

Chronic and Episodic Stress in Children of Depressed Mothers

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The goal of this study was to examine chronic and episodic stress in children of mothers with and without a history of major depressive disorder (MDD) during the children's lives. Participants were 255 mothers selected according to their history of MDD (present vs. absent during child's life) and their children (age 8–14; 53% girls, 81% Caucasian). Mothers' and children's histories of MDD were assessed using diagnostic interviews, and their depressive symptoms were assessed via self-report measures. Children's levels of chronic and episodic stress were assessed using a semistructured contextual threat interview. Children of mothers with a history of recurrent MDD, compared to single MDD or no depression, experienced more chronic stress within several domains including peers, mother–child relations, and other family member relations as well as greater episodic dependent interpersonal stress. Each of these group differences was maintained after excluding children with a history of MDD themselves and controlling for their current depressive symptoms. However, only the group difference in chronic peer stress was maintained when controlling for mothers' current depression. The results suggest that children exposed to recurrent maternal MDD experience higher levels of both chronic and episodic stress, at least some of which they contribute to themselves (dependent interpersonal stress) and which is at least partially independent of the effects of children's depression. In addition, much of this stress is associated primarily with current depression in the mother, though it appears that chronic peer stress may remain elevated even after the remission of maternal depression.

Countless studies have now documented the increased risk for depression observed among children of depressed mothers (for reviews, see Beardslee, Versage, & Gladstone, 1998; Goodman et al., 2011). However, much less is known about the specific mechanisms through which depression is passed from generation to generation. One of the clearest contributors to depression onset is negative life events (Kessler, 1997; Mazure, 1998). Although typically conceptualized within a unidirectional framework in which negative life

events increase risk for the development of depression, Hammen (1991) proposed in her stress generation theory that depressed individuals may also contribute to the generation of additional stressors in their lives, creating a vicious cycle of depression risk. There is growing support for the stress generation theory with a number of studies now showing that depressed adults, adolescents, and children experience prospective increases in negative life events, particularly negative interpersonal events that are at least partially dependent on the actions of the individual (termed “dependent interpersonal events”; for reviews, see Hammen, 2006; Liu & Alloy, 2010).

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There is also evidence that children of depressed mothers exhibit more stress than children of nondepressed mothers both inside and outside the home (e.g., Burke, 2003; Herr, Hammen, & Brennan, 2007; Hipwell, Murray, Ducournau, & Stein, 2005; Lovejoy, Graczyk, O'Hare, & Neuman, 2000), suggesting a possible mechanism of risk in the intergenerational transmission of depression. Consistent with the stress-generation literature more broadly, this research has focused primarily on interpersonal stress and has generally shown that children of depressed mothers, compared to children of nondepressed mothers, exhibit higher levels of both chronic and episodic interpersonal stress (e.g., Adrian & Hammen, 1993; Carter & Garber, 2011; Gershon et al., 2011; Hammen & Brennan, 2001). Despite the strengths of this research, important questions remain.

First, few studies have focused on different domains of chronic stress, and it is unclear whether the effects may be stronger for some domains of chronic stress versus others (e.g., mother-child relations, relations with other family members, relations with peers, or school behavior). This type of investigation is important because children of depressed mothers may not uniformly experience high levels of stress across all interpersonal domains. For example, a child may get along quite well with their family members but have significant difficulties with their peers. Therefore, specifically examining stress severity within different domains may help to identify specific areas of dysfunction, which would also represent specific targets for intervention. A second key question is whether children of depressed mothers are actually more likely to contribute to the generation of stress in their lives as opposed to simply experiencing more stress that is perhaps due more to the actions of their mothers or to situations outside their own control. This type of specificity is difficult to determine with chronic stressors, which may result from dynamic and complex patterns of influence over time. To examine this issue more precisely, therefore, research is needed on episodic stressors, for which the contextual information can be more precisely defined. Third, research has tended to focus on maternal depression broadly defined, and no study of which we are aware has directly examined levels of chronic or episodic stress among children of depressed mothers as a function of the characteristics or course of maternal depression. This said, we do know that children of mothers with more severe and chronic major depression are at higher risk for psychopathology and exhibit poorer functioning than children of mothers with shorter and less severe depression (for a review, see Hammen, 2009). In addition, there is evidence that women with recurrent depression generate more episodic stress in their lives than women who had experienced only a single episode of depression (Harkness,

Monroe, Simons, & Thase, 1999). However, no studies have examined the effects of maternal depression recurrence on stress generation in offspring. Fourth, it is unclear whether elevated levels of stress in children of depressed mothers are at least partially independent of the effects of depression in the children themselves. We have evidence from one study that currently depressed adolescents who have a depressed mother experienced higher levels of chronic interpersonal and episodic dependent interpersonal events than did depressed adolescents whose mothers were not depressed (Hammen & Brennan, 2001). However, it is not clear whether these differences would be observed among children with no history of depression themselves. This is important because, if observed, it would suggest that stress generation may be a mechanism of risk for the intergenerational transmission of depression. Finally, a key question that has not been addressed in previous research is whether the effects are due to current depression in the mothers or whether they would be observed among children of currently nondepressed mothers who have a history of (recurrent) MDD during the child's life.

The goal of this study, therefore, was to examine levels of chronic and episodic stress in the lives of children of mothers with a history of major depression addressing each of these gaps in the literature. We focused specifically on children 8 to 14 years of age in this study because (a) rates of negative life events, particularly interpersonal events, increase during this time (Ge, Lorenz, Conger, Elder, & Simons, 1994; Rudolph & Hammen, 1999) and (b) because it is before the window of highest risk for depression onset (Hankin et al., 1998), which is important for determining whether heightened levels of chronic and episodic stress in children of depressed mothers is at least partially independent of depression in the children themselves. We predicted that children of mothers with a history of MDD (particularly recurrent MDD) during the children's lives would exhibit higher levels of chronic stress (particularly interpersonal stress) and episodic stress (particularly dependent interpersonal stress) compared to children of never depressed mothers and that these effects would be observed even among children with no current or past depression themselves and even after accounting for the impact of mothers' current depression. Finally, exploratory analyses were conducted to examine the potential moderating effect of child age and/or sex based on evidence that adolescent girls may experience greater levels of interpersonal stress than preadolescent girls or boys of either age (e.g., Rudolph & Hammen, 1999). We should note, however, that these findings were based solely on demographic differences in rates of stress and did not take into account the potential role of maternal depression history. Therefore, although we

tested for moderation effects, we made no specific hypotheses.

METHOD

Participants

A total of 255 children between the ages of 8 and 14 and their biological mothers were recruited from the community for participation in this study. If more than one child was available in the age range, one child was chosen at random for participation. Mothers were selected according to their history of major depressive disorder (MDD; present vs. absent during child's life) as defined by the *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition (DSM-IV)*; American Psychiatric Association, 1994). Mothers in our sample either experienced at least one major depressive episode during their child's life ($n = 129$) or had no lifetime diagnosis of any *DSM-IV* mood disorder and no current Axis I diagnosis ($n = 126$). Mothers were excluded from either group if they exhibited symptoms of schizophrenia, an organic mental disorder, an alcohol or substance dependence within the last 6 months, or a history of bipolar disorder. For children in our sample, the average age was 10.89 years ($SD = 1.91$), 53% were female, and 81% were Caucasian. The average age of mothers in our sample was 40.36 years ($SD = 6.83$, range = 31–55), 87% were Caucasian, and 61.1% had a degree in higher education (associates degree or higher). In addition, 73.5% of the mothers were currently married, and 17.7% were either separated or divorced. The annual family income ranged from \$0–\$5,000 to more than \$115,000, and the median income was \$50,001 to \$55,000.

Measures

The Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1995) 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 current *DSM-IV* Axis I disorders in mothers and their children, respectively. Two separate trained interviewers administered the SCID-I and the K-SADS-PL to mothers and children, respectively. As previously noted, 129 mothers had experienced at least one major depressive episode. Of this group, 58 had experienced recurrent MDD during their child's lifetime and 24 had a current diagnosis of MDD. In terms of children's diagnoses, 18 met lifetime criteria for MDD (two current). To assess interrater reliability, a subset of 20 SCID and K-SADS interviews from this project

were coded by a second interviewer, and kappa coefficients for diagnoses of MDD and anxiety disorders in mothers and children were excellent (all κ s = 1.00).

Mothers' and children's current symptoms of depression were assessed with the Beck's Depressive Inventory–II (BDI–II; Beck, Steer, & Brown, 1996) and the Children's Depression Inventory (CDI; Kovacs, 1981), respectively. Both measures have demonstrated excellent reliability and validity in previous research (e.g., Beck et al., 1996; Kovacs, 1981, 1985; Smucker, Craighead, Craighead, & Green, 1986). In the current study, both the BDI-II and the CDI exhibited good internal consistency ($\alpha = .94$ and $\alpha = .86$, respectively).

Children's levels of chronic and episodic stress were assessed with the UCLA Life Stress Interview for Children (LSI-C; Adrian & Hammen, 1993), which is a semistructured interview modeled after contextual threat interviews (Brown & Harris, 1978). Children and their mothers were interviewed separately about the occurrence of both chronic and acute events occurring in the child's life during the previous 6 months across a variety of domains. Any events reported were then probed to elicit information about the event including its timing and duration and associated circumstances. The domains of chronic stress assessed are academic stress, school behavior, peer relations, mother–child relations, and relations with other family members. After probing for specific factual and contextual information in each domain, interviewers assigned a rating of chronic stress severity ranging from 1 to 5 on behaviorally specific scales for each domain, such that a score of 1 indicated superior functioning and a score of 5 indicated severe stressful conditions. Ratings based on mothers' and children's reports of chronic stress in each domain were averaged and used in all analyses. To assess interrater reliability of chronic stress ratings, a subset of 30 interviews were rated by independent coders and yielded the following intraclass correlations for each of the domains: academic = .83, school behavior = .69, peer = .87, mother–child = .75, other family members = .75. In addition to probing for information about chronic stress, the LSI-C was also used to assess for the occurrence of episodic stress within the previous 6 months. Objective information was obtained about each reported episodic stressor and presented to a team of coders who were blind to any subjective experiences the child may have reported during the interview. The coders assigned each episode of stress a negative impact stress score on a scale of 1 to 5, where a score of 1 was indicative of no stress and a score of 5 indicated that the reported episode was characterized by severe stress impact. The coding team also assigned a dependence score to each event to indicate the extent to which the child contributed to the generation of the stress. The stress dependence scores ranged from 1 to 5, where a

score of 1 indicated that the stressor was completely independent of the child's actions (e.g., a relative passing away) and a score of 5 indicated that the stressor was completely generated by the child (e.g., a child failing a test because he or she didn't study). Episodic events with dependence scores of 3 ("mixed") or higher were considered to be dependent events. Finally, each episodic event was coded as interpersonal or achievement (or both). Using this information, we computed episodic stress scores in three domains—dependent interpersonal events, independent interpersonal events, and non-interpersonal/achievement events—which reflected the sum of the objective impact rating for each event type. To avoid inflating episodic stress scores by inclusion of events rated as having no impact (score = 1), we recoded the objective impact scores from a scale of 1–5 to a scale of 0–4 prior to computing the totals in each domain.

Procedure

Potential participants were recruited from the community through a variety of means (e.g., television, newspaper and bus ads, flyers). Mothers responding to the recruitment advertisements were initially screened over the phone to determine potential eligibility. Upon arrival at the laboratory, mothers were asked to provide informed consent, and children were asked to provide assent to be in the study. Next, the mother was administered the K-SADS-PL by a trained interviewer. 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, the mother was administered the SCID-I by a separate interviewer. Mothers and children were then interviewed separately with the UCLA LSI-C. Finally, mothers completed the BDI-II, and children completed the CDI. The data reported are from the initial assessment of an ongoing longitudinal study, and families were compensated a total of \$75 for their participation in this part of the study. The project was approved by the university's internal review board.

Data Analysis Plan

Analyses for this project followed a three-stage approach. First, we tested demographic differences in each of the forms of chronic and episodic stress (child age, sex, race, family income). Any of the variables demonstrating significant relations with stress were included as covariates in further analyses. Second, we used a series of univariate analyses of variance to test for mother MDD group differences in each form of chronic and episodic stress. Third, we evaluated the robustness the significant relations observed by examining whether they would be maintained after (a) excluding children with a

history of MDD themselves and statistically controlling for children's current depressive symptom levels, and (b) excluding mother-child dyads in which the mother met criteria for MDD in the 6 months prior to the assessment and statistically controlling for mother's current depressive symptom levels.

RESULTS

An initial inspection of the data revealed several variables that exhibited significant skew ($z > 3.29$; cf. Tabachnick & Fidell, 2007). These variables were transformed prior to further analysis to satisfy assumptions of normality (square root: BDI-II, CDI, Chronic Stress: Peer, Chronic Stress: Other Family Members, Episodic Stress: Independent Interpersonal, Episodic Stress: Non-interpersonal; log10: Chronic Stress: Academic; inverse: Chronic Stress: School Behavior, Chronic Stress: Mother-Child, Episodic Stress: Dependent Interpersonal).¹ Next, given the presence of some missing data (BDI-II: 4.7%, CDI: 1.2%, family income: 3.1%), we examined whether the data were missing at random, thereby justifying the use of data imputation methods for estimating missing values (cf. Schafer & Graham, 2002). Using each of the study variables in the model, we found that Little's missing completely at random test (Little & Rubin, 1987), for which the null hypothesis is that the data are missing completely at random, was nonsignificant, $\chi^2(99) = 106.58, p = .28$, supporting the imputation of missing values. Therefore, maximum likelihood estimates of missing data were created and used in all subsequent analyses (see Schafer & Graham, 2002). Means and standard deviations for all study variables are presented in Table 1. To facilitate comparisons with other studies, values presented in the table are based on untransformed data.

We then tested for potential demographic differences in each of the forms of chronic and episodic stress (child age, sex, race, family income). We found that child age was significantly positively correlated with chronic school behavior stress ($r = .13, p = .03$) as well as with episodic noninterpersonal stress ($r = .20, p = .002$). There was also a significant sex difference in chronic school behavior stress, $t(253) = 2.39, p = .02, r_{\text{effect size}} = .15$, with boys reporting more stress than girls. In terms of racial differences, we found that Caucasian children, compared to children from other racial groups, experienced significantly less chronic stress in the domains

¹The only variable that did not reach our criteria for normality ($z < 3.29$) following transformation was episodic noninterpersonal stress ($z = 9.88$ for square root transformation). However, because the square root transformation resulted in a lower skew statistic than any of the other transformations, this is what was used for all analyses.

TABLE 1
Means and Standard Deviations for Study Variables

	<i>Nondepressed Moms^a</i>	<i>Moms With Single MDD^b</i>	<i>Moms With Recurrent MDD^c</i>
Child Age	10.92 (1.81)	11.04 (2.03)	10.62 (1.96)
Child Sex (% Girls)	52.40%	46.50%	51.70%
Child Race (% Caucasian)	90.50%	71.80%	72.40%
Family Income (Median)	\$60,001–65,000	\$35,001–40,000	\$35,001–40,000
BDI-II	3.82 (4.92)	12.74 (10.22)	16.77 (11.22)
Child MDD (% Yes)	2.40%	9.90%	13.80%
CDI	4.92 (5.24)	7.95 (6.44)	7.29 (6.07)
CS: Academic	1.91 (0.67)	2.20 (0.76)	2.03 (0.75)
CS: School Behavior	1.67 (0.43)	1.82 (0.53)	1.94 (0.67)
CS: Peer	2.26 (0.58)	2.44 (0.65)	2.70 (0.64)
CS: Mother–Child	2.07 (0.46)	2.22 (0.64)	2.42 (0.66)
CS: Other Family Members	2.35 (0.54)	2.71 (0.75)	2.81 (0.72)
ES: Dependent Interpersonal	0.53 (0.80)	0.87 (1.05)	1.33 (1.60)
ES: Independent Interpersonal	1.21 (1.25)	1.77 (1.90)	1.85 (2.46)
ES: Noninterpersonal	0.15 (0.30)	0.16 (0.37)	0.22 (0.41)

Note: MDD = major depressive disorder; BDI-II = Beck Depression Inventory-II; CDI = Children's Depression Inventory; CS = chronic stress; ES = episodic stress.

^a*n* = 126.

^b*n* = 71.

^c*n* = 58.

of school behavior, $t(253) = -2.57$, $p = .01$, $r_{\text{effect size}} = -.16$; peer, $t(253) = -2.94$, $p = .004$, $r_{\text{effect size}} = -.18$; mother–child, $t(253) = -2.10$, $p = .04$, $r_{\text{effect size}} = -.13$; and other family members, $t(253) = -2.27$, $p = .02$, $r_{\text{effect size}} = -.14$, as well as less episodic independent interpersonal stress, $t(253) = -3.66$, $p < .001$, $r_{\text{effect size}} = -.22$. Finally, family income was significantly negatively correlated with all five domains of chronic stress: academic, $r = -.30$, $p < .001$; school behavior, $r = -.22$, $p < .001$; peer, $r = -.30$, $p < .001$; mother–child, $r = -.19$, $p = .002$; and other family members, $r = -.40$, $p < .001$. Family income was also significantly negatively correlated with episodic dependent interpersonal, $r = -.18$, $p = .004$; episodic independent interpersonal, $r = -.29$, $p < .001$; and episodic noninterpersonal, $r = -.12$, $p = .05$, stress. Given these results, child age was included as a covariate in analyses for which chronic school behavior stress or episodic noninterpersonal stress served as an independent variable; child sex was included as a covariate in all analyses for which chronic school behavior stress served as the dependent variable; child race (Caucasian vs. all other racial groups) was included as a covariate for all analyses in which chronic stress in the domains of school behavior, peer relations, mother–child relations, and relations with other family members, as well as episodic independent interpersonal stress, served as the dependent variable; and family income was included as a covariate in all analyses for which any of the forms of chronic or episodic stress served as the dependent variable.

Next, we examined the link between maternal MDD history (none vs. single episode vs. recurrent) and

children's levels of chronic and episodic stress, statistically controlling for the influence of the relevant demographic variables as just described. We found significant mother MDD group differences in levels of chronic stress across several domains: peer, $F(2, 250) = 6.00$, $p = .003$, $\eta_p^2 = .05$, mother–child, $F(2, 250) = 4.21$, $p = .02$, $\eta_p^2 = .03$; and other family members, $F(2, 250) = 5.22$, $p = .006$, $\eta_p^2 = .04$. Post hoc tests revealed that children of mothers with recurrent MDD during the children's lives experienced more chronic peer stress than children of mothers with only a single episode of MDD ($p = .006$) or no depression history ($p = .001$), with these latter two groups not differing ($p = .72$). Similar results were observed for chronic stress in mother–child relations, with children of mothers with recurrent MDD experiencing more chronic stress than children of mothers with only a single episode of MDD ($p = .03$) or no depression history ($p = .005$), again with these latter two groups not differing ($p = .66$). Finally, in terms of chronic stress in relationships with other family members, children of mothers with recurrent MDD or a single episode of MDD experienced higher levels of stress than did children of mothers with no history of depression ($ps = .002$ and $.049$, respectively), with the two MDD groups not differing significantly ($p = .25$). In contrast to these significant effects, there were no significant group differences in chronic stress within the academic, $F(2, 251) = 1.45$, $p = .24$, $\eta_p^2 = .01$, or school behavior, $F(2, 248) = 1.76$, $p = .18$, $\eta_p^2 = .01$, domains. For episodic stress, we found significant group differences for dependent interpersonal episodic stress, $F(2, 251) = 4.93$, $p = .008$, $\eta_p^2 = .04$, but not for independent

interpersonal, $F(2, 250) = 0.16$, $p = .85$, $\eta_p^2 = .001$, or noninterpersonal, $F(2, 251) = 1.07$, $p = .35$, $\eta_p^2 = .01$, episodic stress. Post hoc tests revealed that children of mothers with recurrent MDD during the children's lives generated more interpersonal episodic stress than children of mothers with no depression history ($p = .002$), with children of mothers with a single episode of MDD during their lives not differing significantly from either of the other groups ($ps > .10$).

We then conducted follow-up analyses to determine whether the mother MDD group differences were due simply to the presence of depression in the children. We found that, even after excluding children who had a history of MDD themselves ($n = 18$) and statistically controlling for the influence of children's current depressive symptoms, the significant mother MDD group differences were maintained for chronic stress with peers, $F(2, 231) = 6.56$, $p = .002$, $\eta_p^2 = .05$; mother-child, $F(2, 231) = 4.16$, $p = .02$, $\eta_p^2 = .04$; and other family members, $F(2, 231) = 3.55$, $p = .03$, $\eta_p^2 = .03$; as well as episodic dependent interpersonal stress, $F(2, 232) = 3.48$, $p = .03$, $\eta_p^2 = .03$. Next, to determine whether the effects were independent of mothers' current depression, we reconducted the analyses excluding mothers who met criteria for MDD during the 6 months prior to the assessment (i.e., the time point covered by the Life Stress Interview; $n = 40$) and statistically controlling for mothers' current depressive symptoms. The mother MDD group difference was maintained for chronic stress with peers, $F(2, 209) = 4.32$, $p = .02$, $\eta_p^2 = .04$. However, the mother MDD group differences in mother-child, $F(2, 209) = 0.63$, $p = .54$, $\eta_p^2 = .006$, and other family member, $F(2, 209) = 0.17$, $p = .85$, $\eta_p^2 = .002$, chronic stress as well as episodic dependent interpersonal stress, $F(2, 210) = 1.47$, $p = .23$, $\eta_p^2 = .01$, were reduced to nonsignificant.²

Finally, a series of exploratory analyses was conducted to determine whether any of the relations between mother MDD history and children's levels of chronic or episodic stress were moderated by children's age and/or sex. Specifically, the analyses of variance just described were repeated, this time including all two- and three-way interactions among mothers' MDD history, child age, and child sex were examined, with the

same covariates just described included in the models. None of these analyses yielded significant interaction effects including either child age or child sex.

DISCUSSION

The primary goal of the current study was to evaluate levels of chronic and episodic stress across a number of domains experienced by children with and without a history of exposure to maternal depression. We predicted that children of mothers with a history of MDD, particularly children whose mothers experienced recurrent MDD during the children's lives, would experience more severe chronic stress and would generate more episodic stress, particularly within interpersonal domains, than children of mothers with no history of depression. Our secondary goal was to determine whether the differences in chronic and episodic stress would be maintained among children with no depression themselves and even after taking into account mothers' current depressive symptoms and diagnoses. Supporting our hypothesis, we found that children of mothers with a history of recurrent MDD during their child's lifetime experienced more severe chronic stress than children of never depressed mothers within each of the interpersonal domains (peer relations, mother-child relations, and relations with other family members) but not in the academic or school behavior domain. Specifically, children who had been exposed to maternal recurrent MDD experienced more severe chronic stress within the peer and mother-child domains than children of mothers with no depression history or only a single MDD episode during their children's lives. Children of mothers with any history of MDD during their child's lifetime (recurrent or single MDD) experienced more severe chronic stress with other family members. This is consistent with previous work showing that daughters of depressed mothers experience elevated stress within interpersonal domains, but not within the academic domain (Gershon et al., 2011). In terms of episodic stress, we found that children who had been exposed to multiple episodes of maternal MDD generated more interpersonal stress in their lives than those who had never been exposed to maternal depression. This is in line with previous findings that have indicated that children of depressed mothers generate more episodic interpersonal stress in their lives than nondisordered mothers (Adrian & Hammen, 1993; Carter & Garber, 2011). These group differences were observed even among children with no history of depression themselves and even after statistically controlling for their current depressive symptom levels, suggesting that the effects are not simply a consequence of children's own depression in this high risk sample.

²Because this could be considered a very conservative test, we also conducted two additional separate sets of analyses in which we (a) statistically controlled for mothers' current depressive symptom levels in one set of analyses and (b) excluded mothers with current MDD in the second set of analyses. The pattern of results was virtually identical to that just reported. The only difference was that the link between recurrent maternal MDD and children's episodic dependent interpersonal stress was maintained after statistically controlling for mothers' current depressive symptoms in the full sample, $F(2, 250) = 3.24$, $p = .04$, $\eta_p^2 = .03$, but not when excluding mothers with current MDD, $F(2, 211) = 1.91$, $p = .15$, $\eta_p^2 = .02$.

Although the precise mechanisms of action for these effects remain unclear, there is evidence that children of depressed mothers exhibit lower social competence (Ashman, Dawson, & Panagiotides, 2008), lower levels of peer social support (Lewinsohn, Olino, & Klein, 2005), and higher levels of peer conflict (Hipwell et al., 2005). Furthermore, a recent study illustrated that mothers with depressive symptoms offer less cognitive restructuring and more cognitive avoidance suggestions to their children when they are dealing with interpersonal stress (e.g., peer victimization; Monti, Rudolph, & Abaied, 2014). These interpersonal deficits in children of depressed mothers along with the modeling of poor coping strategies for dealing with interpersonal stress may be mechanisms through which maternal depression may lead to interpersonal stress generation in their offspring. Future research is needed to test this hypothesis.

Another potentially important finding that we should highlight is that, of the forms of chronic and episodic stress examined, only the group difference in chronic peer stress was maintained when we excluded mothers with a major depressive episode in the 6 months prior to the assessment (the period covered by the stress interview) and statistically controlled for the influence of mothers' current depressive symptoms. We should note that the effect sizes for the group differences in chronic mother-child and other family member stress and dependent interpersonal stress decreased dramatically when we excluded mothers with current depression, suggesting that the nonsignificant effects are not due simply to a reduction in sample size. Therefore, these results suggest the unique role played by current maternal depression in contributing to chronic stress within the family. They also suggest that elevated levels of stress outside the family (i.e., chronic peer stress) may remain even after the remission of maternal depression. These results suggest potentially important differences in peer stress versus other types of chronic stress in lives of children of depressed mothers, especially those who have been exposed to multiple major depressive episodes. Specifically, whereas other studies have found that levels of interpersonal stress within the family (i.e., with children and other family members) remain elevated even after mothers' depression remits (e.g., Hammen & Brennan, 2002), we found that only the impairments in peer function were maintained once we excluded dyads in which mothers met criteria for MDD in the previous 6 months (the time frame covered by the chronic stress interview) while statistically controlling for the influence of mothers' current symptoms. Although the precise reason for this difference in findings is unclear, it could be due to the difference in sample size (812 women in Hammen & Brennan, 2002, versus 255 in the current study), which allowed greater statistical power in

Hammen and Brennan's (2002) study, or a difference in the age of the samples in the two studies (15 year olds in Hammen & Brennan vs. a mean age of 10.89 years in the current study). Indeed, although we did not find any significant moderating effects of child age in this study, it is clear that familial conflict increases as children age into adolescence. Therefore, future research is needed to determine whether more stable chronic family stress would be observed for adolescents whose mothers have a past history of depression. It is also possible, however, that stress within families is reduced once mothers' depression remits. This is consistent with previous research showing that women's relations with their children and their partners improve as their depression improves (Beach, Jones, & Franklin, 2009; Foster et al., 2008). Another point worth remembering is that although the mothers and their children may both contribute to stress within the family, mothers have less of a direct impact on children's relations with their peers, which may be more due to more stable interpersonal problems in the children themselves. Therefore, any improvement in mothers' functioning would be expected to have a greater impact on stress within the family than on stress outside the family.

Despite these caveats, the current results provide clear evidence to support the hypothesis that difficulties in children's peer relations remain even after mothers' depression remits. These findings suggest that peer chronic stress may be a relatively stable risk factor for depression in children of mothers with a history of MDD. This implies that high-risk children may display stable impairments in their social interactions that contribute to elevated levels of chronic stress within the peer domain. This interpersonal dysfunction within the peer domain may persist even when their mothers are not currently exhibiting symptoms of depression, thereby transferring risk for depression to offspring even after maternal depression has fully remitted. These findings extend interpersonal stress models of intergenerational transmission of depression by highlighting peer chronic stress as a stable risk factor for depression in children of depressed mothers (Hammen, Shih, & Brennan, 2004).

One strength of the current study was that it compared chronic and episodic stress experienced by children of mothers with and without a history of major depression within their child's lifetime. By specifying that mothers in the "high-risk" group had a depressive episode during their child's life, the study was able to ensure that all of these children were directly exposed to depression. As other samples examining maternal depression have included participants with past depressive episodes not limited to their child's lifetime, the current study possessed a higher risk sample. Furthermore, the current study further broke down the higher risk sample into children exposed to a single episode

of depression and children exposed to recurrent depression. However, as the study compared groups solely with and without depression, the study was also unable to draw inferences about whether the differences in stress experienced by the children in these groups was due directly to the exposure to maternal depression itself or if there were other variables influencing these differences. It is possible that other variables such as global impairment in the mothers or maternal criticism play a more salient role in elevated levels of chronic stress in children than depression itself. To address this limitation, future studies should include additional control groups (e.g., mothers with chronic illnesses). Past studies that have included additional control groups in this way have been able to make more specific conclusions about how children of depressed mothers differ from children of nondisordered and chronically ill mothers (Hammen et al., 1987).

In addition to the study's strengths, we should also note its limitations, which provide important areas of future research. The primary limitation is the cross-sectional design of the study, as we could not determine whether mothers' depressive symptoms predicted prospective increases in children's levels of chronic or episodic stress, nor could we determine whether these increases in stress increased children's risk for developing depression themselves. Future longitudinal research is needed to test these hypotheses. Second, as previously noted, although we were able to document elevated rates of chronic and episodic interpersonal stress in children of depressed mothers, future research is needed to focus on the potential mechanisms of these effects.

In summary, this study provides support for the interpersonal stress model of intergenerational transmission of depression by highlighting interpersonal chronic stress, particularly within the peer domain, and episodic interpersonal stress generation as stable risk factors in children of depressed mothers, especially in children who have been exposed to recurrent maternal depression. These results demonstrate that the elevated levels of interpersonal stress observed in children of depressed mothers are not due simply to the presence of depression in the children themselves. The results also highlight the role of current maternal depression and its impact chronic stress within the family (both in terms of mother-child stress and stress with other family members). Important, though, elevated levels of peer stress appeared to be independent of current maternal depression, suggesting that a history of maternal depression may be associated with longer term deficits in children's peer relations. Therefore, in addition to targeting mothers' current depression, intervention efforts in high-risk children should focus specifically on children's relations with their peers.

ACKNOWLEDGMENTS

We thank Ashley Johnson, Lindsey Stone, Sydney Meadows, Michael Van Wie, Andrea Hanley, Katie Burkhouse, Mary Woody, and Anastacia Kudinova for their help in conducting assessments for this project.

FUNDING

This work was supported by the National Institute of Child Health and Human Development [Grant Number HD057066] and National Institute of Mental Health [Grant Number MH098060] awarded to B. E. Gibb.

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