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Expressed Emotion-Criticism and Risk of Depression Onset in Children

Katie L. Burkhouse¹, Dorothy J. Uhrlass¹, Lindsey B. Stone¹, Valerie S. Knopik², and Brandon E. Gibb¹

¹Binghamton University (SUNY)

²Division of Behavioral Genetics, Rhode Island Hospital and Department of Psychiatry and Human Behavior, Brown University

Abstract

Objective—The primary goal of the current study was to examine the impact of maternal criticism (expressed emotion-criticism; EE-Crit) on the prospective development of depressive episodes in children. In addition to examining baseline levels of EE-Crit, we also sought to determine whether distinct subgroups (latent classes) of mothers could be identified based on the levels of EE-Crit they exhibited over a multi-wave assessment and whether that latent class membership would predict depression onset in children. Finally, we examined whether EE-Crit and maternal depression would independently predict children's depression risk or whether EE-Crit would moderate the link between maternal depression and children's depression onset.

Method—Children of mothers with or without a history of major depression (N=100) were assessed five times over 20 months. Children completed the Children's Depression Inventory (CDI) and mothers completed the Five Minute Speech Sample and the Beck Depression Inventory (BDI) at the baseline assessment, and at 2, 4, and 6 month follow-up assessments. Children and mothers completed diagnostic interviews assessing children's onsets of depressive episodes at the 20 month follow-up.

Results—Latent class analysis of the 4 waves of EE-Crit assessments revealed two distinct groups, exhibiting relatively lower versus higher levels of EE-Crit across the first 6 months of follow-up. EE-Crit latent class membership predicted children's depression onset over the subsequent 14 months. This finding was maintained after controlling for mother's and children's depressive symptoms during the initial 6 months of follow-up. Finally, maternal depression did not moderate the link between EE-Crit and childhood depression onset.

Conclusions—Continued exposure to maternal criticism appears to be an important risk factor for depression in children, risk that is at least partially independent of the risk conveyed by maternal depression. These results highlight the importance of a modifiable risk factor for depression—repeated exposure to maternal criticism.

Keywords

Maternal Criticism; Child Depression; Intergenerational Transmission; Vulnerability; Risk

Correspondence concerning this article should be addressed to Katie L. Burkhouse, Department of Psychology, Binghamton University (SUNY), Binghamton, NY 13902-6000. kburkho1@binghamton.edu.

⁴Because all new onsets of depression occurred among children of depressed mothers, mother MDD status could not be used as a covariate in our analysis. Therefore, we examined whether EE-Crit latent class would continue to predict depression onset in children when we limited our sample to children of depressed mothers. Even among these 48 children, EE-Crit latent class membership based on levels of criticism during the first 6 months of follow up significantly predicted onset of depressive episodes over the remaining 14 months of follow up, *Wald* = 7.89, *p* < .01, *OR* = 6.81.

Depression during childhood and adolescence is associated with impairment in social, emotional, and academic domains as well as with a wide range of psychiatric and physical health problems in adulthood (Harrington, 1996). Given accumulating evidence regarding the high prevalence rate, chronic course, and debilitating consequences of childhood depression (Avenevoli, Knight, Kessler, & Merikangas, 2008), a better understanding of the etiology of depression in youth is needed. More specifically, the identification of contextual factors that contribute to the onset of childhood depression may help to improve prevention and intervention efforts for this population.

One potentially important influence is the family environment of the child. Expressed emotion (EE) is a measure of the affective climate of the family that offers promise in understanding depression risk in children. EE is a robust predictor of relapse for a variety of disorders including depression (for a review, see Butzlaff & Hooley, 1998). Although initially used to study relapse rates among adults, researchers have begun to use measures of EE in studies of children and adolescents (e.g., Nelson, Hammen, Brennan, & Ullman, 2003; Schwartz, Dorer, Beardslee, Lavori, & Keller, 1990). These studies have suggested that one component of EE, criticism (EE-Crit), has the strongest validity among pediatric samples. Specifically, studies have found that mothers of depressed children exhibit higher levels of EE-Crit than mothers of nondepressed children (e.g., Asarnow, Tompson, Hamilton, Goldstein, & Guthrie, 1994; Asarnow, Tompson, Woo, & Cantwell, 2001; Hirshfeld, et al., 1997; Silk et al., 2009). There is evidence from one study showing that EE-Crit predicts risk for a future depressive episode in children (Silk et al., 2009). Despite the strengths of this literature, a key limitation is that past research has focused on levels of EE-Crit assessed at a single time point. It is unclear, however, whether a single assessment is adequate to capture the general level of maternal criticism to which the child is exposed. In the current study, therefore, we assessed mothers' levels of EE-Crit four times over 6 months to determine whether we could identify distinct latent classes of mothers exhibiting high versus low levels of EE-Crit over time and whether latent class membership would exhibit stronger predictive validity than levels of EE-Crit obtained from a single assessment.

When evaluating the influence of family factors on children's depression risk, one has to consider the role of family history of depression, which is one of the strongest predictors of depression risk in children (for reviews, see Goodman, 2007; Hammen, 2009). There is also evidence for a link between maternal depression and EE-Crit, with depressed mothers more likely to exhibit EE-Crit than nondepressed mothers (e.g., Nelson et al., 2003; Tompson et al., 2010). A key question, therefore, is whether EE-Crit predicts unique risk for depression onset in children above and beyond that contributed by maternal depression. Although there is evidence from one study that levels of EE-Crit predicted risk for a future depressive episode in a sample of children at high risk for depression due to a family or personal history of past depression (Silk et al., 2009), this study did not formally control for the influence of maternal depression because all of the children were at high risk. Therefore, the extent to which EE-Crit may contribute unique risk for depression in children remains unclear. Another possibility is that EE-Crit and maternal depression may interact to predict children's depression risk. That is, EE-Crit may moderate the link between maternal and child depression such that children of depressed mothers who are also exposed to high levels of EE-Crit are at particularly elevated risk of developing depression themselves. In the current study, we tested both of these possibilities.

Method

Participants

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

Measures

The Schedule for Affective Disorders and Schizophrenia – Lifetime Version (SADS-L; Endicott & Spitzer, 1978) and the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) were used to assess for current DSM-IV Axis I disorders in mothers and their children, respectively. Both measures are widely used diagnostic interviews with well-established psychometric properties (Endicott & Spitzer, 1978; Kaufman et al., 1997). The SADS-L and K-SADS-PL were administered by separate interviewers. For the K-SADS-PL, mothers and children were interviewed separately. As noted above, 52 mothers met criteria for MDD during their child's life. Of these 52 mothers, 8 were currently depressed at the baseline assessment and 28 had a history of recurrent depression during their lives. The K-SADS-PL was used to assess for clinically significant episodes of major and minor depression in children.¹ At the initial assessment, 21 children met lifetime criteria for a depressive diagnosis (current = 4, past = 17). The depression section of the K-SADS was also administered at the follow-up assessment to determine whether children met criteria for major or minor depression during the follow-up period and, if so, the date of onset. During the follow-up, 9 children, all from the high-risk group, met criteria for a new depressive episode (4 with a first onset and 5 with a recurrence, none of whom met criteria for a current episode at the initial assessment). A subset of 20 SADS-L and 20 K-SADS-PL interviews from this project were coded by a second interviewer and kappa coefficients for depressive diagnoses were excellent ($\kappa = 1.00$).

Mothers' and children's symptoms of depression were assessed using the Beck Depression Inventory- II (BDI-II; Beck, Steer, & Brown, 1996) and Children's Depression Inventory (CDI; Kovacs, 1981), respectively. Numerous studies have supported the reliability and validity of both measures (e.g., Beck et al., 1996; Kovacs, 1981). In the current study, both

¹Consistent with Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978), as well as past research studies of youth focusing on diagnoses of minor depression (e.g., Stone, Hankin, Gibb, & Abela, 2011), criteria for minor depression included the presence of a criterion A symptom plus at least one symptom from criterion B, which lasted for at least two weeks and resulted in clinically significant impairment.

the BDI-II and the CDI exhibited good internal consistency (α s = .92-.93 and .76-.86, respectively, across all time points).

The Five Minute Speech Sample (FMSS; Magaña et al., 1986) was used to assess mothers' levels of Expressed Emotion-Criticism (EE-Crit). To administer the FMSS, the mother is asked to speak for five uninterrupted minutes about her child and how the mother and child get along together. The response is audiotaped and coded by a certified rater for levels of EE-Crit. Mothers are rated as high on EE-Crit if any of the following three criteria are met: their initial statement about the child is negative, they report a negative relationship, or they report one or more criticisms as defined by the FMSS coding system. Mothers are rated as borderline critical if they express dissatisfaction with the child not severe enough to be rated as a criticism. Responses to the FMSS were assigned values of 2, 1, and 0 to reflect high, borderline-high, and low EE-Crit, respectively.² A number of studies have supported the reliability and validity of the FMSS EE-Crit subscale (e.g., Asarnow et al., 2001; Magaña et al., 1986). In this study, the FMSS was administered and coded by individuals blind to the other study variables. Coders were trained to reliability standards by the creator of the FMSS (Ana Magana-Amato). All samples were independently coded by two raters and when discrepancies arose, a third rater was consulted and a consensus rating was reached. To assess inter-rater reliability, a subsample of 20 FMSSs was also coded by the creator of the FMSS (Ana Magana-Amato) and inter-rater agreement for the three-level EE-Crit classifications was excellent (95% agreement). Baseline (Time 1) levels of maternal criticism yielded the following classification: high ($n = 16$), borderline ($n = 25$), and low ($n = 59$).

Procedure

Potential participants were recruited from the community through a variety of means (e.g., newspaper and bus advertisements, flyers). Mothers responding to the recruitment advertisements were initially screened over the phone to determine potential eligibility. Those reporting either significant depressive symptoms during the child's lifetime or no lifetime history of depression were invited to participate in the study. Upon arrival at the laboratory, mothers were asked to provide informed consent and children were asked to provide assent to be in the study. Next, the mother completed the Five Minute Speech Sample (FMSS) and was administered the K-SADS-PL interview by a research assistant. During this time, the child completed the CDI and other assessments in a separate room. After completing the K-SADS-PL with the mother, the same interviewer then administered the K-SADS-PL to the child. While children were being administered the K-SADS-PL, the mother completed the BDI-II, and was then administered the SADS-L by a separate interviewer. Short-term follow-up assessments occurred 2, 4, and 6 months after the initial assessment, during which mothers completed the FMSS and BDI-II and children completed the CDI. Families were invited to participate in a follow-up assessment approximately 20 months after the initial assessment ($M = 20.25$ months, $SD = 4.89$) at which point they were administered the depression section of the K-SADS to assess for the onset of depressive diagnoses since the initial assessment. Families were compensated \$125 for their participation. All study procedures were approved by the University's Internal Review Board.

²Although we examined baseline levels of EE-Crit continuously, we also ran analyses to determine if our results would differ by dichotomizing EE-Crit to examine low vs. borderline and high (c.f., Hooley & Parker, 2006) and also low and borderline vs. high. Our baseline EE-Crit results did not differ based on how we classified EE-Crit.

Results

Of the 100 mother-child pairs participating in the initial assessment, 90, 89, and 90 participated at the 2, 4, and 6-month follow-ups, respectively, and 75 families participated in the 20-month follow-up. Given the presence of missing data, we examined whether the data were missing at random, thereby justifying the use of data imputation methods for estimating missing values (Shafer & Graham, 2002). Little's missing completely at random (MCAR) test, for which the null hypothesis is that the data are MCAR (Little & Rubin, 1987) was non-significant, $\chi^2(426) = 428.62, p = .46$, supporting the imputation of missing values. Given these results, maximum likelihood estimates of missing data were created and used in all subsequent analyses (see Shafer & Graham, 2002), giving us an effective sample size of 100 children for all analyses.³

Next, to determine whether there were distinct groups of mothers exhibiting high versus low EE-Crit over the first 6 months of follow-up (4 assessments), we conducted latent class growth analysis using Mplus software (Version 5.1; Muthen & Muthen, 2008). We first fit a model that allowed for only 1 class. This was compared to our second model which tested for two classes. Note that for each of the model comparisons, the comparison is made to the fit of a model with one less class (i.e., a two class model is compared to a one class model, and a 3-class model is compared to a two class model, etc.). Model comparisons were made using the following fit indices: entropy (a measure of classification uncertainty - higher value indicates more distinguishable classes), Bootstrapped Likelihood Ratio Test (BLRT), Vuong-Lo-Mendell-Rubin Likelihood Ratio Test (VLRT) and the Lo-Mendell-Rubin Adjusted Likelihood Ratio Test (LRT). BLRT is considered the gold standard. As shown in Table 1, we tested models that allowed for up to four classes. The results suggest that there are 2, and possibly 3, classes that exist within our data; however, the size of the third class is very small ($n = 5$). The creation of the third class inflated the relationship between two of the classes ($r_{Class1-Class3} = .16$) and entropy was slightly lower, indicating more classification uncertainty. Based on these results, it appears that the most parsimonious model is the 2 Class model (entropy = .79, BLRT < .001). The low EE-Crit class composed 79% of the sample and, of these mothers, 41.8% of mothers exhibited low EE-Crit at all four time points, with the remaining mothers being rated as either borderline (41.8%) or high (16.4%) during at least one time point. The remaining 21% of our sample were in the high EE-Crit class. Of these mothers, 23.8% exhibited high EE-Crit at all four time points, with the remaining mothers being rated as either borderline (33.3%) or low (42.9%) during at least one time point. EE-Crit latent class membership was significantly related to baseline EE-Crit ratings, $\chi^2(2, N = 100) = 34.84, p < .001, r_{effect\ size} = .59$. Mothers' MDD history (yes versus no) was not significantly related to their baseline EE-Crit ratings, $\chi^2(2, N = 100) = 1.92, p = .38, r_{effect\ size} = .04$, or EE-Crit latent class, $\chi^2(1, N = 100) = 1.05, p = .31, r_{effect\ size} = .10$. Analyses were then conducted to determine whether any of the study variables were related to children's age or gender. None of these analyses was significant.

Next, using survival analyses, we tested the hypothesis that levels of EE-Crit would predict prospective onsets of depressive episodes. Because we focused on levels of EE-Crit during the first 6 months of follow-up, the one child that had an onset of MDD before the 6-month follow-up was excluded from these analyses as were the 4 children who met criteria for current MDD at Time 1. The final sample consisted of 95 children, of whom 9 (9.4%) experienced a depressive episode during the follow-up. In these analyses, we examined the impact of EE-Crit latent class and baseline levels of EE-Crit in separate survival analyses.

³We focused on estimated data because this provides more reliable parameter estimates than those obtained with pairwise or listwise deletion of missing data (Shafer & Graham, 2002). This said, we should note that the pattern of significant findings was identical when we focused only on participants with complete data.

Although T1 EE-Crit did not predict depression onset in children, EE-Crit latent class significantly predicted the onset of children's depressive diagnoses between the 6- and 20-month follow-up, $Wald = 7.73, p < .01$, odds ratio (OR) = 6.60 (see Figure 1), and was a significantly better predictor than was T1 EE-Crit ($z = 3.45, p < .001$). To determine whether the predictive validity of EE-Crit would be maintained even after accounting for the influence of maternal depression, we entered mothers' average levels of depressive symptoms during the first 6 months of follow-up in the second step of the survival analysis. Maternal depressive symptoms was a significant predictor in this step, $Wald = 13.08, p < .01, OR = 3.45$. Importantly, the predictive validity of EE-Crit latent class remained significant, $Wald = 4.15, p < .05, OR = 4.51$, suggesting that the influence of EE-Crit on children's risk for depression is at least partially independent of the impact of maternal depression. Further, the predictive validity of EE-Crit was maintained even when statistically controlling for the influence of children's average depressive symptom levels during the first 6 months of follow-up, $Wald = 5.78, p < .05, OR = 5.47$, suggesting that the results were not due simply to children's concurrent depressive symptoms during the first 6 months. Finally, although we also examined whether maternal depression moderated the link between EE-Crit (T1 or latent class) and childhood depression onset, these analyses were not significant (lowest $p = .88$).

Discussion

The primary aim of this study was to test the hypothesis that maternal criticism (EE-Crit) would predict the onset of new depressive episodes in children during a 20-month follow-up period. We also sought to determine whether EE-Crit would predict unique risk for children's depression onset above and beyond that contributed by maternal depression. Using latent class analysis to examine patterns of EE-Crit exhibited by mothers over 4 assessments during the first 6 months of follow-up, we found evidence for two distinct latent classes, with 79% of our sample classified as low criticism and the remaining 21% of our sample classified as high criticism. Children of these highly critical mothers were significantly more likely to develop a diagnosis of depression during the follow-up than were children of mothers expressing low criticism. Importantly, these results were maintained even after statistically controlling for mothers' (and children's) levels of depressive symptoms during the first 6 months of follow-up (assessed concurrently with EE-Crit) suggesting that maternal criticism predicts depression onset in children above and beyond that contributed by maternal depression. Latent class membership was also a stronger predictor of depression onset than levels of EE-Crit exhibited at the initial assessment, which did not significantly predict children's depression risk.

These results suggest that a single assessment of EE-Crit may not be sufficient for capturing typical levels of maternal criticism within the home. Specifically, although baseline measures of criticism and latent class memberships of criticism were significantly related, only latent class membership based on levels of criticism exhibited during four assessments over six months significantly predicted children's risk for future depression onset. That is, findings suggest that chronic or continuous exposure to maternal criticism based on latent class analysis, as opposed to isolated incidents of high levels of criticism, appears to be particularly detrimental in predicting which children are likely to experience a depressive episode, even among an already high-risk sample. Although the precise mechanism for this relation remains unclear, a possible explanation for these findings is that chronic elevations of maternal criticism may increase the frequency and intensity of children's negative affect and may interfere with the development of adaptive skills for regulating emotions, which typically are acquired and refined in the context of parent-child interactions (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Another possibility is that continuous maternal criticism may foster the development of negative cognitive styles in children. Rose &

Abramson (1992) hypothesized that verbal abuse may exert particularly salient impact by fostering the development of negative inferential styles in children. If maternal criticism is conceptualized as a less severe form of negative parent-child interaction, future researchers may consider exploring whether the development of children's negative cognitions mediates the link between chronic maternal criticism and children's risk for depression.

We should also highlight the nonsignificant findings from our study. First, we found no support for the idea that EE-Crit may moderate the link between maternal and child depression. This said, however, the current sample may not have allowed adequate power for a true test of this hypothesis. For example, because only children of mothers with a history of MDD developed depressive diagnoses during the follow-up, we could not include mother MDD history as a predictor in our models and had to rely on maternal depressive symptoms instead. Future research, therefore, is needed to determine whether maternal MDD moderates the link between EE-Crit and childhood depression onset. Second, levels of EE-Crit (baseline or latent class groups) were not significantly associated with mother's history of MDD. This is in contrast to the majority of previous research, which has found support for this relation (e.g., Nelson et al., 2003; Tompson et al., 2010). However, as we reported in previous analyses with this sample (Gibb, Uhrlass, Grassia, Benas, & McGeary, 2009), levels of EE-Crit during the first 6 months of our follow-up were significantly related to concurrent levels of depressive symptoms, suggesting that maternal criticism may be driven more by the presence of current depression than a past history of MDD.

The current study has several notable strengths including the multi-wave prospective design, high-risk sample, and inclusion of diagnostic interviews, which allowed us to accurately assess onsets of depressive episodes in children of depressed mothers. Further, by using the prospective design, we were able to use latent class analysis to identify distinct groups of mothers based on levels of EE-Crit displayed over time. Despite these strengths, however, limitations of the current study should be noted as they may provide directions for future research. First, given the size of our sample, we were unable to examine potentially important moderating variables (e.g., child gender or age). Second, the base rate of new depression diagnoses during the follow-up was relatively low, which precluded us from examining first onsets versus recurrences individually. Therefore, future studies are needed to determine if maternal criticism predicts first onset of MDD in children. Third, the current study did not consider potential bidirectional relations between childhood depression and maternal criticism. That is, research suggests that when children behave in difficult ways, parents may respond with more difficult parenting behaviors (e.g., Forbes et al., 2008). Future research, therefore, should examine how maternal criticism and child depression may operate within a viscous cycle of risk. Finally, our study focused exclusively on mothers and their children. However, it is also clear that fathers influence youth's risk versus resilience to depression (Pargas, Cristina, Brennan, Hammen, & Le Brocque, 2010). Future research, therefore, is needed to understand the impact of fathers, not only in terms of potentially exacerbating or mitigating the effects of maternal criticism, but also in terms of better understanding the potential impact of paternal criticism.

Implications for Research, Policy, and Practice

The current results have a number of potentially relevant implications. Most importantly, children exposed to repeatedly high levels of maternal criticism were at the highest risk for depression onset during the follow-up. This study, therefore, highlights a potential modifiable risk factor for childhood depression – repeated exposure to maternal criticism. These findings suggest that clinical interventions for youth, even those already at high-risk for depression, may be strengthened by specifically targeting levels of maternal criticism. The development of intervention programs teaching mothers more adaptive ways of interacting with their child may help to reduce depression risk in youth.

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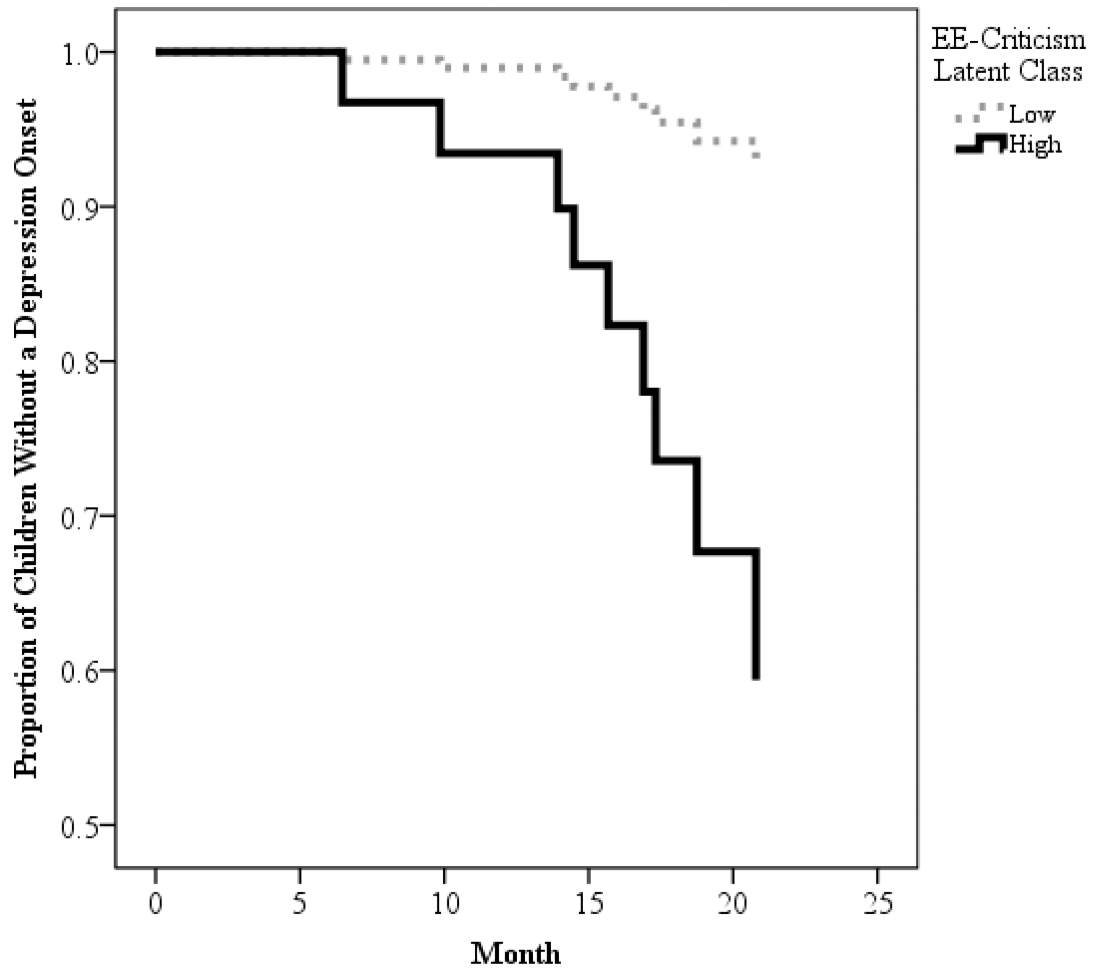


Figure 1. Results of survival analysis predicting time to depression onset among children of depressed mothers as a function of latent class EE-Criticism.

Table 1

Results of Latent Class Growth Analyses

Model	Individuals per Class				Comparative Indices					
	Class 1	Class 2	Class 3	Class 4	AIC	SS-BIC	Entropy	VLTR	LRT	BLRT
Class 1	100	-	-	-	705.41	703.75	-	-	-	-
Class 2	79	21	-	-	651.51	648.19	0.79	0.002	0.003	<0.001
<i>r_{Class1-Class2}</i> = .08										
Class 3	67	5	28	-	639.67	634.69	0.78	<0.001	<0.001	<0.001
<i>r_{Class1-Class3}</i> = .16										
Class 4	24	69	5	2	642.79	636.16	0.83	0.59	0.60	0.50

Abbreviations: BLRT - Bootstrapped Likelihood Ratio Test for (K versus K-1 classes); VLRT - Vuong-Lo-Mendell-Rubin Likelihood Ratio Test; LRT - Lo-Mendell-Rubin Adjusted Likelihood Ratio Test for (k versus K-1 classes).