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Brooding Rumination and Risk for Depressive Disorders in Children of Depressed Mothers

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Abstract The goal of the current study was to examine the role of brooding rumination in children at risk for depression. We found that children of mothers with a history of major depression exhibited higher levels of brooding rumination than did children of mothers with no depression history. Examining potential mechanisms of this risk, we found no evidence for shared genetic influences (*BDNF* or 5-HTTLPR) or modeling of mothers' rumination. However, we did find that children with a history of prior depressive disorders exhibited higher current levels of brooding rumination than children with no depression history. Importantly, children's brooding predicted prospective onsets of new depressive episodes over a 20-month follow-up even when we statistically controlled for depressive symptom levels at the initial assessment, suggesting that the predictive effect of brooding rumination in children was not due simply to co-occurring depressive symptoms.

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According to the response styles theory (Nolen-Hoeksema 1991; Nolen-Hoeksema et al. 2008), a tendency to ruminate in response to a depressed mood (i.e. a ruminative response style) is hypothesized to increase both the duration and severity of depressive reactions. Since its introduction, the response style theory has garnered considerable support for predicting the onset and maintenance of depression in adults (for a review, see Nolen-Hoeksema et al. 2008). Although early work focused on rumination generally, more recent research has focused on two distinct components of rumination, brooding and reflective pondering (e.g., Armey et al. 2009; Schoofs et al. 2010; Treynor et al. 2003). Brooding is defined as "a passive comparison of one's current situation with some unachieved standard" where as reflection is defined as "a purposeful turning inward to engage in cognitive problem-solving to alleviate one's depressive symptoms" (Treynor et al. 2003, p. 256). There is growing evidence that brooding and reflective rumination are distinct constructs and that brooding represents a more maladaptive form of rumination than reflection, with stronger links to depression and suicide attempts (e.g., Armey et al. 2009; Grassia and Gibb 2008, 2009; Schoofs et al. 2010; Treynor et al. 2003; for a review, see Nolen-Hoeksema et al. 2008).

Given the strong support for the role of rumination in depression risk among adults, researchers have begun to examine whether it may also help explain risk for depression among youth. There is now considerable support for the cross-sectional relation between rumination and depressive symptoms among children and adolescents as well as initial evidence that rumination may predict prospective changes in depressive symptoms in this age group (for reviews, see Abela and Hankin 2008; Rood et al. 2009). There is also evidence from one recent study that

rumination predicts the onset of depressive episodes in adolescents (Abela and Hankin 2011). This said, although there is evidence for the two-factor structure of rumination in youth (as with adults), with brooding rumination generally being more strongly related to depressive symptoms than reflection (Burwell, and Shirk 2007; Lopez et al. 2009; Verstraeten et al. 2010), the majority of past research has focused on rumination generally and no study of which we are aware has examined the link between brooding rumination specifically and depressive diagnoses in youth.

When examining risk factors for depression in children, an important question is whether these factors can help to explain why depression is so strongly transmitted across generations. Children of mothers with a history of major depression are 3–4 times more likely to meet criteria for a major depressive disorder (MDD) by early adulthood than are individuals in the general population (for reviews, see Goodman 2007; Hammen 2009). However, the mechanisms by which this risk is conveyed are not well understood, though they likely include genetic, environmental, and psychological influences. Theorists (e.g., Goodman 2007; Goodman and Gotlib 1999) have suggested that the development of a cognitive vulnerability to depression may be a final common pathway in the intergenerational transmission of depression.

In the current study, therefore, we predicted that children of depressed mothers would exhibit significantly higher levels of brooding rumination than children nondepressed mothers. We also tested three potential mechanisms for this relation – genetic influences, modeling of parental cognitions, and cognitive "scarring" from children's prior depression. First, in terms of genetic influences, there is some evidence that the Val66Met polymorphism (rs6265) in the brain-derived neurotrophic factor (*BDNF*) gene may contribute to differences in rumination (Beevers et al. 2009a; Hilt et al. 2007; Juhasz et al. 2011), though the direction of this relation has differed across studies. Specifically, one study found that the presence of the *BDNF* Met allele was associated with *lower* levels of rumination, compared to Val/Val homozygotes, in adolescent girls; however, no relation was observed between *BDNF* and rumination in their mothers until the authors focused specifically on mothers with adult onset depression, among whom the *BDNF* Met allele was associated with *higher* levels of rumination (Hilt et al. 2007). In a separate study of adults with no history of major depression, Met allele carriers exhibited *higher* levels of rumination than Val/Val homozygotes (Beevers et al. 2009a). Most recently, in a large community sample, carriers of the Met allele exhibited *lower* levels of rumination than Val/Val homozygotes (Juhasz et al. 2011). Therefore, although there is some evidence for a relation between *BDNF* genotype and rumination, the direction of this relation is unclear and may depend, in part, on the age or depression history of the sample.

A second potential mechanism is that children may model the depressive cognitions expressed by their parents (see Goodman 2007; Goodman and Gotlib 1999). Thus, for example, a child who sees his or her mother respond to a depressed mood by ruminating may start employing this response style him or herself. Although no study of which we are aware has examined the correlation between mothers' and children's levels of rumination, a number of studies have examined potential modeling effect for other forms of depressive cognitions. These studies, however, have yielded mixed results, with some studies finding evidence for a significant relation between parent and child cognitions, while others find no relation (for reviews, see Abela and Hankin 2008; Gibb and Coles 2005).

A third possibility is that elevated levels of rumination in children of depressed mothers may represent "scarring" effects of earlier depressive episodes. According to the "scar hypothesis" (Lewinsohn et al. 1981), episodes of depression may lead to lasting psychological changes, such as increasingly negative cognitions, which place one at risk for recurrence of depression in the future. Within the context of the intergenerational transmission of depression, one would not expect the magnitude of the effect of depressive episodes on rumination to differ for children with and without a familial history of depression. However, scarring effects should be more common among children of depressed mothers to the extent that they are more vulnerable to early onset depression than children of nondepressed mothers. There is considerable evidence that depression predicts prospective changes in various forms of depressive cognitions, including rumination (e.g., Gibb et al. 2006; Nolen-Hoeksema et al. 1986, 1992; Nolen-Hoeksema et al. 2007; but see Beevers et al. 2007). Within this context, however, an important question is whether elevated levels of rumination merely reflect a consequence or correlate of earlier depression or whether it also predicts the future onset of depressive episodes in currently nondepressed children.

In the current study, we sought to examine each of these questions. We focused specifically on brooding, rather than reflective, rumination given evidence reviewed above that these are distinct forms of rumination and that brooding is more strongly related to depression in children and adults than reflection (e.g., Armey et al. 2009; Burwell, and Shirk 2007; Grassia and Gibb 2008; Lopez et al. 2009; Schoofs et al. 2010; Treynor et al. 2003). We predicted that children of mothers with a history of MDD would exhibit higher levels of brooding rumination than children of mothers with no depression history. We also examined the three potential pathways to rumination highlighted above. First, we examined the role of *BDNF* genotype. Based on previous research, we expected that *BDNF* genotype would predict differences in children's levels of rumination but, given

previous mixed results, we made no hypotheses about the direction of this relation. To test the specificity of any observed results, we also examined a functional polymorphism (5-HTTLPR) in promoter region of the serotonin transporter gene (*SLC6A4*) that has been linked to other forms of information-processing biases including attention and memory biases (e.g., Beevers et al. 2009b; Gibb et al. 2011; Hayden et al. 2008), but does not appear to be related to rumination (Beevers et al. 2007). Given this, we did not expect 5-HTTLPR genotype to predict differences in levels of brooding rumination. Second, we tested the modeling hypothesis that there would be a significant relation between children's and their mothers' levels of rumination. Third, we tested the scar hypothesis. Specifically, to the extent that children of depressed mothers are at risk for early-onset depressive disorders and these disorders leaving lasting effects on children's cognitions, we predicted that children with a history of depression would exhibit higher current levels of rumination than children with no depression history. We also tested the specificity of these potential scarring effects to children's history of depressive versus anxiety and disruptive behavior disorders.

In addition to examining developmental antecedents of rumination, we also tested the predictive validity of children's brooding rumination for the development of new depressive episodes over a 20-month follow-up. To the extent that brooding rumination represents a risk factor for depression in children rather than simply a correlate or consequence of prior depression, it should prospectively predict the onset of new depressive episodes. Finally, we should note that the response styles theory was originally developed to help explain the gender difference in depression (Nolen-Hoeksema 1991). However, although we tested for gender differences and gender moderation in each of our analyses, we made no specific hypotheses given the mixed findings observed in previous studies of children and evidence that stable gender differences in rumination may not develop until adolescence (for a meta-analytic review, see Rood et al. 2009).

Method

Participants

Participants in this study were 100 mothers and their children drawn from the community. To qualify for inclusion in the "depressed" group (n=52), mothers were required to meet criteria for MDD during the child's lifetime according to the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (*DSM-IV*; American Psychiatric Association 1994). To qualify for inclusion in the "nondepressed" group (n=48), mothers were required to have no lifetime diagnosis of any *DSM-IV* mood disorder. Exclusion criteria for both groups included symptoms of schizophrenia, organic mental disorder, alcohol or substance abuse within the last 6 months, or history of bipolar disorder. Children's participation was limited such that no more than one child per mother could participate and all children were between the ages of 8-12 years. If more than one child was available within this age range, one child was chosen at random for participation. The average age of mothers in our sample was 38.56 years (SD=6.66, Range= 26–53) and 88% were Caucasian. The median family income was $50,000–55,000 and, in terms of education level, 45% of the mothers had graduated from college. For the children in our sample, the average age was 9.97 years (SD=1.32), 59% were girls, and 82% were Caucasian. Maternal history of MDD was not significantly related to children's age, sex, or race (Caucasian vs. non-Caucasian). Descriptive statistics for the sample are presented in Table 1.

Measures

The Schedule for Affective Disorders and Schizophrenia-Lifetime Version (SADS-L; Endicott and Spitzer 1978) and the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL; Kaufman et al. 1997) were used to assess for lifetime histories of *DSM*-IV Axis I disorders in mothers and their children, respectively, at the initial assessment. The depression section of the K-SADS was also used to assess for depressive diagnoses among children during the follow-up. Both measures are widely used diagnostic interviews with well-established psychometric properties (Angold 1989; Endicott and Spitzer 1978; Kaufman et al. 1997). Separate interviewers administered the SADS-L and K-SADS-PL and were kept blind of the other's diagnoses in order to prevent biases. For the K-SADS-PL, mothers and children were interviewed separately. As noted above, 52 mothers met criteria for MDD during their child's life (8 met criteria for current MDD). Twenty eight mothers met lifetime criteria for one or more anxiety diorders (social phobia = 18; panic disorder = 7; posttraumatic stress disorder = 6; generalized anxiety disorder = 5; acute stress disorder = 2 [Note: 9 women met criteria for more than one anxiety disorder]). At the initial assessment, 21 children met lifetime criteria for major or minor depression (current MDD = 4, past MDD = 7, past minor depression = 10). Sixteen children met lifetime criteria for an anxiety disorder (separation anxiety disorder = 7; social phobia = 6; obsessive-compulsive disorder = 2; generalized anxiety disorder = 1; panic disorder = 1; posttraumatic stress disorder =1 [Note: 2 children met criteria for more than one anxiety disorder]), and 10 met lifetime criteria for a

Table 1 Descriptive statistics

	Depressed Moms (n=52)	Nondepressed Moms (n=48)	$r_{effect\ size}$
Mom age	39.12 (6.73)	40.04 (6.61)	−0.07
Mom race (% Caucasian)	84.6%	97.7%	−0.11
Child age (years)	10.08 (1.38)	9.85 (1.26)	0.09
Child sex (% female)	55.77%	62.50%	−0.07
Child race (% Caucasian)	76.92%	87.50%	−0.14
Mom Current MDD (% yes)	15.38%	0.00%	0.28**
Mom Anxiety Disorder (% yes)	30.77%	18.75%	0.14
RRS-Brooding	11.90 (3.06)	9.21 (2.33)	0.45**
Child RRS Brooding	10.45 (3.17)	9.26 (2.49)	0.20*
Child *BDNF* (% Met carriers)	34.62%	27.08%	0.08
Child *5-HTTLPR* (% S or L_G carriers)	76.92%	77.08%	−0.00
CDI	7.86 (6.40)	5.49 (5.76)	0.20*
Child Depressive Disorder (% yes) [a]	36.54%	4.17%	0.40**
Child Anxiety Disorder (% yes) [a]	25.00%	6.25%	0.26*
Child Behavior Disorder (% yes) [a]	15.38%	4.17%	0.19
Child Prospective Depressive Disorder during follow-up (% yes)	15.39%	0.00%	0.28**

MDD = Major Depressive Disorder. *BDI-II* = Beck Depression Inventory-II. *CDI* = Children's Depression Inventory. *BDNF* = brain-derived neurotrophic factor gene. *5-HTTLPR* = functional polymorphism in the in promoter region of the serotonin transporter gene (*SLC6A4*). *RRS-Brooding* = Ruminative Response Scale-Brooding Subscale. [a] Lifetime diagnoses assessed at Time 1

* p<0.05. ** p<0.01

disruptive behavior disorder (attention-deficit/hyperactivity disorder = 7; oppositional defiant disorder = 4 [Note: 1 child met criteria for both disorders]). The depression section of the K-SADS was also administered at the follow-up assessment to determine whether children met criteria for a depressive disorder during the follow-up period and, if so, the date of onset. During the follow-up, 8 children met criteria for a new depressive episode (2 with a first onset and 6 with a recurrence, none of whom met criteria for a current episode at the initial assessment). A subset of 20 SADS-L and 20 K-SADS-PL interviews from this sample were coded by a second interviewer and kappa coefficients for diagnoses included in this study were: mother MDD (κ=1.00), mother anxiety disorder (κ=0.78), child MDD (κ=1.00), child any anxiety disorder (κ=0.76), and child any disruptive behavior disorder (κ=1.00).

The brooding subscale from the Ruminative Response Scale (RRS-B; Treynor et al. 2003) was used to assess levels of brooding rumination in children and mothers. The RRS is a self-report questionnaire that asks participants to rate the frequency with which they think or do certain things when they feel sad, down, or depressed (e.g., "Go some place alone to think about your feelings"). The statements are rated on a 4-point Likert-type scale from *almost always* to *almost never*. The brooding subscale is composed of 5 items and has exhibited good reliability and validity in adult (e.g., Grassia and Gibb 2008, 2009; Joormann et al. 2006; Treynor et al. 2003) and youth (e.g., Kuyken et al. 2006; Nolen-Hoeksema et al. 2007; Papadakis et al. 2006) samples. In the current study, the internal consistency of the RRS-Brooding subscale was .62 for children and .77 for mothers.

Genomic DNA was isolated from buccal cells and saliva using a modification of published methods (Lench 1988; Meulenbelt et al. 1995; Spitz et al. 1996; Freeman et al. 1997). The cheeks and gums are rubbed for 20 s with three sterile, cotton-tipped wooden swabs. The swabs are placed in a 50- ml capped polypropylene tube containing lysis buffer (500 μl of 1 MTris–HCl; pH 8.0; 500 μl of 10% sodium docecyl sulfate; and 100 μl of 5 M sodium chloride). The participants then rinse out the mouth vigorously with 10 ml of distilled water for 20 s and this was added to the 50-ml tube. Samples were stored at 4°C until the DNA was extracted. The Val66Met polymorphism (rs6265) was genotyped using Taqman assay C___11592758_10 (Applied Biosystems) using an ABI 7,300 Real time PCR system. We combined the Val/Met and Met/Met groups to form a Met-carrier group (n=31) that was compared to Val homozygotes (n=69). The assay for 5-HTTLPR is a modification of that used by Lesch et al. (1996). The primer sequences are: forward, 5′-GGCGTTGCCGCTCTGAATGC-3′ (fluorescently labeled), and reverse, 5′-GAGGGACT GAGCTGGACAACCAC-3′ with yield products of 484 or 528 bp. Allele sizes are scored by two investigators independently and inconsistencies were reviewed and rerun when necessary. To distinguish between the S, L_A, and L_G

fragments, the PCR fragment was digested with MspI by methods described in Wigg et al. (2006). Consistent with previous research (e.g., Zalsman et al., 2006), three groups of participants were formed based on their genotyping: (a) S′S′: children with two copies of the lower expressing *5-HTTLPR* alleles (SS, SL_G, or L_GL_G), n=26, (b) S′L′: children with one copy of a lower expressing *5-HTTLPR* allele (SL_A or L_GL_A; n=51), and (c) L′L′: children homozygous for the higher expressing L_A allele (L_AL_A; n=23).

Finally, children's symptoms of depression were assessed at the baseline assessment using the Children's Depression Inventory (CDI; Kovacs 1981). Numerous studies have supported the reliability and validity of the CDI (Kovacs 1981, 1985; Smucker et al. 1986) and it exhibited good internal consistency in the current study (α=0.86).

Procedure

Potential participants were recruited from the community through a variety of means (e.g., newspaper and bus ads, flyers). Mothers responding to the recruitment advertisements were initially screened over the phone to determine potential eligibility. Those reporting either significant depressive symptoms during the child's life or no significant lifetime symptoms of depression were invited to participate in the study. Upon arrival at the laboratory, mothers were asked to provide informed consent and children were asked to provide assent to be in the study. Once consent was obtained, the mother was administered the K-SADS-PL interview by a research assistant and the child completed questionnaires in a separate room supervised by a second research assistant. After completing the K-SADS-PL with the mother, the same interviewer then administered the K-SADS-PL to the child. While children were being administered the K-SADS-PL, mothers completed a series of questionnaires and were then administered the SADS-L by a separate interviewer. Participation in this initial assessment took approximately 3 h, which included frequent breaks for children to minimize fatigue effects. Families were invited to participate in a follow-up assessment approximately 20 months after the initial assessment (M=20.25 months, SD=4.89) at which point they were administered the depression section of the K-SADS to assess for the onset of depressive diagnoses since the initial assessment. Families were compensated a total of $100 for their participation in this study.

Results

Of the 100 mother-child pairs participating in the initial assessment, 75 families participated in the follow-up. Given the presence of missing data, we examined whether the data were missing at random, thereby justifying the use of data imputation methods for estimating missing values (cf. Schafer and Graham 2002). Little's missing completely at random (MCAR) test, for which the null hypothesis is that the data are MCAR (Little and Rubin 1987) was non-significant, $\chi^2(872)$= 830.57, p=0.84, supporting the imputation of missing values. Given these results, maximum likelihood estimates of missing data were created and used in all subsequent analyses (see Schafer and Graham 2002). Preliminary analyses were then conducted to determine whether any of the study variables were related to children's age or sex. The only significant finding was that children of mothers with a history of an anxiety disorder were younger than were children of mothers without a history of an anxiety disorder (M=9.52, SD=0.96 versus M=10.12, SD=1.40), $F(98)$=3.98, p=0.05, η_p^2=0.04. There was no significant sex difference in children's levels of brooding rumination, $F(1, 98)$=2.04, p=0.16, η_p^2=0.02, nor was children's brooding rumination significantly correlated with their age, r=0.14, p=0.17.

Next, we tested the hypothesis that children of mothers with a history of MDD would exhibit higher levels of brooding rumination than children of mothers with no depression history. This hypothesis was supported, $F(1, 98)$=4.27, p= 0.04, η_p^2=0.04. The impact of mother MDD on children's rumination also varied as a function of children's degree of exposure to maternal depression such that children of mothers with recurrent MDD during their lives exhibited higher levels of brooding rumination than children of mothers with only a single MDD episode, $F(1, 50)$=4.49, p=0.04, η_p^2=0.08. Similarly, children's levels of brooding rumination were significantly correlated with the total duration of mothers' MDD during their lives, r=0.32, p=0.03, as well as the overall proportion of children's lives that their mothers were in an MDD episode, r=0.36, p=0.01. Importantly, children's levels of brooding rumination were not significantly related to the recency of mothers' MDD (number of years since last episode), r=-0.03, p=0.83, suggesting that the results are not due only to recent exposure to maternal depression. In addition, children's levels of rumination remained significantly related to mothers' MDD characteristics (recurrence, duration, proportion of child's life) when statistically controlling for children's current depressive symptom levels and when omitting children with a current depressive disorder (all ps<0.05) suggesting that the relations are not due simply to current depression in the children.

We then examined the specificity of children's brooding rumination to mothers' histories of MDD versus anxiety disorders. Because families were chosen for inclusion in this study based on mothers' MDD history, we included MDD status (yes versus no) as a covariate in this analysis. Children's levels of brooding rumination were not significantly related to their mothers' history of anxiety disorders, $F(1, 97)$=0.11, p=0.75, η_p^2=0.001.

Next, we examined the three proposed mechanisms for the development of brooding rumination in children of depressed mothers: (i) influence of candidate genes, (ii) modeling of mothers' brooding rumination, and (iii) history of past depressive episodes. Focusing first on potential genetic influences, we found no relation between children's levels of rumination and either their *BDNF*, $F(1, 97)=0.08$, $p=0.78$, $\eta_p^2=0.001$, or 5-HTTLPR, $F(2, 96)=0.38$, $p=0.96$, $\eta_p^2=0.001$, genotypes. Also polymorphisms in these genes did not moderate the link between maternal MDD history and children's levels of brooding rumination (both ps>0.18; $\eta_p^2<0.02$).[1] Second, we also found no support for the modeling hypothesis. Specifically, the relation between mothers' and children's levels of brooding rumination was not significant, $r=0.15$, $p=0.14$, nor did the magnitude of this relation differ significantly for children of mothers with versus without a history of MDD, $F(1, 96)=0.01$, $p=0.92$, $\eta_p^2=0.0001$. In terms of the third potential mechanism, we found that, even after statistically controlling for the influence of mother MDD, children with a history of depressive diagnoses exhibited higher current levels of brooding rumination than those with no depression history, $F(1, 97)=6.13$, $p=0.02$, $\eta_p^2=0.06$. This relation was maintained even after we removed the 4 children who met criteria for current depression, $F(1, 93)=3.98$, $p<0.05$, $\eta_p^2=0.04$. Supporting the specificity of children's brooding rumination to depression versus other disorders, it was not significantly related to children's lifetime diagnoses of anxiety disorders, $F(1, 97)=2.84$, $p=0.10$, $\eta_p^2=0.03$, or disruptive behavior disorders, $F(1, 97)=0.86$, $p=0.36$, $\eta_p^2=0.009$.[2]

Second, survival analyses were used to test the hypothesis that children's levels of brooding rumination would predict prospective onset of depressive episodes over the follow-up. For theses analyses, the 4 children with a current depressive episode at Time 1 were excluded leaving us with 96 children, of whom 8 (8.3%) experienced a depressive episode during the follow-up. As predicted, children's initial levels of brooding rumination predicted depressive disorder onset during the follow-up, *Wald*=7.43, $p=0.006$, odds ratio (*OR*)=1.34. This relation was maintained even after we statistically controlled for children's levels of depressive symptoms at the initial assessment, *Wald*=3.99, $p<0.05$, *OR*=1.26, suggesting that it was not due simply to co-occurring depressive symptoms at Time 1. We then sought to determine whether this effect would be maintained after taking into account the potential influence of mother depression status. Because all new onsets of depression occurred among children of depressed mothers, mother MDD status could not be used as a covariate in our analysis. Therefore, we examined whether the predictive validity of child brooding rumination would be maintained even when we limited our sample to children of depressed mothers (i.e., those children already at heightened risk for depression due to their mothers' depression history) who were not in a depressive episode at the initial assessment. Even among these 49 children, brooding rumination still predicted onset of depressive disorders over the follow-up, *Wald*=4.12, $p=0.04$, *OR*=1.23.

Finally, exploratory analyses were conducted to determine whether any of the links examined were moderated by children's age or sex. The only significant finding was that children's age moderated the link between brooding rumination and their history of depressive disorders, $F(1, 95)=5.72$, $p=0.02$, $\eta_p^2=0.06$, such that the link between history of depressive disorders and higher levels of brooding rumination was significant among relatively older (i.e., 10-12 year olds), $F(1, 56)=9.60$, $p=0.003$, $\eta_p^2=0.15$, but not younger (i.e., 8–9 year olds), $F(1, 38)=0.13$, $p=0.73$, $\eta_p^2=0.003$, children. The relation between brooding rumination and history of depressive disorders in relatively older children in our sample was maintained even after we excluded children with current depressive diagnoses and controlled for their current depressive symptoms, $F(1, 51)=5.92$, $p=0.02$, $\eta_p^2=0.10$, suggesting that it is not due simply to the presence of current depression.[3]

[1] Children's 5-HTTLPR and *BDNF* genotypes were also not significantly related to children's lifetime depressive diagnoses or current depressive symptoms assessed at T1 nor did they predict depression onset during the follow-up

[2] Though not significant, we should note that the relation between children's brooding rumination and their lifetime anxiety disorders was in the opposite direction from what may have been expected, such that children with a history of anxiety disorders had somewhat lower levels of brooding rumination (*M*=8.72) than children with no anxiety disorder history (*M*=10.07).

[3] Although we focus primarily on children's levels of brooding rumination, we should also note that mothers' levels of brooding rumination were also significantly related to their history of MDD, such that mothers with a history of MDD reported significantly higher levels of brooding rumination than mothers with no MDD history, $F(1,98)=24.36$, $p<0.001$, $\eta_p^2=0.20$. Examining mothers with current MDD, past MDD, and no MDD separately, we found that each of the three groups differed significantly, $F(2,97)=17.42$, $p<0.001$, $\eta_p^2=0.26$, with current MDD associated with the highest levels of brooding rumination (*M*=14.41), followed by past MDD (*M*=11.45), and then no MDD (*M*=9.21). In contrast, mothers' levels of brooding rumination were not significantly associated with their history of anxiety disorders, controlling for the influence of MDD history, $F(2,97)=2.72$, $p=0.10$, $\eta_p^2=0.03$. We also examined potential links between brooding rumination and mothers' *BDNF* and 5-HTTLPR genotypes. Although *BDNF* genotype was not significantly related to mothers' levels of brooding rumination, $F(1,97)=1.90$, $p=0.17$, $\eta_p^2=0.02$, brooding was significantly related to 5-HTTLPR genotype, $F(2,96)=3.50$, $p=0.03$, $\eta_p^2=0.07$, with women carrying 2 copies of the 5-HTTLPR S or L_G allele exhibiting significantly higher levels of brooding rumination (*M*=11.82) than women carrying only 1 copy of these alleles (*M*=10.48) or women homozygous for the L_A allele (*M*=9.68). This result, though consistent with other studies examining 5-HTTLPR and information-processing biases, should be interpreted with caution given that it was not replicated in the children, nor was it found in a previous sample of healthy adults (Beevers et al. 2009a).

Discussion

The primary goal of this study was to examine the role of brooding rumination within the context of the intergenerational transmission of depression. Consistent with prediction, children of mothers with a history of depression during their children's lives exhibited significantly higher levels of brooding rumination than children of mothers with no depression history. The magnitude of this relation was directly related to the degree of children's exposure to mothers' depression (i.e., recurrence, duration) and was specific to maternal depression versus exposure to anxiety disorders in their mothers.

We also tested three potential mechanisms for the link between mother MDD and children's rumination: genetic influences, modeling of mothers' rumination, and "scarring" effects of prior depression exposure. Of these, we only found support for the scar hypothesis. Specifically, we found that children with a history of depressive disorders exhibited significantly higher current levels of brooding rumination than children with no depression history. This relation was maintained even when children meeting criteria for a current depressive disorder were excluded and was specific to child depression versus anxiety or disruptive behavior disorders.

In contrast, there was no relation between children's *BDNF* (or 5-HTTLPR) genotype and their levels of brooding rumination. Although our sample size is somewhat small for genetic association studies, the size of the obtained effects (η_p^2=0.001) suggest that the nonsignificant results were not due simply to our sample size. These results contrast with those from previous published studies, which have suggested a link between *BDNF* genotype and levels of rumination (Beevers et al. 2009a; Hilt et al. 2007; Juhasz et al. 2011). However, even these studies have yielded mixed results regarding the direction of the effect, with one study finding that carriers of the met allele have higher levels of rumination than val/val homozygotes, one study finding the reverse, and one study finding that the pattern differed in mothers versus daughters. Given these mixed findings, future research is needed to determine factors (environmental, demographic, or epistatic) that may moderate the relation between *BDNF* genotype and rumination. In addition, though, it is clear that potential intermediate phenotypes including rumination are influenced by a number of genes so research is needed to identify other genetic influences on this cognitive process.

Our data also did not support the modeling hypothesis, given the nonsignificant relation between mothers' and children's levels of brooding rumination. Although this is the first study of which we are aware to examine the relation between mother and child rumination, our results are consistent with studies examining other forms of cognitive vulnerability, which tend to find nonsignificant relations between parents' and children's own cognitive vulnerabilities (for a review, see Gibb and Coles 2005). This said, there is some evidence that children's explanations for the causes of events may be influenced by parents' explanations for the causes of events in the children's lives, suggesting that modeling effects may occur for other types of cognitive styles linked to depression risk (Gibb and Coles 2005).

A second aim of this study was to test the hypothesis that children's brooding rumination would predict the prospective onset of new depressive episodes during the 20-month follow-up. Consistent with our hypothesis, children's levels of brooding rumination predicted the onset of depressive episodes during the follow-up. Importantly, the predictive validity of children's brooding rumination was established among children not currently depressed at the initial assessment and was maintained even after statistically controlling for the influence of baseline depressive symptoms, suggesting that the effect was not due simply to co-occurring depression. Further, the result was maintained even when we limited our analyses to children of depressed mothers, suggesting that brooding rumination increases risk for depression even among this already high-risk group. This is a crucial finding in that even among children at high risk due to maternal depression, individual differences in levels of brooding rumination helped to predict which children would experience a clinically significant depressive episode over a 20-month period. This is the first study of which we are aware to show that brooding rumination predicts prospective onsets of depression in children and extends recent findings from an adolescent sample (Abela and Hankin 2011). Together, these studies provide strong support for the response style theory's (Nolen-Hoeksema 1991; Nolen-Hoeksema et al. 2008) vulnerability hypothesis in youth.

The current results, therefore, are consistent with the hypothesis that early experiences of depression among children of depressed mothers contribute to the development of brooding rumination via scarring effects, which then leave the children vulnerable to additional episodes of depression in the future. However, our conclusions regarding this developmental model must remain tentative because we only assessed children's rumination once and, therefore, cannot determine whether the initial depressive episode predicted changes in children's levels of rumination. Indeed, one study found no evidence for the scar hypothesis in a sample of adolescents (Beevers et al. 2007). Specifically, although adolescents' levels of rumination did increase during a depressive episode, they were elevated prior to depression onset compared to never depressed adolescents, and returned to baseline following depression remission. Therefore, an alternate interpretation of our

results is that children of depressed mothers exhibit relatively stable elevations in brooding rumination prior to the onset of their first depressive episode, which then stay elevated following remission and increase risk for future onsets. This said, there is evidence from other studies for reciprocal relations between rumination and depressive symptoms in adolescents (e.g., Nolen-Hoeksema et al. 2007). Also, to the extent that ruminative tendencies do not develop into a relatively stable response style during adolescence (cf., Hankin 2008; Nolen-Hoeksema et al. 2008), stronger support for the scar hypothesis may be observed among children, particularly children of depressed parents who are at higher risk for early onset first episodes of depression during childhood (for a review, see Hammen 2009). Future research, therefore, is needed to more fully understand the process by which children of depressed mothers develop a ruminative response style themselves.

The current study exhibited a number of strengths, including the specific focus on the brooding subtype of rumination, the prospective design, and the assessment of diagnoses. Despite these strengths, we should also note some limitations as they provide important areas for future research. First, our study was designed to examine the intergenerational transmission of depression and study inclusion was specifically based on the presence versus absence of maternal history of MDD. Although this approach has several benefits including the increased base rates of depressive diagnoses in children and their mothers, it is unclear whether the current results will generalize to a more representative community sample. Second, we used the brooding subscale of the Ruminative Response Scale (RRS-B; Treynor et al. 2003) to assess levels of brooding rumination in mothers and in children rather than using a scale specifically designed for children (e.g., Children's Response Styles Scale; Ziegert and Kistner 2002). Although our use of the RRS-B for both age groups facilitated our comparisons between them, the internal consistency of the RRS-B in children was fairly low (α = 0.62), which could have reduced power for our analyses. Therefore, the results reported may be an underestimate of the true effects of children's levels of brooding. Third, although we were able to establish the specificity of brooding rumination to depression versus other disorders in terms of lifetime diagnoses at the initial assessment, we did not include measures of other disorders during our follow-up assessments. Future studies, therefore, are needed to determine whether rumination may increase prospective risk for disorders other than depression. These studies should seek to include both children and adolescents given evidence from previous research that the predictive validity of rumination may increase with age (see Rood et al. 2009) and also to determine whether rumination can help to account for the dramatic increase in depression observed during adolescence (cf. Abela and Hankin 2011). Finally, the base rate of new depression diagnoses during the follow-up was relatively low, which precluded us from examining first onsets versus recurrences individually. Also, because prospective onsets of depressive disorders during our follow-up were only observed among children of depressed mothers, we were precluded from testing more complex models of risk, such as whether children's rumination mediates or moderates the link between mother MDD history and depression onset in children. Therefore, although the results support the idea that levels of brooding rumination may help to identify which subgroup of children of depressed mothers are at greater risk for depression themselves, future research is needed to determine whether brooding rumination actually mediates or moderates the link between mother and child depression risk.

In summary, the current results support the role of brooding rumination in the intergenerational transmission of depression. They are consistent with the hypothesis that children of depressed mothers exhibit elevated levels of rumination, in part, because of their early experiences of depression themselves. Importantly, this study is the first to show that brooding rumination predicts prospective onset of new depressive episodes, risk that is at least partially independent of current depression. This may help to explain why children of depressed mothers are at such increased risk for depression themselves. If these results are replicated, it would suggest clinical interventions (prevention or treatment) for depressed or at-risk youth may be strengthened by specifically targeting levels of rumination in children much as researchers are starting to do with depressed adults (e.g., Watkins et al. 2009; Watkins and Moberly 2009). This may help to not only reduce the duration of depression among currently depressed children, but also prevent its occurrence among those most at risk.

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