Maternal Depression, Child Frontal Asymmetry, and Child Affective Behavior as Factors in Child Behavior Problems

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Abstract

**Background:** Despite findings that parent depression increases children’s risk for internalizing and externalizing problems, little is known about other factors that combine with parent depression to contribute to behavior problems. **Methods:** As part of a longitudinal, interdisciplinary study on childhood-onset depression (COD), we examined the association of mother history of COD, child frontal electroencephalogram asymmetry, and affective behavior with children’s concurrent behavior problems. **Results:** Children in the COD group had higher anxious/depressed and aggressive problems than did children in the control group, but this was qualified by a COD-by-asymmetry interaction effect. For COD but not control children, left frontal asymmetry was associated with both anxious/depressed and aggressive child problems. Children with left frontal asymmetry and low affect regulation behavior had higher anxious/depressed problems than did those with high affect regulation behavior. Boys with left frontal asymmetry had higher aggressive problems than did those with right frontal asymmetry. **Conclusions:** In children of mothers with COD, physiological and behavioral indices of affect regulation may constitute risks for behavior problems. **Keywords:** maternal depression, behavior problems, affect regulation, psychophysiology, parent-child interaction. **Abbreviations:** COD = childhood-onset depression.
Maternal Depression, Child Frontal Asymmetry, and Child Affective Behavior as Factors in Child Behavior Problems

Children of parents with depression are at increased risk for developing different forms of psychopathology (Beardslee, Versage, & Gladstone, 1998; Downey & Coyne, 1990). Risk for disrupted functioning may be especially grave for children of parents with childhood-onset depression (COD), a variant of the disorder characterized by greater familiality and chronicity than adult-onset depression (Kessler, Avenevoli, & Merikangas, 2001; Kovacs, Devlin, Pollock, Richards, & Mukerji, 1997). Children of depressed parents are at particular risk for depression (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; Weissman et al., 1987), but diagnosable depressive disorders typically do not appear until late childhood (Fleming & Offord, 1990). In studying young children of depressed parents, it is thus important to identify behavioral and psychophysiological markers of later psychiatric disorders. Symptoms among related psychopathology dimensions, such as depression and aggression, may be valuable in this respect because they reflect the types of problems that children of depressed parents tend to develop (Downey & Coyne, 1990). Similarly, behavioral and physiological indicators of affect regulation may foreshadow later depression-relevant problems with emotions such as sadness and positivity (Kovacs & Devlin, 1998).

What distinguishes the children of depressed parents who develop behavior problems? Affect regulation has been proposed as a link between risk and behavior problems in children of depressed parents (Ashman & Dawson, 2002; Field, 1994). Infants of depressed mothers have difficulty with regulatory tasks such as reducing the frequency and intensity of negative affect or enhancing the frequency of positive affect (Cohn & Campbell, 1992; Forbes, Cohn, Allen, & Lewinsohn, 2004). Children who have internalizing problems or depressive disorders regulate
affect less effectively than do children without such adjustment problems (Eisenberg et al., 2001; Garber, Braafladt, & Weiss, 1995; Silk, Steinberg, & Morris, 2003).

Affect regulation includes physiological and behavioral components (Gross & Muñoz, 1995), both of which are expected to influence pathways to child psychopathology (Cicchetti & Toth, 1998). Frontal electroencephalogram (EEG) asymmetry reflects the balance of brain activation in left and right frontal areas and is considered an important biological correlate of affect regulation and risk for depression (Fox, 1994). Left frontal asymmetry reflects greater activation in left-hemisphere relative to right-hemisphere frontal areas, and right frontal asymmetry reflects the opposite pattern. Left frontal asymmetry is postulated to be associated with approach behavior, including both positive affect and anger, and right frontal asymmetry is postulated to be associated with withdrawal behavior (Davidson, Jackson, & Kalin, 2000; Fox, 1991). Right frontal asymmetry has been the focus of research with children and has been associated with social wariness (Fox et al., 1995; Henderson, Fox, & Rubin, 2001), behavioral inhibition (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001), and crying during maternal separation (Davidson & Fox, 1989). Infants of depressed mothers exhibit right frontal asymmetry (Dawson et al., 1999), and this pattern of asymmetry is presumed to be a trait characteristic of risk for depression. As for behavior problems, there is some evidence to suggest that right frontal asymmetry is linked to early internalizing and externalizing problems (Fox, Schmidt, Calkins, Rubin, & Coplan, 1996). Gender-by-asymmetry interactions may merit consideration: girls with anxiety or externalizing problems have been reported to exhibit right frontal asymmetry, whereas boys with anxiety exhibited left frontal asymmetry (Baving, Laucht, & Schmidt, 2002, 2003). However, relatively few such studies have been carried out in preschool- and school-age children at risk for psychopathology.
Studies with adults suggest that left frontal asymmetry could also play a role in the development of behavior problems. Consistent with the approach-withdrawal perspective, adults with left frontal asymmetry exhibit trait-like anger and aggressive responses to provocation (Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001). Left frontal asymmetry may thus constitute a risk factor for externalizing problems, especially aggression, in children at risk for depression. A previous study with adults from the current sample also lends support to this claim: those with comorbid COD and externalizing disorders had left frontal asymmetry (Miller et al., 2002).

Variability in children’s affect regulation can also be ascribed to external sources (Calkins, 1994), such as parent-child relationships. The mother-child relationship is an important context for learning affect regulation, and difficulties in this context are hypothesized to influence poor behavioral outcomes in children of depressed mothers (Goodman & Gotlib, 1999; Gotlib & Goodman, 2002). Negative and inappropriate affect during mother-child interactions may be especially important in predisposing children to develop poor affect regulation, as indicated by studies with children and depressed adolescents (Eisenberg, Fabes, & Murphy, 1996; Sheeber, Allen, Davis, & Sorensen, 2000; Sheeber & Sorensen, 1998).

Associations between maternal depression and child behavior problems are complex and multiply determined (Cummings, Davies, & Campbell, 2000), and it is not surprising that the two have not been uniformly related. For instance, previous studies have found that depressed mothers and their children do not differ in affect from healthy dyads (Cohn & Campbell, 1992; Seifer, Sameroff, Anagnostopolou, & Elias, 1992). Other factors may be important to consider. First, depression is a heterogeneous disorder, and focusing on a specific subtype, such as COD, may allow relations with behavior problems to emerge. Second, assessing physiology variables
such as frontal asymmetry would broaden the scope to include biologically based risk factors. Finally, considering several risk factors at once and measuring factors independently is promising. Maternal history of depression, children’s frontal asymmetry, and children’s affect regulation behavior may each play a role, but it may be only in the context of multiple risk factors that relations between these factors and child behavior problems are evident. A developmental psychopathology approach indicates that risk factors across domains are linearly (Sameroff, Seifer, & Zax, 1982; Shaw, Winslow, Owens, & Hood, 1998) or multiplicatively (Rutter et al., 1975) associated with an increased risk of child adjustment problems.

The focus of the current study was whether frontal asymmetry and affect regulation behavior contribute in an additive or interactive manner to behavior problems in children of depressed mothers. The families in the study were part of a broader, multimethod, longitudinal study whose overarching goal was to describe the characteristics, development, and utilization of affect regulation among COD offspring. The current study is unique with respect to several features. First, physiology was assessed independent of mother-child interaction. Second, child affect regulation behavior was assessed on dimensions – such as negative affect, positive affect, and appropriate expression – critical to the development of behavior problems in previous literature. We predicted that in children of psychiatrically well mothers with a rigorously defined history of depression, right frontal asymmetry would be associated with high levels of depression-related problems, while left frontal asymmetry would be associated with high levels of aggressive problems, and that children characterized by risk factors across domains would have a higher probability of showing problem behavior.
Method

Participants

Participants were 74 mother-child dyads, 44 of whom had a maternal history of COD. Participating families were part of a longitudinal program project that included several laboratory assessments, including a psychophysiology assessment and a mother-child interaction assessment. Children were between the ages of 3 and 9 years. This age range was selected because of the importance of affect regulation and the increased stability relative to other periods. Other studies using similar procedures for eliciting affect regulation have included children from similar developmental periods and age spans (Cole, 1986). All families who had participated in both assessments were included, but an additional two control families were excluded because the mother developed a psychiatric disorder after study entry. All offspring in the COD group in the target age range were assessed (32 families total). One child per control family was assessed.

Table 1 presents the sample characteristics. Children in the COD and control groups did not differ in age, ethnicity, or handedness. COD and control mothers did not differ in education level, with 80% in the control group and 92% in the COD group having at least a high school diploma. Data analyses for frontal asymmetry hypotheses were also conducted without left-handed participants, confirming that the presence of left-handed children did not influence results.

Participants were drawn from a collaborative longitudinal study on genetic, psychophysiological, and behavioral factors related to the adjustment of offspring of adults with
COD (Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984). Inclusion was based on a maternal history of childhood-onset major depression or dysthymia. COD mothers were recruited from treatment programs at the Western Psychiatric Institute and Clinic in Pittsburgh, Pennsylvania, through prior research studies, or through community advertisements. Control mothers were recruited through a marketing directory, newspaper advertisements, and other studies.

Maternal history of depression was determined through the administration of structured clinical interviews and a review of childhood psychiatric records. Diagnosis of COD was made from clinical interviews conducted when mothers were children and confirmed in subsequent follow-up interviews. COD mothers had received diagnoses of major depressive disorder or dysthymia before age 14 years (n = 39) or bipolar spectrum disorders before age 17 (n = 5). (The results reported below did not differ when families with early-onset bipolar spectrum disorders were excluded). Seventeen COD mothers were participants in a longitudinal, naturalistic follow-up study of COD and had undergone multiple psychiatric assessments over the course of up to 20 years. This subsample was evaluated during childhood using the Interview Schedule for Children and Adolescents (Sherrill & Kovacs, 2000). COD mothers recruited as adults and control mothers were administered the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995). Mothers’ current depressive symptoms were measured with the Beck Depression Inventory (Beck, Steer, & Garbin, 1988), a reliable and valid self-report measure of depressive symptomatology.

*Design, Procedure, and Data Quantification*

*Laboratory visits.* Participants visited two laboratories: a psychophysiology laboratory, in which resting frontal asymmetry was recorded; and a mother-child interaction laboratory, in
which mother-child interaction was observed. Data included in the current study are from a subset of tasks from the two laboratory assessments, as each laboratory visit involved several tasks. Participants completed affect-eliciting tasks in the psychophysiology laboratory and other tasks in the mother-child laboratory. The two laboratory visits occurred within 2 months of each other for 88% of the participants, with 6 months as the greatest lag between the two visits. After the physiology assessment, children were administered an 11-item behavioral version of the Edinburgh Handedness Inventory (Oldfield, 1971) adapted for children. During the mother-child interaction assessment, mothers completed questionnaires about their current depressive symptoms and the child’s behavior.

*Frontal asymmetry.* EEG was recorded during six 30-s resting segments, during which children sat quietly and alternately looked at a small model spaceship or closed their eyes. EEG was recorded with an electrode cap (Electro-Cap International) placed according to standard landmarks. The following sites were included: mid-frontal (F3, F4), lateral frontal (F7, F8), central (C3, C4), anterior temporal (T7, T8), mid-parietal (P3, P4), and occipital (O1, O2). Online recordings were referenced to the vertex (Cz), then re-referenced to a whole-head average. The signal was amplified with a gain of 5000 and bandpass filtered at 1–100 Hz. Data were digitized on-line at a sampling rate of 512 Hz per channel. Electrode impedances were below 5 kOhms, and impedances for homologous sites were within 0.5 kOhms. Vertical and horizontal electrooculogram (EOG) data were used to identify and manually remove eye movement artifact. Artifact rejection was conducted by two trained coders, who visually inspected data from EOG and EEG channels for the entire resting period and manually removed data from epochs that included blinks, horizontal or vertical eye movements, or motor activity. EOG was recorded using tin cup electrodes, with vertical EOG electrodes placed on the suborbital and supraorbital
areas around the right eye, and horizontal EOG electrodes placed on the left and right outer canthi.

EEG data were inspected visually by coders blind to group status, and artifact related to eye movement, gross motor activity, and muscle tension was removed manually. The EEG signal was quantified with discrete Fourier transformation (DFT) using a Hanning window 1-s wide and with 50% overlap. Prior to DFT computation, the mean voltage was subtracted from each data point to eliminate any influence of DC offset. Power (in units of picowatt-Ohms or $\mu V^2$) was computed for 1-Hz frequency bins for frequencies between 1 and 30 Hz. The frequency range of interest was the alpha band, which is putatively inversely related to brain activation (Pfurtscheller, Stancak, & Neuper, 1996). Based on an examination of each participant’s EEG activity in single-Hz bins and developmental findings (Marshall, Bar-Haim, & Fox, 2002), the alpha range was defined as 7–10 Hz for 3–5-year-olds and 8–11 Hz for 6–9-year-olds. Alpha power values (in picowatt-Ohms or $\mu V^2$) for each electrode site were weighted by the number of artifact-free epochs in each segment and averaged across segments. Average values were subjected to a natural-logarithm transformation to normalize distributions (Gasser, Bacher, & Mocks, 1982).

Following a widely used approach (Davidson, Jackson, & Larson, 2000), asymmetry scores were computed as the difference of log-transformed power scores for midfrontal leads (F3 and F4). Dichotomous frontal asymmetry variables were then computed, based on a conservative strategy (Fox et al., 2001) in which direction of asymmetry is considered more meaningful than degree of asymmetry. The underlying construct is conceptualized as dichotomous (Fox, 1991), and continuous scores may be subject to measurement error. Further, findings reported below did not differ when continuous score was included. Thus, participants with asymmetry scores > 0
were classified as left activated (COD n = 20, control n = 15); those with asymmetry scores ≤ 0 were classified as right activated (COD n = 24, control n = 15).

**Affect Regulation Behavior.** Mothers and children engaged in a 25-min series of 4-5 tasks designed to elicit (1) both positive and negative affect in children and (2) comparable levels of emotion for children varying in developmental status. For example, at age 3, tasks involved a dinosaur puzzle; etch-a-sketch (fine-motor drawing board), stack-n-pop (motor and balance), and toss-a-cross (gross motor and hand-eye coordination) games; and exposure to a wiggle ball (a ball with flashing lights that emits shrill sounds). At age 5, tasks included “Hungry Hungry Hippos” (a competitive game involving fine motor skills and speed), a marble game, a naming game (e.g., name things that fly), a shape sorter task, and a tractor treader (a large wheel that turns when a child crawls while inside it). At least one task within each group was selected to provide a provocative or frightening element (e.g., wiggle ball at age 3).

Child affective behaviors were subsequently coded from videotapes by two-person coding teams. The following behaviors were coded within 10-sec intervals: positive affect, negative affect, disruptiveness, task involvement, and task uninvolvement. Additionally, coders rated the following behaviors on 4-point global scales after viewing all tasks: positivity toward mother, negativity toward mother, involvement with tasks, involvement with mother, appropriate affect, inhibition, and sociability. Kappas for coder reliability ranged between .59 and .76, with a mean of .65. These levels are consistent with established reliability expectations for observational codes (Mitchell, 1979). Behaviors occurring on average less than once per dyad were coded by consensus.

Behavior data were reduced using principal components analysis with varimax rotation and a 2-factor solution. The two factors that emerged explained 53% of the variance and
corresponded to task engagement and affect regulation. The affect regulation factor explained 16.5% of the variance (eigenvalue = 1.97, internal consistency alpha = .60) and involved a set of behaviors – positive affect, negative affect (inverse), sociability, and appropriate affect – that contribute to effective modulation of affect in social contexts. Based on our hypotheses and the literature on children at risk for depression, affect regulation was used in models for behavior problems.

Behavior problems. During their visit to the mother-child interaction laboratory, mothers completed the Child Behavior Checklist (CBCL) (Achenbach, 1991, 1998), a parent-report questionnaire on behavior problems. Mothers completed the 2–3-year-old version of the CBCL for 3-year-olds and the 4–18-year-old version for older children. Using software with age and gender norms, raw scores for all standard factors were obtained.

Based on hypotheses, the CBCL factors of anxious/depressed and aggressive problems were the focus of analyses. These factors were moderately correlated (r = .56, p < .01). Both factors have sound reliability (Achenbach, 1991, 1998). Narrow-band rather than broad-band factors (e.g., internalizing) were selected because they involve greater specificity and have lower correlations with factors tapping other problem areas than do broad-band factors.

Data Analysis

Hypotheses were tested using random effects regressions with anxious/depressed and aggressive problems as dependent variables. COD group, frontal asymmetry, affect regulation behavior, and all related 2-way interactions were included as fixed effects. Gender and the gender-by-asymmetry interaction were also included as fixed effects. Family was included as a random effect, and level of current maternal depressive symptoms was included as a covariate. When 2-way interaction effects involved the continuous variable of affect regulation, post-hoc
analyses involved creating high and low groups based on scores +/- .5 SD from the sample mean, graphing two-way interactions, and conducting analyses of variance (ANOVAs) to compare groups. Effect sizes for significant effects were computed in SD units. Statistical power considerations led to the decision not to test the 3-way interaction involving COD X frontal asymmetry X affect regulation behavior.

Because we did not predict age effects, our focus was on age-appropriate affect regulation, and age was unrelated to behavior in preliminary analyses, all models were computed with and without age (in years). Findings did not differ, and age was not included. Also, models were re-computed with parietal rather than frontal asymmetry, confirming that effects were specific to the frontal region.

Results

The analysis for anxious/depressed problems revealed significant effects for COD group, frontal asymmetry, COD X frontal asymmetry, and frontal asymmetry X affect regulation behavior (Table 2). COD children had higher anxious/depressed problems than did control children, but this main effect was qualified by an interaction. Follow-up analysis indicated that for the left frontal group only, COD children had higher anxious/depressed problems than did control children ($F(1,29) = 11.06, p < .01$), and those with low affect regulation behavior had higher anxious/depressed problems ($F(1,29) = 12.18, p < .01$) (see Figure 1). The nature of the frontal asymmetry X affect regulation interaction effects was further examined by creating high and low affect regulation groups. In the left frontal asymmetry group, children with low affect regulation had higher anxious/depressed problems than those with high affect regulation, $d = 1.40$ SD.
The analysis for aggressive problems revealed significant effects for COD group, frontal asymmetry, COD X frontal asymmetry, and gender X frontal asymmetry (Table 2). COD children had higher aggressive problems than did control children, but this main effect was qualified by an interaction. Follow-up analyses (see Figure 2) indicated that for the left frontal group only, COD children had higher aggressive problems than did control children ($F(1,29) = 10.82, p < .01$). Among boys, those with left frontal asymmetry had higher aggressive problems than did those with right frontal asymmetry ($F(1,35) = 7.49, p < .05$).

Discussion

The current study used a multimethod approach to examine factors in behavior problems in children with a parent history of COD. Physiology and behavior were measured in separate laboratories on separate occasions, and unlike other studies of the psychophysiology of risk for depression, children were assessed during early to middle childhood. As predicted, combinations of parent history of depression, child frontal asymmetry, and observer-rated child affective behavior were associated with internalizing and externalizing types of child behavior problems. Children of COD mothers who had left frontal asymmetry had high anxious/depressed and aggressive problems. Children with left frontal asymmetry who displayed low affect regulation behavior had high anxious/depressed problems. Boys with left frontal asymmetry had higher aggressive problems than did those with right frontal asymmetry.
Taken with the literature on maternal depression, our findings suggest that parent depression is a more consistent influence when children exhibit difficulties with affect regulation, either in terms of behavioral or physiological characteristics. Specifically, maternal depression in combination with frontal asymmetry was related to children’s behavior problems. This finding is consistent with findings of another recent study of maternal depression (Dawson et al., 2003), suggesting that frontal asymmetry is a good candidate for a physiological index of affect regulation tendencies in children of depressed parents. Individual differences in affect regulation thus appear to play the postulated role for children of depressed parents (Ashman & Dawson, 2002; Goodman & Gotlib, 1999).

For children with a maternal history of depression and for boys, left frontal asymmetry was associated with aggressive problems. This finding supports claims of the approach-withdrawal model, in which anger is considered an approach emotion (Fox, 1991). It is also consistent with studies reporting an association of left frontal asymmetry with adults’ normal and abnormal externalizing behaviors (Harmon-Jones & Allen, 1998; Miller et al., 2002). As such, our study is the first to link child psychophysiology research with research on the psychophysiology of anger in adults. The finding that left frontal asymmetry was only related to aggressive problems for boys also raises questions about gender differences in the relation of frontal asymmetry to aggression, an issue raised by two previous studies (Baving et al., 2002, 2003) and worthy of further investigation.

Contrary to our expectations, left frontal asymmetry in combination with maternal depression was also associated with depression-related problems. Perhaps in addition to indicating a predisposition to approach-related problems such as aggression, left frontal asymmetry in children of COD mothers serves as a general risk factor for poor affective
flexibility. Although anxious/depressed problems and aggressive problems differ in many respects, both can be viewed as involving difficulties with changing affect appropriately (e.g., enhancing positive affect or modulating anger, respectively). The claim that affective dysregulation may be common to both types of problems is supported by the correlation between anxious/depressed and aggressive problems. Sample characteristics such as age range and level of current maternal symptoms may explain the discrepancy of frontal asymmetry findings with those in previous studies, but it is also possible that frontal asymmetry is most powerfully related to children’s behavior problems when considered within the context of other factors. Our findings on aggression, along with previous findings of left frontal asymmetry in men with COD (Miller et al., 2002) hint that gender is one such factor.

Another finding also suggests that left frontal asymmetry may, in combination with other characteristics, be a risk factor for internalizing types of problems in children. Left frontal asymmetry was related to depression-related problems for children who exhibited low affect regulation behavior during mother-child interaction. This was the case even though children with low affect regulation behavior in isolation did not have higher levels of behavior problems than did those with high affect regulation behavior. It may be that children who have physiological vulnerability to poor affect regulation and also exhibit difficulty in maintaining appropriate affect in social contexts are at greatest risk for affective disorders. A common mechanism may underlie both kinds of tendencies, and repeated experiences of ineffective social exchanges may also serve to exacerbate physiological and behavioral tendencies toward poor affect regulation.

An important strength of the study is its sample. Many of the COD mothers represent a well-characterized, longitudinally assessed group that has been followed since youth. In addition, all COD mothers in the study have a pernicious form of depression. This feature allowed us to
examine children who are at especially high risk for adjustment problems and to focus on parent history of depression while accounting for current symptoms. Few studies of parental depression assess parents’ age of onset, and yet this factor plays a striking role in clinical course and severity (Costello et al., 2002). In the current study, restricting the focus to COD allowed us to reduce heterogeneity in what is a varied form of affective illness.

This study is one of few involving an intense examination of both proximal family influences and trait-like physiological influences on behavior problems, allowing the investigation of complementary rather than competing factors. For instance, although affect regulation behavior on its own was unrelated to anxious/depressed problems, it was relevant in combination with frontal asymmetry. This developmental psychopathology approach provides a step toward a more complete description of the complex, interrelated mechanisms of the pathway from parent to child psychopathology.

These results, unlike those of studies relying strictly on maternal report, cannot be attributed to shared method variance. The assessment of physiology and behavior in separate laboratories and on different days lends strength to the conclusion that COD, affect expression, and frontal asymmetry all contribute to child behavior problems. However, the study’s findings must be considered within the context of its limitations. These include the sample size, age range, and inclusion of multiple children from COD families. Our findings await replication with larger samples drawn from similar populations.

Conclusions

In all, the current study suggests that affect regulation is a fruitful construct for examining risk for psychopathology in children of depressed parents. Using clinical, physiological, behavioral, and parent-report measures of individual differences allowed testing of
hypotheses about the interplay among several factors believed to predispose children to adjustment problems. It will be valuable for future studies to examine the trajectories of children beyond early childhood and to compare children of parents with different subtypes of depression.
Acknowledgements

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Table 1. *Child Characteristics and Study Variables, by COD Group*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>COD</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 30)</td>
<td>(n = 44)</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>5.09 (1.55)</td>
<td>5.06 (1.77)</td>
</tr>
<tr>
<td>Gender (female)</td>
<td>43%</td>
<td>43%</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>European-American</td>
<td>60%</td>
<td>36%</td>
</tr>
<tr>
<td>African-American</td>
<td>27%</td>
<td>32%</td>
</tr>
<tr>
<td>Latino</td>
<td>0%</td>
<td>5%</td>
</tr>
<tr>
<td>Asian-American</td>
<td>3%</td>
<td>0%</td>
</tr>
<tr>
<td>Mixed&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10%</td>
<td>27%</td>
</tr>
<tr>
<td>Frontal asymmetry (left)</td>
<td>54%</td>
<td>46%</td>
</tr>
<tr>
<td>Affect regulation</td>
<td>.17 (.65)</td>
<td>-.12 (1.18)</td>
</tr>
<tr>
<td>Behavior problems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggressive Problems</td>
<td>7.38 (5.13)</td>
<td>12.05 (7.70)</td>
</tr>
<tr>
<td>standard (T)</td>
<td>53.24 (5.53)</td>
<td>58.77 (10.20)</td>
</tr>
<tr>
<td>Anxious/Depressed Problems</td>
<td>1.86 (1.90)</td>
<td>3.37 (2.98)</td>
</tr>
<tr>
<td>standard (T)</td>
<td>50.90 (2.35)</td>
<td>53.70 (5.18)</td>
</tr>
</tbody>
</table>

*Note.* Values are mean (SD) or percentages. Behavior problems are presented as raw and T scores (*M* = 50, *SD* = 10). COD = childhood-onset depression. Two mothers (1 COD) did not complete behavior problem questionnaires.

<sup>a</sup>Primarily mixed European-American and African-American.
### Table 2. Random Effects Regression Analyses for Child Behavior Problems

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient (SE)</th>
<th>F</th>
<th>Coefficient (SE)</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>-.43 (1.19)</td>
<td>1.59</td>
<td>-5.27 (3.90)</td>
<td>.67</td>
</tr>
<tr>
<td>COD</td>
<td>.95 (1.83)</td>
<td>4.16**</td>
<td>-3.92 (7.66)</td>
<td>8.11**</td>
</tr>
<tr>
<td>Frontal asymmetry</td>
<td>.09 (1.33)</td>
<td>4.54*</td>
<td>-2.59 (6.77)</td>
<td>10.79**</td>
</tr>
<tr>
<td>Affect regulation behavior</td>
<td>2.22 (1.51)</td>
<td>.47</td>
<td>-.54 (14.55)</td>
<td>.59</td>
</tr>
<tr>
<td>COD X frontal asymmetry</td>
<td>4.67 (1.69)</td>
<td>7.63*</td>
<td>23.84 (7.30)</td>
<td>10.68**</td>
</tr>
<tr>
<td>COD X affect regulation</td>
<td>-1.40 (1.52)</td>
<td>.85</td>
<td>.26 (15.01)</td>
<td>.54</td>
</tr>
<tr>
<td>Frontal asymmetry X affect regulation</td>
<td>-2.62 (.86)</td>
<td>9.28**</td>
<td>2.51 (14.90)</td>
<td>.24</td>
</tr>
<tr>
<td>Gender X frontal asymmetry</td>
<td>-.08 (1.24)</td>
<td>.00</td>
<td>8.62 (3.93)</td>
<td>4.82*</td>
</tr>
<tr>
<td>Random Effect(a)</td>
<td>5.37 (1.07)</td>
<td>5.00***</td>
<td>50.31 (10.16)</td>
<td>4.95***</td>
</tr>
</tbody>
</table>

Note. COD = childhood-onset depression (in mother). df = 1,50 for all tests. *Test statistic = Wald Z.

* \(p < .05\)  ** \(p < .01\)  *** \(p < .001\)
Figure Captions

Figure 1. Children’s anxious/depressed problems as predicted by (1) COD X frontal asymmetry and (2) affect regulation X frontal asymmetry. Error bars represent 1 SE of the mean.

Figure 2. Children’s aggressive problems as predicted by (1) COD X frontal asymmetry and (2) gender X frontal asymmetry. Error bars represent 1 SE of the mean.
Maternal Depression and Child Problems

**Graph 1:**
- **Y-axis:** Anxious/Depressed Problems
- **X-axis:** Frontal Asymmetry Group (Left, Right)
- **Legend:**
  - Control
  - COD

**Graph 2:**
- **Y-axis:** Anxious/Depressed Problems
- **X-axis:** Frontal Asymmetry (Left, Right)
- **Legend:**
  - low affect regulation
  - high affect regulation
Maternal Depression and Child Problems

Asymmetry Group

Aggressive Problems

Gender

Left Frontal
Right Frontal

Male
Female