "Certain things she does upset me"). Previous studies have shown that the FMSS EE-Crit subscale has strong reliability and validity (Asarnow et al., 2001; Magaña et al., 1986) and levels of criticism assessed with the FMSS are significantly related to levels of criticism, anger, and hostility determined by independent raters during parent-child interactions (Cruise et al, 2011; Hermanns et al., 1989; Kim Park et al., 2008; McCarty et al., 2004; Narayan et al., 2012; Rea & Shaffer, 2016). In the current study, all speech samples were independently coded by two individuals who presented their ratings to a team of other raters. Based on a randomly selected subset of FMSSs, the inter-rater reliability of these initial EE-Crit ratings (low, borderline, high) was moderate (Fleiss's $\kappa = .58$; Altman, 1999). Any discrepancies between the raters were discussed by the team of raters who then generated a consensus EE-Crit rating. These consensus ratings were used for all analyses. For these analyses, a rating of low EE-Crit was assigned a value of 0 (59.20% of all speech sample), a rating of borderline EE-Crit was assigned a value of 1 (21.51% of all speech samples), and a rating of high EE-Crit was assigned a value of 2 (19.28% of all speech samples).

The Children's Depression Inventory (CDI; Kovacs, 1981) is a 27-item self-report questionnaire used to assess levels of depressive symptoms in youth aged 7–17 years old. Previous studies have shown that the CDI has strong reliability and validity in community samples of children ages 8–16 (e.g., Smucker et al., 1986). In this study, the CDI was administered at each assessment and demonstrated good reliability across all five assessment points ($\alpha s = .85–.89$).

Finally, youth's levels of anxiety were assessed with the Multidimensional Anxiety Scale for Children (MASC; March et al., 1997). The MASC has demonstrated excellent reliability and validity in previous research (e.g., March et al., 1997, March & Sullivan, 1999) and good internal consistency across all time points in this study ($\alpha s = .88–.93$).

### Procedure

During the baseline assessment, participating mothers provided informed consent and offspring provided assent. Following the consenting procedures, mothers completed the FMSS. Next, mothers completed the SCID-I while the youth completed the CDI and MASC in a separate room. Participants returned for follow-up assessments 6, 12, 18, and 24 months after the baseline assessment during which the mothers completed the FMSS and youth completed the CDI and MASC. Families were compensated financially for participating in the study. All study procedures were approved by the university's Institutional Review Board.

### Analysis plan

Prior to testing the full transactional mediation model, we first examined the prospective relations between EE-Crit and offspring depressive symptoms using hierarchical linear modeling (HLM). First, we examined the contribution of EE-Crit to residual change in offspring's depressive symptoms between assessments.

The Level 1 model was:

$$\text{CDI}_{ti} = \pi_{0i} + \pi_{1i}(\text{CDI}_{t\text{-}1i}) + \pi_{2i}(\text{EE-Crit}_{t\text{-}1i}) + e_{ti,}$$

which allowed us to determine whether EE-Crit at time T-1 predicted residual change in CDI from time T-1 to time T. We then used a similar analytic approach to examine whether CDI scores at time T-1 predicted residual change in EE-Crit from time T-1 to time T. Although we examined whether maternal history of MDD would moderate either of these relations as part of exploratory analyses, this type of moderation was not expected as part of our overall hypothesized mediation model.

We then tested the full transactional mediation model using a cross-lagged panel model in AMOS. We predicted that, in addition to transactional relations between EE-Crit and offspring's depressive symptoms across the follow-up, (a) levels of EE-Crit occurring during the multi-wave follow-up would mediate the link between mothers' histories of MDD and residual change in offspring's depressive symptoms, and that (b) offspring's levels of depressive symptoms during the follow-up would mediate the link between mothers' histories of MDD and residual change in EE-Crit. This hypothesized model is presented in Figure 1. Following standard model building approaches (Cole & Maxwell, 2003), we first fit an unconstrained model, with values for all paths free to vary. We then tested the equivalence of cross-lagged effects across waves (e.g., whether the path from CDI at a given time point to EE-Crit at the next time point was equivalent across waves of data) using a nested model comparison ($\chi^2$ difference test). Establishing this type of cross-wave equivalence indicates that the influence of one variable on change in the other between waves is equivalent across all the waves of the follow-up, which would be expected given that the

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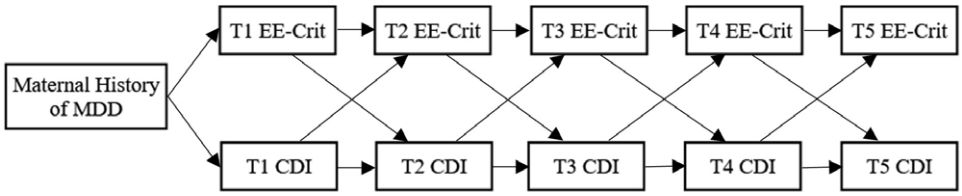


**Figure 1.** Hypothesized transactional mediation model examining relations among maternal history of MDD, maternal criticism, and youth depressive symptoms over time. *Note.* MDD = Major Depressive Disorder; EE-Crit = Expressed Emotion-Criticism; CDI = Children's Depression Inventory.

timing of the Time 1 assessment was not tied to any specific event. Model fit was evaluated using standard fit indices (comparative fit index [CFI], root mean squared error of approximation [RMSEA], and standardized root mean squared residual [SRMR]) with values of CFI > .90, RMSEA < .08, and SRMR < .10 reflecting good fit and values of CFI > .95, RMSEA < .06, and SRMR < .08 reflecting excellent fit (Hu & Bentler, 1999; Kline, 1998). To formally evaluate the two mediation paths – mother MDD predicting residual change in CDI through change in EE-Crit and mother MDD predicting residual change in EE-Crit through change in CDI – across the follow-up, the significance of the indirect effects was examined using 1000 bootstrap samples (cf. Cole & Maxwell, 2003).

## Results

Of the 251 mother–offspring pairs participating in the initial assessment, 211, 197, 170, and 174 participated at the 6-, 12-, 18-, and 24-month follow-ups, respectively. A preliminary inspection of the data revealed that the CDI and MASC exhibited significant skew. Therefore, these variables were transformed prior to further analysis (square root) to satisfy assumptions of normality. Given the presence of missing data, we then examined whether the data were missing at random, thereby justifying the use of data imputation methods for estimating missing values (Schafer & Graham, 2002). Little's missing completely at random test, for which the null hypothesis is that the data are missing completely at random (Little & Rubin, 1987) was nonsignificant, $\chi^2(1759) = 1829.21$, $p = .12$, supporting the imputation of missing values. Given these results, we used the expectation maximization method within SPSS to generate maximum likelihood estimates of missing data, which were used in all subsequent analyses (see Schafer & Graham, 2002). Descriptive statistics and correlations among the variables are presented in Tables 1 and 2, respectively. To facilitate comparisons with other studies, the descriptive statistics presented in Table 1 are based on untransformed variables.

As can be seen in Table 2, the between-wave 6-month stability of EE-Crit ranged from $r = .30$ to .47, and the 6-month stability of offspring's depressive symptoms ranged from $r = .69$ to .74, suggesting greater variability in EE-Crit than in offspring's depressive symptoms across the follow-up. In addition, offspring's gender (coded a 0 for boys and 1 for girls) and age were inconsistently associated with levels of EE-Crit and offspring depression. Specifically, girls reported higher levels of depressive symptoms at Time 5, but not at the other time points. In addition, older offspring age was associated with higher levels of EE-Crit at Time 2, but not at the other time points, and higher levels of depressive symptoms at Time 3, 4, and 5, but not Time 1 or 2.

### *Temporal relations between maternal criticism and youth depressive symptoms*

HLM was then used to determine the temporal relations between EE-Crit and youth depressive symptoms across the 2-year follow-up. Although, as described above, we first focused on EE-Crit at time T-1 predicting residual change in CDI from time T-1 to time T, this effect was not significant, $t(249) = -0.11$, $p = .91$, $r_{\text{effect size}} = .01$. Given this, we examined whether EE-Crit at time T was associated with residual change in CDI from time T-1 to time T and this was significant, $t(249) = 2.48$, $p = .01$, $r_{\text{effect size}} = .15$ (see Table 3). Therefore, EE-Crit appears to be concurrently associated with residual change in youth's depressive symptoms over the previous 6 months rather than predicting future change in depressive symptoms 6 months later. This effect was maintained when offspring age and gender were added as covariates, $t(247) = 2.60$, $p = .01$, $r_{\text{effect size}} = .16$. In neither of these analyses did maternal MDD history moderate the relation between EE-Crit and residual change in CDI (lowest $p = .41$) indicating that the association of EE-Crit with change in youth depressive symptoms over the past 6 months was similar in dyads with and without a maternal history of MDD.

Next, we used a similar analytic approach to examine whether CDI scores predicted residual change in EE-Crit levels, and whether this relation was moderated by maternal depression history. We found that CDI scores at time T-1 predicted residual change in EE-Crit from time T-1 to time T, $t(249) = 2.96$, $p = .003$, $r_{\text{effect size}} = .18$ (see Table 4). This effect was maintained when offspring age and gender were added as covariates, $t(247) = 2.99$, $p = .003$, $r_{\text{effect size}} = .19$. Maternal history of MDD did not significantly moderate this relation, $t(249) = -0.24$, $p = .81$, $r_{\text{effect size}} = .02$, suggesting that youth depressive symptoms predicted prospective increases in EE-Crit 6 months later to a similar degree in mothers with and without a history of MDD.

### *Full mediation model*

We then tested the full transactional mediation model using AMOS. Based on our preliminary HLM analyses, the model depicted in Figure 1 was modified to reflect the fact that EE-Crit at time T (rather than T-1) predicted residual change in CDI from time T-1 to time T. In addition, based on the results of the correlation table (Table 2), offspring age and gender were included as covariates in the model with direct paths from offspring age to T2 EE-Crit and T3-5 CDI as well as a path from offspring gender to T5 CDI. This model provided a marginal fit to the data, $\chi^2(54) = 155.56$, $p < .001$, CFI = .90, RMSEA = .09, SRMR = .09 (cf. Hu & Bentler, 1999; Kline, 1998). Modification indices suggested fit would be improved by allowing the error terms for T4 and T5 CDI to correlate. Because it is not uncommon for error terms for the same measure to be correlated across waves (Cole & Maxwell, 2003), we added a correlation between these error terms to the model. This modified model provided a good fit to the data, $\chi^2(53) = 131.43$, $p < .001$, CFI = .92, RMSEA = .08, SRMR = .08. Finally, we tested a nested model in which the cross-path beta weights (i.e., from EE-Crit to CDI and from CDI to EE-Crit) were required to be equal across waves (cf. Cole & Maxwell, 2003). This model also provided a good fit to the data, $\chi^2(59) = 139.69$, $p < .001$, CFI = .92, RMSEA = .07,

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**Table 2.** Correlations among study variables

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Mom MDD | | | | | | | | | | | | | | | | | |
| 2. Child gender | −.05 | | | | | | | | | | | | | | | | |
| 3. Child age | .00 | −.02 | | | | | | | | | | | | | | | |
| 4. T1 EE-Crit | .13 | −.01 | .02 | | | | | | | | | | | | | | |
| 5. T2 EE-Crit | .14 | −.04 | .18 | .30 | | | | | | | | | | | | | |
| 6. T3 EE-Crit | .08 | −.04 | .05 | .28 | .31 | | | | | | | | | | | | |
| 7. T4 EE-Crit | .04 | −.03 | .08 | .28 | .30 | .47 | | | | | | | | | | | |
| 8. T5 EE-Crit | .19 | .00 | .02 | .21 | .23 | .33 | .39 | | | | | | | | | | |
| 9. T1 CDI | .26 | .03 | .07 | .26 | .28 | .09 | .06 | .18 | | | | | | | | | |
| 10. T2 CDI | .29 | .02 | .10 | .19 | .24 | .06 | .14 | .15 | .69 | | | | | | | | |
| 11. T3 CDI | .26 | .11 | .18 | .11 | .20 | .07 | .14 | .19 | .62 | .74 | | | | | | | |
| 12. T4 CDI | .28 | .08 | .15 | .14 | .22 | .04 | .16 | .10 | .49 | .62 | .70 | | | | | | |
| 13. T5 CDI | .27 | .17 | .18 | .17 | .27 | .06 | .11 | .17 | .47 | .63 | .66 | .70 | | | | | |
| 14. T1 MASC | .22 | .13 | −.11 | −.01 | .03 | −.12 | .00 | .09 | .40 | .27 | .28 | .24 | .30 | | | | |
| 15. T2 MASC | .10 | .09 | .00 | .03 | .06 | −.09 | .09 | .02 | .24 | .31 | .27 | .29 | .23 | .58 | | | |
| 16. T3 MASC | .09 | .06 | −.03 | −.06 | −.01 | −.10 | .05 | −.10 | .20 | .24 | .30 | .27 | .19 | .51 | .69 | | |
| 17. T4 MASC | .13 | .05 | −.09 | −.02 | .13 | −.20 | −.06 | −.15 | .16 | .17 | .19 | .35 | .26 | .43 | .53 | .60 | |
| 18. T5 MASC | .02 | .12 | −.03 | −.08 | .04 | −.21 | −.06 | −.03 | .11 | .28 | .27 | .28 | .40 | .45 | .62 | .65 | .67 |

*Note.* MDD = major depressive disorder; EE-Crit = expressed emotion-criticism; CDI = Children's Depression Inventory; MASC = Multidimensional Anxiety Scale for Children. Correlations > .12 significant at $p < .05$; correlations > .17 significant at $p < .01$; correlations > .21 significant at $p < .001$.

**Table 3.** Maternal criticism predicting change in offspring depressive symptoms

| | *B* | *t* | $r_{\text{effect size}}$ |
|---|---|---|---|
| $CDI_t$ intercept ($\pi_0$) | | | |
| Intercept ($\beta_{00}$) | 0.57 | 9.29** | .51 |
| Mom MDD ($\beta_{01}$) | −0.06 | −0.45 | .03 |
| $EE\text{-}Crit_t$ slope ($\pi_1$) | | | |
| Intercept ($\beta_{10}$) | 0.08 | 2.48* | .16 |
| Mom MDD ($\beta_{11}$) | 0.03 | 0.51 | .03 |
| $CDI_{t-1}$ slope ($\pi_2$) | | | |
| Intercept ($\beta_{20}$) | 0.62 | 21.11** | .80 |
| Mom MDD ($\beta_{21}$) | −0.02 | −0.27 | .03 |

*Note.* CDI = Children's Depression Inventory; MDD = major depressive disorder; EE-Crit = expressed emotion-criticism.
*$p < .05$; **$p < .001$.

**Table 4.** Offspring depressive symptoms predicting change in maternal criticism

| | *B* | *t* | $r_{\text{effect size}}$ |
|---|---|---|---|
| $EE\text{-}Crit_t$ intercept ($\pi_0$) | | | |
| Intercept ($\beta_{00}$) | 0.31 | 6.92** | .40 |
| Mom MDD ($\beta_{01}$) | −0.09 | −1.04 | .07 |
| $CDI_{t-1}$ slope ($\pi_1$) | | | |
| Intercept ($\beta_{10}$) | 0.06 | 2.96* | .18 |
| Mom MDD ($\beta_{11}$) | −0.01 | −0.24 | .02 |
| $EE\text{-}Crit_{t-1}$ slope ($\pi_2$) | | | |
| Intercept ($\beta_{20}$) | 0.25 | 7.17** | .41 |
| Mom MDD ($\beta_{21}$) | 0.10 | 1.43 | .09 |

*Note.* EE-Crit = expressed emotion-criticism; MDD = major depressive disorder; CDI = Children's Depression Inventory.
*$p < .01$; **$p < .001$.

SRMR = .08, and did not fit significantly worse than the unconstrained model in which the paths were free to vary, $\chi^2(6) = 8.26$, $p = .22$. The results of this final model are presented in Figure 2. All the direct paths in the model were significant ($p < .05$). We then examined the significance of the various indirect effects using 1000 bootstrap samples. The total indirect effect of maternal history of MDD through EE-Crit at each wave on residual change in CDI at each wave was significant, *beta* = .01, $p = .03$. Furthermore, the total indirect effect of maternal MDD history through CDI at each wave on residual change in EE-Crit at each wave was significant, *beta* = .08, $p = .003$. We should also note that across wave transactional indirect paths were also significant across all waves. Specifically, the total indirect effect of EE-Crit on future EE-Crit through residual change in CDI was significant, *beta* = .01, $p = .03$, as was the total indirect effect of CDI at each wave on future CDI through residual change in EE-Crit, *beta* = .01, $p = .03$. Overall, this model account for 45.0% of the variance in Time 5 CDI scores.

### *Specificity to youths' depression versus anxiety*

Finally, to evaluate the specificity of the findings to offspring's levels of depression versus anxiety, the analyses were repeated, this time replacing CDI with MASC scores. Focusing first on the

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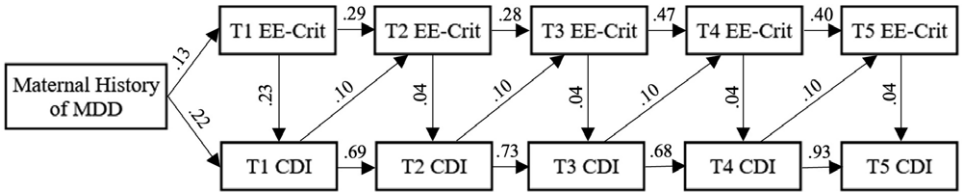


**Figure 2.** Final transactional mediation model examining relations among maternal history of MDD, maternal criticism, and youth depressive symptoms over time. *Note.* MDD = Major Depressive Disorder; EE-Crit = Expressed Emotion-Criticism; CDI = Children's Depression Inventory. Values represent standardized beta weights, and all paths are significant ($p < .05$). Error terms and covariates are not included in the figure to facilitate presentation.

HLM analyses, there was no evidence of temporal relations between EE-Crit and MASC scores across the follow-up, either in terms of EE-Crit predicting residual change in MASC over the next 6 months, $t(249) = -1.12$, $p = .26$, $r_{\text{effect size}} = .07$, or the previous 6 months, $t(249) = 0.48$, $p = .63$, $r_{\text{effect size}} = .03$, or MASC predicting residual change in EE-Crit over the next 6 months, $t(249) = -0.14$, $p = .89$, $r_{\text{effect size}} = .01$, or previous 6 months, $t(249) = 0.15$, $p = .88$, $r_{\text{effect size}} = .01$. In addition, none of these relations were moderated by mothers' histories of MDD (lowest $p = .28$). Therefore, the full transactional mediation model was not examined for offspring's levels of anxiety because these relations are required for evaluating the full model.

## Discussion

The aim of this study was to examine the role of maternal criticism within a transactional mediation model of risk for the intergenerational transmission of depression. In doing so, we sought to integrate two lines of research that have, to date, advanced separately. The first has examined the role of maternal criticism in youth depression risk, though no prior study has formally evaluated whether maternal criticism mediates the link between mothers' history of MDD during their child's lifetime and prospective changes in youth depressive symptoms. The second line of research focuses on interpersonal and stress generation models of risk, which show that depressed individuals may behave in ways that increase risk for future interpersonal stress in their lives. With this line of research, however, only one prior study has examined whether depressive symptoms in youth predict future increases in maternal criticism and it focused on self-reported levels of perceived criticism (Nelemans et al., 2014).

First, we examined temporal relations between EE-Crit and offspring depressive symptoms across the follow-up. Partially supporting our hypothesis regarding the role of EE-Crit in depressive symptom change, we found that levels of EE-Crit at each assessment (time T) were associated with residual change in youth depressive symptoms from the previous timepoint (time T-1) to the current time point (time T), but EE-Crit did not predict prospective change in depressive symptoms 6 months later. It may be that EE-Crit predicts youth depressive symptom change over shorter time intervals but not as far out as 6 months later. Future research is needed to test this possibility. Turning to the reverse direction of influence, we found strong support for the stress generation hypothesis in that youth depressive symptoms predicted prospective increases in EE-Crit 6 months later. This adds to existing research showing that youth depressive symptoms contribute to future interpersonal stressors generally (Connolly et al., 2010; Shapero et al., 2013) and extends this research to demonstrate that youth depressive symptoms predict increases in maternal criticism, specifically. As such, it extends prior research by Nelemans et al. (2014), who showed that adolescent depressive symptoms predict future increases in youth and mother reported levels of maternal criticism to demonstrate that this stress generation effect extends to objectively coded levels of maternal criticism (i.e., EE-Crit). Therefore, the current study is the first to show that youth depressive symptoms predict prospective increases in observer-coded levels of maternal criticism over time. These findings are consistent with interpersonal theories of depression, which acknowledge that not only do interpersonal stressors contribute to depressive symptoms, but also that depressed individuals may elicit interpersonal stress and negative evaluations from others (Coyne, 1976; Hammen, 1991, 1999).

Finally, we tested the full transactional mediation model. Consistent with our hypotheses, we found that levels of EE-Crit at each time point mediated the relation between maternal MDD history and residual change in youth depressive symptoms over the previous 6 months and that youth depressive symptoms at each time point mediated the relation between maternal MDD history and residual change in EE-Crit over the next 6 months. Further supporting the transactional model, residual change in offspring depressive symptoms mediated the link between prior and future EE-Crit and residual change in EE-Crit mediated the link between prior and future offspring depression. Theorists have proposed transactional models of risk for youth psychopathology (Goodman & Gotlib, 1999; Sameroff & Mackenzie, 2003); however, the current findings are the first to document how maternal criticism operates within transactional model of risk for the intergenerational transmission of depression.

Although we primarily focused on a model of risk for the intergenerational transmission of depression, we also examined youth anxiety symptoms to determine whether the model would be specific to youths' symptoms of depression. We found that EE-Crit did not predict changes in youth anxiety during the follow-up nor did youth anxiety predict changes in EE-Crit, suggesting that the transactional model of risk was specific to offspring's risk for depression. This is consistent with prior research suggesting that parental EE-Crit may be specific to offspring depressive symptoms whereas offspring's symptoms of anxiety may be more strongly related to emotional over involvement (EE-EOI), another category of expressed emotion (Asarnow et al., 2001; Hirshfeld et al., 1997; Stubbe et al., 1993).

The current findings have several important implications for future research. First, they demonstrate transactional relations between maternal criticism and offspring depressive symptoms over time, suggesting that these types of reciprocal influences between youth symptoms and maternal behavior may be particularly important to consider when examining risk for depression and methods of reducing this risk. For example, interventions for parents of young children (e.g., In-Home CBT, Enhanced Triple P) which promote responsive, sensitive, and positive

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parenting skills have been implemented among mothers with depression (Goodman & Garber, 2017). Second, these findings could have broader implications that extend outside of the family. Previous research shows that youths' depressive symptoms can contribute to the generation of future interpersonal stress with peers, including peer victimization (Gibb & Hanley, 2010; Morabito et al., 2022). Therefore, it is possible that the transactional model of depression risk demonstrated within parent–offspring relationships is also present in youths' relationships with their peers. Future research should investigate a more inclusive dynamic model of risk where the transactional influence of both parent and peer factors are considered.

The current study exhibited a number of strengths including the multi-wave longitudinal design, which allowed us to examine temporal relations between maternal criticism and youth depressive symptoms over time. Another strength is the focus on objectively coded levels of maternal criticism, which minimizes response bias related to depressive symptoms in either maternal or offspring perceptions of maternal criticism. The study also exhibited some limitations, which provide important avenues for future research. One limitation is that the youth in our sample, who were recruited from the community, had relatively low levels of depressive symptoms. Future research is needed to determine how well the current findings generalize to a more severely depressed sample. Another limitation is that we only focused on mothers and did not account for potential risk that could be contributed from fathers' MDD history or levels of criticism. Future research should examine the role of MDD and/or EE-Crit in fathers, either alone or moderating the impact of mothers. Third, although the stress generation model focuses specifically on dependent stressors (i.e., stressors to which the individual at least partially contributed), our measure of maternal criticism did not allow this type of specificity and additional research is needed to determine the extent to which youth symptoms directly contribute to the elicitation of maternal criticism. Fourth, this study only examined the relation between maternal criticism and offspring's internalizing symptoms but maternal criticism may also be related to other forms of child pathology (e.g., externalizing symptoms), which should be explored in future research. Finally, the participants in this study predominantly identified as White and future research is needed to determine the generalizability of these findings to more racially and ethnically diverse samples.

In conclusion, the current results document the role of maternal criticism within a transactional model of intergenerational risk for depression. In doing so, they combine prior research examining the association between maternal criticism and youth depression with findings from interpersonal theories and the stress generation literature showing how depressive symptoms can contribute to additional interpersonal stress in an individual's life. Notably, EE-Crit was associated with change in offspring depressive symptoms over the previous 6 months rather than predicting change in depressive symptoms 6 months later, suggesting that the temporal influence of EE-Crit may be more proximal and future studies are needed to examine shorter follow-up intervals. This said, the current results suggest that maternal criticism and youth depressive symptoms should be viewed as contributing to a vicious cycle of risk in which both mothers and offspring transactionally influence each other over time, increasing levels of maternal criticism and offspring depression. These findings have broader implications for early intervention/prevention programs as breaking this vicious cycle may be a promising approach for reducing risk for the intergenerational transmission of depression.

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