

Research paper

Experiential avoidance in the vulnerability to depression among adolescent females



William Mellick, Salome Vanwoerden, Carla Sharp*

University of Houston, United States

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ABSTRACT

Background: Although various mechanisms in the maternal transmission of Major Depressive Disorder (MDD) have been investigated, it is unknown whether experiential avoidance (EA) is a vulnerability factor in the development of depression or a consequence of the illness. The present study utilized a high-risk design to determine if EA indeed poses vulnerability to adolescent MDD. Secondly, we examined the means by which adolescent EA may come to pose vulnerability, namely that it explains the relation between maternal EA and adolescent depressive symptoms.

Methods: One-hundred and forty-six biological mother/adolescent daughter dyads comprised three diagnostic groups: mothers with a history of MDD and their depressed daughters (MDD; $n=21$), mothers with a history of MDD and their never-depressed daughters (high-risk, HR; $n=69$), and healthy controls (HCs; $n=56$).

Results: Groups differed on daughter EA such that the MDD group reported greater EA than the HR group, which in turn reported greater EA than HCs. Daughter EA mediated the relation between maternal EA and daughter depressive symptoms after controlling for maternal depressive symptoms.

Limitations: Strengths aside, this study included a relatively small group of depressed mother-daughter dyads and relied on cross-sectional self-report data.

Conclusions: EA appears to serve as a vulnerability factor for adolescent MDD, and the mechanistic role of daughter EA highlights the significance of intergenerational EA in the maternal transmission of depression. Therapeutic approaches may therefore consider reducing the transmission of EA from mothers to daughters.

1. Introduction

Children of mothers with a history of major depressive disorder (MDD) are at a three-fold increased risk of developing psychopathology during childhood or adolescence (Goodman et al., 2011). More specifically, these at-risk children are especially likely to develop affective disorders, most notably major depression (Downey and Coyne, 1990). An extensive body of work has therefore investigated various mechanisms in the maternal transmission of depression that confer vulnerability among youth (i.e., genetic, neurodevelopmental, and psychosocial: Diego et al., 2004; Hammen, 2002; Sullivan et al., 2000). This research has been highly informative; however, upward of 41% of at-risk children still come to experience clinically-significant depressive symptoms (Goodman, 2007). Since MDD often emerges in adolescence (Hankin, 2006), the continued examination of plausible mechanisms during this sensitive period is highly warranted.

Experiential avoidance (EA) refers to an “unwillingness to remain in contact with uncomfortable private events by escaping or avoiding these experiences” (Hayes et al., 1996, p. 1154). This overarching

concept embodies “emotions, thoughts, physiological reactions, action tendencies and urges, memories, or even factual knowledge that would contradict a well-rehearsed view of oneself” (Boulanger et al., 2010, p. 109). As a cross-cutting feature of psychopathology, EA has been linked to the development and symptom presentations of several psychiatric disorders (Chawla and Ostafin, 2007). In relation to depression, theoretical accounts suggest that EA alters an individual’s emotional experience thereby increasing psychological distress and exacerbating depressive symptoms (Hayes et al., 2004). Adult findings converge on a strong positive relation between EA and depression, particularly among women with affective disorders (Spinoven et al., 2014). Although the relation between EA and depression is present even with sub-clinical levels of depression (Tull et al., 2004; Polusny et al., 2004).

Though it has not been in the context of maternal MDD, investigators have explored parental EA and its relation to child psychopathology symptoms. For instance, among parents of clinically anxious youth, maternal EA was positively associated with maternal depressive symptoms and accounted for significant variance in child anxiety symptoms as rated by both parents and clinicians (Cheron et al.,

* Correspondence to: Department of Psychology, University of Houston, 126 Heyne Building, Houston, TX 77204, United States.
E-mail address: csharp2@uh.edu (C. Sharp).

2009). Moreover, maternal EA was negatively associated with familial communication of emotions suggesting that EA disrupts the caregiving environment. Specifically, high-EA mothers may be less likely to attend to their own negative emotional experiences as well as those of their children. Though maternal EA has received more empirical attention in regards to early childhood anxiety and behavior problems (Coyne et al., 2007; Tiwari et al., 2008), similar relations have been demonstrated among depressive mothers and their young children. Specifically, maternal dysphoria, maternal EA, and child internalizing symptoms were all found to be significantly correlated (Coyne and Thompson, 2011). Despite this growing body of literature, there is a lack of research investigating these relations among parents and their adolescent children.

Nonetheless, it is evident that EA and depression are positively related in adolescents as they are in adults. While fewer studies have been conducted, findings with both clinical and community adolescent samples have mirrored adult findings (Biglan et al., 2015; Greco et al., 2008; Howe-Martin et al., 2012; Venta et al., 2012). Moreover, the relation between EA and depression appears to be stronger among adolescent girls as compared to boys (Howe-Martin et al., 2012; Venta et al., 2012).

Clearly, EA has emerged a strong correlate of depressive symptoms in both adults and children. The question therefore arises whether EA is a vulnerability factor in the development of depression or a consequence of the illness. If EA were a vulnerability factor, it should be present in the non-depressed biological offspring of depressed mothers. In this case, the biological offspring of depressed mothers may acquire similar depressotypic characteristics, including EA, through social learning and/or genetic transmission (Goodman, 2007). However, if EA is a consequence of adolescent depression, EA will correlate with adolescent depressive symptoms but will be absent in the non-depressed biological offspring of depressed mothers. By utilizing a high-risk research design, investigators can examine the above questions effectively in order to determine the vulnerability status of psychological processes such as EA (Gotlib et al., 2010). Positive results would justify a treatment focus on EA in children at risk for developing depression by virtue of being the biological offspring of a depressed mother. Against this background, the first aim of the current study was to compare levels of EA in three groups of daughters: currently depressed daughters of mothers with a history of depression, never-depressed daughters of mothers with a history of depression, and healthy daughters of never-depressed mothers. Daughters rather than sons were specifically recruited because they possess greater vulnerability to depression and the female preponderance of MDD begins to emerge during adolescence (Goodman et al., 2011). Moreover, girls have reported significantly greater EA than boys across adolescent studies highlighting the importance of examining EA among females specifically (Howe-Martin et al., 2012; Venta et al., 2012).

A separate but related question was whether maternal EA, regardless of the status of maternal depression would relate to depressive symptoms in daughters, and whether this relation would be mediated by daughter EA. Previous research has demonstrated a positive association between maternal EA and child internalizing problems among depressive mothers and their pre-school-aged children (Coyne and Thompson, 2011). This may also hold for adolescence. If so, a putative model for understanding the relation of maternal EA to daughter depressive symptoms is to suggest that maternal EA increases levels of EA in daughters, which in turn increases levels of depressive symptoms in daughters. Therefore, the second aim of the current study was to examine a mediation model by which the relation between maternal EA and daughter depressive symptoms was mediated by daughter EA. In order to show specificity in these relations, as EA and depressive symptoms often co-occur, we also tested an alternate mediation model in which daughter EA served as the dependent variable and daughter depressive symptoms was the mediator. In both instances, we controlled for maternal depressive symptoms in order to

examine maternal EA's direct influence. Positive results in this regard would justify a focus on reducing the transmission of EA from mothers to daughters through various therapeutic approaches.

2. Methods

2.1. Participants

A total of $N=146$ biological mother/adolescent daughter dyads comprised three groups who differed in diagnostic status: mothers with a history of MDD and their depressed daughters (MDD: $n=21$), mothers with a history of MDD and their never-depressed daughters (high-risk, HR; $n=69$), and mothers and daughters free from any psychiatric history (healthy controls, HCs; $n=56$). Among mothers with a history of MDD, nine in the MDD group (42.9%) and twenty-eight in the HR group (40.6%) were experiencing a current episode of major depression. Comorbidity history among MDD mothers included generalized anxiety disorder ($n=6$), obsessive compulsive disorder ($n=5$), specific phobia ($n=5$), social phobia ($n=5$), and alcohol dependence ($n=4$). Comorbidity history among MDD daughters included obsessive-compulsive disorder ($n=6$), post-traumatic stress disorder ($n=4$), social phobia ($n=4$), attention-deficit/hyperactivity disorder ($n=4$), and oppositional defiant disorder ($n=2$). Among HR mothers, comorbidity history included alcohol dependence ($n=14$), specific phobia ($n=7$), generalized anxiety disorder ($n=6$), anxiety disorder not otherwise specified ($n=6$), social phobia ($n=5$), obsessive compulsive disorder ($n=2$), and pain disorder ($n=1$). Comorbidity history among HR daughters included obsessive-compulsive disorder ($n=4$), post-traumatic stress disorder ($n=1$), social phobia ($n=2$), attention-deficit/hyperactivity disorder ($n=2$), and oppositional defiant disorder ($n=2$). The overall sample was diverse with 57% identifying as Hispanic and a racial breakdown as follows: 40.4% Caucasian, 21.9% African-American, 13.7% Multiracial, 3.4% American Indian or Alaskan Native, 0.7% Asian, and 0.7% Pacific Islander with 19.2% choosing not to identify.

Inclusion criteria required participants to possess English fluency, girls were required to be between 10 and 16 years-old and possess a fifth-grade reading level as determined by the Wide Range Achievement Test 4 (WRAT4; Wilkinson and Robertson, 2006), and both mothers and daughters had to meet appropriate psychiatric criteria for dyad group assignment. Girls between ages 10–16 years were recruited given the focus on at-risk status of adolescents. We purposefully sought to maximize the possibility of recruiting biological offspring of depressed mothers who have not yet developed depression in order to compare them with already depressed and non-depressed groups. If participants failed to meet full criteria, had a psychotic disorder or any learning disability or mental retardation, they were excluded. Participants responded to community advertisements (57%), and were recruited from local inpatient and outpatient clinics for adolescents (43%).

2.2. Measures

2.2.1. Diagnostic status

The Structured Clinical Interview for DSM-IV TR Axis I disorders (SCID-I; First et al., 2002) was conducted to determine whether mothers had a history of MDD during their daughter's lifetime. For the assessment of MDD and comorbid psychiatric disorders, all modules were used. Moderate to excellent inter-rater reliability has been found for the SCID-I (mean kappa=0.71; Lobbetael et al., 2011). Inter-rater reliability on audiotaped SCID-I interviews used for the present study, with raters blind to the diagnosis of the mother, found Kappa to be 1.00 for current diagnosis of depression and 0.81 for past diagnosis of depression (Sharp et al., 2014).

The NIMH Diagnostic Interview Schedule for Children – Version IV (DISC-IV; Shaffer et al., 2000) was conducted with daughters to

determine the presence or absence of MDD and other psychiatric disorders. The DISC-IV is a structured and comprehensive interview for use with children and adolescents ages 9–17. The computer-based interview consists of “yes/no” questions and determines whether DSM-IV diagnostic criteria are met algorithmically, and therefore inter-rater reliability is not required. High test-retest reliability for diagnoses on the DISC-IV has been evidenced in clinical samples, i.e., MDD, κ .92 (Shaffer et al., 2000).

2.2.2. *Experiential avoidance*

Mothers completed the Acceptance and Action Questionnaire (AAQ; Hayes et al., submitted for publication) which consists of ten items, including “When I feel depressed or anxious, I am unable to take care of my responsibilities” and “I’m not afraid of my feelings”. Responses are scored on a seven-point Likert scale, with lower scores typically indicating greater EA. However, we chose to rescale the measure so that higher scores indicated greater EA to aid interpretation of results (i.e., positive relations between key variables). The AAQ has demonstrated good psychometrics in other adult studies (Hayes et al., submitted for publication). Internal consistency in the present sample, as measured by Cronbach’s alpha (α), was 0.73.

Daughters completed the Avoidance and Fusion Questionnaire for Youth (AFQ-Y; Greco et al., 2008) which was adapted from the AAQ. The AFQ-Y consists of seventeen items, including “I push away thoughts and feelings that I don’t like” and “My life won’t be good until I feel happy”. Items are rated on a five-point Likert scale, with higher scores indicating greater EA. In the present study $\alpha=0.88$, which is comparable to other adolescent studies (i.e., Howe-Martin et al., 2012).

2.2.3. *Depressive symptoms*

Mothers reported depressive symptoms over the past two weeks using the Beck Depression Inventory-II (BDI-II; Beck et al., 1996), a 21-item self-report questionnaire. Both internal consistency ($r=.92$) and stability ($r=.93$) have been demonstrated for the BDI-II (Beck et al., 1996). For the current study, $\alpha=0.94$.

Daughters reported depressive symptoms over the past two weeks using the Mood and Feelings Questionnaire (MFQ; Angold et al., 1987), a 33-item self-report questionnaire for use with children and adolescents. Criterion validity and re-test reliability for the MFQ has been supported (Vander Stoep et al., 2012; Wilkinson et al., 2013). In the current study, $\alpha=0.94$.

2.3. *Procedures*

This study was approved by the appropriate institutional review board. Those interested in the study were required to complete phone screens, and if eligible were invited to participate. Mothers and daughters completed structured clinical interviews to determine dyad group assignment. MDD group membership required mothers to have a lifetime diagnosis or recurrent MDD since their daughter’s birth and daughters to have an MDD history; HR membership required mothers to have a lifetime diagnosis or recurrent MDD since their daughter’s birth and daughters to have never been depressed; and lastly HC group membership required both mothers and daughters to be free of any history of psychiatric disorder (current or lifetime). Mothers and daughters were administered structured clinical interviews and completed self-report measures separately in private. All interviews were conducted by doctoral graduate students and clinical research assistants whom had completed training under the supervision of the study’s principal investigator. Data collection for the present study began in 2010 and was completed in 2012.

2.4. *Data analytic strategy*

All statistical analyses were performed using SPSS version 19.0

(Chicago, IL). Chi-Square tests for independence and Analyses of Variance (ANOVAs) were conducted to examine group differences. Preacher and Hayes (2008) test of the indirect effect was used for mediation analyses, and tolerance and variance inflation factors (VIF) were examined beforehand to ensure multicollinearity was not present. To ease interpretation of mediation model path coefficients, standardized values were presented.

3. Results

3.1. *Participant characteristic*

The average age in years was 12.98 for daughters ($SD=1.92$) and 40.12 for mothers ($SD=7.12$), respectively. Families reported an average annual household income of \$41.4K ($SD=\$4.1K$). There were no significant differences between dyads on sociodemographic variables: daughter age, $F(2, 145)=0.33, p=0.721$; mother age, $F(2, 145)=0.120, p=0.887$; annual household income, $F(2, 129)=0.412, p=0.663$; Hispanic ethnicity, $\chi^2(2, N=146)=3.76, p=0.449$; or race, $\chi^2(2, N=146)=9.78, p=0.660$. There were also no significant differences among participants on key variables of interest based on recruitment source.

Table 1 summarizes the clinical characteristics of daughter and mothers as well as group comparison results. As expected, there were

Table 1
Participant characteristics and group comparison results.

	MDD n=21	High-Risk n=69	HC n=56	p	Group difference
Clinical variables					
Daughter					
AFQ-Y	32.29 (12.51)	20.33 (12.40)	14.83 (8.92)	< .001	MDD > HR > HC
MFQ	27.43 (12.24)	15.46 (12.05)	9.80 (8.46)	< .001	MDD > HR > HC
Mother					
AAQ	35.57 (13.03)	33.03 (12.97)	24.05 (10.08)	< .001	MDD, HR > HC
BDI-II	19.28 (17.25)	16.21 (12.24)	7.35 (8.65)	< .001	MDD, HR > HC
Demographics					
Daughter age					
	13.29 (1.85)	12.90 (1.90)	12.96 (2.00)	0.721	
Mother age					
	40.76 (7.82)	39.91 (7.10)	40.25 (7.01)	0.887	
Familial Income					
	37.50K (20.24)	39.30K (20.94)	40.54K (35.50)	0.667	
Hispanic ethnicity					
Yes	(47.6%)	(44.9%)	(63.2%)	0.449	
No	(47.6%)	(55.1%)	(33.3%)		
Race					
Black	6 (28.6%)	15 (21.7%)	11 (19.6%)	0.660	
White	10 (47.6%)	24 (34.8%)	26 (46.4%)		
Multiracial/other	4 (19.0%)	12 (17.4%)	5 (8.9%)		
Asian	0	1 (1.4%)	0		
American Indian/ Alaskan Native	0	3 (4.3%)	2 (3.6%)		

Data are mean (standard deviation). AFQ-Y=Avoidance and Fusion Questionnaire for Youth; MFQ=Mood and Feelings Questionnaire; AAQ=Acceptance and Action Questionnaire; BDI-II=Beck Depression Inventory-II; Age=age in years; Familial income=Yearly familial income. A total of n=15 mothers chose not to report family income, n=28 chose not to identify race (MDD, n=1; HR, n=14; HC, n=13), and n=3 chose not to identify as Hispanic or not (MDD, n=1, HC, n=2). Note: p-values of One-Way ANOVAs and chi-square tests of independence; AAQ scores were rescaled so higher scores indicate greater EA.

significant differences between daughters on depressive symptoms, $F(2, 145)=20.30, p < .001, \eta^2=.221$. Compared to HR and HCs, daughters in the MDD group reported significantly greater symptoms of depression, $p < .001$, whereas HR and HC daughters also significantly differed, $p=.012$. For mothers, a consistent pattern emerged for EA and depressive symptoms where the MDD and HR groups did not statistically differ from each other but differed from HCs; EA, $F(2, 145)=11.42, p < .001, \eta^2=.138$, and depressive symptoms, $F(2, 145)=11.71, p < .001, \eta^2=.141$.

3.2. Group differences in adolescent EA

A One-Way ANOVA was performed to determine whether adolescent EA was associated with vulnerability to MDD. Adolescent EA was therefore entered as a between-subject factor compared across the three diagnostic groups of daughters. All groups were found to significantly differ, $F(2, 145)=18.61, p < .001, \eta^2=.210$. Specifically, the MDD group reported greater EA than the HR group, $p < .001$, who in turn reported greater EA than HCs, $p=.019$.

3.3. Adolescent EA as a mediator of the relation between maternal EA and adolescent depressive symptoms

Prior to performing mediation analyses, we examined the bivariate correlations between all variables included in the tested models. As shown in Table 2, all relations between variables of interest were statistically significant, $p's \leq .001$. Analyses were then performed with the full sample to test whether daughter EA mediated the relationship between maternal EA and daughter depressive symptoms, controlling for maternal symptoms of depression (see Fig. 1). Using Preacher and Hayes' (2008) test of the indirect effect, the mean of the indirect effect of daughter EA across all bootstrap samples estimated at .2159 with a confidence interval that did not include zero, $CI=.0450-.4200$ (Preacher and Hayes, 2008). Thus, adolescent EA was shown to serve as a mediator, such that greater maternal EA was related to higher adolescent EA and subsequently, greater depressive symptoms in daughters. Maternal depressive symptoms was a nonsignificant covariate, $p=.156$.

Given the cross-sectional design of our study, we conducted post-hoc mediation analyses to examine the possibility that daughter depressive symptoms may have an indirect effect on the relation between maternal EA and daughter EA. Maternal EA was again entered as the independent variable; however, in this instance, daughter EA served as the dependent variable and daughter depressive symptoms as the mediator, controlling for maternal depressive symptoms. This alternate model revealed a nonsignificant indirect effect of daughter depressive symptoms with a confidence interval that included zero, $CI=-.0329$ to $.3590$.

4. Discussion

This is the first investigation of the role of EA in the maternal transmission of major depression. Two key findings emerged from this study; first, EA appeared to pose vulnerability for MDD in never-

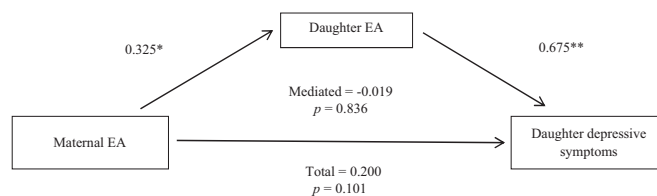


Fig. 1. Mediation model of the effect of maternal EA on daughter depressive symptoms through daughter EA. Note: Values are standardized path coefficients controlling for maternal symptoms of depression (BDI-II). Maternal EA=AAQ total; Daughter EA=AFQ-Y total; Daughter depressive symptoms=MFQ total. * $p \leq .01$ ** $p \leq .001$.

depressed high-risk adolescent girls. Second, adolescent EA mediated the relation between maternal EA and adolescent depressive symptoms, even after controlling for maternal depressive symptoms. Greater levels of maternal EA were associated with greater levels of daughter depression, and daughter EA served as a mechanism by which these variables were associated. Though we cannot infer causal relations, this suggests EA to be transmitted inter-generationally and to increase adolescents' levels of depression. The directionality of this relation was provided further support by results from our alternate mediation model, which revealed a nonsignificant indirect effect of daughter depressive symptoms on the relation between maternal EA and daughter EA. Taken together, our findings suggest that focusing therapeutic efforts on reducing maternal and daughter EA may in turn reduce risk for depression, particularly among adolescents predisposed to the disorder.

Given that EA was found to be significantly elevated among the high-risk daughters as compared to healthy controls makes EA a plausible vulnerability factor for depression. The tiered fashion of EA across the groups of daughters suggests that elevated EA is present prior to major depressive onset and is further exacerbated post-onset, yet we acknowledge that results do not allow for definitive conclusions. Depressive symptoms were also significantly greater across groups and we are unable to infer temporal relations between EA and depressive symptoms, which is particularly relevant among HR daughters. However, this notion that EA may increase adolescent vulnerability is in line with longitudinal findings in affective-disordered adults where EA has been shown to remain present over two-year's time, increasing with disorder occurrence and decreasing during remission (Spinhoven et al., 2014). As we alluded, though we cannot disentangle from our cross-sectional data whether high EA presupposed initial MDD onset among the depressed daughters, we suspect this to be the case. Future longitudinal research with never-depressed high-risk youth is needed to support or refute this working hypothesis.

The extent to which EA remains static in the vulnerability to depression is also to be determined; however, it may be malleable over time through new learning experiences or environmental changes. Many vulnerability models for depression are stress-diathesis based whereby stressful events occur and elicit cognitive vulnerability processes resulting in depression (Ingram et al., 2011), and EA may operate in a similar fashion. Indeed, among never-depressed community adult females, EA has been shown to interact with life stress in predicting increases in longitudinal depressive symptoms, even after controlling for baseline depressive symptoms (Shallcross et al., 2010). This work has yet to be extended to youth, but the potential for an EA x life stress interaction among high-risk daughters may be particularly likely vis-à-vis exposure to maternal depression and the familial stress associated with it (Hammen, 2002; Monroe and Reid, 2009). Whether EA also interacts with other known vulnerabilities to youth depression, in the presence of life stress, is worthy of investigation as certain constellations of vulnerability may pose greater risk than others.

Taking a developmental perspective, certain aspects of EA may be more or less influential in increasing depression vulnerability across childhood and adolescence. Whereas we considered EA as a unitary construct, it has been conceptualized as being multifaceted, consisting

Table 2
Bivariate correlations among mediation model variables.

	1	2	3	4
1. Daughter EA	–			
2. Maternal EA	.35*	–		
3. Daughter depressive symptoms	.70*	.31*	–	
4. Maternal depressive symptoms	.27*	.76*	.30*	–

Daughter EA=AFQ-Y total; Maternal EA=AAQ total (rescaled); Daughter depressive symptoms=MFQ total; Maternal depressive symptoms=BDI-II total.

* $p \leq .001$.

of cognitive, affective, and behavioral components (Boulanger and Hayes, 2010; Chawla and Ostafin, 2007). Although cognitive vulnerabilities to depression, such as dysfunctional attitudes and rumination, have been evidenced in as early as middle childhood (Hankin, 2006), it is possible or even likely that non-cognitive components of EA (i.e., behavioral) may have greater influence for vulnerability at these ages. Use of measures that tap into the multiple facets of EA, such as the Multidimensional Experiential Avoidance Questionnaire (MEAQ; Gámez et al., 2011), may help shed light on this matter.

The fact that adolescent EA mediated the relation between maternal EA and adolescent depressive symptoms highlights the potential role of intergenerational EA in promoting adolescent depressive symptoms. It is well understood that children learn to model their mother's cognitive and behavioral strategies for regulating emotion through social learning. As such, children at risk for depression are likely to demonstrate cognitions, affect, and behaviors similar to those of their depressed mother (Goodman, 2007). For instance, findings have shown high-risk children to exhibit negative self-schemas and attributional styles, greater hopelessness, and poorer emotion regulation strategies as compared to children of non-depressed mothers (Jaenicke et al., 1987; Murray et al., 2001; Silk et al., 2006). Such depressotypic characteristics have been found to longitudinally predict increased depressive symptoms among at-risk children (Morris et al., 2008), and the extent to which EA may be similarly predictive is worthy of investigation.

The acquisition of EA may begin as early as infancy, whereby the child acquires emotion-regulation skills as a function of their mother's response to their own negative emotional states (Vanwoerden et al., 2015). These processes may be further augmented with the child's continued development of language and cognition in conjunction with parenting practices. By pre-school age, parents take an active role in helping their children label and manage their negative emotional states (Gottman et al., 1996). Experimentally-avoidant mothers may have limited capacity in these domains regardless of their history of MDD. High-EA mothers may abruptly end their child's aversive behavior without explanation or ignore the behavior altogether as a means to regulate their own negative internal states when emotionally-charged situations arise, and these parental styles may be internalized by the child (Shea and Coyne, 2011). During parent-child interaction, EA may be particularly high among depressed mothers independent of their degree of dysphoria (Shahar and Herr, 2011). Thus, depressed mothers' EA may influence high-risk children even in the absence of a current maternal major depressive episode.

This study has limitations that must be acknowledged. For one, we included a relatively small group of depressed mother/daughter dyads; however, this is largely a reflection of our strict exclusion criteria paired with the rigor required for recruitment for the employed study design. In investigating EA as a trait marker, it would have been worthwhile to examine whether findings held if we specifically considered mother/daughter dyads that were not currently depressed. In doing so, we could further clarify that EA is transmitted intergenerationally independent of current depression. However, the limited size of the MDD group precluded us from performing such sub-analyses due to lack of power. Given that the present high-risk sample was likely heterogeneous in terms of depression vulnerability, findings are preliminary and in need of replication. Furthermore, while all mothers in the MDD and high-risk groups had a history of MDD some were experiencing current depression while others were not; however, similar proportions in each group were currently depressed. Shared method variance may have influenced results given our reliance on self-report measures (Podsakoff et al., 2003). Importantly, the cross-sectional nature of this study does not allow for causal inferences such that we cannot definitively conclude whether maternal EA has a causal effect on daughter's tendencies to experientially avoid. Rather, as developmental psychopathology literature has taught us, the relations between parent and child functioning often consist of transactional processes with

dynamic outcomes (Cummings et al., 2002). Whether the AAQ and AFQ-Y measure the same psychological construct has been called into question (Schmalz and Murrell, 2010). Thus, there may have been important discrepancies in EA measurement between mothers and daughters. Finally, given that we limited our study of adolescents to females we are unsure how our findings may generalize to boys at risk for MDD.

Limitations notwithstanding, this study had many notable strengths including: the rigor that went into group assignment, its high-risk design, and the fact that it is the first to investigate EA in the vulnerability to initial onset MDD. Moreover, this is the first study to examine the concurrent relation between maternal and adolescent EA as well as the relation between maternal EA and adolescent depressive symptoms.

Given that this study was specific to major depression, future investigations should seek to examine the role of EA in the intergenerational transmission of psychopathology more broadly. In doing so investigators may also consider levels of analysis beyond interview and self-report. For instance, reward-related paradigms have been used to examine the relation between EA neural correlates and performance-based behavioral avoidance in healthy adults (Schlund et al., 2011). Extending similar methodology for a more integrated study of intergenerational EA with putative behavioral phenotypes or endophenotypes may enhance our understanding of vulnerability to various psychopathologies.

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