EXPRESSED EMOTION AND MATERNAL DEPRESSION: THE ROLE IN PREDICTING ADOLESCENT DEPRESSION

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ELANA C. GOLDMINTZ-GOTFRIED

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APPROVED:

Jami F. Young, Ph.D.

Brenna Bry, Ph.D.

DEAN:

Stanley Messer, Ph.D.
ABSTRACT

Background: Expressed emotion (EE) and maternal depression are important risk factors for youth depression and functioning. The present study examined these two risk-factors, both concurrently and longitudinally, within a sample of adolescents who were at risk for developing a depressive disorder as a function of elevated depression symptoms.

Methods: Fifty-seven adolescents and their mothers were drawn from a randomized trial of Interpersonal Psychotherapy-Adolescent Skills Training (IPT-AST). Assessments were conducted at baseline, post-intervention, and at 6-, 12-, and 18-month follow up. First, rates of critical EE were examined in this subclinical sample. Second, an ANCOVA framework was used to examine the relationship between EE, maternal depression, and adolescent depression and functioning. Results: Rates of critical EE in our sample were comparable to rates of critical EE in other at-risk and clinical samples. At baseline and post-intervention, no significant relationships were found between critical EE and adolescent depression and functioning. Maternal depression was related to one of the two measures of adolescent depression at baseline, but not to functioning. Both maternal depression and critical EE predicted adolescent depression and functioning at the 6- and 12-month follow up, however maternal depression was a more enduring risk factor in the longer term. Conclusions: Results suggest that although critical EE remains an important predictor of later adolescent depression and functioning, maternal depression may be a stronger risk-factor beyond the one year follow-up.
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CHAPTER I

Introduction

Child and adolescent depression is a frequent and recurrent problem that is associated with comorbid disorders and significant social and emotional impairment, as well as future depressive episodes (Bardone, Moffitt, Caspi, & Dickson, 1996; Lewinsohn, Rohde, Klein, & Seeley, 1999; Puig-Antich et al. 1993). Likewise, depressed children and adolescents are at a higher risk for attempted and completed suicides (Wagner, 1997). Thus, a thorough understanding of the factors that contribute to the pathogenesis of child and adolescent depression is critical. The development and maintenance of depression involves a complex interplay between inherited genetic predisposition and socio-environmental factors (Cicchetti & Toth, 1998). Interpersonal theories of depression have emphasized family environment as one important socio-environmental factor in the development of child and adolescent depression (Coyne, 1976; Lewinsohn, 1974). Consistent findings reveal that families of children and adolescents with depressive disorders are characterized by low levels of warmth, support, and communication (Messer & Gross, 1995; Puig-Antich et al., 1985; Sheeber & Sorensen, 1998), and high levels of parent-child criticism and conflict (Gotlib, Lewinsohn, & Seeley, 1998; Puig-Antich et al., 1993). However, the early literature was inconclusive as to whether these interpersonal factors precede depression and act as a risk factor, or whether they follow as a consequence of the depression.
Recently, numerous prospective and longitudinal studies have found that interpersonal factors, such as poor family relationship quality, predict the onset of depression (Reuter, Scaramella, Wallace, & Conger, 1999; Rice, Harold, Shelton, & Thapar, 2006; Sagrestano, Paikoff, Holmbeck, & Fendrich, 2003). Reuter and colleagues (1999) found that over a 4-year period, changes in disagreements between parents and adolescents predicted changes in adolescents’ internalizing symptoms. Likewise, Sheeber, Hops, Alpert, Davis, and Andrews (1997) found a causal influence of conflictual family environments on the development of adolescent depression. Finally, in a recent study of high-school females, Eberhart and Hammen (2006) looked at multiple predictors of depression symptoms and future episodes and found that one of the best predictors was conflictual or unsupportive relationships with family members. These studies serve as evidence that interpersonal difficulties, particularly in familial relationships, serve as risk factors for the development of depression.

**Expressed Emotion**

Expressed emotion (EE) is one index of familial affective climate that has emerged from the literature, which shows strong promise in helping to understand risk, course, and maintenance of childhood and adolescent depression. EE refers to critical, hostile, or emotionally over-involved attitudes that are observed in the relationship between relatives and a family member with a psychiatric illness (Vaughn & Leff, 1976). The EE construct is comprised of two elements: criticism and emotional over-involvement (EOI). The criticism dimension reflects critical and hostile attitudes, while the EOI dimension reflects intrusive over-concern. The operational definition of the attitudes that comprise the construct of EE originated in the schizophrenia research. The
level of EE expressed by a relative about the patient with schizophrenia at the time of admission proved to be the best single predictor of post-discharge relapse 9 months later (Brown, Birley, & Wing, 1972). Since this initial study, EE attitudes have been found to predict the course of schizophrenia (Vaughn & Leff, 1976), bipolar disorder (Miklowitz, Goldstein, Nuerchterlein, Snyder, & Mintz, 1988) and unipolar depression (Butzlaff & Hooley, 1998; Hooley & Teasdale, 1989) in adult populations. Thus, researchers in the area of child and adolescent psychology and psychiatry have begun to investigate the application of these findings to childhood disorders and their pathogenesis.

**EE and Child Diagnoses**

Within the child and adolescent literature, EE has been studied in the context of a number of disorders. Hibbs et al. (1991) found that parents of children and adolescents with disruptive behavior disorders and obsessive-compulsive disorder were rated significantly higher on levels of EE than normal controls. Others have found high rates of maternal EE in children and adolescents with attention-deficit hyperactivity disorder (ADHD), conduct disorder, depression, and substance abuse (Schwartz, Dorer, Beardslee, Lavori, & Keller, 1990). Using a large community sample, Stubbe, Zahner, Goldstein, and Leekman (1993) found that over one half of the families with high levels of EE had a child with a diagnosable condition as compared to families rated low on EE; however, high EE was not related to any specific child disorder. These findings suggest that EE may be a nonspecific correlate of child and adolescent psychopathology.

Alternatively, some studies have found EE to be a correlate of specific forms of child psychopathology. Asarnow and colleagues (Asarnow, Tompson, Hamilton, Goldstein, & Guthrie, 1994) found that amongst the three diagnostic categories of
“depressed” (including major depression, and dysthymia), “schizophrenic” (including schizophrenia, and schizotypal personality disorder), and “normals,” depressed children were the most likely to have families who rated high on EE. In their next study, Asarnow and colleagues (Asarnow, Tompson, Woo & Cantwell, 2001) examined EE in children and adolescents from inpatient and outpatient settings who fell within three diagnostic groups: depressed, current ADHD, and those with no lifetime diagnosis of either depression or ADHD. Results demonstrated that depressed youth were more likely to have parents who were rated high on criticism and overall EE than children with ADHD or controls. These findings were true regardless of age (children versus adolescents) or severity of psychopathology (inpatient versus outpatient). The EOI dimension yielded inconclusive findings, which is consistent with other research that has failed to find correlates of the EOI dimension (McCarty & Weisz, 2002).

These initial studies suggest that EE, particularly critical EE, may be a specific correlate of child and adolescent depression. This is further supported by recent findings by Silk et al. (2009) who found that 76% of mothers of currently depressed participants were rated as either borderline or high on criticism on an EE paradigm. However, two recent studies found no association between critical EE and child internalizing disorders, but rather a link between critical EE and child externalizing disorders (McCarty & Weisz, 2002; Nelson, Hammen, Brennan, & Ullman, 2003).

EE as Predictor of Outcome and Course

EE has also been examined as a predictor of clinical course of depressive episodes. Relapse rates were higher for depressed adults who returned to high EE families post-discharge than those who returned to low EE families (Hooley & Teasdale,
asarnow, goldstein, tompson, and guthrie (1993) extended these findings to
children and found that ee was predictive of 1-year post-discharge outcomes for children
with major depressive disorder and dysthymic disorder. children returning to high ee
homes were less likely to recover. this was true regardless of moderating variables such
as treatment regimen, chronicity, and severity of diagnosis. however, other studies have
not found a consistent relationship between ee and depression course. for instance,
mccleary and sanford (2002) found that high ee was not predictive of persistent
depression for the overall cohort of depressed adolescents, but found some evidence that
ee may predict one-year clinical outcomes for only a subgroup of depressed adolescents
without comorbid adhd.

maternal psychopathology and ee

it is difficult to interpret the differing results of the studies that have examined ee
in child and adolescent populations (asarnow et al. 1993; asarnow et al., 1994; asarnow
et al., 2001; mccarty & weisz, 2002; mccleary & sanford, 2002; nelson et al., 2003).
one possible interpretation is that those studies which did not find a relationship between
ee and adolescent depression (mccarty & weisz, 2002; nelson et al., 2003) included
measures of maternal psychopathology and expressed emotion. early research suggests
that high ee is more common in parents who have their own axis i or axis ii disorders
(hibbs et al., 1991), suggesting that these two variables are highly correlated. to tease
out this issue, several studies examined the relationship between maternal depression, ee,
and adolescent depression using cross-sectional designs (mccarty & weisz, 2002;
nelson et al., 2003; tompson, pierre, boger, mckowen, chan, & freed, 2010).
McCarty and Weisz (2002) investigated the concurrent relationship between maternal psychopathology, both dimensions of EE, and child psychopathology. Results revealed that critical EE was not significantly associated with child depression. Instead, they found that maternal psychopathology predicted maternal criticism, which partially predicted child externalizing disorders. Moreover, they found that a better predictor of child internalizing disorders was maternal psychopathology. Likewise, Nelson and colleagues (2003) found that both critical EE and the degree of maternal depression separately predicted child functional impairment and externalizing disorders. Although maternal depression was predictive of internalizing disorders, maternal criticism was not.

These findings raise significant questions about the true predictive power of EE in child and adolescent depression. When maternal depression is taken into account, the relationship between EE and internalizing disorders seems less clear than in those studies which did not consider parental psychopathology (Asarnow et al., 1994; Asarnow et al., 2001).

A recent study by Tompson and colleagues (Tompson, et al., 2010) comes closest to addressing this question by examining the concurrent relationship between youth psychopathology, EE, and maternal depression in a sample of 171 youth ages 8-12. They found that neither maternal depression history nor overall EE alone predicted child rated depression. However, the interaction between critical EE and maternal depression history strongly predicted child depressive symptoms. In effect, Tompson and colleagues (2010) suggest that the most powerful effects in youth are observed in the presence of both high critical EE and maternal depression. Future research is required to better understand how these two variables either individually or jointly predict adolescent depression.
Another possible explanation for the discrepant findings is that the populations studied varied in severity of depression. Those which found an association between EE and child and adolescent depression (Asarnow et al., 1993; Asarnow et al., 1994) utilized inpatient samples, while those that did not (McCarty & Weisz, 2002; Nelson et al., 2003; Tompson et al., 2010) utilized outpatient samples. Only one study (Asarnow et al., 2001) utilized both. To examine whether there are differential rates of EE in adolescents with varying levels of depression, and to determine whether EE is a significant predictor of major depressive disorder (MDD) across these samples, Silk et al. (2009) recruited four groups of youth: (a) those with a current episode of MDD, (b) those with a past history of MDD, (c) nondepressed children and adolescents at high familial risk for developing MDD, and (d) nondepressed controls. They found that mothers of children and adolescents with current depression, remitted depression, and high familial risk for depression all had significantly higher rates of critical EE than mothers of low-risk controls. In addition, critical EE across the first three groups predicted future depressive episodes and there were no significant differences between the currently depressed group and those who were at high-risk for depression or in remission (Silk et al., 2009). These findings suggest that EE is a predictor of depression even in those adolescents who are not currently depressed.

EE and Overall Functioning

Another area which requires further clarification is how EE and maternal depression contribute to overall child and adolescent functioning. Previous literature has documented the relationship between maternal depression and child functioning (Shaffer
et al. 1983; Mufson, Gallagher, Dorta & Young, 2004), however, little is known about its relationship to EE. Nelson et al. (2003) found that both critical EE and maternal depression independently predicted child functional impairment as measured by the chronic stress interview developed by Hammen and colleagues (Hammen, Adrian, Gordon, Burge, Jaenieke & Hiroto, 1987). High critical EE was also an intervening variable between maternal depression and child functional impairment. In addition, Asarnow et al. (2001) found that high critical EE was associated with poor GAF scores across groups. To our knowledge, these are the only studies which have examined the relationship between EE and functional impairment. Further research is necessary to examine these relationships and how they evolve over time.

Current Study

The current study has four aims. The first is to examine the rates of maternal EE in adolescents who are at risk for depression as a function of elevated depression symptoms. The second aim of the study is to examine the concurrent relationships between EE, maternal depression, and adolescent depression and functioning. The third aim is to determine whether critical EE or maternal depression is a better predictor of later depressive symptoms in this at-risk sample. The fourth aim is to examine the relationship between maternal depression, critical EE, and later adolescent functioning.

Hypotheses

On the basis of the prior research on EE and adolescent depression, four hypotheses will be tested. First, there will be similar rates of critical EE in this subclinical sample as have been found in clinically depressed adolescent samples. Second, baseline adolescent depression symptoms and functioning will be significantly correlated with
baseline critical EE and baseline maternal depression symptoms. Third, baseline critical EE and maternal depression will be predictive of adolescent depression symptoms post-intervention and at follow-up. Fourth, baseline critical EE and maternal depression will be predictive of adolescent overall functioning post-intervention and at follow-up.
Participants for this study were drawn from a randomized trial that compared different preventive interventions, Interpersonal Psychotherapy-Adolescent Skills Training (IPT-AST; Young & Mufson, 2003) and usual school counseling (SC) for adolescents with subthreshold depression symptoms. The methods of the larger study are described in detail elsewhere (Young, Mufson, & Gallop, 2010). Briefly, a two-stage procedure was used to identify adolescents with elevated symptoms of depression in three single-sex schools in New York City, two girls’ schools and one boys’ school.

Recruitment began in November 2005 and ended in February 2007. The first stage was a classroom-based screening in which parents of students in the 9th and 10th grades received letters from the school administration about the screening. Parents were asked to send back a letter of refusal if they did not wish for their child to participate. If a letter was not received, another letter was sent to the parents, allowing them to refuse participation for a second time. On the day of the screening, adolescents were informed of the procedures, and those that wished to participate signed a screening assent form. Six-hundred forty-two adolescents were screened using the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977). Adolescents who received a score between 16 and 39 were eligible to be approached for the prevention program. Eligible adolescents and their
families were contacted to schedule an in-person meeting to describe the prevention program. During these meetings, procedures for the eligibility evaluation and the prevention program were reviewed, and informed consent and assent were obtained. One third of the 237 adolescents contacted \( N = 79 \) agreed to participate in the program.

Those adolescents with consent and assent completed a structured diagnostic interview, the Schedule for Affective Disorders and Schizophrenia for School-Aged Children (K-SADS-PL) (Kaufman, Birmaher, Brean, & Rao, 1997), and the Children’s Global Assessment Scale (CGAS) (Schaffer et al., 1983) to determine eligibility. Since this was a prevention study, adolescents were deemed eligible to participate if they had at least two subthreshold or threshold depressive symptoms on the K-SADS-PL, did not meet criteria for a current depressive episode, and had a CGAS score equal to or greater than 61. Exclusion criteria included a current diagnosis of any mood disorder or other various disorders. This resulted in 21 adolescents who were excluded from the program: 4 did not have enough depressive symptoms, 10 met criteria for a current depression diagnosis, suicidal ideation, or self-harm behaviors, and 7 met criteria for the other exclusionary diagnoses. One adolescent left the school after the eligibility evaluation was completed but prior to randomization.

The resulting sample consisted of 57 adolescents, aged 13 to 17 in the 9th and 10th grades. The average age was 14.51 \( (SD = 0.76) \) years. The sample was 59.65% female and a majority of the sample (73.68%) identified themselves as Hispanic. Racially, 61.40% of the sample was white, 35.08% was African American, and 31.5% was biracial. Over half of the sample (70.18%) lived in a single-parent household with 29.07% reporting a gross household income of $25,000 or less. Randomization to intervention
condition was done using a table of random numbers. Over the two years of recruitment, 36 adolescents were randomized to IPT-AST and 21 were randomized to SC.

Assessment

All adolescents and their parents completed assessments at baseline, post-intervention, 6, 12, and 18 month follow-up points. These assessments were conducted by independent evaluators (IE) who were blind to intervention conditions. The domains assessed for this study were: 1) adolescent depression symptoms on the CES-D and the Children’s Depression Rating Scale-Revised (CDRS-R; Pozanski & Mokros, 1996), 2) adolescent overall functioning on the Children’s Global Assessment Scale (CGAS) (Shaffer, et al., 1983), 3) parental depression symptoms on the CES-D, and 4) parental expressed emotion on the Five Minute Speech Sample (FMSS) (Magana et al., 1986). For all but three adolescents in the study, the parental measures were completed by the adolescent’s biological mother. The remaining parents were two fathers and one stepmother, who were the primary caretakers of the adolescent. Given the preponderance of mothers in this sample, parental EE and depression will be referred to as maternal EE and maternal depression.

Measures

Depression symptoms

Adolescent and maternal depression symptoms were assessed using the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977). The CES-D is a 20-item self-report measure that assesses the presence of depressive symptoms over the course of the past week. Both parents and adolescents completed the CES-D at baseline.
Adolescents also completed the CES-D at post-intervention as well as 6, 12, and 18 months post-intervention.

Adolescents were also assessed using the Children’s Depression Rating Scale-Revised (CDRS-R; Pozanski & Mokros, 1996). The CDRS-R is a 17-item clinician-rated instrument which assesses depressive symptoms in four domains: cognitive, somatic, affective, and psychomotor. The assessment is completed by a trained interviewer and the score is converted to a $t$ score, with higher scores indicating more symptoms.

*Overall Functioning*

Adolescent functioning was assessed using the Children’s Global Assessment Scale (CGAS; Shaffer et al., 1983). The CGAS is a clinician rated global measure of social and psychiatric functioning for children ages 4–16 years. The independent evaluator assigned a CGAS score at each assessment point. Higher scores indicate higher overall functioning.

*Expressed Emotion*

The Five Minute Speech Sample (FMSS; Magana et al., 1986) is a brief procedure used to assess expressed emotion attitudes from a speech sample recorded by a family member. Parents were instructed to speak for 5 minutes into a tape recorder, without interruption, about their adolescent and how they get along together. A certified rater, who was blind to all other diagnostic information about the parent and their child, coded the recordings. Each sample was coded according to the criteria developed by Magana et al. (1986) and Magana-Amato (1993).

According to this scoring system, a total EE score is based on scores across two dimensions: criticism and emotional over-involvement (EOI). In accordance with the
manual scoring system (Magana-Amato, 1993) a sample is scored as high on criticism if the mother expresses a negative initial statement, a statement indicating a negative relationship with the child, or one or more criticisms as defined by the coding system. In addition to a categorical determination of high, low or borderline critical EE, the coding system scores the number of critical comments made during the speech sample. Thus, although EE is typically measured categorically, theoretically, it represents a continuous quality of the mother’s attitude towards her child (Nelson et al., 2003). Moreover, the literature has suggested that the FMSS may underestimate critical EE (Hooley & Parker, 2006). We looked at rates of EE in our sample using the categorical dimension of critical EE. Due to both our small sample size and the fact that categorical dichotomization often leads to misleading results due to loss of power (MacCallum, Zhang, Preacher, & Rucker, 2002), we used the number of critical comments as our measure of critical EE in the remaining analyses.

**Analyses**

Data were collected at baseline, post-intervention, and 6-month, 12-month and 18-month follow-up assessments of a randomized controlled trial which evaluated the efficacy of different prevention programs for depression in at-risk adolescents. First, we examined rates of critical EE in our sample. Second, utilizing an analysis of covariance (ANCOVA) framework, we examined the relationship between critical EE (as measured by the number of critical comments made by the mother), baseline maternal depression, and adolescent baseline depression symptoms and functioning. ANCOVA under this setting allows us to control for the effect of intervention and school. Third, we examined parent depression and critical EE as predictors of child depression (CES-D and CDRS-R)
and overall functioning (CGAS) at each of the time points, while controlling for baseline scores, school, and intervention condition.

The primary outcome analyses reported in Young et al. (2010) used analysis of covariance (ANCOVA) to examine mean differences between the intervention conditions at post-intervention and follow-up, controlling for baseline scores and school. We augmented the ANCOVA model to include these two additional predictors. First, we looked at each predictor separately. When both maternal depression and critical EE were significant, we created an additional ANCOVA model which included both predictors. For participants with missing data, we imputed missing data based on the last available observation, as was done in Young, et al. (2010).

The partial association between the outcome and the predictors, while controlling for other covariates and factors, is assessed through the ANCOVA model (Lipsitz, Leong, Ibrahim, & Lipshultz, 2001). The effect size index, partial eta-squared, corresponds to the squared partial correlation coefficient of the predictor with the outcome controlling for the additional predictors in the model. Similar to the thresholds for the Cohen’s d effect size, we have values of 0.01, 0.06, 0.14, as marking small, medium, and large effects for the partial eta-squared. The R-squared index, referred to as the coefficient of determination, quantifies the percentage of variance of the outcome explained by the model. To quantify how each additional predictor contributes to the model, we will summarize the R-squared for the outcome model, the R-squared when each of the two predictors is included in the ANCOVA model, and the R-squared when both predictors are simultaneously included in the ANCOVA model.
While normality of the outcome measure is an assumption of the ANCOVA model, normality of the predictors is not a necessary requirement. An important quality for the predictors is whether a unit increase in the predictor is clinically meaningful. For the number of critical statements, the unit increase in this measure is clinically meaningful as it represents an increase in the number of critical statements. For maternal depression, the positive skew in the measure provides concern about the linearity assumption of this predictor and the interpretability of a unit increase; therefore, we implemented a square-root transformation which appeared to normalize the predictor.
CHAPTER III

Results

Rates of EE and Baseline Maternal Depression

Mothers were rated as high, low, or borderline on levels of EE. In our subclinical sample, 38.60% \((n = 22)\) of mothers were rated high in critical EE, 33.30% \((n = 19)\) were rated borderline, and 28.10% \((n = 16)\) were rated as low in critical EE. Studies have differed in whether they count the borderline group as high EE or low EE. In line with the recommendations and procedures of previous studies, (Magana-Amato, 1993; Silk et al., 2009; Stubbe, et al., 1993) which have included borderline scores in the high EE category, we also examined rates of critical EE when the high and borderline groups were combined. When borderline scores were included in the high category, 71.90% \((n = 41)\) of our sample scored high on critical EE. Regarding maternal depression, mothers in the current study reported only mild symptoms of depression at baseline as measured by the CES-D \((M = 11.82, SD = 8.74)\)

Baseline Associations

There were no significant associations between critical EE (as measured by the number of critical statements made during the speech sample) and adolescent depression and functioning at baseline, while controlling for intervention assignment and school. For maternal depression, we found a significant association with adolescent depression as measured on the CDRS-R \((F(1,52) = 4.08, p = 0.04)\), but the association did not replicate
for the CES-D ($F(1,52) = 1.75, p = 0.19$). There was no significant association between maternal depression and adolescent functioning ($F(1,52) = 0.22, p = 0.64$) at baseline.

**EE, Maternal Depression, and Adolescent Depressive Symptoms**

**Post Intervention**

*CDRS-R*. The original outcome model, which included baseline scores, school, and intervention condition, explained 21.74% of the variance in post-intervention CDRS-R scores. Maternal depression was not a significant predictor of post-intervention CDRS-R scores (see Table 1 for details). Critical statements however, was a significant predictor of post-intervention CDRS-R scores ($F(1,50) = 4.22, p = 0.04$). The partial eta-squared for critical statements was 0.07 corresponding to a medium to large effect. The inclusion of critical statements in the model accounted for 5.99% of the variance, resulting in a total R-squared of 27.73%.

*CES-D*. The original outcome model explained 18.30% of the variance in post-intervention CES-D scores. Neither maternal depression nor critical statements significantly predicted post-intervention CES-D scores.

**Six Month Follow-up**

*CDRS-R*. The original outcome model explained 9.47% of the variance in CDRS-R scores at the 6-month follow-up. There was a trend towards significance for maternal depression ($F(1,50) = 3.72, p = 0.06$), while critical statements was a significant predictor of adolescent CDRS-R scores at the 6-month follow-up ($F(1,50) = 5.19, p = 0.03$). The partial eta-squared for critical statements was 0.09, corresponding to a medium to large effect size. Inclusion of critical statements in the model accounted for 5.85% of the variance, resulting in a total R-squared of 17.82%.
CES-D. The original outcome model explained 8.53% of the variance in CES-D scores at the 6-month follow-up. Both maternal depression ($F(1,50) = 5.13, p = 0.02$) and number of critical comments ($F(1,50) = 5.91, p = 0.02$) significantly predicted 6-month CES-D scores. Maternal depression accounted for 8.53% of the variance resulting in a net R-squared of 16.88%. Critical statements accounted for 9.34% of the variance resulting in a total R-squared of 17.87%. When maternal depression and critical statements were simultaneously entered into the model both maternal depression ($F(1,50) = 5.33, p = 0.02$) and critical statements ($F(1,50)=6.00, p = 0.01$) remained significant predictors of 6-month CES-D scores. With both predictors in the model, the partial eta-squared was 0.09 for parent depression and 0.10 for critical statements, corresponding to medium to large effects. The inclusion of both measures simultaneously in the model accounted for 17.26% of the variance resulting in a net R-squared of 17.79%. The net increase when including both variables does not equal the sum of the net increase for each; therefore, both measures share some common variance explained by the outcome.

Twelve Month Follow-up

CDRS-R. The original outcome model explained 9.77% of the variance in CDRS-R scores at the 12-month follow-up. Neither maternal depression nor critical statements were significant predictors of 12-month follow-up CDRS-R scores.

CES-D. The original outcome model explained 4.53% of the variance in CES-D scores at the 12-month follow-up. Only maternal depression was a significant predictor of 12-month follow-up CES-D scores ($F(1,50) = 4.38, p = 0.04$). Partial eta-squared for maternal depression was 0.08, corresponding to a medium to large effect. The inclusion
of maternal depression in the model accounted for 7.55% of the variance, resulting in a total R-squared of 12.08%.

**Eighteen Month Follow-up**

**CDRS-R.** The original outcome model explained 10.70% of the variance in CDRS-R scores at the 18-month follow-up. Only maternal depression was a significant predictor of 18-month follow-up CDRS-R scores ($F(1,50) = 4.50, p = 0.03$). Partial eta-squared for maternal depression was 0.08, corresponding to a medium to large effect. Inclusion of maternal depression in the model accounted for 7.24% of the variance, resulting in a total R-squared of 17.94%.

**CES-D.** The original outcome model explained 8.30% of the variance of CES-D scores at the 18-month follow-up. Only maternal depression was a significant predictor of 18-month follow-up CES-D scores ($F(1,50) = 7.39, p < 0.01$). Partial eta-squared for maternal depression was 0.12, corresponding to a medium to large effect. The inclusion of maternal depression in the model accounted for 11.60% of the variance, resulting in a total R-squared of 19.90%.

**EE, Maternal Depression, and Overall Functioning**

**Post Intervention**

The original outcome model explained 35.96% of the variance in post-intervention CGAS scores. Neither maternal depression nor critical statements were significant predictors of post-intervention CGAS scores.

**Six Month Follow-up**
The original outcome model explained 17.07% of the variance in CGAS scores at the 6-month follow-up. Maternal depression \( F(1,50) = 4.68, p = 0.03 \) was a significant predictor of 6-month follow-up CGAS scores, while there was a trend towards significance for critical statements \( F(1,50) = 2.93, p = 0.09 \). Including maternal depression in the model significantly improved the model fit, accounting for 6.97% of the variance, corresponding to a net increase of 24.04%. The partial eta-squared for maternal depression was 0.08, corresponding to a medium to large effect.

**Twelve Month Follow-up**

At the 12-month follow-up assessment, the original outcome model explained 6.67% of the variance in CGAS scores. Both maternal depression \( F(1,50) = 7.17, p = 0.01 \), and critical statements \( F(1,50) = 14.55, p < 0.01 \) significantly predicted 12-month follow-up CGAS scores. Inclusion of maternal depression in the model accounted for 11.51% of the variance, corresponding to a total R-squared of 18.18%. The inclusion of critical statements in the model accounted for 20.72% of the variance, resulting in a total R-squared of 27.39%. When maternal depression and critical statements were simultaneously entered into the model both maternal depression \( F(1,50) = 8.77, p = 0.04 \) and critical statements \( F(1,50) = 16.22, p < 0.01 \) remained significant. With both predictors in the model, the partial eta-squared was 0.15 for maternal depression and .24 for critical statements, each corresponding to large effects. The inclusion of both measures simultaneously in the model accounted for 31.55% of the variance resulting in a total R-squared of 38.22%.

**Eighteen Month Follow-up**
The original outcome model explained 10.27% of the variance in CGAS scores at the 18-month follow-up. Only maternal depression was a significant predictor of 18-month follow-up CGAS scores \((F(1,50) = 4.19, p = 0.04)\). Partial eta-squared for maternal depression was 0.08, corresponding to a medium to large effect. The inclusion of maternal depression in the model accounted for 6.82% of the variance, resulting in a total R-squared of 17.09%.
CHAPTER IV

Discussion

The purpose of the current study was to examine the relationship between critical EE, maternal depression, and adolescent depressive symptoms and overall functioning concurrently, and over time. Our first hypothesis was that there would be similar rates of critical EE in our subclinical sample as there have been in clinically depressed adolescent samples. We found this to be true. In the two studies conducted by Asarnow and colleagues, 26% (Asarnow et al., 1994) and 49% (Asarnow et al., 2001) of mothers with depressed children scored high on critical EE. These rates are similar to our rates (39%) of mothers who scored high on critical EE. Likewise, when borderline EE was included in the high category, 72% of mothers rated high on EE. This is comparable to the rates of critical EE in the 3 high-risk groups in the Silk et al. (2009) study. They found that while 34.1% of mothers in the control group scored high on critical EE, 61.9% of mothers with children and adolescents with high familial risk, 83.3% of those with children and adolescents with a past history of MDD, and 75.9% of those with children and adolescents with current MDD, scored high on critical EE, when the borderline category is included in the high EE category.

Our second hypothesis was that there would be a significant relationship between baseline critical EE (as measured by the number of critical statements), baseline maternal depression, and baseline adolescent depression and functioning. Contrary to our
hypothesis, we did not find any relationship between baseline critical statements and baseline adolescent depression. As discussed in the introduction, previous research on the concurrent relationship between EE and adolescent depression has been inconclusive. Although some research (Asarnow et al., 1994; Asarnow et al., 2001) has found a specific relationship between critical EE and adolescent depression, other studies (McCarty & Weisz, 2002; Nelson et al., 2003) have not. In these studies, a stronger relationship was found between critical EE and externalizing disorders. Interestingly, the studies which have failed to find a relationship between critical EE and depression (McCarty & Weisz, 2002; Nelson et al., 2003) have used outpatient settings, whereas studies of inpatient or combined inpatient/outpatient settings have found a stronger relationship between EE and adolescent depression (Asarnow, et al., 1994; Asarnow, et al., 2001). This suggests that EE may be a more salient risk factor for more depressed and clinically impaired adolescents than it is for mildly depressed or subthreshold adolescents.

Surprisingly, we only found a significant relationship between baseline maternal depression and baseline adolescent depression, on one measure but not the other. Using a similar subclinical population, Tompson et al. (2010) found that current maternal depression was only associated with mother’s reports of their children’s internalizing symptoms, but not children’s reported symptoms. Our findings are inconsistent however, with previous research which has demonstrated a strong association between maternal depression and increased risk of depression in youth (Beardslee, Versage, & Gladstone, 1998; Cummings & Davies, 1994; Goodman & Gotlib, 1999; Nelson et al., 2003). The discrepant findings may be accounted for by the fact that mothers in the current study reported only mild symptoms of depression as measured by the CES-D, while the other
studies consisted of more clinical parent samples. Our sample also differs from the “at risk” samples in the Tompson et al. (2010) and Silk et al. (2009) studies, in that both of these samples were chosen because a parent had a current or prior episode of depression. The relationship between parental depression and child depression may be strongest in those families where a parent has a diagnosis of depression, rather than just an elevated depression score. This is supported by Forehand, McCombs, and Brody (1987) who found a stronger relationship between parental depression and child adjustment when parental depression was identified through diagnostic interviews rather than self-report questionnaires.

We next looked at post-intervention follow-up scores. To our knowledge, this is the first study to examine the effects of EE in this population within the context of a prevention intervention. There was no significant relationship between baseline maternal depression and post-intervention adolescent depression and functioning. Moreover, baseline critical statements predicted adolescent depressive symptoms on only one measure of depression but not the other, and did not predict overall adolescent functioning. Intervention condition was a strong predictor of post-intervention depression and functioning scores, accounting for 18 to 36% of the variance in scores. Thus, our limited findings may be due in part, to the large effects of the intervention. We next conducted exploratory analyses to examine the interaction of these variables and intervention condition to examine the interaction of these variables and intervention condition. Neither maternal depression nor critical statements moderated intervention effects in the current study. In contrast, prior studies have examined the role of parent depression (Garber et al., 2009) and EE (Miklowitz et al., 2009) in the context of
interventions for adolescent mood disorders and found that these variables moderated intervention outcome. Our study may have been underpowered to detect interaction effects. Future intervention studies should examine whether EE and/or parental depression moderate intervention outcome.

Our study expanded on previous research by examining the longitudinal effects of EE and parental depression on adolescents’ depressive symptoms 6 months, 12 months and 18 months post-intervention. Results demonstrated the unique influence of both predictors on adolescent depression at different time points. At the 6-month follow-up, both baseline critical statements and parental depression either approached significance or significantly predicted adolescent depressive symptoms, although critical statements was a stronger predictor than parental depression. At the 12-month follow-up, baseline parental depression became a stronger predictor of adolescent depression symptoms.

Most of the previous longitudinal studies of EE in child and adolescent samples (Asarnow, et al., 1993; McCleary & Sanford, 2002) have utilized a 12 month follow-up point. Thus, our 6- and 12-month follow-up data, which were collected 9 and 15 months post-baseline, are most similar to the time frame used in previous research. Similar to these studies, we found a significant relationship between critical statements and adolescent symptoms almost one year later. Our findings are noteworthy because they demonstrate that critical EE remains an important predictor of later depression, even in a subclinical sample.

Data from the 18-month follow-up revealed that in the long-term, maternal depression is a more stable and stronger predictor of adolescent depressive symptoms than is critical EE. As stated earlier, this is the first study to look at the predictive validity
of these two variables, across time, within a subclinical population. Not only were the adolescents in the current sample subclinical, but so were the majority of the parents. The cross-sectional study by Tompson et al. (2010) found that the interaction of past maternal depression diagnoses and EE was significantly associated with adolescent depression; neither EE alone nor current maternal depression symptoms were significant predictors. Our findings that EE was not predictive of adolescent depression at the 18-month follow-up, combined with those by Tompson et al. (2010) suggest that critical EE may be predictive of later adolescent depression only within the context of more severe maternal psychopathology.

Our study also added to the previous literature by examining the relationship between adolescent functioning and EE. To our knowledge, the current study is the first to examine these relationships longitudinally. Our results demonstrated that EE and maternal depression predict overall functioning at different time points. The relationship between these variables follows the same trend as their relationship to adolescent depression. The one discrepancy is at the 12-month follow-up when both maternal depression and critical statements predicted overall functioning, whereas only maternal depression predicted depression symptoms. These findings suggest that EE may continue to predict overall functioning beyond the time it predicts depressive symptoms. The long-lasting effects of parental depression on child and adolescent functioning is well documented in the literature (Beardslee et al., 1998; Cummings & Davies, 1994). Our study adds to the literature by identifying the difference in predictive power of one risk factor relative to another over time. Our results suggest that both EE and maternal
depression are important predictors of adolescent’s functioning over time, although maternal depression has more long-lasting effects.

The reason that parental depression remains a significant risk factor for later depression and functioning while EE does not, requires exploration in future research. The diathesis-stress model of depression postulates that the interaction of genetics and socio-environmental factors contributes to the etiology and pathogenesis of depression (Cicchetti & Toth, 1998; Rao, Hammen, & Poland, 2010). Parental depression likely acts as a genetic and socio-environmental risk factor (Goodman & Gotlib, 1999). Critical EE, on the other hand, may be best understood as a socio-environmental trait of a family unit (Miklowitz, 2004). According to Miklowitz’s transactional model, critical EE is the result of emotional disturbances in the family climate which become more entrenched over time. Silk et al., (2009) found that even when depression remitted, critical EE endured. Thus, consistent with Miklowitz (2004) they speculate that a depressive episode may have a “scarring effect” on family relationships (Silk et al., 2009).

Given that the majority of parents and adolescents in our sample had no previous depressive episode, critical EE may not have been as entrenched as it is in families with a psychiatrically ill member. In fact, the original study by Vaugh and Leff (1976) defined EE as an attitude which was observed between relatives and a family member with a psychiatric illness. The EE construct may not be as powerful in families without a diagnosis. Rather, parental depression, which likely has a larger genetic component, may be a more stable risk factor for later adolescent depression in subclinical populations.

Several limitations of our study are noteworthy. First, our small sample size limited our ability to detect small to moderate effects in the association between critical
EE, maternal depression, and child depressive symptoms. While small effects may be clinically meaningful, we were underpowered to detect these small differences. Likewise, we were underpowered to detect interaction effects between these variables if they existed. Second, given our small sample size, we used a continuous measure of critical EE in our ANCOVA analyses. We may have found different, and possibly more long-lasting, relationships between critical EE and adolescent depression had we utilized the more traditional high vs. low dichotomy. Third, over two-thirds of our sample was Hispanic and a large percentage came from low socio-economic backgrounds. The inclusion of this population is an asset to our study since many of the other studies which have examined EE in the context of adolescent depression have utilized predominantly Caucasian populations (Asarnow et al., 1993; Asarnow et al., 1994; McCarty & Weisz, 2002; Nelson et al., 2003; Tompson et al., 2010; Silk et al., 2009). However, it is unclear whether the results of this study can be generalized to adolescents of other ethnicities and socio-economic backgrounds. Fourth, our study is also limited by the inclusion of mostly mothers and their adolescents. Future research on EE and adolescent depression would benefit from inclusion of paternal EE to examine the differential relationships with adolescent depressive symptoms. Fifth, we recognize the selection bias that may exist within our sample. Parents who consented and participated in the study may be more aware of psychological symptoms and/or have a more positive view of psychological treatment than those who did not wish to participate in the study and were thereby not represented. Finally, our sample was comprised of adolescents who had elevated symptoms of depression, but were not currently depressed. Our finding that maternal depression was a stronger predictor of later depression symptoms than critical EE may be
limited to adolescents with subclinical depression. Future research should examine whether the long-term relationship between maternal depression, critical EE, and adolescent depression and functioning is consistent among depressed adolescents, adolescents at risk for depression because of maternal depression, and adolescents with subclinical depression.

Conclusions

To date, the association between critical EE and child and adolescent depressive disorders remains unclear; but the literature is beginning to shed light on these complex relationships by teasing out other important contributing variables such as maternal depression. Our study adds another piece of the puzzle by examining these variables concurrently and over time. To our knowledge, this is the first study to do so with a subclinical sample, identified because of the adolescents’ symptoms, rather than the presence of parental depression. It is also the first study to examine the longitudinal relationship of these variables to adolescent functioning. Results from our study replicate previous findings that EE is predictive of one-year clinical outcomes for adolescents with depression. Second, our study demonstrates that over time both critical EE and maternal depression are independently predictive of adolescent depression and functioning, but that maternal depression is a more enduring predictor over a longer time period. Finally, our findings suggest that critical EE is likely to be a stronger predictor of future adolescent depression in the context of more severe child and maternal psychopathology. Given that maternal depression is an enduring predictor of adolescent depression over time, continuing efforts should be made to develop and study selective preventive interventions for adolescent depression.
REFERENCES


Table 1
Summary of EE and Maternal Depression as Predictors

<table>
<thead>
<tr>
<th>Predictor</th>
<th>CDRS-R</th>
<th>CES-D</th>
<th>CGAS</th>
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<tr>
<td></td>
<td>F Value</td>
<td>p value</td>
<td>Partial Eta Squared</td>
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<td>Baseline</td>
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<td>EE</td>
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</tr>
<tr>
<td>Depression</td>
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<td>0.04</td>
<td>0.07</td>
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<tr>
<td>Post Intervention</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>EE</td>
<td>4.22</td>
<td>0.04</td>
<td>0.08</td>
</tr>
<tr>
<td>Depression</td>
<td>0.06</td>
<td>0.81</td>
<td>0.01</td>
</tr>
<tr>
<td>6 Month Follow-up</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>EE</td>
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<tr>
<td>Depression</td>
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<td>12 Month Follow-up</td>
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<td>EE</td>
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<tr>
<td>Depression</td>
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<td>0.08</td>
</tr>
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</table>

Note: All results control for intervention and school. Results post baseline control for the baseline assessment.