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The Impact of Maternal Depression on Adolescent Adjustment : The Role of Expressed Emotion

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Abstract

The present study evaluated the role of expressed emotion (EE) as a predictor of child symptomatology and functional impairment in a sample of nearly 800 adolescent children of mothers with varying histories of depression or who were nondepressed. Structural equation modeling was used to test the hypothesized associations in half of the sample, and all models were cross-validated on the other half of the study sample. Results indicated that EE criticism and degree of maternal depression both had independent predictive associations with youths' externalizing symptoms and functional impairment. In addition, high EE criticism served as an intervening variable between maternal depression and child functioning (externalizing symptoms and functional impairment). Results are discussed in terms of the mutual effects of depressed mothers and dysfunctional youths on each other.

The purpose of the present study is to address unresolved issues about expressed emotion (EE) as a risk marker for children's negative outcomes and to test the hypothesis that degree of maternal depression predicts maternal EE, which functions as an intervening variable between maternal depression and child disorder and dysfunction.

The construct of negative EE (high EE) has been empirically successful as a predictor of relapse in schizophrenia and other adult psychopathology including mood disorders (e.g., Butzlaff & Hooley, 1998; Coiro & Gottesman, 1996). Although lacking a clear theoretical basis (e.g., Coiro & Gottesman, 1996; Hooley, 1985), EE is thought to reflect the emotional atmosphere of the family. An assumption of the EE construct is that the way parents/partners talk about their psychiatrically impaired relative to an interviewer mirrors the way the relative is treated on a daily basis. Negative attitudes reflecting high EE may serve as stressors that increase the likelihood that the vulnerable individual will experience symptomatology or relapse (Butzlaff & Hooley, 1998; Coiro & Gottesman, 1996). More recent studies have also begun to focus on more specific processes consistent with high EE; specifically, current studies have distinguished between different subtypes of high EE-criticism and emotional overinvolvement (EOI).

EE research has also been extended to child samples and has been identified as a correlate or risk factor for child psychopathology (e.g., Hibbs et al., 1991; Kershner, Cohen, & Coyne, 1996; Schwartz, Dorer, Beardslee, Lavori, & Keller, 1990). Several studies have found that maternal EE criticism is an especially potent predictor of children's disorders (e.g., Asarnow, Tompson, Hamilton, Goldstein, & Guthrie, 1994; Asarnow, Tompson, Woo, & Cantwell, 2001; Goodman, Adamson, Riniti, & Cole, 1994; Hirshfield, Biederman, Brody, Faraone, & Rosenbaum, 1997; Vostanis, Nicholls, & Harrington, 1994).

Despite strong evidence of its association with children's disorders, there are several conceptual and empirical gaps in the use of EE in predicting children's outcomes. Conceptually, as noted, it is generally assumed that high EE is a stressor that provokes increased symptoms in the target. However, the construct of EE has an unclear association with caretaker symptomatology; thus, the EE constructs of criticism or emotional overinvolvement might simply reflect symptomatology in

the caretaker that itself might be the predictor of the target's symptomatology. Although some studies have found no association of EE status with current maternal symptoms or diagnoses (e.g., Kershner, et al., 1996; Stubbe, Zahner, Goldstein, & Leckman, 1993), several other studies did find a significant relationship (e.g., Goodman et al., 1994; Hibbs et al., 1991; Hirshfield et al., 1997). Further, although some studies have found associations of high EE with diverse forms of child psychopathology, the results of other studies (Hirshfield et al., 1997; Stubbe et al., 1993) suggest that the subtypes of EE may be differentially associated with child disorders-with EE EOI related to internalizing disorders and EE criticism related to externalizing disorders. Further study of whether EE independently contributes to negative child outcomes beyond the effects of maternal disorder and, in addition, whether the subtypes of EE contribute differentially to children's internalizing and externalizing symptomatology is clearly warranted.

The conceptual and empirical gaps may be attributed in part to methodological limitations and lack of sufficient research. The EE construct has been primarily investigated with clinical (treated) or high-risk (parental psychopathology) samples, and the sample sizes in most studies have been small. Generalization to community samples needs to be explored, given that treatment-seeking families are commonly loaded with multiple psychopathological, marital, and stress circumstances that might contribute to symptomatology and relapse (Hammen, Rudolph, Weisz, Rao, & Burge, 1999). EE has been studied in samples varying widely in age and has not been adequately tested with sufficiently large samples of boys and girls to determine whether the effects are equal for both genders.

In addition to evaluating children's symptomatology associated with parental EE, researchers may also find impairment in functioning an important outcome to study. It is possible that living in a high EE family reduces exposure to certain developmental experiences and impedes the acquisition of appropriate skills such as functioning in social and academic roles. It is conceivable that even in the absence of diagnosable psychopathology, children in high EE environments might display impaired role performance that portends subsequent maladjustment. Few studies have examined the effects of maternal EE on aspects of child role functioning other than symptomatology (Asarnow et al., 2001; Seifer, Sameroff, Baldwin, & Baldwin, 1992). Research on children's outcomes needs to expand to include a focus not just on disorder and diagnoses but also on performance in relevant roles (e.g., Angold, Costello, Farmer, Burns, & Erkanli, 1999).

The present study attempted to address a number of these unresolved empirical and conceptual issues in the context of a large community study of adolescent children of mothers with varying histories of depression or who were nondepressed. Children of depressed parents are, of course, a group known to be at high risk for the development of depressive and other disorders (see Beardslee, Versage, & Gladstone, 1998, for a review). It is often hypothesized that a key mechanism of risk is maladaptive parenting. A number of studies have demonstrated negative and problematic mother-child interactions between depressed women and their children compared with those of nondepressed mothers (e.g., Cummings & Davies, 1994; Goodman & Gotlib, 1999; Kaslow, Deering, & Racusin, 1994; NICHD Early Child Care Research Network, 1999). However, most studies have been descriptive, mainly demonstrating that depressed mothers interact with their children in a relatively negative manner. These studies suggest that when depressed, mothers' parenting is relatively impaired and generally reflects criticism and/or rejection or withdrawal, inconsistency, and disengagement-and that such qualities have adverse effects on children. Although we generally concur with the view that depression disrupts parenting, we believe that intimate relationships for many depressed women are impaired even when they are not specifically in a depressive episode (e.g., Hammen & Brennan, 2003). Children's long-term exposure to critical or disengaged mothers is highly likely to result in problems of emotional and behavioral regulation.

To date, few studies have actually linked the quality of parent-child relationships to outcomes in children. Some multivariable studies have evaluated predictors of children's outcomes, but not all have statistically tested whether parenting quality mediates the effect of degree of parental depression on youths' outcomes or whether depression is an independent predictor (e.g., Canino, Bird, Rudio-Stipec, Bravo, & Alegria, 1990; Fendrich, Warner, & Weissman, 1990; Goodman, Brogan, Lynch, & Fielding, 1993; Harnish, Dodge, & Valente, 1995; Warner, Mufson, & Weissman, 1995).

The present study proposes to test several hypotheses using structural equation modeling

(SEM) in a community sample of over 800 women varying in history of depression and their 15-year-old children. The sample was sufficiently large to conduct analyses on an estimation sample and a cross-validation sample to increase confidence in the reliability of the results. On the basis of findings of prior studies of EE and of children of depressed mothers, the following hypotheses were tested:

Degree of maternal depression has a predictive association with EE (both subtypes, criticism and EOI). EE has an independent association with child outcome beyond any effects due to the degree of maternal depression. We specifically predicted that subtypes of EE have differential associations: EE criticism would be related to externalizing symptoms, whereas EE EOI would be specifically associated with internalizing symptoms. Both subtypes of EE would be related to child functional impairment. In addition to independent effects, EE serves as an intervening variable in the association between degree of maternal depression and youths' outcomes. We also predict that the same structural models apply for boys and girls.

Method

Participants

Women and their 15-year-old children were selected to represent families with mothers who had a probable history of depression (varying degrees of severity and chronicity) or who were nondepressed. These adolescents and their mothers were drawn from a large birth-cohort study, the Mater-University of Queensland Study of Pregnancy (MUSP), which investigated children's physical, cognitive, and psychological health at age 5 as a function of pregnancy, obstetric conditions, birth weight, and psychosocial conditions. The current study used information collected from the original MUSP study to identify women who were likely to vary in their histories of depression. The investigators' earliest access to the original MUSP birth-cohort sample was when the children were 15 years old.

The initial MUSP birth-cohort sample consisted of 7,775 mothers and their children born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital in Brisbane, Queensland, Australia (Keeping et al., 1989). When the youths were 13 years old, 5,277 (68%) of the original sample had been retained. From these individuals, 991 families were targeted for inclusion (using a sampling algorithm that is detailed later) in the present study at age 15. A final sample of 816 families was located and consented to participate in the present study.

The goal of the present investigators (Constance Hammen and Patricia A. Brennan) was to obtain a sample of women with histories of depression that varied in severity and frequency, along with a nondepressed sample. The mothers in the MUSP study had reported depressive symptoms on the Bedford-Foulds Delusions-Symptoms-States Inventory (DSSI; Bedford & Foulds, 1977, 1978; see *DSSI* section) at four assessment periods: at their first clinic visit (mean gestation 18 weeks), when their child was born (3-4 days after delivery), when their child was 6 months old, and when their child was 5 years of age. Using the DSSI and a sampling algorithm, we targeted women who were likely to have varied depression histories – our intent was to obtain a sample with the greatest differences in maternal depression. Specifically, women were targeted who reported severe depression at two or more times; severe depression only once; moderate depression two or more times but never severe; and low depression at all assessment periods. Again, the point of this selection algorithm was simply to include varied profiles of depression to yield continuous variables of severity and chronicity (later measured using the Structured Clinical Interview for *DSM-IV;* SCID-IV; Spitzer, Williams, Gibbon, & First, 1995; described in the *SCID-IV* section). Of the 991 recruited, 816 consented and were included (82%); of those not included, 68 families could not be located, 103 declined to participate in this wave, 3 included a child with a hearing or visual impairment that precluded participation, and 1 child had died. Children in this subsample *(n* = 816) were not significantly different from the original birth cohort in terms of gender, $\chi^2(1, N = 816) = 0.53$, $p = .48$; income measured in dollars, $t(7147) = 0.81$, $p = .42$; or mother's education measured in years, $t(7612) = 1.70$, $p = .09$.

The present sample included 414 boys and 402 girls, mean age 15 years, 2 months (range: 14-16 years, 95.4% were within 2 months of age 15, *SD* = 0.29). The overall sample included 92% Caucasians; median family income was in the level of Australian middle- and working-class socioeconomic status (AU $35,000-$45,000; approximate United States equivalent of $19,600-

$25,300), median mothers' education was Grade 10 (approximately equivalent to a U.S. high school graduate), and the mothers' mean age at the time of the youths' 15-year follow-up was 41 years. Mothers' marital status included 76.8% who were currently married or cohabiting, and overall, 64.8% were currently married to the biological father of the target child.

Procedure

Interviews were conducted in the family home at a time when both the mother and child were available. Interviewers were clinically trained advanced graduate students in psychology who were blind to the mother's current depression status and history. A team of two interviewers conducted confidential parent and child interviews separately and privately. Between interviews, the participants also completed a battery of questionnaires as noted below. The mother and child gave written informed consent (assent), and were paid for their participation, which lasted approximately 3.5 hr.

Maternal Measures To better reflect the enormous heterogeneity of depression, six depression variables served as indicators of a latent variable, maternal depression, that encompassed facets of both past maternal depression and current depression as a comprehensive variable for testing the proposed model. It was reasonable to consider past and current depression together as one latent factor because of the large covariance that existed between the factors (i.e., history of depression and current depression, r =.93).

SCID-IV Current and lifetime depressive diagnoses and significant depressive symptomatology were assessed using the SCID-IV. One third (33%) of mothers met criteria for at least one current or past major depressive episode. Four percent had significant current or past depressive symptoms. Interrater reliability of SCID-IV depression diagnoses for the current project was based on a random sample of 52 cases; weighted kappa for current depressive diagnostic status was.85 and weighted kappa for lifetime depressive diagnostic status was .81.

Indices of depressive status obtained from the SCID-IV included ratings of current and past severity, defined on a 0-2 scale: *0 (no symptoms), 1 (significant subsyndromal symptoms),* and 2 *(major depressive episode);* indices of chronicity included the number of previous major depressive episodes or significant subsyndromal episodes and the total duration of previous diagnoses and/or symptoms of depression.

DSSI Maternal history of severe depressive symptoms was also measured using the Depression Scale of the DSSI. The DSSI Depression Scale is a seven-item, self-report screening measure on which each item is rated on a 1-5 scale. The DSSI is similar to other depressive screening measures and contains some overlapping items with the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1978) and the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). As part of the MUSP study, mothers had been administered the DSSI Depression scale four times: during pregnancy, immediately (3-4 days) after the birth of her child, when the child was 6 months old, and when the child was 5 years old. The number of depressive symptoms endorsed by the mother at each of these administrations was summed to obtain a total history-of-depression severity score.

BDI Current maternal depressed mood was assessed using the BDI, a 21-item self-report questionnaire designed to measure the severity of depressive symptoms in adults. The BDI is well validated as a measure of depression severity and has high internal consistency, with a mean coefficient alpha of.81 (Beck, Steer, & Garbin, 1988).

Maternal EE The Five-Minute Speech Sample (FMSS; Magana et al., 1986) was used to measure maternal EE. In the FMSS, a mother is asked to speak for 5 min into a taperecorder, without interruption, about her child and how they get along together. Raters, who were blind to all other information about the mothers and their children, scored the recordings. The FMSS was coded

according to the criteria developed by Magana et al. (1986). Using this coding system, raters base a categorical rating of high EE on a high score on either of two dimensions: criticism or EOI. A speech sample is scored as high EE on the criticism dimension if mothers express a negative initial statement, negative relationship, or one or more criticisms as defined by the FMSS coding scheme. A high EE rating on the EOI dimension is given if the mother expresses self-sacrificing and/or overprotective behavior, displays excessive emotion (e.g., crying), or states five or more positive remarks combined with a strong statement of affection or excessive detail about past events. Mothers who do not meet the above criteria are categorized as low EE. By these criteria, 54 mothers were rated as high EE EOI and 96 mothers were rated as high EE critical.

However, it is possible that using the traditional criteria for low and for high EE (primarily developed for use with adult children) may obscure important information when used with children. It is unknown whether children are more sensitive to lower levels of EE criticism or EOI. Therefore, borderline high EE, which is traditionally included in the low-EE category, was also coded. Thus, EE ratings (for both criticism and EOI) in this study were based on a 0-3 scale, on which 0 = *low EE, 1 = borderline-high EE,* and 2 = *high EE.* Mothers were rated as borderline-high critical if they expressed dissatisfaction with their child but the dissatisfaction was not extreme enough to be rated as a criticism (Magana et al., 1986). In addition, mothers were rated as borderline-high EOI if they exhibited moderate levels of self-sacrificing and/or overprotective behavior or if they expressed any one of the following: excessive details about the past, statements of love or willingness to do anything for the child, or exaggerated praise (Magana et al., 1986).

Raters for this study were trained by the research group (Magana et al., 1986) that developed the measure. Following training, a reliability study compared interrater reliability of the raters with an expert from the research group that developed the instrument; weighted kappa for EE criticism ranged from.69 – .79 *(Mdn =.70),* and weighted kappa for EE-EOI ranged from.72 – .91 *(Mdn = .74).*

Child Measures

Analyses included latent variables with three measured variables each for internalizing and externalizing symptoms based on interviewer, parent, and child reports as noted later. The broadband constructs were used to construct meaningful latent variables.

Schedule for Affective Disorder and Schizophrenia for School-Age Children (Version 5; KSADS) The K-SADS (Orvaschel, 1995) was used to obtain systematic diagnoses of current child disorders. Interview data collected with the child were supplemented with information obtained in a separate interview with the mother. A research team made a final best estimate diagnosis based on all available information concerning the child's symptoms and impairment. All interviewers were blind to maternal diagnostic status and EE ratings. A second interviewer rated tape-recorded K-SADS interviews of 75 children; mean kappa for current diagnostic status (across the various diagnoses) was.78 (range: .67 to 1.0).

The total number of internalizing diagnoses and externalizing diagnoses for which a child met criteria was computed. Diagnoses included in the internalizing category were major depression, dysthymia, eating disorders, and anxiety disorders. Diagnoses included in the externalizing category were conduct disorder and oppositional defiant disorder. Attention deficit/hyperactivity disorder was not included because this disorder does not load clearly on either externalizing or internalizing factors (Achenbach, 1991). On the basis of the K-SADS interview, the average number of externalizing disorders per child was.04 (range: 0-1) 4.8% of the children had at least one externalizing disorder. The average number of internalizing disorders per child was 0.14 (range: 0-4)- with 10.5% of the children meeting diagnostic criteria for one or more internalizing disorders.

Child Behavior Checklist (CBCL) The CBCL (Achenbach, 1991) is a standardized parent-report measure that lists child behavior problems that mothers rate on a 3-point scale as 0 *(not true), 1 (somewhat/sometimes true),* or 2 *(very/often true).* Items were summed to obtain a total score for internalizing symptoms (i.e., withdrawn, somatic complaints, and anxiety/depression) and externalizing symptoms (i.e., delinquent and aggressive behavior).

Youth Self-Report Form of the CBCL (YSR) The YSR (Achenbach, 1991) is analogous to the CBCL that is administered to mothers. The YSR has been found to be a reliable and valid measure of symptomatology for children over age 11 (Achenbach, 1991).

Children's functional impairment Children's level of impairment in major life domains was measured using the chronic stress interview developed by Hammen and colleagues (Hammen, Adrian, et al., 1987; Hammen, Gordon, et al., 1987). The interview was designed to assess the level of a child's general functional impairment experienced in several domains, including the following: close friendships, social life, academic performance, and school behavior. An additional scale, relationship with family members, was not included in the present analyses.

On the basis of information provided by the participant regarding the past 6 months, each area was systematically assessed and rated by the interviewer on a 5-point scale of impairment, ranging from 1 *(optimal circumstances)* to 5 *(the most stressful and impaired circumstances).* Interviewers were blind to all diagnostic and EE information. A composite general-functioning score was obtained by adding impairment levels across the various content domains. The mean intraclass correlation coefficient (SPSS two-way mixed effects model; judges were considered fixed effects, participants were considered random effects, design was completely crossed) for these child functioning variables was.80 *(n* = 48; range:.63 social life to.94 academic performance).

Analysis of Assumptions and Estimation Methods

Because the software used for our SEM analyses (EQS; Bentler, 2002) requires that there be no missing data, cases were discarded if an entire instrument or procedure was not available. Accordingly, 74 cases were not included because of the following missing items: FMSS *(n* = 41, mainly because of equipment failure or inaudible tapes), BDI *(n = 23),* CBCL *(n = 21),* YSR *(n* = 8), mother depression duration *(n* = 2), mother chronic stress *(n = 2),* and youth functional impairment *(n* = 1; some dyads did not complete multiple measures). The excluded group *(n = 74)* did not significantly differ from the individuals in the remaining sample *(n* = 742) on any demographic variables (ethnicity, child gender, marital status, family income, or maternal employment) or any of the measured variables. In addition, 3 mothers were eliminated from the analyses because they had extremely high scores for the duration of past depressive episodes variable (extremely high univariate outliers; *z*s = 6.9, 10.5, and 17.6, $p < .01$). The resulting exclusion group *(n* = 77) did not significantly differ from the inclusion group *(n* = 739) on any demographic variables or clinical variables except duration of depression. The means and standard deviations for all study variables are presented in Table 1 and correlations among variables are presented in Table 2.

Because of univariate and multivariate nonnormality of the data, estimation methods were used that account for nonnormality (maximum likelihood [ML] robust; Byrne, 1994). ML is used with continuous data. However, there is uncertainty as to whether the low, borderline, and high dimensions of EE are linear. Given this, we also estimated the models considering EE as categorical using the arbitrary distribution generalized least squares method (AGLS), which allows analysis of categorical variables. Fit indices were similar across the ML and AGLS methods, and the results were the same in terms of significance and magnitude of effect. Given that results were consistent across both methods (when EE was defined as continuous or categorical), we have opted to report the ML results because this method is able to adjust for nonnormal data. Robust statistics are not available for the AGLS method, and it has been found that ML robust statistics perform better than uncorrected AGLS statistics when the normal distribution assumption is violated (Bentler, 2002).

TABLE 1
Means and Standard Deviations for Measured Variables: Total Sample

Variable	*M*	*SD*
Child		
CBCL, externalizing symptoms	8.11	8.38
CBCL, internalizing symptoms	6.46	6.56
Functional impairment	9.36	1.84
K-SADS no. of diagnoses, externalizing	0.04	0.21
K-SADS no. of diagnoses, internalizing	0.14	0.45
YSR, externalizing symptoms	14.63	9.04
YSR, internalizing symptoms	11.22	8.52
Mother		
BDI	6.94	7.40
Chronic stress	12.11	1.97
DSSI	11.62	3.01
EE, criticism	0.40	0.69
EE, emotional overinvolvement	0.45	0.62
Major depression, current severity	0.12	0.44
Major depression, lifetime maximum severity	0.70	0.93
Major depression, total duration (months)	4.14	10.19
Major depression, total no. episodes	0.68	1.09

Note. For all questionnaire measures (i.e., Child Behavior Checklist [CBCL], Youth Self-Report Form of the CBCL [YSR], Beck Depression Inventory [BDI], Delusions-Symptoms-States Inventory [DSSI]), the problem of missing data was reduced by using the mean for all items answered, rather than using the sum of all items. However, to be consistent with standard reporting practices regarding these measures, the values in the table above are the mean item score and standard deviation multiplied by the number of items on the measure. K-SADS = Schedule for Affective Disorder and Schizophrenia for School-Age Children (version 5); EE = expressed emotion.

TABLE 2
Correlations of Measured Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. CBCL, Externalizing	—														
2. CBCL, Internalizing	.655**	—													
3. Child functional impairment	.455**	.343**	—												
4. K-SADS, Externalizing	.542**	.364**	.384**	—											
5. K-SADS, Internalizing	.121**	.256**	.224**	.125**	—										
6. YSR, Externalizing	.436**	.239**	.373**	.299**	.194**	—									
7. YSR, Internalizing	.196**	.296**	.240**	.104**	.264**	.591**	—								
8. BDI	.290**	.357**	.150**	.162**	.136**	.129**	.100**	—							
9. DSSI	.230**	.252**	.145**	.145**	.024	.099**	.051	.374**	—						
10. EE, criticism	.344**	.162**	.195**	.188**	.109**	.150**	.026	.151**	.089**	—					
11. EE, EOI	−.079*	.018	−.040	−.061	.078*	−.090*	−.007	.071*	−.025	−.076*	—				
12. MDE, current severity	.161**	.116**	.104**	.120**	.049	.088*	.034	.292**	.132**	.074*	.013	—			
13. MDE, lifetime severity	.158**	.157**	.122**	.092*	.136**	.098**	.038	.258**	.213**	.091*	.029	.312**	—		
14. MDE, total duration	.080*	.084*	.036	.018	.061	.014	−.021	.129**	.126**	.055	.008	.166**	.541**	—	
15. MDE, total episodes	.164**	.191**	.146**	.107**	.116**	.090*	.025	.234**	.211**	.084*	.032	.286**	.826**	.616**	—

Note. CBCL = Child Behavior Checklist; K-SADS = Schedule for Affective Disorder and Schizophrenia for School-Age Children (version 5); YSR = Youth Self-Report Form of the CBCL; BDI = Beck Depression Inventory; DSSI = Delusions-Symptoms-States Inventory; EE = expressed emotion; EOI = emotional overinvolvement; MDE = major depressive episode. * $p < .05$. ** $p < .01$.

Three test statistics are reported for the models: the Satorra-Bentler scaled chi-square (S-B χ^2), the comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA; Browne & Cudeck, 1993; Hu, Bentler, & Kano, 1992; Satorra & Bentler, 1988; Ullman, 2001). It should be noted that with large sample sizes, trivial differences between the matrices are often significant (as represented by the S-B χ^2). Thus, two other test statistics were also evaluated – a comparison-based fit index (CFI) and a residual-based fit index (RMSEA). The CFI compares the fit of the estimated model to the independence model (values range from 0 to 1). CFI values of .95 or higher are accepted as indicating a good-fitting model (Hu & Bentler, 1999). RMSEA compares the fit of the estimated model to the perfect model (values range from 0 to 1). For the RMSEA, values .05 or lower are accepted as indicating good model fit (Browne & Cudeck, 1993). Model modification, to improve model fit, was evaluated through the Lagrange multiplier test (LM).

Cross-Validation

Given novel hypotheses and variables, we undertook a cross-validation approach to enhance the reliability of results (as a test of internal validity) and reduce the chance of Type I error (Shaddish, Cook, & Campbell, 2002). After randomly splitting the study sample, one half of the sample was used to conduct initial tests of the associations and make any necessary model modifications, and the other half was used to formally test (and cross-validate) the proposed associations. These two samples did not significantly differ from each other on any demographic or measured variables. Fit indices are presented for each sample. However, for brevity in reporting, path coefficients are presented for only the cross-validation sample.

Results

Analysis Overview

When SEM is used, hypotheses are tested at the level of the construct rather than at the level of a single measured variable-thereby reducing the effect of measurement error associated with specific instruments. SEM techniques also allow relationships among sets of constructs to be examined, and this technique can control for rater bias. Rater bias is the systematic tendency of raters to judge that if some symptoms are present, other symptoms (or aspects of a disorder) are also present (Garber, Quiggle, Panak, & Dodge, 1991). Rater bias may be present especially when measured variables are drawn from the same individual reporting on the same measure at the same time point. Several indicators in the study met these criteria. Therefore, residuals were correlated for maternal past depression duration, past severity of depression, and past number of depressive episodes (all mothers' report on the SCID-IV of past depression). In addition, correlated residual paths were added for mothers' report of child internalizing and externalizing symptoms on the CBCL and for children's report of internalizing and externalizing symptoms on the YSR.

Before addressing the specific study hypotheses, we needed to assess the fit of the proposed model. Our hypothesized model (which includes both direct and indirect effects) was tested first. To determine whether this model was the best fitting and most parsimonious model, we subsequently compared this overall model with two other models-one which contained only direct effects and the other which contained only indirect effects (see alternative models in *Model Fit in Modification Sample* section). The best-fitting model was retained and then tested with the cross-validation sample. Thus, study hypotheses were tested with both the model modification and cross-validation samples. Because similar results were obtained, study hypotheses are addressed using the results from the cross-validation sample.

Model Fit in Modification Sample

Figure 1 depicts the measurement and structural portions of the overall model-the predicted relations between the maternal depression factor, EE variables, and child outcomes. The figure shows that there were six indicators of the maternal depression factor and three indicators for both the child externalizing and internalizing symptoms factors. We predicted that maternal depression would be

related to both subtypes of EE as well as related to all types of child outcome (i.e., child functional impairment and symptomatology). Furthermore, we hypothesized predictive paths from the EE subtypes to specific types of symptomatology on the basis of the results of previous studies finding that EE criticism was differentially related to externalizing disorders and EE EOI was related to internalizing disorders (Hirshfield et al., 1997; Stubbe et al., 1993). We predicted that both subtypes of EE would be related to child functional impairment. A residual covariance was predicted between the subtypes of EE. Residual covariances were also predicted between the factors of internalizing and externalizing symptoms because previous research has found a strong association among these types of disorders in children (Garber et al., 1991; Weiss & Weisz, 1988). Residual covariances were also predicted between both types of child symptomatology and child functional impairment. It should be emphasized that this overall model includes *both* direct and indirect effects.

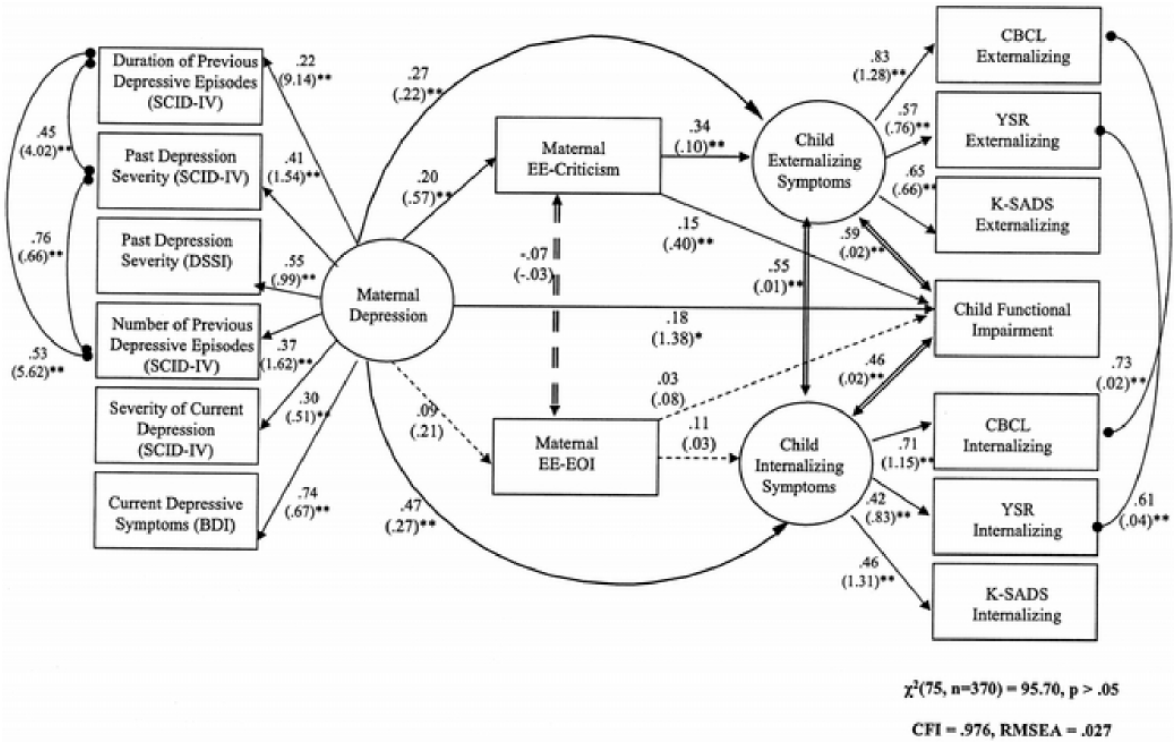

Figure 1. Effects of expressed emotion (EE) and maternal depression on child symptoms and functioning. Circles represent latent variables and rectangles represent measured variables. Single-arrow lines depict direct effects. Broken lines depict a hypothesized relationship that was nonsignificant. Absence of a line implies no direct effect was hypothesized. Double lines with double arrows represent a covariation between residuals. Lines with circles instead of arrows on the ends indicate the covariance of residuals to control for rater bias. Residuals were estimated, but not included in the figure. Unstandardized values are indicated in the parentheses. SCID–IV = Structured Clinical Interview for *DSM–IV;* CBCL = Child Behavior Checklist; YSR = Youth Self-Report Form of the CBCL; DSSI = Delusions–Symptoms–States Inventory; K-SADS = Schedule for Affective Disorder and Schizophrenia for School-Age Children (Version 5); EOI = emotional overinvolvement; BDI = Beck Depression Inventory; CFI = comparative fit index; RMSEA = root-mean-square error of approximation. **p* < .05. ***p* < .01.

Test statistics indicated a good fit with the data: S-B $\chi^2(75, N = 369) = 113.44$, $p < .05$; CFI =.962; and RMSEA =.037. The LM test did not suggest any modifications that would significantly improve the original model.

To more fully determine whether the overall model best represented the data and was the most parsimonious (good-fitting) model possible, we compared it with alternative models that contained only direct or only indirect effects. On the direct effects model (i.e., no mediation), the paths between

maternal depression and EE (both criticism and EOI) were fixed to zero: S-B χ^2 (77, N = 369) = 104.20, p <.05; CFI =.969; RMSEA =.031. A chi-square difference test was then conducted following the procedures outlined by Satorra (2000) to determine whether the constrained model (direct effects only) was significantly different from the full model (direct and indirect effects). Constraining these paths to zero significantly degraded the model, S-B $\chi^2_{difference}$(2, N = 369) = 7.57, p <.05. This indicated that the overall model was significantly better than one that only included direct effects.

Another model was run that only included indirect effects (a full mediation model). In this model, the direct paths from maternal depression to child externalizing symptoms, child internalizing symptoms, and child functional impairment were fixed to zero, S-B χ^2(78, N = 369) = 125.87, p <.05; CFI =.945; RMSEA =.041. A chi-square difference test between this model and the overall model (with both direct and indirect effects) was again significant, SB $\chi^2_{difference}$(2, N = 369) = 71.74, p <.05, indicating that EE is not a full mediator of the relationship between maternal depression and child symptoms and functional impairment. Thus, the overall model was retained, as it provided a significantly better fit with the data than a model containing only direct or indirect effects.

It was also possible that the overall model would fit differently if the factor of maternal depression was composed solely of indicators of current depressive symptoms or history of depression. These additional models were run. Analyses indicated that similar structural models applied (all paths were consistent with the overall model currently presented) when the construct of maternal depression was composed solely of indicators of current depressive symptoms or history of depression. Because there were no significant differences between these models and because of the large covariance present between the factors of history of depression and current depression (see *Maternal Measures* section), the initial factor of maternal depression was retained (representing both history and current symptoms).

Finally, although we hypothesized specific paths from EE criticism to externalizing symptoms and from EE EOI to internalizing symptoms on the basis of results from previous studies (Hirshfield et al., 1997; Stubbe et al., 1993), the general literature on children of depressed mothers, as well as other EE studies (e.g., Asarnow et al., 2001), also suggests a link between criticism and internalizing disorders. Therefore, another exploratory analysis was conducted where additional paths were added from EE criticism to child internalizing disorders and from EE EOI to child externalizing disorders. When these paths were added, the model retained adequate fit. Results indicated that EE criticism continued to have a significant association with externalizing symptoms but a nonsignificant effect on internalizing symptoms. EE EOI had a nonsignificant relationship with both child internalizing and externalizing symptomatology. Thus, the more parsimonious original overall model was again retained.

Cross-Validation Sample

Fit of the retained model.

The measurement and structural portions of the model are represented in Figure 1. This overall model also demonstrated good fit when tested with the cross-validation sample: S-B χ^2 (75, N = 370) = 95.70, p >.05; CFI =.976; and RMSEA =.027. Significance tests for each parameter (indicated beside the unstandardized coefficients) are indicated in Figure 1. All paths in the cross-validation sample were consistent with those obtained in the model modification sample.

All hypothesized indicators significantly loaded on their respective latent factor (i.e., maternal depression, child externalizing symptoms, or child internalizing symptoms). Maternal depression was significantly associated with EE criticism (standardized coefficient =.20), but was not significantly associated with EE EOI (standardized coefficient =.09, *ns).* There was a nonsignificant covariance between the residuals of EE criticism and EE EOI (standardized coefficient = –.07, *ns).* However, the hypothesized covariances between all other residuals (including those representing rater bias) were significant.

Thus, the results suggested that degree of maternal depression is related to EE – however, only to the EE criticism subtype. Given this relation, we next analyzed whether EE had a significant predictive relationship with child outcome, beyond any effects due to degree of maternal depression.

Independent prediction of child symptomatology and functional impairment from maternal depression and EE

As depicted in Figure 1, the maternal depression factor had a significant independent effect on all indices of child outcome: externalizing symptoms (standardized coefficient =.27), internalizing symptoms (standardized coefficient =.47), and functional impairment (standardized coefficient =.18). Maternal EE criticism had a significant effect on both child externalizing symptoms (standardized coefficient =.34) and child impaired functioning (standardized coefficient =.15); these effects were independent beyond any effects due to maternal depression (or EE EOI). However, maternal EE EOI did not have a significant association with either child internalizing symptoms (standardized coefficient =.11, *ns)* or child functional impairment (standardized coefficient =.03). R^2 was .22 for externalizing symptoms, .24 for internalizing symptoms, and .07 for child functional impairment. Thus, results suggest that both EE criticism and maternal depression separately predict both child externalizing symptoms and functional impairment. In addition, maternal depression also predicted internalizing symptoms.

Intervening effect of EE criticism

In addition to the direct effects, we also predicted that EE would serve as an intervening variable between maternal depression and youth outcomes (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Figure 1 presents only direct effects. Analysis of indirect effects revealed two significant indirect relations (not depicted in Figure 1). Specifically, there was a significant indirect relation between maternal depression and both child externalizing symptoms (standardized coefficient for parameter indirect effect =.07, p <.05) and child functional impairment (standardized coefficient for parameter indirect effect =.03, p <.05). Specifically, the indirect effect (through EE criticism) of maternal depression on child externalizing symptoms accounted for 20% of the overall relation between maternal depression and child externalizing symptoms; the indirect effect also accounted for 15% of the total effect between maternal depression and child functional impairment (procedures outlined in Holmbeck, 2002; percentage of effect accounted for by indirect effect = unstandardized coefficient indirect effect/unstandardized coefficient total effect).

Moderation effect of gender.

Multiple sample analysis was used to evaluate gender as a moderator (Bentler, 2002). The model was tested to determine whether boys and girls represent a single population or two different populations (with different structural models applying). Only one small interaction between population membership and structural model was observed: The path from the latent variable of child externalizing symptoms to K-SADS externalizing disorders could not be constrained to be equal for boys and girls without significantly degrading model fit (fully constrained model: S-Bχ^2[172, N = 370] = 214.85, p <.05; model constrained except for path from latent variable of child externalizing symptoms to K-SADS externalizing disorders: S-B χ^2[171, N = 370] = 197.12, p >.05; S-B $\chi^2_{\text{difference}}$[1, N = 370] = 8.39, p <.05; Satorra, 2000). These results indicate that the externalizing factor is slightly different for girls and boys~ with K-SADS externalizing disorders loading more strongly on the externalizing factor for boys (standardized coefficient =.69) than for girls (standardized coefficient =.58); otherwise, the model is the same for both genders.

Discussion

The present study used SEM to address several questions concerning the role of maternal EE in the association between extent of mothers' depression and adolescent symptom and functional outcomes. The results indicated that both EE criticism and degree of maternal depression (indexed by both symptom history and current depression) separately predicted both child externalizing symptoms and functional impairment. In addition, maternal depression also predicted internalizing symptoms. As predicted, EE criticism served as an intervening variable and accounted for a large

portion of the indirect effect between maternal depression and child outcomes, with the indirect effect accounting for 20% of the relation between maternal depression and child externalizing symptoms and nearly 15% of the association between maternal depression and youth functional outcomes. EOI was not associated with maternal depression and was not predictive of any child outcome. The hypothesized model was found to be the same for boys and girls. Also, similar results occurred when the latent variable of maternal depression was composed solely of indicators of degree of current depressive symptoms or history of depression.

The results are consistent with the view that maternal depression is a significant risk factor for symptomatology, disorder, and impaired functioning in offspring and with the commonly held hypothesis that depression's effect is at least partially mediated by quality of the interaction between mothers and their children (e.g., Cummings & Davies, 1994; Goodman & Gotlib, 1999; Kaslow et al., 1994). Similar to the relatively few other studies that have tested the associations among maternal depression, parenting, and child outcomes, the effects were not entirely mediated by quality of parental attitudes, and maternal depression also had direct associations with children's outcomes (e.g., Canino et al., 1990; Fendrich, Warner, & Weissman, 1990; Warner, Mufson, & Weissman, 1995).

The analyses indicated that high EE criticism is not simply an expression of maternal depression but rather has its own independent association with child outcome. In addition, EE criticism is also related to child functional impairment, beyond the effects of maternal depression or other child symptomatology (overlap between symptomatology and functional impairment was statistically removed). The analyses also indicated that the EE subtype of EOI did not generally predict children's outcomes. This resonates with the majority of previous studies that have found no significant predictive effects for EE EOI (e.g., Asarnow et al., 1994, 2001; McCarty & Weisz, 2002). However, this finding is inconsistent with the findings of two studies that found an association between EE EOI and separation anxiety and other anxiety disorders (Hirshfield et al., 1997; Stubbe et al., 1993); as such the possibility of specific associations of overinvolvement and anxiety disorders cannot be ruled out. The current study indicated that EE criticism had a specific association with child externalizing symptoms. This is consistent with previous research indicating a strong association between maternal EE criticism and externalizing psychopathology in children (Asarnow et al., 1994; Goodman et al., 1994; Hirshfield et al., 1997; Stubbe et al., 1993; Vostanis et al., 1994). The findings are also consistent with the broader literature on externalizing disorders in children, which find robust associations between punitive, negative-parenting practices and disruptive behavior disorders (see Hinshaw & Anderson, 1996, for a review).

The present study did not observe a significant association between maternal criticism and internalizing disorders, as some studies have previously reported (e.g., Asarnow et al., 2001). It might be that use of an adolescent sample and statistical controls for effects of maternal depression and general functional impairment resulted in lack of such patterns. However, perhaps externalizing behaviors were seen as more under the youth's control than depressive or other internalizing symptoms-especially when many mothers had themselves experienced depressive disorders. It is also possible that depressive disorders in the youth were associated with varied factors (e.g., such as peer and other environmental influences) not included in the present analyses and not with just maternal depression and mother-child interaction dysfunction. In another study of the children in the present sample, Hammen and Brennan (2001) observed that depressed youths of depressed mothers had significantly different social characteristics than depressed youths of never-depressed mothers. Such patterns are consistent with the hypothesis that different predictors or correlates of youth depression may distinguish subgroups of children of depressed or never-depressed mothers.

Although the findings of the present study are consistent with the view that degree of maternal depression is a cause of maternal criticism, which in turn leads to higher levels of child externalizing symptoms and functional impairment, the present design does not permit clarification of the direction of effects. It is possible, and indeed even likely, that there is a mutual effect of maternal critical attitudes and child maladjustment on each other (e.g., Hammen, Burge, & Stansbury, 1990). We might speculate that maternal depression, perhaps especially as it occurs early in the child's life (as was true of many of the women in the present sample), may have dysfunctional effects on the quality of the

mother-child interaction and elicit various forms of child maladjustment, and over time, such behaviors in turn might provoke and maintain negative maternal attitudes (e.g., Cummings & Davies, 1994; Goodman & Gotlib, 1999; Kaslow et al., 1994; NICHD Early Child Care Research Network, 1999).

Although it is possible to speculate about mechanisms by which maternal criticism may be predictive of youth externalizing difficulties and dysfunction, it is beyond the scope of the current study to clarify the processes. It will be an important issue for future research to determine how criticism eventuates in disruptive behavior disorder and to address the question of whether maternal depression is a common or unique component in different populations of youths who have externalizing disorders.

The current results are not intended to be a complete model of the pathways linking degree of maternal depression to children's risk for disorders and maladjustment. In fact, the current results indicate that there are likely other factors that are playing a role in mediating the relationship between maternal depression and child maladjustment. Various factors not included in the present model may be vitally important to an overall understanding of the process. For example, in other analyses we are exploring the specific contributions of maternal depression severity, chronicity, and timing (Brennan et al., 2000; Hammen & Brennan, 2003) as well as the role of paternal psychopathology and father-child relationship quality (Brennan, Hammen, Katz, & LeBrocque, 2002) on child functioning.

As noted, the causal associations among variables cannot be determined in the present study, and a longitudinal study currently underway will eventually help to address the predictive association between maternal attitudes at her child's age 15 and changes in the youth's symptomatology over time. However, in the current study, the consistent association of EE criticism with a construct of maternal depression that is composed of several indicators of history of depression provides some tentative support for the depicted direction of associations. In addition, these associations also held when the factor of maternal depression was composed *entirely* of indicators of maternal history of depression. Future studies can better explore the reciprocal influences between mother and child through examination of discrete interactions (a molecular approach) or through the examination of longitudinal data beginning in early childhood.

Another limitation of the current study includes its exclusive focus on 15-year-old youths in a community sample. Thus, generalization to other age groups and clinical populations may not be possible. However, the results are generally consistent with those previously reported from clinical samples with children of varying ages. It is acknowledged that the theoretical foundation of EE is not strong, and there continues to be speculation about what it is measuring (e.g., Hooley, 1985). In addition, although the measurement of EE is largely categorical, theoretically it represents a continuous quality of the mothers' attitudes toward their child. In this study, we have included the borderline dimension of EE in our analyses because we believed solely using the low/high categorization may obscure important information when used with children (rather than with adults). Given the more continuous scale of EE that we used (low, borderline, and high), we opted to use the ML data analysis method because it adjusts for nonnormal data. Although there is some concern about the appropriateness of this analysis given that our measurement of EE is barely continuous (on a 3-point scale), results for the depicted models were the same even when categorical analyses were used (see *Analysis of Assumptions and Estimation Methods* section).

The present study attempted to disentangle maternal EE from maternal depression (current symptomatology and history), but further research is needed to help determine what EE reflects and its impact over time. Overall, the current study confirms and extends prior research on the EE construct as a correlate of clinical and functional outcomes. The results raise the possibility that interventions aimed at improvement in the parent-child relationship might help reduce youngsters' risk for disorders in families with mothers with depressive symptomatology.

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