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RESEARCH ARTICLE

Transactional patterns of depressive symptoms between mothers and adolescents: The role of emotion regulation

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Abstract

Background: Depression is a highly prevalent, debilitating disorder that runs in families. Yet, empirical support for bidirectional mechanisms linking mother–adolescent depression symptoms remains limited. This study examined longitudinal bidirectional relations among emotion regulation (ER) constructs and depressive symptoms among mother–adolescent dyads over time. Pathways for girls and boys were explored separately, given extant research on sex differences in the intergenerational transmission of depression.

Methods: Adolescent ($n = 232$; $M = 15.02$ years, $SD = 0.95$; 44% female)–mother dyads, drawn from a longitudinal study on the development of risky behaviors, completed annual assessments of depressive symptoms and facets of ER over 4 years. Panel modeling examined lagged and cross-lagged effects of mother–adolescent depressive symptoms and ER constructs over time, in a multigroup model of boys and girls.

Results: Among girls, higher baseline maternal depression scores predicted increased adolescent ER difficulties (std. est. = $-.42$, $p < .001$) in turn, predicting increased adolescent depressive symptoms (std. est. = $-.33$, $p = .002$) and subsequent maternal ER difficulties (std. est. = $.39$, $p = .002$). The indirect effect of maternal depressive symptoms→adolescent ER→adolescent depressive symptoms→maternal ER was significant (ind. eff. = $.10$, 95% confidence interval [$>.001$, $.19$]) for girls, but not boys.

Conclusion: Implications for interrupting intergenerational cycles of depressive symptoms and emotion dysregulation are discussed.

KEYWORDS

depression, emotion regulation, parent–child relations

1 | INTRODUCTION

Adolescence is a period of significant upheaval across familial, biological, and psychological domains. Rates of depression rise dramatically between 15 and 18 (Hankin et al., 1998; Weinberger et al., 2018) and differences in the prevalence of depression between boys and girls begin to emerge, such that women are twice as likely as men to be diagnosed with a depressive disorder by early adulthood (Shore et al., 2018). Increased rates of depression are particularly troubling as early onset is associated with more severe dysfunction,

including risky behaviors, suicide, and substance use (Kessler, 2012; Shore et al., 2018), and recurrent depressive episodes into adulthood (Lallukka et al., 2019).

1.1 | Maternal and adolescent depressive symptoms

Extensive research demonstrates both environmental and genetic factors contribute to the intergenerational transmission of depression

within families (Goodman, 2020). These factors can also be reciprocal, meaning that parent and child characteristics mutually influence depression symptomatology over time (Goodman & Gotlib, 1999). Processes linking maternal-offspring depression become most apparent by adolescence, when risk factors interact with increasing stress exposure (Rudolph, 2002). Studies also suggest patterns of parent-child mutual influence may be different in boys and girls; however, the preponderance of these studies utilize child samples and cross-sectional data, preventing the mapping of longitudinal associations across a critical period of development for depression (Connell & Goodman, 2002).

1.2 | Emotion regulation

The capacity to regulate emotions within the context of distress predicts the severity of depression course across the lifespan (Aldao et al., 2010). Research suggests that, like depression, emotion regulation (ER) is influenced by genetic (Hawn et al., 2015) and environmental factors, suggesting multiple pathways linking parent and child ER capacity (McRae et al., 2017). Additionally, maternal depressive symptoms are consistently linked to impaired ER in offspring (Maughan et al., 2007). Research suggests that children of depressed mothers, particularly girls, have early deficits in cognitive and behavioral ER strategies (Silk et al., 2006). Given that depression can interfere with parents' abilities to model ER and healthy coping (Goodman & Gotlib, 2002), offspring may observe their depressed parent engaging in maladaptive emotional reactions and model these behaviors (Brand & Klimes-Dougan, 2010). Thus, ER may be a crucial piece of the transmission puzzle.

One important component of ER is one's ability to persist in goal-directed activity, rather than avoid or withdraw, in the face of distress (i.e., distress tolerance; DT). DT is conceptualized as a dispositional ER ability, capturing the capacity of a person to withstand distressing states, and closely linked to the deployment of ER strategies (Naragon-Gainey et al., 2017). DT represents an important part of the negative reinforcement cycle of depression maintenance, such that avoiding distress provides temporary relief, but reinforces patterns of withdrawal behavior associated with depression (Felton et al., 2019). DT has been associated with subsequent youth depressive symptoms, both directly and as moderated by stressful events (Felton et al., 2017, 2019). Studies have also found that adolescent girls are particularly impaired in maintaining approach behaviors in the face of distress compared to boys (Neumann et al., 2010) and a positive relation between maternal DT and DT in daughters (Daughters et al., 2014). Parent modeling of distress responses may become increasingly important across adolescence, as youth are exposed to more frequent stressors (Arnett, 1999; Rudolph, 2002) and less receptive to direct parent guidance (Katz & Hunter, 2007). Research suggests that depressed mothers demonstrate more maladaptive responses to child-related distress (Cummings et al., 2013) which, in turn, may decrease children's own tolerance of distress in their environments and promote depressive symptoms. Thus, DT represents

a facet of ER that is likely relevant in understanding the development of depression during adolescence.

1.3 | Transactional processes linking parent-to-child-to-parent effects

Although a majority of research on the intergenerational transmission of depression focuses on downstream effects of parent symptoms on child outcomes, a smaller literature has probed relations in the opposite direction (i.e., child-to-parent). Most have looked at the role of infants' and young children's temperament as a predictor of maternal depression (Slagt et al., 2016). Studies examining these relations in older children/adolescents tend to support youth depression as a driver of maternal symptomatology. For instance, Garber et al. (2011) found that child depressive symptoms were associated with parental depressive symptom. One potential mechanism may be the tendency of depressogenic behaviors in youth to overwhelm maternal ER capacity, eliciting negative reactivity in parents (Schwartz et al., 2011). Indeed, Shortt et al. (2015) found that parents of depressed adolescents are more likely to respond to youths' distress/anger with their own anger; however, this was examined cross-sectionally, precluding researchers from drawing conclusions regarding temporality. Other research highlights numerous and negative familial and parental outcomes following the onset of adolescent depression for girls specifically, including decreases in family cohesion, adaptability, and communication between mother and father (Russell et al., 2019). This distress, in turn, may cumulatively serve to decrease mothers' adaptive ER abilities. Prospective research is needed to examine whether adolescent depressive symptoms drive maternal emotion dysregulation, especially among distinct groups of boys and girls, to elucidate important differences in transactional relations between maternal-daughter/son depressive symptomatology.

1.4 | Current study

The current study examined transactional influences (specifically, parent-to-child-to-parent effects) of maternal and adolescent depressive symptoms and ER capacity in boys and girls across 4 years using panel modeling (see Figure 1). Based on existing literature, we hypothesized that maternal depressive symptoms would predict changes in adolescents' ER ability (defined as distress intolerance), in turn predicting increases in adolescent depressive symptoms. We also hypothesized that increases in adolescent depressive symptoms would predict worsening maternal ER. Although the effect of sex on direct relations between maternal depressive symptoms on rates of adolescent depression is unclear, maternal depressive symptoms appear to impact girls' ER specifically (Silk et al., 2006) and, in turn, girls depressive symptoms may have a specific negative impact on family functioning (Russell et al., 2019). Thus, we hypothesized that links between these constructs would be significant among girls but not boys.

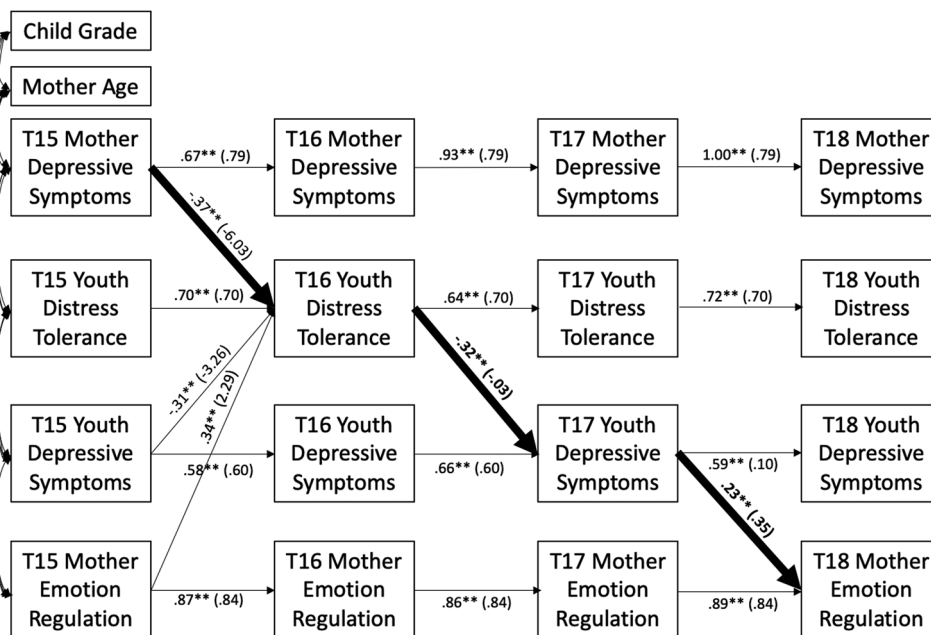


FIGURE 1 Mediation model and standardized estimates of the longitudinal relations between mother and adolescent depressive symptoms and facets of emotion regulation in female adolescents. Mediation pathway is shown in bold. Note: Only significant pathways are shown to reduce visual clutter

2 | MATERIALS AND METHODS

2.1 | Participants and procedures

Adolescent–mother dyads were drawn from the final four waves of a longitudinal study investigating the development of risky behaviors (masked for review). Eligible youth were: (1) 9–13 years old; (2) proficient in English; and (3) willing to participate in annual assessments. Given our focus on development across middle adolescence, the current study's baseline corresponds to wave 5 of the larger study. At this time, mothers were, on average, 46 years old (range: 30–59) and adolescents were approximately age 15 (age range: 13–17, grades: 8–12). For clarity, time point labels moving forward reflect the average adolescent age at each wave (T15, T16, T17, and T18). Of the original sample ($N = 277$), 206 participated at T15, 177 at T16, 153 at T17, at 151 at T18 for the current study. At baseline, youth were, on average, 15.02 years old ($SD = 0.95$; 44% female; 55% White/Caucasian, 36.9% Black/African American, 0.5% American Indian or Alaska Native, 1% Asian, and 6.4% other race/ethnicity). The majority of mothers (97.4%) were biologically related to the adolescent.

Assessments were completed in the laboratory, where participants provided informed consent and assent at each wave. Youth and parents were assessed in separate rooms and responses were not shared between respondents. Study procedures were IRB approved, and adolescents and parents received up to \$40 at each assessment.

2.2 | Measures

2.2.1 | Demographics

Mothers reported their own and their adolescent's age, grade, sex, and race at each time point. Adolescents' grades and mothers' ages were included in all models as covariates.

2.2.2 | Depression symptoms

Maternal and adolescent depression symptoms were measured using two, parallel, self-report measures: the Center for Epidemiological Studies-Depression (CES-D; Olsson & von Knotting, 1997) and the Center for Epidemiological Studies-Depression Child (CES-DC; Weissman et al., 1980), respectively. These 20-item self-report measures capture symptom frequency across the past week. Response options range from 0 (*rarely/none of the time*) to 3 (*most/almost all of the time*). Scores are summative with higher scores indicating greater depression symptoms. At baseline (T15), 10.3% of mothers and 29.1% of adolescents reported clinically significant depressive symptoms, using standard criteria ($CES-D \geq 16$; Lewinsohn et al., 1997), consistent with other community-based studies (Olsson & von Knotting, 1997; Vilagut et al., 2016). In this study, Cronbach alpha (α) ranged from .87 to .89 for mothers and .89 to .90 for adolescents.

2.2.3 | Maternal emotion regulation

A variety of facets of maternal ER was measured using the 36-item Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2003). Mothers rated how often each item applied to them using a 5-point Likert scale from 1 (*almost never*) to 5 (*almost always*). Total scores reflect a sum of items, with higher scores indicating more maternal emotion dysregulation (Gratz & Roemer, 2003). In this study, Cronbach alpha (α) ranged from .92 to .93.

2.2.4 | Adolescent emotion regulation

Adolescent ER was operationalized by a behavioral measure of DT: The Behavioral Indicator of Resiliency to Distress (BIRD; Lejuez et al., 2006). The computerized task requires youth to click on 1 of 10 boxes designated by a dot. Participants who click on the correct box earn a point; those who click the wrong box hear a loud and unpleasant noise and are not awarded a point. The BIRD task is split into three levels of increasing difficulty. Participants are told at the beginning of the task that they may quit the last level at any point, but will then not earn additional points. The amount of time participants persist in the final level is used as a behavioral measure of DT, with lower values indexing lower DT. The BIRD demonstrates significant test-retest reliability (Cummings et al., 2013) and has been shown to elicit significant increases in negative affect (Amstadter et al., 2012). Moreover, the BIRD demonstrates both concurrent and prospective validity (Danielson et al., 2010; Daughters et al., 2009).

2.3 | Data analytic plan

All analyses were conducted in *Mplus* 8 (Muthén & Muthén, 1998–2017). A structural equation model depicting transactional relations between maternal-adolescent depressive symptoms, maternal ER, and adolescent DT across four waves of data was examined. Given variability in children's grade and maternal age at baseline and research suggesting associations with depressive symptoms (e.g., Eshbaugh, 2008), child grade and mothers' age were added as covariates at baseline. Full Information Maximum Likelihood estimation methods addressed missing data within and across waves, yielding less biased parameter estimates relative to listwise and pairwise deletion or single imputation approaches under missing at random assumptions (Little & Rubin, 1989). Model fit was evaluated using standard criteria: χ^2 statistic, Comparative Fit Index (CFI), Standardized Root Mean Square Residual (SRMR), and Root Mean Square Error of Approximation (RMSEA).

To examine proposed relations among boys and girls, a series of nested models were evaluated. Utilizing an iterative model-building approach and starting with the full sample without constraints, we compared models with increasingly constrained parameters. Given our goal of examining patterns of relations in separate groups of boys and girls, we evaluated significant change in model fit across three

indicators: $\Delta \chi^2$, $\Delta \text{CFI} \geq .01$ and $\Delta \text{RMSEA} \geq .015$ (Chen, 2007; Cheung & Rensvold, 2002) and elected to utilize the most parsimonious model consistent with project aims that demonstrated adequate fit. Successive models and their fit indices are reported in the Supporting Information.

All pathways demonstrating transactional (mother-to-child-to-mother) relations between constructs of interest were examined in our final model using bootstrapping, which involved generating 10,000 randomized samples with a 95% bias-corrected confidence interval [CI]. CIs that do not include zero suggest the indirect effect is statistically significant.

3 | RESULTS

3.1 | Preliminary analyses

Skew and kurtosis were examined across all variables and found to be within appropriate limits (≤ 3.00). Little's Missing Completely at Random (MCAR) test was not significant: $\chi^2(895) = 904.79$, $p = .40$, supporting the assumption that data could be considered MCAR. Descriptive statistics for maternal and adolescent depressive symptoms, ER and DT for boys and girls are reported in Table 1. Next, a series of *t*-tests (Table 1) and correlations (Table 2) were computed. Boys and girls significantly differed on depressive symptoms and age, with girls being younger and more depressed (with the exception of T18). Maternal depressive symptoms significantly correlated with same-wave difficulties in ER at every time point (correlations ranged from .56 to .64); adolescent depressive symptoms were significantly associated with DT task performance at T16 only.

3.2 | Transactional effects model

Iterative models and their respective fit indices are presented in Supporting Information. Our final, most parsimonious, model examined relations in boys and girls separately utilizing multigroup analyses (figures with all path estimates are depicted in Supporting Information). The model included constraints across the stability paths and variances of same constructs to over time and between groups, demonstrating adequate model fit: $\chi^2(174) = 293.53$, $p < .001$; CFI = .90; SRMR = .08; and RMSEA = .07 (90% CI = .06 to .09).

In the girls' model, baseline levels of depressive symptoms, maternal ER, and maternal depressive symptoms predicted changes in DT at T16 (Figure 1). Change in DT at T16 then predicted changes in adolescent depressive symptoms at T17 which, in turn, predicted changes in maternal ER at T18.

In the boys' model, T15 maternal ER predicted changes in T16 maternal depressive symptoms. Lower rates of depressive symptoms at T16 predicted worse maternal ER at T17, and worse maternal ER at T16 also predicted higher adolescent depressive symptoms at T17.

Across all pathways, only one transactional (i.e., parent-to-child-to-parent) effect emerged: among girls, there was a significant

TABLE 1 Means, SDs, and *t*-tests of key study variables

Measure	<i>M (SD)</i>		<i>t</i> (<i>df</i>)	<i>p</i>	Cohen's <i>d</i>
	Boys	Girls			
T15 Mother CES-D	8.81 (7.93)	9.68 (7.98)	0.76 (193)	.450	0.11
T16 Mother CES-D	8.96 (8.60)	8.30 (7.89)	-0.51 (168)	.614	-0.08
T17 Mother CES-D	7.67 (7.65)	8.69 (8.18)	0.78 (147)	.434	0.13
T18 Mother CES-D	8.54 (8.67)	8.09 (6.09)	-0.35 (140)	.731	-0.06
T15 Mother DERS	62.02 (16.39)	62.28 (16.14)	0.11 (194)	.910	0.02
T16 Mother DERS	60.19 (17.21)	60.91 (15.80)	0.28 (168)	.784	0.04
T17 Mother DERS	60.33 (14.24)	61.70 (17.67)	0.52 (147)	.601	0.09
T18 Mother DERS	61.16 (18.49)	58.90 (15.54)	-0.76 (138)	.446	-0.13
T15 Adolescent CES-D	12.15 (8.37)	15.93 (10.39)	2.82 (168)	.005	0.40
T16 Adolescent CES-D	11.34 (8.21)	16.11 (11.01)	3.15 (128)	.002	0.48
T17 Adolescent CES-D	12.06 (8.73)	15.58 (10.82)	2.18 (127)	.031	0.35
T18 Adolescent CES-D	11.85 (8.67)	14.71 (10.36)	1.81 (125)	.073	0.29
T15 Adolescent BIRD	197.50 (112.92)	202.64 (112.14)	0.31 (182)	.757	0.05
T16 Adolescent BIRD	205.38 (114.33)	199.27 (110.44)	-0.31 (131)	.756	-0.05
T17 Adolescent BIRD	185.55 (118.25)	174.87 (121.19)	-0.51 (126)	.615	-0.09
T18 Adolescent BIRD	184.33 (125.22)	183.25 (122.03)	-0.05 (116)	.962	-0.01

Abbreviations: BIRD, Behavioral Indicator of Resiliency to Distress; CES-D, Center for Epidemiological Studies - Depression; DERS, Difficulties in Emotion Regulation.

TABLE 2 Intercorrelations of key study variables in male and female adolescents

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. T15 Mother CES-D	1.00	.70**	.61**	.62**	.59**	.46**	.54**	.59**	.09	.09	.20	-.06	-.04	.18	-.03	-.13
2. T16 Mother CES-D	.61**	1.00	.52**	.65**	.57**	.57**	.50**	.59**	.12	.16	.17	-.05	.03	.23	.03	.03
3. T17 Mother CES-D	.54**	.45**	1.00	.71**	.44**	.26*	.50**	.57**	.02	.08	.17	-.16	.06	.27*	.01	-.04
4. T18 Mother CES-D	.63**	.72**	.62**	1.00	.52**	.47**	.50**	.54**	-.01	.07	.14	.01	-.16	.17	-.16	-.14
5. T15 Mother DERS	.56**	.63**	.29*	.60**	1.00	.82**	.76**	.75**	.05	.24*	.15	-.01	-.08	.12	<.01	.02
6. T16 Mother DERS	.63**	.64**	.43**	.61**	.90**	1.00	.75**	.79**	.12	.26**	.28*	.21	.01	.14	.04	.06
7. T17 Mother DERS	.63**	.55**	.56**	.54**	.86**	.84**	1.00	.77**	.03	-.03	.03	-.13	.01	.26	-.05	-.10
8. T18 Mother DERS	.45**	.55**	.51**	.59**	.71**	.74**	.80**	1.00	.04	.13	.12	<.01	-.14	.24	-.11	.03
9. T15 Adolescent CES-D	.20	-.06	.17	-.09	.12	.11	.15	.16	1.00	.53**	.19	.20	-.01	.08	.01	.07
10. T16 Adolescent CES-D	.14	.07	-.12	-.05	.10	.06	-.01	.06	.53**	1.00	.31**	.38**	.05	-.08	.12	.29*
11. T17 Adolescent CES-D	.29*	.23	.27*	.10	.23	.23	.29*	.42*	.59**	.55**	1.00	.47**	.09	.01	.03	-.02
12. T18 Adolescent CES-D	.11	<.01	.12	.08	.09	.05	.04	-.04	.48**	.38*	.39**	1.00	.13	.06	.01	.05
13. T15 Adolescent BIRD	.04	-.17	.11	.12	-.07	-.15	-.06	-.04	.11	-.02	-.17	-.08	1.00	.49**	.47**	.34*
14. T16 Adolescent BIRD	-.22	-.03	-.08	.07	.01	-.01	-.05	.13	-.19	-.38**	-.47**	-.24	.61**	1.00	.28	.50**
15. T17 Adolescent BIRD	-.10	-.04	-.16	.12	.05	-.07	-.20	.07	.05	-.08	-.08	-.12	.39**	.43**	1.00	.64**
16. T18 Adolescent BIRD	<.01	-.08	<.01	.16	.03	.10	-.11	-.02	-.18	-.14	-.15	.01	.53**	.65**	.43**	1.00

Note: Correlations for male adolescents are displayed above the diagonal, correlations for female adolescents are displayed below the diagonal. T1-T4 correspond to waves 1 through 4.

Abbreviations: BIRD, Behavioral Indicator of Resiliency to Distress; CES-D, Center for Epidemiological Studies - Depression; DERS, Difficulties in Emotion Regulation.

p* < .05; *p* < .01.

indirect effect from T15 maternal depressive symptoms to T18 maternal ER, via T16 adolescent DT and T17 adolescent depressive symptoms (std. ind. eff. = .03, $SE = .02$, 95% CI [$<.001$, .07]).¹ Additional (but not transactional) indirect pathways indicating links between mother's pathology and adolescents' depression symptoms were also significant; however, these were not explicitly transactional in nature and are reported in Supporting Information.

4 | DISCUSSION

Identifying parent-to-child-to-parent pathways that sustain the occurrence of depression among mothers and adolescents is important to disrupting the intergenerational transmission of depression. In the current study, relations between maternal and adolescent depressive symptoms and ER factors were evaluated across four years in separate models of boys and girls. Findings are consistent with literature highlighting associations between maternal-child depressive symptomatology (see Goodman [2020] for review), and expand existing research to suggest the important role of ER constructs broadly in the development of depressive symptomatology among mother-adolescent dyads. First, results suggest two ways that mother characteristics impact child vulnerabilities: for girls, maternal depressive symptoms were associated with girls' DT and for boys, maternal ER predicted increases in boys' depressive symptoms. Second, only girls' depressive symptoms were associated with increases in maternal ER. Finally, our findings support several mediation models demonstrating unidirectional effects between parent and child; however, only one (maternal depressive symptoms → girls' DT → girls' depressive symptoms → maternal ER), showed specific transactional pathways between mothers and daughters.

This study is among the first to examine processes linking maternal risk factors to adolescent depression-related outcomes in separate groups of boys and girls. Results suggest that maternal depressive symptoms may shape girls' ability to tolerate distress, and points to a mechanism by which mothers impact their daughters' responses to stress. Among boys, the direct effects of maternal ER on boys' depressive symptoms are consistent with extant research (Crespo et al., 2017), suggesting that mothers with worse ER capacity may model or socialize depressive responses to stress, although these relations were found for boys only. Indeed, these nuanced findings were possible given our multigroup modeling approach that examined pathways separately in boys and girls, rather than collapsing across sexes. These results add to the heterogeneous literature on the effects of maternal risk and pathology on boys' and girls' depressive symptoms. They also suggest that an important avenue for future inquiry is the examination of sex-specific effects of mothers'

engagement in behaviors characteristic of depression and emotion dysregulation. Considerably less attention has been paid to the role of adolescent psychopathology in predicting mothers' ER capacity, as most research to date has focused on the impact of infant/early child temperament on maternal depression (e.g., Slagt et al., 2016). Our results suggest that female adolescents' depressive symptoms may contribute to mothers' own risk for psychopathology by increasing maternal impairment in ER. Perhaps adolescents' depressive symptoms cause distress in parents, as depressed adolescents may engage in a variety of distressing behaviors (e.g., aggression, oppositionality; Knox et al., 2000; Leadbeater et al., 2012), and report overall worse parent-child relationship quality (Withers et al., 2016). This, in turn, may overtax mothers' ER capacity and eventually increase the risk for future parental depressive symptoms (Withers et al., 2016). Indeed, qualitative work examining maternal responses to adolescent depression highlights parental feelings of uncertainty, instability, helplessness, and frustration in the face of adolescent depression (Armitage et al., 2020). Curiously, neither boys' depressive symptoms nor DT predicted changes in maternal outcomes. It may be that mothers are less sensitive to internalizing symptoms among adolescent boys compared to other forms of pathology (Gross et al., 2008). Alternatively, these relations may be specific to certain developmental stages. Indeed, our results indicate that girls' depressive symptoms were linked to maternal ER only at the final wave. It is possible that boys' depressive symptomatology is more impactful at different developmental stages, consistent with other research suggesting child effects may be more pronounced at specific points during development (Gross et al., 2008).

4.1 | Clinical implications

Although considerable attention has been paid to the developmental course of adolescent depression, less research has focused on changes in adult depression as it specifically relates to parenting a depressed adolescent and possible mechanisms of risk. Indeed, many interventions for depressed adolescents include family components, but do not consider the impact of children's pathology on parental pathology and how this may sustain depression within the family system. Our findings suggest the importance of considering maternal psychopathology in the conceptualization of female adolescent depression specifically and treating both mother and daughter concurrently to promote recovery (Compas et al., 2009).

4.2 | Strengths, limitations, and future directions

This study's longitudinal design allowed us to test maternal predictors of adolescent psychopathology and the subsequent impact on mothers' own mental health. These findings support temporal associations of these constructs and point to specific, modifiable risk factors (e.g., DT, ER) that can be targeted by interventions. We also utilized an ecologically valid behavioral measure of DT, which avoided (a)

¹We also examined the stationarity of each significant pathway in boys and girls separately. We found that all significant pathways from T15 to T16 were statistically equivalent to the relation between the same constructs at subsequent waves for boys. Assumptions for stationarity did not hold for all relations among girls. Specifically, we found that the effect of maternal depressive symptoms on daughters' DT and the effect of daughters' depressive symptoms on mothers' ER were not statistically equivalent across waves.

relying on adolescents' perceptions of their responses to abstract stressful situations and (b) depression-related biases. Finally, these processes were examined in an ethnically diverse sample of youth, increasing the generalizability of findings to other populations.

Several limitations of this study suggest potential avenues for future research. First, as with all empirical studies, replication of these findings with larger samples is critical to ensure that findings are not specific to this sample (Maxwell et al., 2015). Moreover, while our use of a behavioral indicator of DT was a strength, we relied on self-report measures of depressive symptoms and maternal emotion dysregulation, which may be influenced by reporter bias effects and shared method variance. Future studies should consider multi-informant and multimethod approaches to assessing symptomology and mechanisms. Second, we did not utilize the same measures of ER in adults and adolescents. Our assessment focused on adolescent DT as a specific facet of ER, rather than ER broadly. Future inquiry could consider parallel measures of ER in adolescents and mothers. Third, it is possible that mothers and children with more severe depressive symptoms and ER difficulties did not enroll in the study, resulting in selection bias and limiting the generalizability of these findings to other samples with higher clinical needs. Fourth, we do not have information regarding the adolescents' history of exposure to maternal depression. Given findings suggesting stronger relations between maternal and child pathology at younger, relative to older, ages (see Goodman, 2020), future research could incorporate the exposure frequency, severity, and length of time to maternal depressive symptoms. Our findings reflect a relatively brief (4 years) period of development. Given that the causal structure of transactional relations between mothers and daughters changed over time, these findings may not generalize to younger ages (when ER capacity may be broader; Zimmermann & Iwanski, 2014) nor to older ages when depressive symptoms begin to plateau (Kessler et al., 2005). Thus, future research is needed to understand the impact of specific developmental processes on these unfolding relations. Fifth, this study did not examine the contributions of biological factors (including genetic and epigenetic predictors). Examination of these relations is important to untangle the relative environmental and biological effects of these relations. Given research suggesting paternal depressive symptoms were concurrently associated with symptomology in sons but not daughters (Andreas et al., 2018), future research can similarly examine father-adolescent transactional effects. It is possible that transmission of ER and depressive symptoms may be different among fathers given potential differences in the way fathers respond to child behaviors (Cassano et al., 2007).

5 | CONCLUSIONS

Transactional influences (from parent-to-child-to-parent) of ER and depressive symptoms among mother-adolescent dyads is an important contributor to the perpetuation of psychopathology within families. Findings from the current study replicate existing models of

parent and child depressive symptoms, and extend this literature to illustrate specific pathways linking ER processes between mothers and daughters. These findings highlight the transactional processes and mechanisms that serve to maintain depression within the family context and suggest potential targets for family based treatments for depression.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

DATA AVAILABILITY STATEMENT

Data are available from the corresponding author upon reasonable request.

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