Trajectories of Maternal Depression over Seven Years: Relations with Child Psychophysiology and Behavior

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Abstract

Trajectories of Maternal Depression over Seven Years: Relations with Child Psychophysiology and Behavior

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Maternal depression is associated with increased risk for child emotional and behavior problems as well as atypical child psychophysiology, including relative right frontal brain activation, elevated heart rate, low vagal tone, and elevated stress hormone levels. The mechanisms through which children develop a vulnerability to behavior problems remain unclear. Chronicity and severity of maternal depression as well as the presence of other risk factors in the social environment, such as marital conflict, have been found to predict more adverse child outcomes. This study explored the relation between the longitudinal course of maternal depression during the child's early life (birth – 6 years) and children's psychophysiology and behavior at age 6 ½ years. One-hundred fifty-nine children of depressed and non-depressed mothers were followed from infancy through age 6 ½ years. Growth mixture modeling was used to identify classes of depressed mothers based on the longitudinal course of the mother's depression. Three classes were identified: mothers with stable low depression, mothers with initial moderate to high followed by decreasing depression, and mothers with chronic moderate to high depression. Six and one half year old children of chronically depressed mothers were found to have elevated externalizing behavior problems, decreased social competence, reduced frontal brain activation (higher EEG alpha power), and higher respiratory sinus arrhythmia (RSA) reactivity. Children of mothers with decreasing and stable low depression were found to have increased hyperactivity and attention problems compared to children of non-depressed mothers. Contextual risk factors, such as family conflict and stressful life events, were found to mediate the relation between maternal depression and child behavioral outcomes.

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DEDICATION

For my parents, Ruth and Michael Ashman, whose lifelong support contributed to this accomplishment

Introduction

Research has consistently found an association between maternal depression and increased risk for psychopathology among children of depressed mothers (Downey & Coyne, 1990; Goodman & Gotlib, 1999; Murray & Cooper, 1997; Radke Yarrow, 1998; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). Compared to children of non-depressed mothers, children of depressed mothers are six times more likely to suffer from depression (Downey & Coyne, 1990). The risk, however, is not limited to depression or other mood disorders. Children of depressed mothers are also more likely to suffer from other types of psychopathology, including attention deficit hyperactivity disorder (ADHD; Orvaschel, Walsh-Allis, & Ye, 1988), conduct disorder (CD; Hammen, Burge, Burney, & Adrian, 1990), anxiety disorders (Fendrich, Warner, & Weissman, 1990; Weissman et al., 1997), and substance abuse (Weissman et al., 1997). While a large body of research documents the association between maternal depression and child psychopathology, relatively less research has chronicled how the course, timing, and severity of the mother's depression affect outcomes for children of depressed mothers. Furthermore, little research has examined the mechanisms underlying the transmission of risk from mother to child.

This study proposes to address some of these unanswered questions by adopting a developmental and psychophysiological approach to the investigation of risk for psychopathology in children of depressed mothers. This longitudinal study of 159 mother-child dyads explores the impact of maternal depression on children's psychophysiological and behavioral development. The goals of this study are 1) to identify patterns of change in maternal depressive symptoms over time that may predict

differences in child psychophysiology and behavior, 2) to compare child behavior and psychophysiology in 6 ½ –year-old children of mothers with different patterns of depression over time, and 3) to examine the contributions of other risk factors in the social environment (e.g. stressful life events, family conflict) that often accompany maternal depression to the prediction of child behavior problems. Psychophysiological measures, taken when the children were 6 ½ years of age, include frontal and parietal electroencephalographic activity (alpha EEG power), autonomic activity (heart rate and respiratory sinus arrhythmia (RSA) as a measure of vagal tone), and salivary cortisol. Child behavioral measures include measures of child mental health and social competence.

Diagnosis of Maternal Depression and Issues of Classification

Research investigating the relation between maternal depression and child outcomes is complicated by variations in the definition and diagnosis of maternal depression as well as by differences in the populations studied. Many studies of maternal depression rely on self-report symptom checklists to assess maternal depression (Alpern & Lyons-Ruth, 1993; Cohn, Matias, Tronick, Connell, & Lyons-Ruth, 1986; Field et al., 1985; Lundy et al., 1999), while others use clinical research diagnostic criteria (Anderson & Hammen, 1993; Cicchetti, Rogosch, & Toth, 1998; Essex, Klein, Miech, & Smider, 2001; Klimes Dougan et al., 1999; Seifer, Dickstein, Sameroff, Magee, & Hayden, 2001). Use of self-report measures may overdiagnose "depression" by defining depression based on symptoms that also characterize other psychiatric disorders, as well as general life stress (Campbell & Cohn, 1991). These studies may also fail to identify some mothers who meet research diagnostic criteria for depression (Campbell & Cohn, 1991).

Alternatively, clinical diagnostic criteria may obscure differences between mothers who are asymptomatic and mothers who have subthreshold symptoms of depression. A further complication that reduces comparability among studies involves the use of high-risk, multi-problem families versus low-risk samples. Many studies with significant findings include families with multiple risk factors in addition to maternal depression (Abrams, Field, Scafidi, & Prodromidis, 1995; Alpern & Lyons-Ruth, 1993; Cohn et al., 1986; Field et al., 1988). As a result, it is unclear to what extent the findings can be attributed to maternal depression per se versus other contextual risk factors, such as low socioeconomic status (SES) or marital discord, or comorbid psychiatric conditions, such as substance abuse (Carter, Garrity Rokous, Chazan Cohen, Little, & Briggs Gowan, 2001; Rutter, 1990).

Regardless of the diagnostic method employed or population studied, most research on maternal depression approaches diagnostic classification as a unitary construct, defining mothers as depressed or non-depressed based on reaching a certain threshold in terms of diagnostic criteria or a cut-off score on a symptom checklist. While valuable for classification in cross-sectional research designs, this conceptualization of depression is problematic for longitudinal investigations of the risk of maternal depression for child outcome. In these studies, the course and timing of the mother's depression may also be significant factors in the prediction of child psychopathology. For instance, women with more chronic depression may have less energy to devote to parenting and, as a consequence, be less available to their children. In addition, chronic depression exposes children to a longer duration of maternal disengagement and negative affect. A further weakness of the typical approach to diagnostic classification is that it

ignores the potential clinical significance of subthreshold levels of depressive symptoms. As argued by Lewinsohn and most other investigators in the field, depressive symptoms are best conceptualized as a continuum and the clinical significance of the symptoms has never been shown to depend on crossing a particular threshold (Lewinsohn, Solomon, Seeley, & Zeiss, 2000).

Before the past decade, factors such as timing and chronicity of the mother's depression were frequently ignored in studies of maternal depression. Even in some recent longitudinal studies, maternal depression is defined based on a single assessment of self-reported symptoms (Edhborg, Lundh, Seimyr, & Widstroem, 2001) or the presence or absence of a lifetime history of major depressive disorder (Weissman et al., 1997). Given that major depressive disorders are chronic and recurrent disorders (Judd, 1997), it could be argued that a single occurrence of depression is indicative of a lifelong vulnerability and possibly ongoing subclinical characteristics that may compromise mother-child interactions. In the absence of data, however, this is a strong assumption that ignores heterogeneity in clinical course and advances in treatment that could prevent or delay relapse. Furthermore, the absence of maternal depression during the child's lifetime even in the presence of a lifetime history of depression prior to childbirth has not been found to be associated with adverse child outcomes (Murray, 1992; Murray, Kempton, Woolgar, & Hooper, 1993). In these studies, there were no significant differences between mothers with a history of depression prior to childbirth only and the non-depressed comparison group in terms of mother-infant interaction at 2-months or infant outcome at 18-months .

When maternal depression is assessed over time, the most common approach is to assess depressive symptoms at two points in time without addressing the persistence or absence of symptoms in the intervening period of time (Alpern & Lyons-Ruth, 1993; Essex et al., 2001; Gross, Conrad, Fogg, Willis, & Garvey, 1995; Klimes Dougan et al., 1999; Lee & Gotlib, 1991; Zahn Waxler, Iannotti, Cummings, & Denham, 1990). This approach permits comparisons of the child outcomes based on the presence of previous versus concurrent depressive symptoms. For example, Alpern and Lyons-Ruth (1993) found teacher-reported child behavior problems at age 5 differed depending on whether the child's mother had experienced chronic, recent, or previous depressive symptoms. Chronic maternal depressive symptoms were associated with increased hostile child behavior problems, recent depressive symptoms were associated with increased hyperactive behavior, and previous depressive symptoms were associated with higher levels of anxiety at age 5. Essex and colleagues (Essex et al., 2001) also examined timing of maternal depression by comparing outcomes of children exposed to maternal depression during infancy to outcomes of children exposed to maternal depression during the toddler / preschool period. They found diagnosed maternal depression during infancy was associated with increased child behavior problems, especially internalizing problems, during kindergarten, while maternal depression during the toddler / preschool period was associated with increased externalizing behavior problems for girls. The limitations of the two time point research design are evident when you consider two "chronic" depressed mothers might differ dramatically in terms of length of depressive episodes and severity of symptoms. Furthermore, the presence of subthreshold symptoms over a long period of time might also have important impacts on child development.

In the past decade, longitudinal research methods have improved and researchers are increasingly including more rigorous assessments of maternal depression at multiple time points and gaining a more precise description of the course, timing, and severity of the mother's depression. Several recent studies have suggested adverse child outcomes are related to the severity or chronicity (or recurrence) of maternal depression as opposed to the absence or presence of a diagnosis (Brennan et al., 2000; Campbell, Cohn, & Meyers, 1995; Luoma et al., 2001; NICHD Early Child Care Research Network, 1999). For example, Brennan and colleagues (Brennan et al., 2000) assessed maternal depressive symptoms at four time points and child outcomes at age 5 years in a large, community sample (nearly 5000 participants). Compared to other studies, their large sample size included enough variability to permit the creation of orthogonal measures of chronicity and severity, which are typically confounded in depression, and to assess timing by identifying mothers who experienced depressive symptoms at only a single time point. Mothers with more chronic and severe depression had children with more severe behavior problems, especially when the chronicity and severity occurred in combination. Although this study represents an advance in the assessment of maternal depression over time, it is still limited by the sole use of self-report measures and the failure to examine the interval between assessments.

The issue of timing of maternal depression may be important to the extent that the depression interferes with the child's achievement of specific developmental tasks crucial to that period. For instance, maternal depression early in the child's development may interfere with development of a secure attachment or important self-regulatory skills. A number of studies have found enduring effects of earlier maternal depression that

improves or remits (Alpern & Lyons-Ruth, 1993; Fergusson & Lynskey, 1993; Hay et al., 2001; Lee & Gotlib, 1991). For example, Hay et al. (2001) found that children of mothers with postpartum depression that did not recur had significantly lower IQ scores than would be expected given their parents' cognitive abilities. Other studies have not corroborated this finding, however (Kurstjens & Wolke, 2001). Lee and Gotlib (1991) found school-age children of depressed mothers continued to display internalizing problems at a ten-month follow-up even if their mother's symptoms had improved. In an earlier study, Ghodsian and colleagues (Ghodsian, Zajicek, & Wolkind, 1984) found an association between early maternal depression and later child behavior problems. It is unclear, however, whether these continued decrements in child functioning are due to maternal depression per se or to the persistent effects of other adversity associated with maternal depression (e.g. family conflict).

In some cases, concurrent or recent maternal depression appears to predict more negative child outcomes than depression that occurs earlier in the child's life (Luoma et al., 2001; Sinclair & Murray, 1998). For example, Luoma and colleagues (Luoma et al., 2001) reported an association between concurrent maternal depressive symptoms and low social competence and adaptive functioning in 8 – 9-year-old children. Sinclair and Murray (1998) found that children of mothers with more recent depression were rated as less mature, more emotionally aroused, and more distractible. Caplan and colleagues (Caplan et al., 1989) found that only concurrent maternal depression was associated with behavior problems in 4-year-olds, and Carro and colleagues (Carro, Grant, Gotlib, & Compas, 1993) found that mother's current depressive symptoms accounted for the effects of earlier maternal and paternal depression on child behavior problems at age 2 -

3-years-old. The absence of longitudinal relations between earlier maternal depression and child outcomes may indicate that early maternal depression exerts some of its effects through its association with other variables, such as social disadvantage, family discord, or less sensitive parenting behavior. These problems may persist even after the depression remits. Caplan et al. (1989) found that marital conflict and the father's psychiatric history had more powerful influences on child behavioral outcomes than maternal depression per se. Hammen, Burge, and Adrian (1991) reported a temporal association between mother and child depressive episodes. The majority of child diagnoses occurred in close proximity to maternal depressive episodes, suggesting that child and mother may have both had reactions to family stressors and/or the presence of a depressed family member interferes with adaptive coping.

Although timing, severity, and chronicity of maternal depression appear to have associations with child outcomes, few longitudinal studies have explicitly modeled individual change in maternal depressive symptoms over time. A recent exception is a study by Munson, McMahon, and Spieker (2001) in which the authors used hierarchical linear modeling to model individual change in maternal depressive symptoms over time as related to changes in child externalizing behavior problems. Munson and colleagues (Munson et al., 2001) found that, for children with avoidant insecure attachment in infancy, when mothers reported an increase in their depressive symptoms, there was an associated increase in children's externalizing behavior problems during preschool and early childhood. This study was limited, however, by the use of mother-report for both the measure of maternal depression and the measure of child behavior problems. Furthermore, self-report measures of depression do not assess the interval between

assessments and do not distinguish between depressive symptoms and clinically diagnosed depression. More research is needed to ascertain the impact of changes in maternal depression over time on child outcomes.

Maternal Depression and Child Adjustment

Despite variations in methodology, maternal depression has consistently emerged as a significant risk factor for children's socioemotional development. Researchers have investigated the association between maternal depression and a range of child outcomes from the neonatal period through adolescence. The following review examines three categories of adjustment problems observed in children of depressed mothers: (1) adjustment problems in infancy and toddlerhood (e.g. insecure attachment and emotion regulation problems) that may indicate a vulnerability to emotional or behavioral problems later in life, (2) problems in psychological functioning, and (3) clinical diagnoses. Within each category, the existing literature is reviewed with particular attention to the methodologies used and the populations studied.

Adjustment Problems in Infancy

One means of examining the development of risk for psychopathology in children of depressed mothers is to study very young infants and toddlers of depressed mothers. Researchers have studied behaviors of infants of depressed mothers as early as the neonatal period. Compared to neonates of non-depressed mothers, neonates of depressed mothers exhibit poorer orientation, abnormal reflexes, less oral exploration, and less optimal levels of excitability, withdrawal, and irritability (Abrams et al., 1995; Hernandez Reif, Field, Del Pino, & Diego, 2000; Jones et al., 1998; Lundy et al., 1999). During interactions with their mothers, infants of depressed mothers display more

frequent negative emotions and fewer positive emotions, vocalize less, and have lower activity levels (Dawson, Frey, Self et al., 1999; Field, 1995). Research also demonstrates that infants of depressed mothers, on average, have poorer mental and motor development, and higher levels of emotional difficulties (Murray & Cooper, 1997). Findings have been similar for both infants of mothers with diagnosed depression and infants of mothers with elevated depressive symptoms. Studies of low SES and community samples have also had similar results. Although longitudinal research is lacking, it is possible that these disturbances in infant behavior indicate a vulnerability to the development of emotional and behavioral problems later in life.

Attachment. Disturbances in the parent-child attachment relationship have been extensively studied as a risk factor for later emotional and behavioral adjustment problems, particularly depression (Cummings & Cicchetti, 1990). In addition, disorganized attachment has been associated with later externalizing problems (Van IJzendoorn, Schuengel, & Bakerman–Kranenburg, 1999). As such, researchers have been interested in whether infants at risk for development of adjustment problems, such as infants of depressed mothers, might develop insecure attachments as a precursor to later psychopathology. Several studies have found an association between maternal depression and insecure attachment relationships (Lyons Ruth, Connell, Grunebaum, & Botein, 1990; Martins & Gaffan, 2000; Radke Yarrow, Cummings, Kuczynski, & Chapman, 1985; Rosenblum, Mazet, & Benony, 1997; Teti, Gelfand, Messinger, & Isabella, 1995; Van IJzendoorn et al., 1999). In a sample of low-income, teenage mothers, Dawson and colleagues (Dawson, Grofer Klinger, Panagiotides, Spieker, & Frey, 1992) found a significant association between attachment disorganization ratings

and maternal depressive symptoms. Two recent meta-analyses have found small, but significant associations between maternal depression and insecure, particularly disorganized, attachment classifications (Martins & Gaffan, 2000; Van IJzendoorn et al., 1999). Finally, Rosenblum and colleagues (Rosenblum et al., 1997) found associations between specific subtypes of maternal depression and different types of insecure attachment. Mothers with irritable / stressed depression were more likely to have infants with insecure-ambivalent attachment, whereas mothers with dull / slow depression were more likely to have infants with insecure-avoidant attachment. Rosenblum and colleagues (Rosenblum et al., 1997) theorized that infants of dull / slow depressed mothers develop insecure-avoidant attachments as an emotion regulation strategy in response to the withdrawn behaviors of their mothers.

The association between maternal depression and insecure attachment is complicated by inconsistencies in findings and variability among research participants. In a recent meta-analysis, Martins and Gaffan (2000) reported significant heterogeneity among studies, despite their use of narrow criteria for inclusion in the analysis. Among seven studies of middle-class depressed mothers without concomitant psychopathology, four studies reported less secure attachment among infants of depressed mothers, and three reported the opposite or no effect (Martins & Gaffan, 2000). Focusing on disorganized attachment, Van IJzendoorn and colleagues (Van IJzendoorn et al., 1999) found an association between maternal depression and increased likelihood of disorganized attachment for clinical samples, but not community samples. The apparent discrepancies in findings may relate to variability in age of the children, family SES, or severity of the mother's depression (e.g. clinical vs. community samples). Infants are

most likely to develop insecure attachments when the mother's affective disorder is severe and/or chronic (Teti et al., 1995), bipolar (DeMulder & Radke Yarrow, 1991; Radke Yarrow et al., 1995), comorbid with other psychiatric disorders (Carter et al., 2001), or combined with other risk factors, such as low SES or marital dissatisfaction (Lyons Ruth et al., 1990; Shaw & Vondra, 1995). Thus, research suggests there is a slightly higher risk for insecure, particularly disorganized attachment, among children of more severely depressed mothers; however, the relation between maternal depression and insecure infant attachment is likely complex and merits further research. Maternal depression is likely to be one of several risk factors that collectively contribute to the development of insecure attachment. In addition, further research is necessary to determine whether infants of depressed mothers who exhibit insecure attachments are more likely to develop psychopathology later in life.

Psychological Functioning

Research examining the psychological functioning of children of depressed mothers has found these children are at risk for a range of socioemotional problems. Measures of psychological functioning typically include parent-report, teacher-report and/or child self-report questionnaire measures, such as the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983).

Compared to children of non-depressed mothers and/or medically-ill mothers, preschool and school-aged children of mothers with depression or elevated depressive symptoms have been found to have increased rates of both internalizing and externalizing problems as reported by parents (Alpern & Lyons-Ruth, 1993; Anderson & Hammen, 1993; Caplan et al., 1989; Carro et al., 1993; Carter et al., 2001; Cicchetti et al., 1998;

Field et al., 1996; Lang et al., 1996; Lee & Gotlib, 1991; Philipps & O' Hara, 1991), teachers (Alpern & Lyons-Ruth, 1993; Gross et al., 1995; Wright, George, Burke, Gelfand, & Teti, 2000), and the children themselves (Lee & Gotlib, 1991). Elevated levels of emotional and behavior problems in children of depressed mothers have been observed both at home (Alpern & Lyons-Ruth, 1993; Anderson & Hammen, 1993; Murray, Sinclair, Cooper, Ducournau, & Turner, 1999) and at school (Alpern & Lyons-Ruth, 1993; Anderson & Hammen, 1993). Children of depressed mothers are also more likely to report suicidal ideation, plans, and/or attempts (Klimes Dougan et al., 1999). In addition to increased symptoms of emotional and behavior problems, children of depressed mothers have been found to exhibit deficits in social competence (Anderson & Hammen, 1993; Carter et al., 2001; Gross et al., 1995), lower performance on cognitive tests (Sharp et al., 1995), poorer problem-solving skills (Jones, Field, & Davalos, 2000), academic problems (Anderson & Hammen, 1993), less creative play and less positive responses to the social approaches of other children (Murray et al., 1999), and school adjustment problems (Wright et al., 2000).

Gender differences. Research also suggests maternal depression may have differential effects on the psychological functioning of boys vs. girls (Duggal, Carlson, Sroufe, & Egeland, 2001). In one study, Duggal and colleagues (Duggal et al., 2001) found that maternal depressive symptoms in childhood were predictive of adolescent depression for girls, but not boys. For males, lack of supportive early care was more predictive of depression in adolescence. Fergusson and colleagues (Fergusson, Horwood, & Lynskey, 1995) also found that maternal depression in middle childhood was related to adolescent depression for girls, but not boys; however they also found that this

association was indirect through the association of maternal depression with family adversity and social disadvantage. Researchers studying younger children have found the opposite effect. At age 30 months, Carter and colleagues (Carter et al., 2001) found maternal depression was associated with problem behaviors in boys, but not girls. For 30-month-old girls, the quality of early interactions was more relevant. It is not surprising to find variability in findings by child developmental level. In adolescence, maternal depression may have a stronger association with daughters' depression because of sex-linked heredity, gender identification, and/or the tendency for mothers to use their daughters as comfort objects and confidantes (Duggal et al., 2001). In contrast, during childhood, boys may be more vulnerable because of differences in how boys and girls learn to regulate emotions (Carter et al., 2001).

Longitudinal research. Over the past ten to fifteen years, researchers have increasingly adopted a longitudinal perspective in the study of the impact of maternal depression on child adjustment (Alpern & Lyons-Ruth, 1993; Anderson & Hammen, 1993; Brennan et al., 2000; Gross et al., 1995; Hay et al., 2001; Luoma et al., 2001; NICHD Early Child Care Research Network, 1999; Sinclair & Murray, 1998; Wright et al., 2000). The assessment of maternal depression and child behavior problems over time permits a more comprehensive investigation of the relations between maternal depression and child psychological functioning. Researchers can begin to answer questions about the relation between the timing, severity, and chronicity of the mother's depression and child functioning, as well as developmental questions about how maternal depression affects children at different stages of life (see review above).

A few studies have also examined the effects of maternal depression on the continuity and change in child adjustment problems over time. Anderson and Hammen (1993) found school-age children of unipolar depressed mothers had a higher incidence of chronic behavior problems over a two-year period compared to children of bipolar mothers and children of mothers with no psychiatric diagnosis. Recent advances in longitudinal data analysis, such as hierarchical linear modeling, allow for examination of the correlates of individual developmental trajectories in child behavior problems. Using this technique, Munson and colleagues (Munson et al., 2001) examined developmental trajectories in child externalizing behavior problems from preschool through third grade and found that maternal depressive symptoms predicted both the level of externalizing problems at age 9 and the rate of change in externalizing problems. Specifically, mothers who reported higher levels of depressive symptoms had children with higher levels and higher rates of increase of externalizing behavior problems. When examining disruptive behavior problems from preschool to age 6 years, Spieker, Larson, Lewis, Keller, and Gilchrist (1999) found that maternal symptoms of depression and anxiety predicted the absolute level, but not the rate of change in child behavior problems.

Clinical Diagnoses

Children of depressed mothers are also more likely to suffer from a diagnosed clinical disorder, especially depression. Evidence of increased risk for a diagnosed psychological disorder in children of depressed mothers was first discovered when these children were used as a comparison group for children of schizophrenic mothers (Downey & Coyne, 1990). Later, controlled studies of children of depressed parents confirmed their elevated risk for a psychiatric diagnosis. In a 10 year longitudinal

follow-up study, Weissman et al. (1997) found that, compared to children and young adults of non-depressed parents, children and young adults with at least one depressed parent were approximately 3 times more likely to have a lifetime diagnosis of major depression or phobia and nearly 5 times more likely to have a lifetime diagnosis of panic disorder or alcohol dependence. Children of depressed mothers have also been found to have increased rates of ADHD (Orvaschel et al., 1988) and are more likely to have multiple diagnoses (Hammen et al., 1990).

The peak incidence for specific disorders has been found to vary with age in children of depressed mothers (Weissman et al., 1997). In general, anxiety disorders have an earlier onset, peaking between the ages of 5 and 10 years, while depressive disorders tend to occur between ages 15 and 20 years (Weissman et al., 1997). While this pattern exists for children of both depressed and non-depressed mothers, there is some evidence that children of unipolar depressed mothers develop depressive disorders earlier than children of non-depressed, bipolar, or medically-ill mothers (Hammen et al., 1990). Although prepubertal depression is uncommon, children of depressed mothers may be more likely to develop prepubertal depression (Weissman et al., 1997).

Summary: Maternal Depression and Child Adjustment

Children of depressed mothers display increased rates of adjustment problems from the neonatal period through adolescence. Adjustment problems include early markers of risk, such as insecure attachment and emotional regulation problems, internalizing and externalizing symptoms, poorer social competence, cognitive and academic problems, and psychiatric disorders, such as depression, anxiety disorders, and conduct problems. Although the negative outcomes associated with being raised with a

depressed mother are well documented, the type and severity of the impact appear to vary with a number of factors including child factors, such as age and gender, the chronicity and severity of the mother's depression, and the presence or absence of associated risk factors, such as low SES. Additional research is needed to further understand the risks to children of both sexes at different developmental levels, as well as whether the risk is associated with maternal depression per se or other factors in the child's environment. In addition, the question remains as to how this risk is conferred from mother to child. Researchers have hypothesized several possible mechanisms of transmission. Before turning to these, however, the literature on the relation between maternal depression and the psychobiological systems related to emotion expression and regulation is reviewed.

Maternal Depression and Alterations in Psychophysiological Systems Related to Emotion Regulation and Expression

Research suggests maternal depression relates not only to increased rates of child emotional and behavior problems, but also to alterations in the psychophysiological systems associated with emotion regulation and expression, which themselves have been associated with affective disorders in adulthood. Alterations in these systems are reflected in altered levels of cortical activity, autonomic nervous system activity, and stress hormone secretion. In this section, literature is reviewed that relates measures of each of these psychophysiological responses to individual differences in emotion regulation and expression. Then, the association between maternal depression and alterations in these psychophysiological indices is examined.

The Prefrontal Cortex

Researchers have long recognized the role of the prefrontal cortex in the expression and regulation of emotion (Davidson, 2000; Marshall & Fox, 2000). Evidence suggests that the different hemispheres of the frontal lobe mediate the expression of different types of emotions (Fox, 1991). Typically, the left frontal region has been found to be associated with the expression of positive affect or "approach" emotions and behaviors, such as joy and interest, whereas the right frontal region has been associated with the expression of negative affect or "withdrawal" emotions and behaviors, such as sadness (Marshall & Fox, 2000). Some of the earliest studies of the relation between brain and emotion examined the effects of brain damage on emotion expression and regulation. In these studies, left frontal lobe lesions tended to result in depression and catastrophic reactions, whereas right frontal lobe lesions tended to result in apathy or euphoria (Gainotti, 1969, 1972; Heller, 1990; Robinson & Benson, 1981; Robinson, Kubos, Starr, Reo, & Price, 1984; Robinson & Stetela, 1981). Related findings have been reported in studies of emotional reactions following sodium amytal injections (see Marshall & Fox, 2000 for a review). When injected into one of the carotid arteries, sodium amytal results in the loss of brain functions on the side of the injection. In these studies, left-sided injections resulted in emotional behaviors reflecting negative affect, including "crying, pessimistic statements, feelings of nothingness, indignity or despair, and inability to hold back fears or negative thoughts" (Marshall & Fox, 2000). In contrast, right-sided injections elicited positive affect and euphoria.

Additional support for the differential mediation of specific emotions by the left and right frontal regions comes from research on frontal EEG activity during the

expression of different emotions (Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Davidson & Fox, 1988, 1989; Dawson, Panagiotides, Grofer Klinger, & Hill, 1992; Finman, Davidson, Colton, Straus, & Kagan, 1989). In these studies, researchers measure brain electrical activity in the alpha frequency band (8 – 13 Hz for adults and lower frequencies for young children) and compare the relative activation of right and left hemisphere regions. Differences in activity between homologous left and right hemisphere regions are posited to reflect relative differences in neuronal activity. Because increased EEG alpha power indicates decreased neuronal activity, decreased EEG alpha power in the left frontal region compared to the right frontal region suggests higher levels of neuronal activity in the left frontal region or relative left frontal asymmetry. Decreased EEG alpha power in the right frontal region compared to the left frontal region indicates higher levels of neuronal activity in the right frontal region or relative right frontal asymmetry.

Findings from EEG studies have tended to support the results of investigations using other methodologies. For example, Wheeler, Davidson, and Tomarken (1993) observed women's frontal EEG patterns in response to emotion-eliciting film clips and found that, for women whose EEG patterns were stable across a 3-week period, relative left frontal asymmetry was associated with more positive emotional responses to the positive film clips, whereas relative right frontal asymmetry was associated with more negative emotional responses to the negative film clips. Jacobs and Snyder (1996) observed similar patterns in men.

Studies of infants and young children suggest that the differential mediation of emotion by the left and right frontal regions may exist very early in life. In a series of

studies, Davidson and Fox (Davidson & Fox, 1982; Davidson & Fox, 1989; Fox, 1991; Fox & Davidson, 1988) reported associations between relative right frontal activation and negative emotions and relative left frontal activation and positive emotions in infants. In one study, for example, they found that infants with relative right frontal activation during a baseline condition were more likely to cry during separation from their mothers (Davidson & Fox, 1989). Similarly, Dawson and colleagues (Dawson, Panagiotides et al., 1992) found that, in 21-month-old infants, relative left frontal activation occurred during the expression of positive emotions, whereas relative right frontal activation occurred during the expression of negative emotions.

Some researchers have theorized that cerebral asymmetries may not simply index the valence (e.g. happy vs. sad) of an emotion, but rather the propensity to use approach vs. withdrawal emotion regulation strategies (Davidson et al., 1990; Dawson, 1994; Dawson, Panagiotides et al., 1992; Fox, 1991; Kinsbourne & Bemporad, 1984; Tucker & Williamson, 1984). These investigators suggest relative left frontal brain activation signals the use of active, organized regulation/coping schemes, such as expressive language, that involve engagement of the external environment. In contrast, they suggest relative right frontal brain activation indicates the use of emotion regulation strategies involving disruption in ongoing activity and withdrawal from the environment. Evidence supporting this notion comes from studies of the association between frontal brain asymmetries and behavioral inhibition as assessed by children's latency to approach novel stimuli. For example, Finman and colleagues (Finman et al., 1989) observed that, compared to uninhibited peers, behaviorally inhibited children displayed greater resting right frontal asymmetry. In a related study by Jones and colleagues (Jones, Field,

Davalos, & Pickens, 1997), three-year-old children with relative right frontal asymmetry were found to be more inhibited. These studies suggest an association between relative right frontal asymmetry and hesitancy to approach novelty which may reflect withdrawal self-regulatory strategies.

Hemispheric asymmetries and depression. The theoretical model associating frontal lobe asymmetries with emotion expression and regulation has been extended to include research examining the role of the frontal lobe in depression. Evidence from EEG studies suggests left frontal hypoactivation is associated with clinical depression (Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991) and that this pattern of brain activity may be a stable trait rather than a state marker for depression (Gotlib et al., 1998; Henriques & Davidson, 1990). For example, Gotlib and colleagues (Gotlib et al., 1998) found that both currently depressed and previously depressed individuals exhibited reduced left frontal brain activation compared to never depressed controls. Field and colleagues (Field, Fox, Pickens, & Nawrocki, 1995) found that, compared to non-depressed mothers, depressed mothers had relative right frontal EEG activation. Jones and colleagues (Jones, Field, Fox, Lundy, & Davalos, 1997) also found depressed mothers had relative right frontal EEG asymmetry compared to non-depressed mothers. Additional evidence for the role of the prefrontal cortex in depression comes from functional neuroimaging studies. One of the most consistent findings among these studies is the association between depression and decreased brain activation, particularly in the left frontal region (Kennedy, Javanmard, & Vaccarino, 1997).

Frontal brain activity in children of depressed mothers. The literature associating the prefrontal cortex with emotion expression and regulation combined with the literature

associating left frontal hypoactivation and depression raises the question of whether children at risk for depression and emotion regulation problems might also exhibit alterations in their frontal brain activity. Several studies have documented a relation between maternal depression and alterations in infants' electrical brain activity. Specifically, researchers have found that infants of depressed mothers exhibit reduced left frontal electrical brain activation during a baseline condition, and during playful interactions with their mothers and a familiar experimenter (Dawson, Frey, Panagiotides, Osterling, & Hessl, 1997; Dawson, Frey, Panagiotides et al., 1999; Dawson, Grofer Klinger, Panagiotides, Hill, & Spieker, 1992; Field, Fox et al., 1995; Jones, Field, Fox, Lundy et al., 1997). This association has been observed as early as the neonatal period. For example, Jones and colleagues (Jones et al., 1998) reported reduced left frontal brain activation in newborns of mothers with depressive symptoms only 1 week after birth. Similar EEG patterns have been observed in 1-month-old (Jones, Field, Fox, Lundy et al., 1997), 3-month-old (Field, Fox et al., 1995), and 13-15-month-old (Dawson et al., 1997; Dawson, Frey, Panagiotides et al., 1999; Dawson, Grofer Klinger, Panagiotides, Hill et al., 1992) infants of depressed mothers. Dawson and colleagues replicated this finding with both low-income, teenage mothers and middle-income mothers without other psychiatric problems (Dawson et al., 1997; Dawson, Grofer Klinger, Panagiotides, Hill et al., 1992). Most studies have found the observed atypical brain activity was specific to the frontal region of the brain (Dawson et al., 1997; Dawson, Grofer Klinger, Panagiotides, Hill et al., 1992; Field, Fox et al., 1995).

There is some evidence to suggest the atypical brain activity observed in infants of depressed mothers is an acquired trait rather than a marker of the infant's concurrent

emotional state. Dawson and colleagues (Dawson, Frey, Self et al., 1999) found that, at the time of EEG recording, there were no observable differences in infants' emotional behavior; however, frontal EEG activity was found to be related to infant behaviors observed on a separate day from the EEG recording. Specifically, reduced left frontal brain activation was associated with lower levels of affectionate behaviors, and increased generalized frontal brain activation was associated with increased negative affect, hostility, tantrums, and aggression. Dawson and colleagues (Dawson, Frey, Self et al., 1999) hypothesized that the atypical brain activity observed in infants of depressed mothers reflects both a tendency to experience more frequent and possibly more intense negative affect, as well as a lack of adequate self-regulatory strategies that might be used to modulate negative emotions.

Although a number of studies have examined the relation between maternal depression and infants' electrical brain activity, few investigators have examined this relation in older children. In one of the few published studies to date, Jones and colleagues (Jones et al., 2000) reported that preschool children of depressed mothers have greater relative right frontal EEG asymmetry than children of non-depressed mothers. Jones et al. (2000) suggest the relative right frontal EEG activation in children of depressed mothers may reflect either a greater propensity to experience negative affect or a tendency to use withdrawal self-regulatory behaviors. At an earlier time point in this investigation, preschool children of mothers with chronic depression were found to have reduced generalized brain activation (Dawson et al., in press), which may reflect decreased engagement with the external environment. To date, no published study has

examined the association between maternal depression and children's electrical brain activity beyond the preschool years.

Although little longitudinal research currently exists, there is some evidence that frontal EEG asymmetry patterns in children of depressed mothers are stable from infancy to preschool, adding additional support to the notion that EEG asymmetry patterns are a trait rather than associated with a particular emotional state. Jones and colleagues (Jones, Field, Davalos et al., 1997) found that 7 of 8 children who exhibited right frontal EEG asymmetry at age 3-6 months exhibited the same frontal EEG pattern at age 3 years. Stability was not as strong for the parietal region.

Biological Stress Systems

In addition to being associated with alterations in brain activity, maternal depression has also been linked to alterations in children's biological stress systems as evidenced by autonomic nervous system activity and stress hormone levels. The association between maternal depression and alterations in children's biological stress systems is of interest because of the association of these systems with emotion regulation and depression. In adults, major depression has been associated with dysregulation of biological stress systems (Heuser, 1998; Nemeroff, 1988; Plotsky, Owens, & Nemeroff, 1998). Specifically, researchers have observed increased levels of stress hormone secretion, hypercortisolemia, in patients with major depression (Plotsky et al., 1998). Furthermore, according to the stress-diathesis model of depression, the onset of major depressive disorder is often preceded by early adverse experiences and other life stressors that, combined with genetic vulnerability, contribute to the development of depression (Plotsky et al., 1998). It is possible that a psychologically stressful early social

environment, such as that which might accompany having a depressed mother, may negatively impact the development of children's biological stress responses and contribute to vulnerability for depression.

The biological consequences of stress and their relation to emotion regulation and child adjustment. The body's biological response to stress involves many physiological structures, including various regions of the brain, as well as the autonomic, immune, and endocrine systems. A complete understanding of biological stress responses requires a comprehensive analysis of all the systems involved and their interactions with one another. Such an analysis goes beyond the scope of this review, but has been described elsewhere (see López, Akil, & Watson, 1999 for a review). In the study of the effects of maternal depression on child development, two components of the stress response have been studied most extensively, and these are neuroendocrine and autonomic responses.

When presented with an actual or perceived threat, an individual experiences both psychological and biological stress responses. The biological response includes a mobilization of the body for action or what is known as the "fight or flight" response. The neuroendocrine component of the "fight or flight" response is mediated by the hypothalamic-pituitary-adrenal (HPA) axis. In humans and non-human primates, activation of the HPA axis by physical or psychological stress initiates a series of physiological events resulting in the release of cortisol, the primary peripheral stress hormone (López et al., 1999). Under normal, non-stressful conditions, cortisol has a circadian rhythm, peaking in the morning upon waking and exhibiting its lowest levels at night. Under stressful conditions, cortisol elevations are superimposed on the regular circadian cycle (Sánchez, Ladd, & Plotsky, 2001). A responsive and adaptive HPA axis

that activates in response to stress and terminates when the threat has abated is essential for survival; however prolonged glucocorticoid secretion as might occur under conditions of chronic stress can have deleterious effects on brain regions that contain high concentrations of glucocorticoid (e.g. cortisol) receptors (Gunnar, 1998; Sapolsky, 1996).

Individual differences in baseline and stress-related cortisol responses have been linked to individual differences in emotion regulation. For example, Stansbury and Gunnar (1994) have identified several emotion regulation strategies that may influence HPA axis activity. These strategies include control, attention regulation, self-soothing, and social support. For example, rats exposed to inescapable electric shocks exhibit significantly higher corticosterone levels than rats exposed to avoidable shocks (Weiss, 1971), and the presence of a familiar social group attenuates the adrenocortical response of squirrel monkeys exposed to maternal separation (Levine & Wiener, 1988). Interestingly, buffering of the adrenocortical response to stress appears to relate to the availability of these emotion regulation strategies rather than the performance of the strategies. Thus, activation of the HPA axis in response to stress may index an individual's perceived ability to cope with the stressor (Stansbury & Gunnar, 1994). For individuals who are vulnerable to emotional problems, the chronic absence of perceived control over stress may affect the development of stress responses.

Research has also found relations between activity of the HPA axis as measured via salivary cortisol levels and child emotional adjustment. Specifically, internalizing behaviors, including anxious and depressive symptoms, have been found to be associated with elevations in baseline cortisol levels and measures of cortisol reactivity (Goodyer, Herbert, Moor, & Altham, 1991; Granger, Weisz, & Kauneckis, 1994; Kaufman et al.,

1997; Scerbo & Kolko, 1994; Smider et al., 2002). In a sample of 7 – 17-year-old clinic-referred children, Granger and colleagues (Granger et al., 1994) found an association between increased salivary cortisol in responses to social challenge (a parent-child conflict discussion task) and child social withdrawal, social anxiety, and social problems. In a large community sample, Smider and colleagues (Smider et al., 2002) found that higher cortisol levels at age 4 ½ years predicted higher mother and teacher reports of internalizing problems and social wariness at the end of kindergarten. Relations between childhood internalizing problems and HPA axis activity or reactivity are not consistently found, however (Birmaher et al., 1996). Associations between childhood internalizing problems and HPA axis dysregulation are most often observed in children with more severe psychopathology, including psychiatric inpatients (Birmaher et al., 1996), depressed abused children who are experiencing ongoing adversity (Kaufman et al., 1997), and severely depressed children (Goodyer et al., 1991). In contrast to positive relations between internalizing problems and HPA axis activity, an inverse relation has been observed between externalizing behaviors, such as aggression, and cortisol levels (McBurnett, Lahey, Rathouz, & Loeber, 2000; Smider et al., 2002). Smider and colleagues (Smider et al., 2002) found a negative correlation between father reports of externalizing behavior problems and boys cortisol levels. In a sample of clinic referred boys, McBurnett and colleagues (McBurnett et al., 2000) found that low cortisol levels were associated with disruptive behavior problems. Relations between disruptive behavior and low cortisol levels are not consistently found however (Scerbo & Kolko, 1994). In a study of comorbid anxiety and conduct disorder, McBurnett et al. (1991) found that children with both conduct disorder and anxiety had higher salivary cortisol

levels than children with conduct disorder alone. Other studies have also found that disruptive behavior disorders in the presence of anxiety are related to higher cortisol reactivity to stress compared to disruptive behavior disorders alone (van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000).

The autonomic component of the "fight or flight" response involves activation of the sympathetic and parasympathetic components of the autonomic nervous system. When an individual is presented with a perceived threat, the sympathetic nervous system mobilizes the body to respond to the stressor by, for example, increasing heart rate and sweating, and inhibiting digestive functions. The parasympathetic nervous system then restores homeostasis by promoting the opposite effects (e.g. decelerating the heart and increasing digestive functions). Sympathetic activity is often measured using heart rate, although heart rate is influenced by both sympathetic and parasympathetic components. A common measure of parasympathetic activity is "vagal tone," a measure of the neural control of the heart by the tenth cranial nerve. Vagal tone is evaluated by quantifying the amplitude of the respiratory sinus arrhythmia (RSA) using heart rate data from an electrocardiogram (Porges, Doussard-Roosevelt, & Maiti, 1994).

Researchers have linked individual differences in emotion expression and regulation with specific variations in heart rate and vagal tone. For example, Field and colleagues (Field, Pickens, Fox, Nawrocki, & Gonzalez, 1995) reported an association between vagal tone and emotional reactivity. Infants with higher vagal tone were found to be more reactive and emotionally expressive. Similarly, Porges et al. (1994) observed correlations between vagal tone and autonomic reactivity, emotion expression, and self-regulation. Higher vagal tone was associated with larger autonomic responses, greater

facial expressivity in 5-month-old infants, and higher soothability in 3-month-old infants. High vagal tone in childhood has been associated with social competence and appropriate emotion regulation (see Beauchaine, 2001 for a review). Decreases in vagal tone in response to challenge have been associated with sustained attention; however, excessive vagal withdrawal is also associated with fight or flight responses that may be maladaptive, such as in the case of panic disorder or aggression (Beauchaine, 2001).

Influence of the early social environment on biological stress systems. According to the stress-diathesis model of depression, early adverse experiences may increase risk for developing depression later in life (Plotsky et al., 1998). It is possible that these early experiences exert their influences through their impact on the development of physiological systems related to emotion regulation and expression. There is ample evidence from both animal and human studies that parent-infant interactions play an important role in the development of infant self-regulatory strategies and stress responses. Furthermore, there is some evidence suggesting the effects of these early interactions may persist into adulthood.

In the animal literature, researchers have extensively studied the effects of variations in the early environment on the HPA axis response to stress. Researchers have found that, in rats, early postnatal handling results in a decreased glucocorticoid response to stress (Francis et al., 1996; Meaney, Aitken, van Berkel, Bhatnagar, & Sapolsky, 1988), whereas longer separations from the mother (3 hours or more) have been demonstrated to lead to elevated glucocorticoid levels in response to stress (Kuhn, Pauk, & Schanberg, 1990; Kuhn & Schanberg, 1998; Pihoker, Owens, Kuhn, Schanberg, & Nemeroff, 1993). Some longitudinal studies have demonstrated these effects can persist

into adulthood (Ladd, Owens, & Nemeroff, 1996; Plotsky & Meaney, 1993). Whether the effects persist, however, appears to depend on the age at which the rat pup is separated from its mother and the chronicity of the separation (Sánchez et al., 2001). In a recent review, Sánchez and colleagues (2001) describe research reporting that neonatal rats exposed to a single 24-hour maternal separation show differential effects on HPA axis functioning later in life depending on the age of exposure. In contrast, researchers have consistently observed long-term consequences of maternal separation in neonatal rats exposed to daily 3-hour separations throughout the neonatal period (postnatal days 2 to 14) (Sánchez et al., 2001). In adulthood, these repeatedly exposed rats demonstrate hyperresponsiveness to psychological stressors as evidenced by increased ACTH and corticosterone secretion and decreased negative feedback inhibition. Behaviorally, these chronically exposed rats exhibited an anxiety-like and depression-like syndrome, anhedonia, and increased ethanol intake (Sánchez et al., 2001). Some researcher have hypothesized that long-term biological consequences of maternal separation and early handling may result from early alterations in glucocorticoid receptor expression in brain regions, such as the prefrontal cortex and hippocampus, that influence negative feedback regulation of HPA axis activity (Francis et al., 1996). Evidence suggests early handling leads to increased receptor expression and increased negative feedback efficacy, while early separation yields the opposite response (Francis et al., 1996).

There is some suggestive evidence that early alterations in the development of the neuroendocrine stress response may be mediated by maternal behaviors. When reunited with their rat pups, previously separated dams delay in retrieving their pups and in initiating maternal behaviors, such as licking, grooming, and nursing, that have been

observed to be necessary for attenuating the stress response of neonatal rat pups (Sánchez et al., 2001). Francis and colleagues (Francis et al., 1996) found a significant negative correlation between the frequency of these maternal behaviors and the magnitude of the neuroendocrine stress response. In addition, Suchecki, Rosenfeld, and Levine (1993) have demonstrated that specific maternal behaviors, such as feeding and stroking, can weaken the adrenocortical response to maternal deprivation. In maternally deprived rat pups, feeding was found to inhibit corticosterone secretion, and stroking to suppress ACTH secretion. Research suggests that the attenuating effects of these maternal behaviors are specific to maternal stimuli, as opposed to non-specific social stimuli (Stanton & Levine, 1990).

Although the findings are less consistent, research with non-human primates (squirrel monkeys and rhesus macaques) has also demonstrated alterations in the neuroendocrine response to stress following maternal separation. In a review, Levine and Wiener (1988) describe research reporting increased levels of plasma cortisol in response to maternal separation in both squirrel monkeys and rhesus macaques. Furthermore, these elevations were found to be significantly and positively correlated with the duration of maternal separation. Interestingly, maternal behaviors alone appear less significant for non-human primates as the presence of familiar peers or visual access to the mother during separation was found to buffer the endocrine effects of maternal separation. In addition, the predictability of the caretaking environment has been found to be important for the development of social behaviors and stress responses in non-human primates (Coplan et al., 1996; Sánchez et al., 2001). For example, unpredictable foraging demands have been found to interfere with normal development. Animals raised in unpredictable

environments were found to exhibit increased responses to stress both behaviorally and physiologically (Coplan et al., 1996; Sánchez et al., 2001). These finding suggest that the availability of coping resources, such as a stable, predictable environment or the presence of peers, can attenuate the adrenocortical response to stress in non-human primates.

In order to study the effects of early deprivation and other adverse experiences on the stress responses of human infants, researchers have had to capitalize on naturally occurring forms of early deprivation that sometimes occur in human populations. In one such study, Gunnar and colleagues (Gunnar, Morison, Chisholm, & Schuder, 2001) tracked the development of children exposed to different amount of profound early deprivation in Romanian orphanages before being adopted into homes in Europe, Canada, and the United States. They found that, approximately six years after adoption, children who had experienced 8 or more months of institutional rearing displayed significantly higher salivary cortisol levels compared to sex, age, and SES matched controls and compared to children who had experienced 4 or fewer months of institutional life. In addition, longer exposure to deprivation was associated with higher evening cortisol levels. Researchers have also found associations between child maltreatment and dysregulation of HPA axis responses to stress both during and following experiences of abuse and neglect (see Glaser, 2000 for a review).

Maternal depression and children's biological stress systems. Animal and human research demonstrating the long term impact of early life stress on physiological stress responses raises the question of whether less profound forms of early stress, such as exposure to maternal depression, may also have long term consequences for the regulation of stress responses. Several studies have found associations between maternal

depression and alterations in infant's biological stress systems. Specifically, neonates of depressed mothers have been found to exhibit higher cortisol and norepinephrine levels, and lower dopamine levels (Lundy et al., 1999). In this same study, mothers' hormone levels during pregnancy were found to predict infants' neonatal hormone levels (Lundy et al., 1999), suggesting a possible prenatal mechanism for the impact of maternal depression on infant's hormonal stress responses (see below). Similarly, a study by Field and colleagues (Field et al., 1988) found that infants of depressed mothers had higher cortisol levels than infants of non-depressed mothers.

Few studies have examined the effects of maternal depression on children's stress responses beyond infancy. At an earlier time point in this study, Hessl and colleagues (Hessl et al., 1996, May) measured salivary cortisol levels in 3 ½ year-old children of depressed and non-depressed mothers, both at baseline and in response to a mild laboratory stressor. Compared to preschool children of non-depressed mothers, preschool children of depressed mothers were found to have higher baseline salivary cortisol levels (Hessl et al., 1996, May). In a longer longitudinal follow-up of a sub-group of families from the earlier study, Ashman and colleagues (Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002) found that 7-8-year-old children of depressed mothers exhibited elevated baseline cortisol levels only if they also were reported to have elevated levels of internalizing symptoms. Thus, in older children, the existing research does not support a simple main effect of maternal depression on children's hormonal stress responses.

In light of animal research suggesting that early life stress can have long-term effects on HPA axis-mediated stress responses (e.g. Ladd et al., 1996; Plotsky & Meaney,

1993), the above researchers further analyzed the data in order to explore the relation between maternal depression during specific years of the child's life and the child's salivary cortisol levels. At age 3 ½ years, results indicated that the number of months of prenatal depression was the best predictor of preschool children's baseline salivary cortisol levels. (Ashman & Dawson, 2002) This finding is consistent with earlier animal research given that the CNS maturity during the early postnatal life of a rat pup corresponds approximately to the CNS maturity of a 24-week gestational age human fetus (Graham, Heim, Goodman, Miller, & Nemeroff, 1999). In contrast, Ashman and colleagues (Ashman et al., 2002) found that maternal depression in the first two years of the child's life was the best predictor of cortisol elevations at age 7. These findings are tentative and require replication; however, both studies suggest early exposure to maternal depression, either prenatally or in the early postnatal years, may have a lasting impact on children's physiological stress systems.

In addition to finding relations between maternal depression and children's hormonal stress responses, studies have found alterations in autonomic activity in infants of depressed mothers. Specifically, researchers have found that infants of depressed mothers had higher heart rate and lower vagal tone than infants of non-depressed mothers. (Dawson et al., 2001; Field et al., 1988; Field, Pickens et al., 1995; Jones et al., 1998) Such findings have been observed as early as the neonatal period. For example, Jones et al. (1998) found that neonates of depressed mothers had lower vagal tone than neonates of non-depressed mothers. In a related study, infants of dysphoric mothers, diagnosed using the Beck Depression Inventory, were found to have lower heart rate variability during interactions with a stranger. Interestingly, these variations in heart rate

variability were predictive of both internalizing and externalizing problems at the preschool stage (ages 3-5 years) (Field et al., 1996). In another study, Field and colleagues (Field, Pickens et al., 1995) found that, compared to infants of non-depressed mothers, infants of depressed mothers had lower vagal tone at 6-months, but not at 3-months. Field et al (1995) suggest that the infants of depressed mothers failed to exhibit the typical developmental increase in vagal tone between 3 and 6-months of age.

Summary: Maternal Depression and Alterations in Psychophysiological Systems Related to Emotion Regulation and Expression

Maternal depression has been associated with alterations in children's biological systems related to emotion regulation and expression. Alterations in these systems are manifested in atypical frontal brain activation, stress hormone levels, and autonomic responses. Research has yet to make clear how alterations in these biological systems relate to the adjustment problems observed in children of depressed mothers, or how these systems may relate to possible mechanisms of transmission of risk from mother to child. Researchers have hypothesized several possible mechanisms of transmission of risk. The next section examines each of these mechanisms in turn.

Possible Mechanisms Underlying the Risk for Psychopathology in Children of Depressed Mothers

As is apparent from the above literature review, much has been written about the association between maternal depression and adverse child outcomes both in terms of child behavior and psychophysiology. Relatively less research has examined the possible mechanisms that might account for the negative outcomes observed in children of depressed mothers. Among the studies that have examined mechanisms of risk, four

candidates have emerged as possible mechanisms for the transmission of risk from mother to child. These are 1) genetic vulnerability, 2) prenatal exposure to biochemical alterations in the intrauterine environment, 3) disturbed mother-child interactions, and 4) contextual risk factors associated with maternal depression, such as marital conflict and life stress (Goodman & Gotlib, 1999). The following section evaluates the existing literature to support each of these mechanisms.

Genetic Vulnerability

The genetic vulnerability hypothesis suggests children of depressed mothers have increased rates of depression because they inherit genes that directly increase their vulnerability to depression. Another form of the genetic hypothesis suggests children inherit a vulnerability to personality traits or specific patterns of behavior that subsequently increase vulnerability to depression and other behavior problems. Support for the heritability of depression comes from twin, adoption, and family studies. These studies have found that adult first-degree relatives of a person with unipolar depression have a significantly increased risk for major depression compared to the risk in the general population (20-25% vs. 7%) (see Nurnberger, Goldin, & Gershon, 1986; Tsuang & Faraone, 1990 for reviews).

Although evidence for the heritability of depression exists, the strength of the genetic contribution is questionable and appears to vary with a number of factors including the timing and severity of the parent's depression and the gender of the child (Goodman & Gotlib, 1999). For example, major depression has been found to be associated with higher heritability than less severe, sub-clinical depression, which may be more influenced by environmental factors (Goodman & Gotlib, 1999). With respect to

the timing of the parent's depression, Weissman and colleagues (Weissman et al., 1984; Weissman, Warner, Wickramaratne, & Prusoff, 1988) found that adults with early-onset depression (before age 20) were more likely to have a child with depression, suggesting a greater heritability for early-onset depression compared to depression experienced later in life. Heritability may also be stronger for girls than for boys (Goodman & Gotlib, 1999). Finally, as described above, children of depressed mothers are vulnerable to a variety of adverse outcomes, including depression, anxiety disorders, conduct problems, and alcoholism. Thus, the risk appears nonspecific, adding to the notion that genetic factors alone are unlikely to fully account for the high risk for affective disorders and behavioral disturbances found in children of depressed mothers (Moldin, Reich, & Rice, 1991; Todd, Neuman, Geller, Fox, & Hickok, 1993).

Prenatal Exposure to Biochemical Alternations in the Intrauterine Environment

A second model for how maternal depression exerts its effects on children involves alterations in the intrauterine environment. This model, primarily proposed by Tiffany Field and colleagues, suggests that alterations in uterine blood flow or maternal neuroendocrine levels during pregnancy may impact the development of the fetus and increase the child's vulnerability to psychopathology later in life (Field, 1995; Lundy et al., 1999). Specifically, these researchers posit that elevated levels of norepinephrine and cortisol associated with depression may cross the placental barrier and either directly influence neonatal behavior or indirectly influence neonatal behavior and neuromotor development by affecting intrauterine blood flow (Lundy et al., 1999).

Evidence for a prenatal mechanism comes from research examining the psychophysiology and behavior of very young infants of depressed mothers. It is argued

that less optimal behaviors present very early in infancy most likely reflect prenatal or genetic influences because the infant has had so little opportunity to interact with the environment. Recently, several studies have reported less optimal behaviors and psychophysiology in newborns of depressed mothers (Abrams et al., 1995; Hernandez Reif et al., 2000; Jones et al., 1998; Lundy et al., 1999). Behaviorally, newborn infants of depressed mothers have been found to have less optimal scores on the Brazelton in terms of orientation, activity levels, and levels of excitability and withdrawal (Abrams et al., 1995; Jones et al., 1998; Lundy et al., 1999). Hernandez Reif and colleagues (Hernandez Reif et al., 2000) also reported that newborns of mothers with elevated depressive symptoms spent less time exploring oral stimuli. Physiologically, newborn infants of depressed mothers have been found to exhibit relative right frontal EEG asymmetry and lower vagal tone (Jones et al., 1998), as well as elevated levels of cortisol and norepinephrine and lower dopamine levels (Lundy et al., 1999). Interestingly, in the study by Lundy and colleagues (Lundy et al., 1999), the mother's prenatal norepinephrine and dopamine levels predicted the neonate's norepinephrine levels, dopamine levels, and performance on the Brazelton.

Field (Field, 1995; Field, 2000) has suggested the prenatal effects of maternal depression on infant development may be compounded by postnatal mother-infant interactions. As will be discussed below, researchers have identified different interaction styles among depressed mothers and these less optimal styles of interacting may compound the prenatal effects of maternal depression on the infant (Field, 2000). It is also possible that the more sympathetically aroused infant may be more difficult to interact with for an already depressed mother. This possibility highlights the need for

research examining the transactional relationship between mother and child. The next section describes the parenting styles and interaction patterns of depressed mothers.

Disrupted Mother-Child Interactions

In studying the interaction patterns of depressed mothers with their infants, researchers have identified two prominent styles of interacting characterized by withdrawn versus intrusive or insensitive behaviors on the part of the mother (Lovejoy, Graczyk, O' Hare, & Neuman, 2000). Withdrawn depressed mothers tend to express less positive and more negative affect, talk less, and disengage more from their infants (Cohn et al., 1986; Cohn & Tronick, 1989; Field, 1986, 1995; Field et al., 1988). Intrusive or insensitive depressed mothers are engaged with their infant in a negative manner that appears angry or irritable (Cohn et al., 1986; Dawson & Ashman, 2000; Hart, Field, del Valle, & Pelaez Nogueras, 1998; Jones, Field, Fox, Davalos et al., 1997). Intrusive behaviors include poking, restraining, directing the infants' attention, thrusting or withdrawing a toy, or otherwise failing to follow the infant's lead (Hart et al., 1998). There is some evidence to suggest negative maternal behaviors on the part of depressed mothers may be state dependent as they are associated more with current than lifetime depression (Lovejoy et al., 2000).

Researchers have also observed less optimal parenting behaviors in depressed mothers beyond infancy. Jameson and colleagues (Jameson, Gelfand, Kulcsar, & Teti, 1997) observed that depressed mothers were less adept at repairing breaks in interactions with their toddlers and spent less time in coordinated interactions with their toddlers compared to non-depressed mothers. Jameson et al. (1997) posit that depressed mothers may be less inclined to repair child-initiated breaks in interaction because they either lack

the energy to repair the interaction or interpret the break as a rejection. In a study of school-age children and adolescents, Fendrich and colleagues (Fendrich et al., 1990) found that children of depressed parents reported higher rates of "affectionless control"—low caring coupled with overprotection—in their parents.

Although patterns of disturbed mother-child interactions have been observed in families with a depressed mother, maternal depression is not invariably associated with disturbed mother-child interactions. Researchers have observed significant variability in interactions between depressed mothers and their children (Cox, Puckering, Pound, & Mills, 1987; Lovejoy et al., 2000). Some of the observed variability appears to be associated with the chronicity of the mother's depression, individual child characteristics such as age and developmental level, and the presence or absence of associated risk factors such as low SES and unemployment (Cohn, Campbell, Matias, & Hopkins, 1990; Field et al., 1996; Hart et al., 1998; Lovejoy et al., 2000; NICHD Early Child Care Research Network, 1999; Tronick & Weinberg, 2000). Chronic depression or depression that occurs in the context of other risk factors may impair parenting to a greater extent because women with chronic depression or a stressful family environment may have less energy to devote to parenting. Several recent studies have found that chronically depressed mothers and unemployed or low SES depressed mothers interact less optimally with their children (Campbell et al., 1995; Cohn et al., 1990; Field et al., 1996; NICHD Early Child Care Research Network, 1999). For example, a recent longitudinal study by the NICHD Early Child Care Research Network (1999) reported that chronically depressed women who also had low income-to-needs ratios were least sensitive to their young children during videotaped mother-child interaction sessions. From a

developmental perspective, there is some evidence that young children, especially infants, experience the most impaired parenting from depressed mothers (Lovejoy et al., 2000).

Several research studies have reported that depressed mothers display more negative interactive behaviors with their sons compared to their daughters (Hart et al., 1998; Tronick & Weinberg, 2000). Tronick and Weinberg (2000) found that depressed mothers were more affectively negative with their sons, and Hart et al. (1998) found that depressed mothers were more intrusive with their sons. In addition, Tronick & Weinberg (2000) found that sons appeared more affected by negative maternal behaviors; compared to daughters, infant sons of depressed mothers were less emotionally positive and less likely to use self-regulation strategies.

While these data suggest that depressed mothers interact differently than non-depressed mothers, research has not yet demonstrated that these behaviors are specific to depression or have a causal link to adverse child outcomes (Downey & Coyne, 1990). Researchers have hypothesized that the disrupted patterns of interaction observed between depressed mothers and their young children may interfere with the child's development of emotion regulation and thus increase a child's vulnerability to developing depression and other psychopathology. Tronick and Gianino (1986) have proposed a "Mutual Regulation Model" in which the depressed mother's inadequate responding to the infant's signals leads to poorly coordinated interactions and increased negative affect on the part of the infant. The infant may initially attempt to engage the parent using "approach" self-regulation strategies, such as signaling the mother, but after repeated

failures, the infant may turn to less effective, "withdrawal-type" self-regulatory strategies, such as thumb-sucking and gaze aversion.

Few studies have directly examined whether maternal behaviors directly increase risk for negative outcomes in children of depressed mothers. Some recent research, however, has provided evidence to support the impact of early interactions on child psychophysiological and behavioral outcomes. Zahn-Waxler and colleagues (Zahn Waxler et al., 1990) reported that a mother's early parenting behaviors, especially her ability to assume the child's perspective and exert respectful control, predicted children's externalizing problems at age 5 years. Carter and colleagues (Carter et al., 2001) found that the mother's emotional availability during infancy predicted later internalizing symptoms for girls. Two recent longitudinal studies have found that maternal behaviors partially mediate the relation between maternal depressive symptoms and child outcomes (NICHD Early Child Care Research Network, 1999; Spieker et al., 1999). The NICHD Early Child Care Research Network (1999) found that observed maternal sensitivity mediated the effects of maternal depression on measures of young children's cognitive and linguistic development. Maternal sensitivity also appeared to buffer the negative effects of maternal depression on young children's expressive language and cooperative behaviors; sensitive maternal behaviors were associated with better outcomes in the children of depressed mothers. In a study of older preschool and early school age children, Spieker and colleagues (Spieker et al., 1999) found that maternal negative control, including physical punishment and verbal yelling and threatening, partially mediated the relation between maternal symptoms of depression and anxiety and child disruptive behavior problems.

Contextual Factors

The above research suggests genetic and prenatal factors as well as disruptions in mother-child interactions may contribute to vulnerability to psychopathology in children of depressed mothers. While these factors are important, they do not tell the entire story. Children of depressed mothers frequently live in a stressful context characterized by high family stress, marital conflict, family conflict, lack of social support, and/or financial stress, such as low SES or unemployment. These contextual risk factors may contribute directly or indirectly to the development of child behavior problems (Adrian & Hammen, 1993; Cicchetti et al., 1998; Coyne, 1976; Emery, Weintraub, & Neale, 1982). Several studies have found that children of depressed mothers are exposed to more stressful life events (Adrian & Hammen, 1993; Cicchetti et al., 1998; Hammen, 2002), including interpersonal stressors to which they may have contributed, such as family and peer conflict (Adrian & Hammen, 1993). The presence of associated risk factors may compound risk for negative outcomes in children of depressed mothers, or, alternatively, account for the relation between maternal depression and child outcomes. Thus far, the literature has provided support for both possibilities. However, because much of the research is cross-sectional and because depression and life stress tend to co-occur, these studies have not established whether contextual risks precede or follow the development of maternal depression.

Some evidence suggests contextual risk factors predict child outcome above and beyond the direct effects of maternal depression (Duggal et al., 2001; Seifer et al., 1996). For example, using a multiple risk model which included contextual risk measures such as life events, family size, mother education, father absence, and minority status, Seifer

and colleagues (Seifer et al., 1996) found significant associations between child social competence and contextual risk factors even after controlling for the effects of maternal depression. Multiple contextual risks were associated with more negative outcomes. Maternal depression still remained a significant predictor in the model. Other studies suggest contextual risks partially account for the relation between maternal depression and child outcomes (Cicchetti et al., 1998; Dawson et al., in press; Fergusson et al., 1995). Cicchetti and colleagues (Cicchetti et al., 1998) reported that families with depressed mothers have more stressful life circumstances (e.g. more stress and daily hassles, less marital satisfaction) and that these contextual risk factors mediate the relation between maternal depression and child behavioral outcomes, particularly externalizing behavior problems. In a longitudinal study, Fergusson and colleagues (Fergusson et al., 1995) found that, for adolescent girls, the relation between maternal depression and adolescent depressive symptoms was partially accounted for by contextual risks, such as stressful life events and marital conflict. Research on marital conflict in particular has also found that the relation between parental affective illness and children's behavior at school can be largely accounted for by marital discord (Emery et al., 1982). Other research has not found support for the role of contextual risks in the relation between maternal depression and child outcome. Carro and colleagues (Carro et al., 1993) found that marital satisfaction and social support did not moderate the relation between maternal depression and child behavior at age 2 – 3-years-old. However, their measures of contextual risk factors were assessed during the postpartum period and not contemporaneously with measures of child behavior problems.

To understand the mechanism through which contextual risk factors and, more specifically life stress, may affect the association between maternal depression and child outcomes, Hammen and colleagues (Adrian & Hammen, 1993; Hammen, 1988, 1992, 1996, 2002) have proposed a developmental psychopathology model of depression in which children's maladaptive social skills and cognitions about self-worth and self-efficacy serve as mediators between maternal depression and the development of depression in children. In this model, the presence of a depressed parent and the stressful context of children's lives impairs the development of adaptive social skills and contributes to the generation of more interpersonal stress and conflict. Specifically, depressed parents are hypothesized to model inadequate coping skills and poor interpersonal problem solving which their children acquire. Subsequently, interpersonal difficulties in the lives of both the depressed mother and the child contribute to the generation of additional stressful life events which precipitates further depression. Such a model emphasizes the need for research examining the contextual risk factors in children's lives as well as the transactional relationship between children and their environments.

Summary: Possible Mechanisms Underlying the Risk for Psychopathology in Children of Depressed Mothers

Researchers have investigated a number of possible mechanisms for the transmission of maternal psychopathology from mother to child. The most widely researched of these mechanisms are 1) genetic vulnerability, 2) prenatal alterations in the intrauterine environment, 3) disrupted mother-child interactions, and 4) contextual risk factors. While there is evidence to support each of these mechanisms separately, it is

more likely that these mechanisms interact or compound each other in some way to produce negative outcomes in children of depressed mothers. In examining each of these possible mechanisms, it is clear more research is needed to elucidate the relative impact of each level of risk on children of depressed mothers as well as how these risk factors might interact to produce a more comprehensive model of risk for psychopathology in children of depressed mothers.

Purpose and Goals

The present study used existing data from a 6-year longitudinal study of children of mothers with and without a history of depression in order to address some of the unanswered questions from the literature. The original research project, funded by the National Institute of Mental Health (Geraldine Dawson, Principle Investigator; No. MH47117), permitted longitudinal data collection on maternal depression, contextual risk and child outcomes from 14 months to 7 years of age. Extensive behavioral and psychophysiological child outcome data were collected at 14 months, 3 ½ years, and 6 ½ years of age. Child outcome data have been analyzed and published from the first two major time points. The present study focuses on the third major time point (6 ½ years of age). The uniqueness of this dataset lies in its comprehensive assessment of the mother's depression over time and its adoption of a psychophysiological approach to the study of the development of emotion regulation and psychopathology.

The present study had three main goals. The first goal was to identify patterns of maternal depression over the child's first 7 years of life that might be predictive of differences in child psychophysiology and behavioral outcomes at age 6 ½ years. Although many studies have examined the relation between maternal depression and child adjustment problems, this study advanced the existing literature because it used a month-by-month assessment of the mother's depression to chronicle the intervals between assessments. To date, most longitudinal studies of maternal depression have failed to gather detailed information about the mother's depression over time, depicting maternal depression as a relatively homogenous construct that influences child outcome through its absence or presence. It was hypothesized that heterogeneity in depressive

symptoms over time would exist and different patterns of change in maternal depression would predict different child outcomes. Specifically, it was predicted that at least four different trajectories of maternal depression would emerge from the data: mothers with a relatively high and stable level of chronic depression, mothers whose depression remitted early in the child's life, mothers who experienced relatively infrequent, minor depression throughout the study period, and mothers who experienced no depression during the child's lifetime.

The second goal of this study was to examine differences in child behavior, electrical brain activity, and stress responses in 6 ½ –year-old children of mothers with different patterns of depression over time. Few studies to date have examined the association between the early social environment and alterations in biological systems related to emotional development in children of depressed mothers beyond infancy and toddlerhood. During infancy, researchers observed reduced left frontal brain activity and increased heart rate among children of depressed mothers (Dawson et al., 2001; Dawson et al., 1997). During the preschool years, Dawson and colleagues found reduced brain activation across all brain regions assessed, including left and right frontal and parietal scalp regions (Dawson et al., in press). With the exception of one study (Jones, Field, Davalos et al., 1997), the relation between maternal depression and children's atypical brain activity and stress responses has not been studied longitudinally to see if the observed differences are transient or enduring, or whether they are predictive of children's social and emotional competence later in life.

Given the existing literature, it was hypothesized that six-year-old children whose mothers had experienced more stable, chronic, and severe depression would exhibit

elevated levels of emotional and behavior problems, decreased social competence, and atypical stress responses and brain activity from regions related to emotion regulation. Specifically, it was expected that children of depressed mothers would exhibit reduced generalized frontal brain activation and elevated stress responses as reflected in increased heart rate and elevated salivary cortisol levels. A secondary hypothesis was that there would be some enduring effects of early maternal depression that remitted earlier in the child's life particularly with respect to internalizing problems, suggesting a possible sensitive period in the development of emotion regulation and the biological systems underlying emotion regulatory behaviors. It was also expected that children's psychophysiological responses would be related to their behaviors. Previous research has found associations between aggressive behavior and low autonomic arousal (low heart rate and low vagal tone), low cortisol levels, and reduced right frontal brain activation (Raine, 2002; Raine, Venables, & Mednick, 1997; Ryan, 1998). Depression and internalizing symptoms have been associated with relative right frontal EEG asymmetry, elevated cortisol levels, lower vagal tone, and higher vagal reactivity (Fox, Schmidt, Calkins, Rubin, & Coplan, 1996; Ryan, 1998; Schmidt, Fox, Schulkin, & Gold, 1999). Based on previous research, children with relative right frontal EEG asymmetry and elevated stress responses as reflected in higher cortisol, higher heart rate, and lower RSA were expected to display more anxious and withdrawn behaviors and depressive symptoms. Children with reduced right frontal brain activation, lower cortisol levels, lower heart rates, and lower RSA were expected to have higher levels of externalizing, especially aggressive behavior problems.

The final goal of this study was to examine the combined influence of maternal depression, child psychophysiology, and contextual risk factors on child behavior at age 6 ½ years. Contextual factors measured were family conflict, stressful life events, parenting stress, social support, and marital satisfaction. Child behavior outcomes were modeled as a joint function of maternal depression, contextual risk factors, and child psychophysiology at age 6 ½ years. Psychophysiological measures were hypothesized to index emotional and behavioral self-regulation. Based on previous research, (Cicchetti et al., 1998; Fergusson et al., 1995), including an analysis of the same sample when the children were 3 ½ years old (Dawson et al., in press), it was expected that contextual risk factors and child frontal brain activity would partially mediate the association between maternal depression and child behavior outcomes.

Methods

Participants

Participants were part of a sample of 159 mother-child dyads participating in a longitudinal study of the effects of maternal depression on children's psychobiological and emotional development. Mothers were initially recruited when the children were 14-months-old, from a variety of sources, including the Psychology Department infant subject pool, newspaper advertisements, community hospitals and clinics. Prior to entry in the study, mothers and children were carefully screened for medical and psychiatric problems. Mothers were excluded if they reported substance abuse, serious medical conditions, attendance in special education classes, serious psychiatric disturbance other than depression (e.g. bipolar disorder, mania, psychosis), immanent suicide, significant pregnancy or birth complications and/or contact with Child Protective Services. Infants were excluded if they weighed less than 5 lbs at birth, were born more than 3 weeks premature or late, had a history of chronic seizures, central nervous system infection, head injury, prolonged hospitalization or chronic medical condition, surgery, physical malformations, sensory or motor problems, prenatal drug exposure, foster care, and/or were taking medications.

Participants were followed longitudinally over the next 5 years during which there were four additional contact points: ages 24 months, 3 ½ years, 4 ½ years, and 6 ½ years. Of the 159 mother-child dyads originally recruited into the study, 138 (86.8%) participated at the 6 ½ year contact. In addition, at the 6 ½ year contact, one child was living with her father and non-biological stepmother. This non-biological mother was also interviewed at the last contact, bringing the total number of participating mothers to

139. Of the 21 mother-child dyads who did not participate at the 6 ½ year contact, ten could not be located for contact, nine refused to participate, and two could not schedule the interview before the end of data collection. Compared to mothers who participated at the 6 ½ year contact, non-participating mothers were more likely to have been depressed (85.7% vs. 52.2% of participating mothers; χ^2 (1) = 8.35, p = .004) and less likely to have been married (52.4% vs. 93.5% of participating mothers; χ^2 (1) = 29.26, p < .001) at initial contact. Non-participating mothers were also younger at initial contact, with non-participating mothers having a mean age of 27.0 years (SD = 5.44) and participating mothers having a mean age of 31.1 years (SD = 4.54; t (157) = -3.79, p < .001), and of a lower socioeconomic status (SES) as measured by the Hollingshead (1975) at initial contact, with non-participating mothers having a mean SES of 36.8 (SD = 16.56) and participating mothers having a mean SES of 45.4 (SD = 11.07; t (157) = -3.11, p = .002).

Most of the mothers who participated at the age 6 ½ year contact were Caucasian, married, middle-class, and college-educated. One-hundred twenty-two (88.4%) mothers were Caucasian. The remaining mothers were Asian-American (.7%), Hispanic (.7%), Native-American (.7%), multi-ethnic (1.4%), and other (1.4%). Ten mothers (6.5%) did not identify themselves as belonging to a particular ethnic group. Seventy-nine percent of mothers were married to the child's biological father at the time of the age 6 ½ year interview. 66.7% of mothers were working outside the home. Mean Hollingshead (1975) estimate of SES was 47.1 $\pm$ 10.9. Mothers had an average of 2 children (range 1-6).

Of the 138 children who participated, 78 (56.5%) were girls and 60 (43.5%) were boys. Most children (68.1%) were attending the first grade at the time of testing. 21% were in second grade, 10.1% were in kindergarten, and .7% were not in school. Most children (73.9%) were attending public schools. 20.3% were attending private schools and 5.8% were home-schooled.

Diagnosis of Depression and Depression Groupings

Mothers were interviewed about their depressive symptoms using the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First, 1989), the Center for Epidemiological Studies Depression Questionnaire (CES-D; Radloff, 1977), and a modified interview version of the Longitudinal Interval Follow-up Evaluation (LIFE; Keller et al., 1987; Keller, Shapiro, Lavori, & Wolfe, 1982). Using these measures, mothers were interviewed about their symptoms of depression on five separate occasions, when their children were 14-months-old, 24-months-old, 3 ½ years old, 4 ½ years old, and 6 ½ years old. At each contact point, mothers were interviewed retrospectively about the time period between the current and most recent contacts.

Structured Clinical Interview for DSM-III-R. The SCID is a semistructured interview used to diagnose mental disorders based on DSM-III-R criteria. The SCID is administered by trained clinicians who are familiar with DSM-III-R diagnostic criteria and classification. In this study, trained clinical psychology doctoral students administered the mood disorders and psychotic screening modules to participating mothers. Diagnostic interviewers were carefully trained and interrater reliability on the SCID was excellent; percent agreement on diagnosis ranged from 90 – 100 percent.

Center for Epidemiological Studies Depression Scale. The CES-D is a 20-item self-report scale of depressive symptoms with scores ranging from zero to 60. Scores greater than or equal to 16 are considered to be within the clinical range (Myers & Weissman, 1980). The CES-D has been shown to have high internal consistency (alphas .85 - .90) in both psychiatric and community populations and good concurrent and construct validity (Radloff, 1977).

Longitudinal Interval Follow-up Evaluation. The modified LIFE provided a month-by-month assessment of the mother's depression (non-depressed, subthreshold depression, or major depression) since the child's conception. Recent research has shown the LIFE is a reliable measure of the longitudinal course of psychiatric disorders (Warshaw, Dyck, Allsworth, Stout, & Keller, 2001). Definitions of subthreshold and major depression were based on SCID criteria. Subthreshold depression was diagnosed when the mother had experienced depressed mood or anhedonia most of the day, nearly every day for at least 2 weeks plus at least 2 other symptoms of depression OR when the mother had experienced depressed mood or anhedonia for at least ½ of a two-week period plus three other symptoms of depression.

At the initial contact when children were 14-months-old, data from all three measures were combined to assign mothers to groups based on their depression status. Of the 159 mothers in the study, 90 were categorized as depressed and 69 were categorized as non-depressed at initial contact. Mothers in the non-depressed group had CES-D scores less than 9 and reported no current major depression or history of depression since the child's conception on the SCID or LIFE.

At the 6 ½ year assessment, 14 (10.1%) mothers received a current SCID diagnosis of depression. Thirteen mothers received a current diagnosis of major depression and one subthreshold depression. Eleven of the 14 depressed mothers had also received a current diagnosis of mood disorder when their children were 14-months-old (major depression = 6, depression in partial remission = 4, dysthymia = 1).

Procedures and Measures at Age 6 ½ Years

The age 6 ½ years assessment took place at the Center on Human Development and Disability at the University of Washington. Mothers and children participated in three laboratory visits: 1) a diagnostic and neuropsychological assessment during which mother and child were interviewed separately by trained graduate student research assistants, 2) an assessment of child psychophysiology during which the child watched several emotion-eliciting video clips while his/her brain activity and heart rate were monitored, and 3) a videotaped mother-child interaction session. Child questionnaires were completed in interview format during the diagnostic visit. All mother questionnaires were completed between visits with the exception of the Family Environment Scale (Moos, 1974), which was completed at the mother-child interaction visit. Teachers were contacted after the first visit for completion of questionnaires assessing child behavior and adjustment at school. Fathers were contacted after the third visit for completion of questionnaires assessing child behavior, father mental health, and family functioning. Fathers who agreed were also contacted by telephone for completion of a brief telephone interview regarding family mental health history.

Families were not always able to complete all three visits because of scheduling difficulties. One hundred eight mother-child dyads completed all three laboratory

sessions. Six families completed only the first visit. Two families completed visits one and two, six families completed only visits one and three, and one family completed only visit two. In addition, in order to minimize attrition, mothers who had moved out of the area were contacted for long distance telephone interviews. Sixteen mothers who no longer lived within commuting distance of the university were interviewed by telephone and completed questionnaire packets through the mail.

Assessment of Child Psychophysiology

Child electrical brain activity, heart rate, and respiratory sinus arrhythmia (RSA). Psychophysiological testing took place in Dawson's Developmental Psychophysiology Laboratory at the Center on Human Development and Disability at the University of Washington. In order to make the room more attractive to 6 ½-year-old children, lab assistants decorated the room to resemble a space capsule. During the EEG/ECG session, children sat in a chair across from a black curtain behind which there was a video camera for recording child affect and behavior. A television monitor was located in front of the black curtain. Children's EEG and ECG were recorded during six conditions. In the first condition (Baseline 1), children watched while soap bubbles cascaded over the curtain for 90 seconds. This baseline condition was followed by the presentation of four brief film clips designed to elicit specific emotions (happy, sad, neutral, and anxious/scared). The clips included segments from the movies, "Andre" (happy), "Where the Red Fern Grows" (sad), "Fantasia" (neutral), and "Jurassic Park" (anxious/scared). All segments were accompanied by music, but spoken language was absent or minimal. Children were asked not to talk while watching the videos. The order of presentation of the video clips was counterbalanced among the children. After each video clip, the video was paused

while the experimenter administered a brief emotion self-report instrument designed to assess the child's self-reported mood during the film clip. After the emotion self-report, there was a 15-second recovery period during which the children viewed a neutral star field on the screen. Following the presentation of all four video clips, the children viewed another 90-seconds of cascading bubbles (Baseline 2). During the entire session, the mother was seated behind her child during the EEG recording and asked to remain quiet and maintain a neutral expression if her child looked toward her. Analysis of the emotion self-report instrument indicated that, for the majority of children, their self-reported emotion matched the emotion elicited during the film clip (Hibbeln, 2002).

EEG recording methods. Children were entertained with a cartoon video while the experimenter prepared the child's scalp for EEG recording. The experimenter used Omni-prep as an abrading compound prior to attaching individual silver-silver-chloride electrodes to the scalp with Grass EEG cream. EEG was recorded from six scalp locations: left and right frontal, parietal, and temporal (F3, F4, P3, P4, T3, T4; International 10-20 system). Active EEG leads were referenced to linked right and left mastoid electrodes for which identical impedances were obtained by matching the lower impedance to the higher one using a potentiometer. A forehead electrode served as ground. Eye movements were recorded for offline removal of ocular artifact via electrodes placed on the external canthus and supra orbit positions. All impedances were under 5 Kohms, measured before and after testing.

EEG was recorded on a Grass Neurodata Acquisition System (Model 12). The high pass active filter was set at 1 Hz and the low pass filter at 30 Hz. Analog-to-digital

conversion was based on a 512 points / sec sampling rate. Digitized data were stored continuously on an IBM PC-AT.

EEG editing and analysis. James Long Co. (Caroga Lake, New York) EEGEDIT software was used to visually edit EEG for motor artifacts, including EOG. Analyses required a minimum of 15 sec of artifact-free EEG.

Discrete Fourier analyses were performed on all artifact-free EEG data using custom-made software developed by James Long Co. (Caroga Lake, New York). Analyses were based on one-second epochs and used a Hanning window of one second, with half-second overlapping windows. The Fourier analyses yielded EEG spectral power in the 7 - 13 Hz band. Analyses focused on the alpha band, 7 - 13 Hz range, a measure of brain activation or arousal (Steriade, 1981), with lower EEG alpha power indicating higher levels of brain activation or arousal. The distribution of EEG spectral power was normalized using natural log transformations. Asymmetry scores (ln right EEG power minus ln left EEG power) were derived to reflect relative activation of homologous right and left hemisphere regions. Because reduced EEG alpha power indicates increased brain activation (Steriade, 1981), positive asymmetry scores indicate relative higher levels of left frontal activation and negative asymmetry scores indicate relative lower levels of left frontal activation.

Heart rate recording and analysis. Heart-rate data were obtained by placing two electrodes on the children, one on the sternum and a second one on the lower left costal region. The ECG signal was then amplified and filtered through a high pass filter set at .1 Hz. The signal was then digitized through an Analog to Digital (A/D) converter at 512 samples per second. The digitized data were stored on a hard disk for off-line processing.

The timing of the R-waves was extracted using a program designed by James Long, Inc. (Caroga Lake, NY). The output of the R-wave detection was edited and corrected by visual inspection. The corrected records were in turn segmented by experimental condition and R-to-R-wave intervals were entered into an analysis program designed by Steve Porges that computed heart rate.

Heart-rate variability was computed from the ECG record. Interbeat intervals (IBI) between R-waves were algorithmically extracted. The records were then visually inspected and corrected for errors. IBI values were then entered into Mxedit for further visual inspection and heart rate variability computation. Mxedit uses the Porges-Bohrer algorithm for band-pass filtering of IBI data that is based on a moving polynomial to remove non-stationarities. Filtering values for children between .24 and 1.04 with a polynomial order of 3 and a coefficient number of 21 were used in each condition analysis. The Porges-Bohrer moving polynomial algorithm estimates respiratory sinus arrhythmia (RSA), which is a measure of vagus nerve activity or vagal tone.

Salivary cortisol collection and assay. Saliva samples were collected from the children on four occasions, twice in the laboratory setting and twice at home. Home samples were collected as a more accurate measure of baseline salivary cortisol levels. In the lab, saliva collection occurred twice during the initial interview visit, at baseline (approximately 20 minutes after arrival at the lab) and 20-30 minutes after a mildly stressful task, the 3-ball task of the Tower of London task of the Cambridge Neuropsychological Test Automated Battery (CANTAB). At home, mothers were instructed to collect samples from their children at a time roughly corresponding to the baseline sample taken at the lab and 30 minutes before bedtime on the same day.

Mothers were instructed to collect samples on an "average" day, when their children woke up and went to bed as close as possible to their regular times. After collecting the samples, mothers answered two questions assessing children's health and sleep on the day of home saliva collection.

Saliva samples (100 – 200 *u*l) were obtained by asking the children to chew on a piece of sugarless gum and then spit through a short straw into the collection tube. After obtaining a sufficient amount of saliva, the lab samples were immediately stored at -20°C until assayed. Mothers stored the home saliva samples in their home refrigerators until ready to return the samples to the lab via surface mail. Upon receipt at the lab, the home saliva samples were immediately frozen. Immediately prior to being assayed, thawed saliva samples were centrifuged at 14,000 rpm for 7 minutes to remove particulate matter. The supernatant was recovered and 0.1 ml aliquots were assayed in duplicate with cortisol radioimmunoassay kits from Pantex (Santa Monica, CA) using a modification of the commercial protocol to optimize the measurement of cortisol in saliva. The minimum sensitivity of the assay was 0.02 *u*g/dl. The intra-assay coefficient of variation was 7.2% and the inter-assay coefficient of variation was 9.9%.

Assessment of Child Emotional and Behavior Problems

Child emotional and behavior problems were assessed using multiple methods and multiple informants. During the first visit, mothers were interviewed using the Diagnostic Interview Schedule for Children IV—Parent Version (DISC-P; Shaffer et al., 1991). This computer-assisted structured interview assesses child behavior problems using DSM-IV criteria. The DISC-P has been shown to have good validity and reliability

(Shaffer, Fisher, Lucas, Dulcan, & Schwab Stone, 2000). Between visits, mothers and 104 fathers completed the Child Behavior Checklist (CBCL; Achenbach, 1979) and the Child Adaptive Behavior Inventory (CABI; Cowan, Cowan, Schulz, & Heming, 1994). Teachers also completed a variety of questionnaires designed to assess child behavior problems and school adjustments. These included the CBCL-Teacher Report Form, CABI, ADHD Rating Scale, and the Child Behavior Scale, peer subscales (see descriptions below).

For two children, both biological and non-biological fathers completed questionnaires. In both cases, the non-biological father was the parent who lived with the child and was the mother's current partner. In these cases, the non-biological father's reports were used because he was the parent who lived with the child and had more contact with the child. For one child, there were two separate teacher ratings completed, one from the first grade teacher and one from the second grade teacher. In this case, the two teacher ratings were averaged to achieve a more representative rating of the child's behavior at school. Finally, at the 6 ½ year-old contact point, one child was living with her biological father and stepmother. Ratings were obtained from both the biological mother, who lived out of state, and the stepmother. For the age 6 ½ child behavior outcomes, the stepmother's reports were used because she was the parent who lived with the child and had more contact with the child.

During the first visit, children were interviewed about their own perceptions of their moods and behaviors. A recent study found that young children are capable of valid reports of depressed mood and feelings (Ialongo, Edelsohn, & Kellam, 2001). Children were interviewed using the Dominic questionnaire (Valla, Bergeron, Berube, Gaudet, &

St-Georges, 1994), the Children's Depression Inventory (CDI; Kovacs, 1985, 1992), the Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978), and the Loneliness and Social Dissatisfaction Questionnaire (Cassidy & Asher, 1992) (see descriptions below).

Child Behavior Checklist. The Achenbach Child Behavior Checklist (CBCL; Achenbach, 1979; Achenbach & Edelbrock, 1983) is a 113-item questionnaire on which parents can report about their child's behavior problems, including both internalizing and externalizing behavior problems. On each item, parents answer on a 3-point scale about whether the item is not true, somewhat true, or very true for their child. The CBCL is standardized and has adequate validity and reliability (Sattler, 1990). Mothers and fathers completed the CBCL and teachers were asked to complete the Teacher Report Form, a teacher version of the CBCL.

Child Adaptive Behavior Inventory. The Child Adaptive Behavior Inventory (CABI; Cowan et al., 1994) is a 106-item questionnaire on which parents and teachers respond to items about a child's behaviors and social and academic competence. Items are rated on a 4-point scale ranging from (1) not at all like this child to (4) very much like this child. The scale yields 22 scale scores, which measure a variety of child attributes, such as intelligence, creativity, task orientation, hyperactivity, introversion, extroversion, and social skills. These scale scores are combined into six factor scores: academic competence, social competence, externalizing—aggressive, externalizing—hyperactive, internalizing—social isolated, and internalizing—anxious and depressed.

ADHD Rating Scale. The ADHD Rating Scale (DuPaul, 1991) is a 14-item questionnaire based on DSM-III-R criteria for attention deficit hyperactivity disorder.

The ADHD Rating Scale yields three scores: a total score, an inattention / restlessness score, and an impulsivity / hyperactivity score. For teacher ratings, alpha reliabilities of these scales ranged from .95 to .96 (DuPaul, 1991). The scale also demonstrated adequate criterion-related validity.

Child Behavior Scale. The Child Behavior Scale (CBS) (Ladd & Profilet, 1996) is a 59-item teacher-report scale used to measure young children's behaviors with peers at school. The scale produces six subscales: Aggressive with peers, prosocial with peers, asocial with peers, excluded by peers, anxious-fearful, and hyperactive-distractible. Only the peer subscales were used in this study. Alphas for the peer subscales ranged from .54 - .72 (Ladd & Profilet, 1996). The CBS also demonstrated adequate validity, correlating in the expected direction with other measures of child behavior with peers (Ladd & Profilet, 1996).

Dominic. The Dominic questionnaire (Valla et al., 1994) is a structured pictorial questionnaire for assessing DSM-III-R diagnoses in children ages 6 to 11-years-old. The Dominic takes into account the short attention spans and cognitive limitations of young children. The Dominic is a set of pictures of a boy or girl (the sex of the child in the pictures is ambiguous) called "Dominic" and children are asked to respond to "yes" or "no" questions about whether they act, think, feel, or are like Dominic. The questionnaire covers symptoms for DSM-III-R diagnoses of attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder, major depressive disorder, separation anxiety disorder, overanxious disorder, and simple phobia. The Dominic has been shown to have adequate reliability and validity given the cognitive limitations of young children (Valla et al., 1994).

Children's Depression Inventory. The Children's Depression Inventory (Kovacs, 1985, 1992) is a 27-item self-report questionnaire assessing symptoms of depression in children. For each item, the child is instructed to choose from three statements which one is the most like him/her. The CDI is the most widely used measure of depression in children (Kazdin, 1990).

The Revised Children's Manifest Anxiety Scale. The Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978) is a 37-item self-report questionnaire assessing symptoms of anxiety in children. Children answer "yes" or "no" about whether a statement describes him/her. A Lie Scale assesses for social desirability.

The Loneliness and Social Dissatisfaction Questionnaire. The Loneliness and Social Dissatisfaction Questionnaire (Cassidy & Asher, 1992) is a 24-item self-report questionnaire assessing children's feelings of loneliness, feelings of social adequacy, appraisal of their current peer relationships, and perceptions of whether their relationship needs are being met. Eight filler items focus on hobbies and interests. This measure has been shown to have adequate reliability when administered to young children (Cassidy & Asher, 1992).

Assessment of Family Contextual Risk Factors

Mothers completed a variety of questionnaire measures designed to assess other risk factors in the social environment that might relate to maternal depression and child outcome, including measures of stressful life events, social support, parenting stress, and marital satisfaction. These measures included the Life Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978), the Dyadic Adjustment Scale (DAS; Spanier, 1976), the Parenting Stress Index (PSI; Abidin, 1990), Family Environment Scale (FES; Moos,

1974), and the Social Support Questionnaire (SSQ; Sarason, Levine, Basham, & Sarason, 1983). A subset of fathers completed the DAS and PSI at the 6 ½ year contact. All measures were also administered at earlier time points in the study. The LES was administered at the 14-month, 3 ½ year, and 6 ½ year assessments. The PSI was administered at the 3 ½ year, 4 ½ year, and 6 ½ year assessments. The FES was administered at the 3 ½ year and 6 ½ year contacts. The DAS and SSQ were administered at all five contact points in the study. In order to take advantage of this longitudinal assessment of the social context, correlations across time points were assessed and measures that demonstrated stability over time were averaged across time points to create a composite measure.

The Life Experience Survey. The LES (Sarason et al., 1978) is a 46-item, self-report measure designed to measure the impact of life changes in the past year. Domains of change include personal events, changes in the makeup of your household, financial changes, changes in work, changes in your partner or relationships, changes in your family, and changes in friends or social events. The scale yields a positive change score, a negative change score, and a total change score. Test-retest reliability coefficients for the negative life change score were .56 and .88 for two separate samples (Sarason et al., 1978). The negative life change score was also found to correlate significantly with measures of anxiety, depression, academic achievement, and personality (Sarason et al., 1978). The LES total negative events score demonstrated moderate stability over the course of the study (mean $\underline{r}$ = .43, range = .34 - .62); therefore, this variable was averaged across the three time points.

The Dyadic Adjustment Scale. The DAS is a 32-item self-report measure that assesses marital or relationship quality. The DAS has been shown to have high internal consistency (alpha = .96 for the total scale score) and good content, criterion-related, and construct validity (Spanier, 1976). Correlations with the Locke-Wallace Marital Adjustment Scale were .86 for married respondents and .88 for divorced respondents (Spanier, 1976). The DAS total dyadic adjustment score demonstrated moderate stability over the course of the study (mean $\underline{r}$ = .72, range = .53 - .81); therefore, this variable was averaged across the five time points.

The Parenting Stress Index / Short Form. The PSI Short Form (Abidin, 1990) is a 38-item self-report measure of parenting stress. This measure was derived from a longer, 126-item scale. The short form contains three subscales: parental distress, parent-child dysfunctional interaction, and difficult child subscales. A global stress score can also be derived from this measure. The total stress score on the PSI Short Form has been found to have a correlation of .94 with the total stress score of the full-length PSI (Abidin, 1990). The short form has been found to have good test-retest reliability (.84) and internal consistency (Abidin, 1990). The PSI total stress score demonstrated moderate stability over the course of the study (mean $\underline{r}$ = .75, range = .69 - .78); therefore, this variable was averaged across the three time points.

Family Environment Scale. The FES (Moos, 1974; Moos, 1990) is a questionnaire designed to assess social and affective dimensions of families. The 63-item version used for this study contains seven subscales: cohesion, expressiveness, conflict, independence, achievement orientation, organization, and control. Respondents are asked to answer "true" or "false" as to whether a particular statement applies to their

family. The FES has been shown to have adequate validity and reliability (Moos, 1974; Moos, 1990). The internal consistency of the conflict subscale used in this study is .75 (Moos, 1990). The FES family conflict subscale score was moderately correlated across the two time points ($r = .51$, $p < .001$); therefore, this variable was averaged across the two assessments.

The Social Support Questionnaire. The SSQ (Sarason et al., 1983) is a self-report measure of perceived number of social supports and satisfaction with the available social support. The SSQ has been shown to have adequate reliability and internal consistency (Sarason et al., 1983). The SSQ total social support score demonstrated moderate stability over the course of the study (mean $r = .64$, range = .45 - .73); therefore, this variable was averaged across the five time points.

Contextual risk composite score. Data from the five contextual risk measures (LES, DAS, PSI, FES, SSQ) were combined to create a composite measure of contextual risk based on a model presented in Cicchetti et al. (Cicchetti et al., 1998). A similar composite was also used at an earlier time point in this study (Dawson et al., in press). For each measure, a total scale score or representative subscale score was selected as a global indicator of the particular domain being measured. The following scale scores were selected as indicators of each domain: LES total negative events scale for stressful life events, DAS total dyadic adjustment scale for marital satisfaction, PSI total stress score for parenting stress, FES family conflict subscale for family conflict, and SSQ total social support score for social support. For each variable a score of one standard deviation above or below the mean was established as a criterion for high risk and variables were transformed into dichotomous indicators of risk. For variables for which

higher scores reflected more adverse circumstances (stressful life events, parenting stress, and family conflict), one standard deviation above the mean was established as the criterion for high risk. For variables for which lower scores reflected more adverse circumstances (marital satisfaction and social support), one standard deviation below the mean was established as the criterion for high risk. If the high risk criterion was met for a particular variable, a score of one was assigned. The risk scores (zero or one) were then summed across the five domains to compute an overall contextual risk composite score, which ranged from zero to five.

Results

Phase 1: Identifying Classes of Mothers Based on Course of Depression

The first goal of the current study was to identify classes of depressed mothers based on variations in initial levels and rates of change in depressive symptoms over time. Latent growth mixture modeling, implemented in Mplus (Muthen & Muthen, 2001), was used to identify homogenous groups of depressed mothers based on specified growth parameters, including each mother's initial level and rate of change in depressive symptoms. Mplus Version 2.13 allows for missing values and uses all available data to estimate the model parameters; therefore, analyses were based on the complete sample of 159 mothers. One a priori maternal diagnostic group, non-depressed mothers (N = 26), was identified and excluded from the latent growth mixture models. This group included mothers who were non-depressed on all measures of depression throughout the study period. The remaining 133 mothers were used to estimate the latent growth mixture models.

Latent Growth Mixture Models

The dependent variable used in the growth mixture models was maternal depressive symptoms as assessed by the LIFE. Although the LIFE provides a month-by-month assessment of the mother's depression, the amount of data in each month (no depression, subthreshold depression, major depression) does not provide enough information for a meaningful assessment of change in the mother's depression over time. Therefore, using the LIFE data, variables were created based on the sum of the number of months of maternal depression experienced during each year of the child's life. Each month was weighted for severity (0 = no depression, 1 = subthreshold depression, 2 =

major depression) before being summed, with months of major depression receiving greater weighting than months of subthreshold depression. The result of this process was variables (which ranged from 0 to 24) representing the average level of maternal depression across each year. Seven variables were created representing frequency and severity of depression during each year from the child's birth through age 7 years.

A visual examination of the LIFE data indicated there might be a significant nonlinear growth pattern to the data, suggesting that fitting a quadratic growth model to the data might be warranted. Therefore, as a preliminary step, two growth curve models were estimated with change in depression over time specified as a function of linear and quadratic growth parameters. Initial status when the child was 1-year-old was selected as the intercept because of interest in modeling how maternal depression changed from study entry. Table 1 presents the results of this analysis. The likelihood ratio test was conducted to compare the fit of the linear vs. the quadratic model. The model including a quadratic growth parameter was found to be significantly different from the linear model (χ^2 diff (4) = 37.89, $p < .01$). Furthermore, the quadratic mean and variance were significant, suggesting that a quadratic model was appropriate for the data. Given these findings, a quadratic growth model was used for the subsequent analyses.

The next set of analyses investigated the existence of two or more subgroups of depressed mothers with varying patterns of initial level and growth trajectory in depression. To determine the optimal number of latent growth classes, three growth mixture models were estimated and compared using Bayesian Information Criterion (BIC) values, which should be minimized in the most appropriate model (Muthen et al., 2002). These models were a two-class model, a three-class model, and a four-class

model. Table 2 presents the results of these analyses. The likelihood statistics and BIC values indicated that the three and four-class models fit better than the two-class model and that the four-class model fit slightly better than the three-class model. Inspection of the growth parameters, however, indicated the three-class model yielded the most interpretable trajectories with reasonable class sizes. In addition, the entropy value decreased slightly from the three to the four-class model suggesting little was gained from the addition of a fourth class.

Mplus provides estimates of the probability of class membership for each individual. Probability estimates range from 0 to 1.0 with a probability of 1.0 reflecting perfect classification. Individuals are assigned to the class for which they have the highest probability of membership. Ideally, the probability of membership in one class will approach 1.0 and other probabilities will be very small, indicating relatively unambiguous classification. This was true for the predicted probabilities for class membership for the three-class solution. Average predicted probabilities for class membership for the three classes ranged from .97 to 1.0.

Final Latent Growth Mixture Model

Table 3 presents the growth parameters and variances for each class in the final three-class growth mixture model. For ease of interpretation, descriptive labels have been added to the classes. Mothers with decreasing depression (25% of the total sample; 30% of depressed mothers) started out with a high frequency and severity of depression during their child's first year of life and decreased to a moderate level during their child's preschool years before beginning to increase again in their child's school years. Mothers with chronic depression (7% of the total sample; 8% of depressed mothers) had, on

average, high frequency and severity of depression throughout the study period. Finally, on average, mothers with stable low depression (52% of the total sample; 62% of depressed mothers) had infrequent and mild depression throughout the study period, averaging about one month of subthreshold depressive symptoms per year. Significant variance in the intercept parameters suggests that, within groups of depressed mothers, there is still significant variation in the average level of depression. Figure 1 presents the average predicted trajectories for each class of depressed mothers.

Validation of Maternal Depression Groups

In order to explore the validity of the empirically-identified maternal depression groups, groups were compared on other measures of depression (CES-D and SCID) used in the study. Valid trajectories should correspond to patterns of change in other variables. In general, this data provided preliminary support for the depression groups. As expected, mothers with chronic depression had average CES-D scores in the clinical range at all major time points while non-depressed mothers and mothers with stable low depression had average CES-D scores below the clinical range at all major time points. Mothers with decreasing depression reported a decrease in depressive symptom severity from a high to a moderate level over the course of the study period (see table 4). A repeated measures analysis of variance (ANOVA) with mother CES-D score as the dependent variable and maternal depression group as the independent variable revealed significant main effects of depression group (F (4, 91) = 22.88, $p < .001$, $\eta_p^2 = .43$) and time point (F (4, 364) = 4.25, $p = .002$, $\eta_p^2 = .05$) and a significant time point by maternal depression group interaction (F (12, 364) = 2.79, $p = .001$, $\eta_p^2 = .08$). This analysis indicated that patterns of CES-D scores over time differed by maternal depression group.

Figure 2 presents the average CES-D trajectories by maternal depression group. Follow-up one-way ANOVAs with post-hoc Tukey tests indicated that, when children were 14-months-old, the mothers with chronic and decreasing depression reported significantly higher levels of symptoms on the CES-D compared to mothers with stable low depression and non-depressed mothers, and mothers with stable low depression reported significantly higher levels of depressive symptoms than non-depressed mothers. Results were similar at the 24-month contact except that mothers with stable low depression and non-depressed mothers were not significantly different from one another. By the age 3 ½ years contact, the CES-D scores of mothers with decreasing depression had decreased and were no longer significantly different from the mothers with stable low depression and both these groups reported a level of symptoms significantly lower than the mothers with chronic depression. At the 4 ½ years and 6 ½ years contacts, mothers with decreasing depression had increased again and were not significantly different from the mothers with chronic depression.

SCID data also appeared to corroborate the findings from the growth mixture models. Non-depressed mothers had no SCID diagnosis while chronically depressed mothers consistently had the highest frequency of major depression diagnoses throughout the study period (see table 5). Mothers with decreasing depression had a high frequency of major and subthreshold depression diagnoses at study entry and their diagnoses decreased in frequency over time. Mothers with stable low depression had a consistently low level of depression diagnoses throughout the study.

Demographics of Maternal Depression Groups

At the age 6 ½ contact, one demographic variable, child's age, differed by maternal depression group (F (3, 134) = 4.36, p = .006). Children of mothers with decreasing depression were significantly younger than children of mothers with stable low depression (Mean age = 82.0 vs. 83.9 months respectively). Other demographic variables did not differ by maternal depression group, including mother's ethnicity, partner status, education or occupation level, or number of hours spent working outside the home. Children of depressed and non-depressed mothers did not differ in terms of sex, number of hours spent in daycare, or number of hours spent in the care of someone other than their mothers. Families did not differ significantly in terms of SES as measured by the Hollingshead (1975) or number of children in the family. Father's ethnicity, education and occupation level also did not differ significantly by maternal diagnostic group.

Phase 2: Child Behavior Constructs

In order to reduce the number of child behavior outcome variables analyzed, the next goal was to create theoretically-driven child behavior constructs using the mother, father, and teacher questionnaire data. Four constructs were created: child internalizing behavior problems, child externalizing-aggressive behavior problems, child externalizing-ADHD behavior problems, and child social competence.

In the first step, variables measuring each construct were identified from the questionnaires completed by mothers, fathers, and teachers, including the CABI, CBCL, ADHD Rating Scale, and the Child Behavior Scale. Only teachers completed the last two scales. CBCL T-scores are sex-normed; therefore, CBCL raw scale scores were used in

order to make the scales more comparable to scales from other measures. The internalizing construct included the CABI Internalizing-Anxious factor score, the CABI Internalizing-Isolated factor score, the CBCL Withdrawn scale score, and the CBCL Anxious / Depressed scale score. The externalizing-aggressive construct consisted of the CABI externalizing-aggressive factor score, the CBCL Aggressive Behavior scale score, and the CBCL Delinquent Behavior scale score. The externalizing-ADHD construct consisted of the CABI externalizing-hyperactive factor score, the CBCL Attention Problems scale score, and teacher report on the ADHD Rating Scale. The social competence construct included the CABI social competence factor score and teacher report on the Child Behavior Scale prosocial with peers subscale. If the distributions of variables were highly skewed, variables were log transformed to meet the assumptions of the statistical analyses.

In order to prevent loss of data for single-mother families, a mean of mother and father report was taken to create a parent scale. Items were standardized before taking their mean to form the parent scales. In families where the mother was the only available reporter, the parent scale included only her report. Mother and father report were significantly correlated for all scales used to create constructs (mean $\underline{r} = .39$, range .20 - .56).

In order to assess the feasibility of creating cross-informant constructs, confirmatory factor analysis (CFA) was conducted using Bentler's EQS Program. Creating constructs across reporters proved intractable; therefore, this approach was abandoned in favor of single reporter constructs. Separate constructs were created for

parent and teacher report. CFA based on the covariance matrix and using maximum likelihood estimation with robust standard errors was used to assess the factors.

Internalizing Behavior Problems

The CFA models for parent and teacher reports of child internalizing behavior problems demonstrated adequate fit. For parent report, the χ^2 (2, N = 131) = 15.86, $p <$ .001, the nonnormed fit index was .80, and the comparative fit index (CFI) was .93. For teacher report, the χ^2 (2, N = 120) = 19.33, $p < .001$, the nonnormed fit index was .79, and the comparative fit index (CFI) was .93. All factor loadings were significant (see Table 6). The four internalizing variables were converted to z-scores to give each equal weighting, and added together to form the internalizing behavior problems construct. These composite scores were created separately for parent and teacher report. Internal consistency (Cronbach's alpha) of the items within the parent and teacher internalizing behavior problems constructs was .84 and .87 respectively. There was a significant correlation between parent and teacher reports of child internalizing problems ($r = .39$, $p < .001$).

Externalizing Behavior Problems

The 2-factor CFA models for parent and teacher reports of child externalizing behavior problems demonstrated adequate fit and confirmed the two hypothesized externalizing factors: externalizing-aggressive and externalizing-ADHD. For parent report, the χ^2 (4, N = 132) = 34.29, $p < .001$, the nonnormed fit index was .83, and the comparative fit index (CFI) was .93. For teacher report, the χ^2 (8, N = 124) = 24.39, $p =$ .002, the nonnormed fit index was .94, and the comparative fit index (CFI) was .97. All factor loadings were significant (see Table 6). The externalizing variables were

converted to z-scores to give each equal weighting, and added together to form the externalizing behavior problems constructs. These composite scores were created separately for parent and teacher report. Teacher report included the ADHD Rating Scale in the externalizing-ADHD construct. Internal consistency (Cronbach's alpha) of the items within the externalizing-aggressive constructs was .88 for parent report and .83 for teacher report. Internal consistency of the items within the externalizing-ADHD constructs was .86 for parent report and .92 for teacher report. There were significant correlations between parent and teacher reports of child externalizing-aggressive ($r = .34$, $p < .001$) and externalizing-ADHD ($r = .38$, $p < .001$) behaviors problems.

Social Competence

CFA was not used to assess the fit of the social competence construct because there was only one indicator, the CABI social competence factor score, for the parent construct, and there were only two indicators, the CABI social competence factor score and the CBS prosocial with peers scale score, for the teacher construct. Internal consistency (Cronbach's alpha) of the items within the teacher-reported social competence construct was .87. There was a significant correlation between parent and teacher reports of child social competence ($r = .38$, $p < .001$).

Phase 3: Analysis of Age 6 ½ Outcomes

The next phase of the study involved analysis of child outcomes at age 6 ½ years and addressed the question: Do children of mothers with different courses of depression differ in their psychophysiological and/or behavioral outcomes at age 6 ½ years-old?

Child Behavior Outcomes

Child behavior constructs. A two-way multivariate analysis of variance (MANOVA) was conducted to determine the effect of maternal depression group and child sex on child behavior constructs. Separate MANOVAs were conducted for parent and teacher reports. Results revealed significant differences among maternal depression groups on the dependent variables for both parent report (Wilks' lambda = .84, F (12, 315) = 1.76, p = .05, η_p^2 = .06) and teacher report (Wilks' lambda = .79, F (12, 260) = 2.00, p = .02, η_p^2 = .08) and significant differences between boys and girls on the dependent variables for both parent report (Wilks' lambda = .89, F (4, 119) = 3.70, p = .007, η_p^2 = .11) and teacher report (Wilks' lambda = .82, F (4, 98) = 5.54, p < .001, η_p^2 = .18).

Follow-up univariate ANOVAs on each of the child behavior constructs revealed significant differences among maternal depression groups on parent report of externalizing-aggressive behaviors (F (3, 124) = 4.41, p = .006, η_p^2 = .10), parent report of externalizing-ADHD behaviors (F (3, 125) = 5.63, p = .001, η_p^2 = .12), parent report of social competence (F (3, 125) = 4.04, p = .009, η_p^2 = .09), teacher report of externalizing-aggressive behaviors (F (3, 116) = 3.29, p = .02, η_p^2 = .08), and teacher report of externalizing-ADHD behaviors (F (3, 118) = 3.47, p = .02, η_p^2 = .08). Significant differences between boys and girls were found for parent report of externalizing-aggressive behaviors (F (1, 124) = 6.12, p = .01, η_p^2 = .05), teacher report of internalizing behaviors (F (1, 112) = 4.51, p = .04, η_p^2 = .04), and teacher report of externalizing-aggressive behaviors (F (1, 116) = 7.12, p = .008, η_p^2 = .06). Boys had higher parent and teacher-reported externalizing-aggressive behaviors and girls had higher teacher-reported internalizing behavior problems.

Post-hoc Tukey tests revealed that children of chronically depressed mothers had significantly higher levels of parent-reported externalizing-aggressive and externalizing-ADHD behavior problems compared to children of non-depressed mothers and mothers with stable low depression. Children of mothers with decreasing and stable low depression also had significantly higher levels of parent-reported externalizing-ADHD behavior problems compared to children of non-depressed mothers. Children of non-depressed mothers had significantly higher parent-reported social competence than children of mothers with decreasing and chronic depression. Compared to children of non-depressed mothers, children of chronically depressed mothers also had significantly higher levels of teacher-reported externalizing-aggressive behavior problems. Figures 3 and 4 display parent and teacher-reported child behavior construct composite scores by maternal depression group.

Diagnostic Interview Schedule for Children (DISC). Of the 138 children who participated at the age 6 ½ year contact, 137 had completed DISC data. In all, 57 children (41.6% of sample) met criteria for one or more psychiatric diagnoses as measured by mother report on the DISC. Thirty-seven children (27.0%) met criteria for one DISC diagnosis. Fourteen children (10.2%) met criteria for two DISC diagnoses. Five children (3.6%) met criteria for three DISC diagnoses, and one child (0.7%) met criteria for four DISC diagnoses. Thirty-six children (26.3% of sample) met criteria for at least one anxiety disorder (separation anxiety disorder, social phobia, specific phobia, agoraphobia, posttraumatic stress disorder) and 36 children (26.3% of sample) met criteria for one or more behavior disorders (oppositional defiant disorder, attention deficit disorder, conduct disorder). No children met criteria for any of the following diagnoses

which were also assessed: generalized anxiety disorder, obsessive compulsive disorder, panic disorder, selective mutism, major depression, mania / hypomania, or dysthymia. Table 7 presents the frequency of diagnosis by maternal depression group. There was no relation between maternal depression group and frequency of diagnosis with an anxiety disorder. There was a significant association between maternal depression group and diagnosis of a behavior disorder (χ^2 (3, N = 136) = 10.98, p = .01). Children of mothers with chronic depression were most likely to be diagnosed with a behavior disorder followed by children of mothers with decreasing depression, children of mothers with stable low depression, and children of non-depressed mothers. Follow-up 2 x 2 chi-square tests indicated that children of chronically depressed mothers were significantly more likely to be diagnosed with a behavior disorder compared to children of non-depressed mothers (χ^2 (1, N = 33) = 7.75, p = .005) and mothers with stable low depression (χ^2 (1, N = 83) = 7.24, p = .007).

Child self-report. A two-way multivariate analysis of variance (MANOVA) was conducted to determine the effect of maternal depression group and child sex on child self-reported behavior problems as measured by the Dominic, Children's Depression Inventory, the Revised Children's Manifest Anxiety Scale, and the Loneliness and Social Dissatisfaction Questionnaire. Results revealed a significant main effect of child sex (Wilks' lambda = .83, F (10, 104) = 2.20, p = .02, η_p^2 = .18). Follow-up univariate ANOVAs revealed significant differences between boys and girls on the Dominic Simple Phobia scale (F (1, 113) = 8.41, p = .004, η_p^2 = .07) and the Dominic Conduct Disorder scale (F (1, 113) = 5.89, p = .02, η_p^2 = .05). Girls endorsed significantly more symptoms of simple phobia and boys endorsed significantly more symptoms of conduct disorder.

Child self-report data was further analyzed by creating groups of children with elevated and low rates of self-reported emotional and behavior problems using a cutoff score of .5 standard deviation above the mean. Low problems were defined as scores at or below the mean. This cut-off was chosen in order to create distinct groups of children with elevated and low behavior composite scores and reasonable sample sizes. There was a significant association between maternal depression group and child report of loneliness and social dissatisfaction (χ^2 (3, N = 107) = 10.46, p = .02). Follow-up 2 x 2 chi-square tests indicated that children of chronically depressed mothers were significantly more likely to report elevated symptoms of loneliness and social dissatisfaction compared to children of non-depressed mothers (χ^2 (1, N = 25) = 5.00, p = .04), children of mothers with stable low depression (χ^2 (1, N = 65) = 9.00, p = .005), and children of mothers with decreasing depression (χ^2 (1, N = 35) = 8.57, p = .006).

Child Psychophysiology

Brain activity. EEG data from the five main conditions (baseline, neutral film, happy film, sad film, and fear film) were analyzed. Of the children who completed the psychophysiology visit, 97 had complete EEG data in all five conditions. The remaining children were missing data from one or more leads in one or more conditions because of motor artifact or technical problems. Loss of EEG data due to motor artifact is very common in this difficult to test age-range. Children from different maternal depression groups did not differ in terms of their familiarity with the various film clips.

A 5 (condition) x 2 (region) x 2 (hemisphere) x 4 (maternal depression group) x 2 (child sex) repeated measures analysis of variance (ANOVA) with EEG alpha power as the dependent variable was conducted. Mauchly's Test of Sphericity was significant

indicating heterogeneity of covariance; therefore, the Greenhouse-Geisser correction was used to correct for potentially inflated p-values. Degrees of freedom and p-values reported are Greenhouse-Geisser corrected where appropriate. The analysis revealed significant main effects of condition (F (3, 268) = 16.79, $p < .001$, $\eta_p^2 = .16$), hemisphere (F (1, 89) = 11.87, $p = .001$, $\eta_p^2 = .12$), and maternal depression group (F (3, 89) = 3.11, $p = .03$, $\eta_p^2 = .09$), a significant condition by maternal depression group interaction (F (9, 268) = 1.96, $p = .04$, $\eta_p^2 = .06$), a significant condition by region interaction (F (2, 207) = 21.42, $p < .001$, $\eta_p^2 = .19$), a significant condition by hemisphere interaction (F (3, 308) = 9.75, $p < .001$, $\eta_p^2 = .10$), and a significant region by hemisphere interaction (F (1, 89) = 6.46, $p = .01$, $\eta_p^2 = .07$). Post-hoc Tukey tests revealed that children of mothers with chronic depression, on average, had lower brain activation (greater EEG alpha power) than children of mothers in the non-depressed group, across all brain regions assessed. Figures 5 and 6 display frontal and parietal EEG alpha power scores by maternal depression group.

In order to understand the significant condition by maternal group interaction effect, individual 2 (region) x 2 (hemisphere) x 4 (maternal depression group) repeated measures ANOVAs were conducted for each condition separately. Results revealed significant main effects of maternal depression group for the baseline (F (3, 102) = 4.87, $p = .003$, $\eta_p^2 = .13$), happy film (F (3, 100) = 2.71, $p = .049$, $\eta_p^2 = .08$), sad film (F (3, 101) = 2.70, $p = .05$, $\eta_p^2 = .07$), and fear film (F (3, 96) = 2.87, $p = .04$, $\eta_p^2 = .08$) conditions. The effect of maternal group was not significant for the neutral film condition (F (3, 102) = 1.87, p = ns, $\eta_p^2 = .05$).

Because previous research has found the association between maternal depression and child brain activity to be specific to the frontal region, separate analyses were conducted by region. Results revealed that, when frontal and parietal regions were analyzed separately, there was a main effect of maternal depression group for the frontal region ($\underline{F}$ (3, 89) = 4.57, $\underline{p}$ = .005, η_p^2 = .13), but not the parietal region ($\underline{F}$ (3, 89) = 1.74, $\underline{p}$ = ns, η_p^2 = .06). Post-hoc Tukey tests revealed that children of mothers with chronic depression, on average, had lower frontal brain activation (greater frontal EEG alpha power) than children of non-depressed mothers, across all conditions.

Because previous research has found EEG laterality differences in infants and young children of depressed versus non-depressed mothers, EEG data were further analyzed using asymmetry scores as the dependent variable. A 5 (condition) x 2 (region) x 4 (maternal depression group) x 2 (child sex) repeated measures ANOVA with EEG asymmetry scores as the dependent variable revealed significant main effects of condition ($\underline{F}$ (3, 308) = 9.65, $\underline{p}$ < .001, η_p^2 = .10) and region ($\underline{F}$ (1, 89) = 6.34, $\underline{p}$ = .01, η_p^2 = .07). Degrees of freedom and p-values reported are Greenhouse-Geisser corrected where appropriate.

Heart rate. A 5 (condition) x 4 (maternal depression group) x 2 (child sex) repeated measures ANOVA with child heart rate (beats / minutes) as the dependent variable was conducted. Mauchly's Test of Sphericity was significant indicating heterogeneity of covariance; therefore, the Greenhouse-Geisser correction was used to correct for potentially inflated p-values. Degrees of freedom and p-values reported are Greenhouse-Geisser corrected where appropriate. This analysis revealed significant main effects of condition ($\underline{F}$ (3, 268) = 12.67, $\underline{p}$ < .001, η_p^2 = .13) and child sex ($\underline{F}$ (1, 87) =

5.15, $p = .03$, $\eta_p^2 = .06$). Within-subjects contrasts with baseline heart rate as the reference category revealed significant increases from baseline heart rate in the neutral film (F (1, 87) = 37.05, $p < .001$, $\eta_p^2 = .30$), sad film (F (1, 87) = 7.89, $p = .006$, $\eta_p^2 = .08$), and fear film (F (1, 87) = 14.97, $p < .001$, $\eta_p^2 = .15$) conditions. There was no significant difference between baseline heart rate and heart rate during the happy film (F (1, 87) = .003, p = ns). On average, girls had high heart rates than boys.

Although children of depressed and non-depressed mothers did not differ in terms of average heart rate, there was a possibility they might differ in terms of heart rate reactivity, change in heart rate from baseline. Heart rate reactivity scores were calculated by subtracting heart rate during each of the emotion-eliciting film conditions from baseline heart rate, yielding scores that indicated the extent of increase or decrease in heart rate from baseline. A 4 (condition) x 4 (maternal depression group) x 2 (child sex) repeated measures ANOVA with child heart rate reactivity scores as the dependent variable revealed a significant main effect of condition (F (2, 201) = 11.95, $p < .001$, $\eta_p^2 = .12$). The Greenhouse-Geisser correction was used for this analysis.

Respiratory Sinus Arrhythmia (RSA). A 5 (condition) x 4 (maternal depression group) x 2 (child sex) repeated measures ANOVA with child RSA as the dependent variable was conducted. Mauchly's Test of Sphericity was significant indicating heterogeneity of covariance; therefore, the Greenhouse-Geisser correction was used to correct for potentially inflated p-values. Degrees of freedom and p-values reported are Greenhouse-Geisser corrected where appropriate. This analysis revealed significant main effects of condition (F (3, 269) = 9.17, $p < .001$, $\eta_p^2 = .10$) and child sex (F (1, 87) = 4.60, $p = .04$, $\eta_p^2 = .05$). On average, RSA was lowest in the neutral and sad film conditions

and highest in the baseline and happy film conditions. Within-subjects contrasts with baseline RSA as the reference category revealed significant decreases from baseline RSA in the neutral film (F (1, 87) = 17.72, $p < .001$, $\eta_p^2 = .17$), sad film (F (1, 87) = 17.91, $p < .001$, $\eta_p^2 = .17$), and fear film (F (1, 87) = 7.37, $p = .008$, $\eta_p^2 = .08$) conditions. There was no significant difference between baseline RSA and RSA during the happy film (F (1, 87) = .421, p = ns). These findings corroborate other research studies that have found decreases in RSA when emotion is manipulated toward the negative (see Beauchaine, 2001 for a review). On average, girls had significantly lower RSA than boys.

Although children of depressed and non-depressed mothers did not differ in terms of average RSA, there was a possibility they might differ in terms of RSA reactivity, change in RSA from baseline. RSA reactivity scores were calculated by subtracting RSA during each of the emotion-eliciting film conditions from baseline RSA, yielding scores that indicated the extent of increase or decrease in RSA from baseline. Higher RSA reactivity scores indicated increased parasympathetic withdrawal during the film conditions. A 4 (condition) x 4 (maternal depression group) x 2 (child sex) repeated measures ANOVA with child RSA reactivity as the dependent variable revealed significant main effects of condition (F (2, 221) = 7.82, $p < .001$, $\eta_p^2 = .08$) and maternal depression group (F (3, 87) = 3.04, $p = .03$, $\eta_p^2 = .09$). The Greenhouse-Geisser correction was used for this analysis. Post-hoc Tukey tests revealed that children of chronically depressed mothers had higher RSA reactivity than children of mothers with stable low depression. Across conditions, children of chronically depressed mothers demonstrated more parasympathetic withdrawal in reaction to the emotion-eliciting film conditions than children of mothers with stable low depression (see Figure 7).

Cortisol. Raw cortisol data were first examined for the presence of potentially contaminated samples. One extreme home bedtime cortisol value (1.77 *u*g/dL) was identified and eliminated from the analysis because of potential contamination. Ten zero values were also changed to .01 *u*g/dL because the minimum sensitivity of the assay was .02 *u*g/dL. This value was chosen because it is halfway between zero and .02. Further examination of the cortisol data revealed the raw values were highly skewed; therefore, variables were natural log transformed to meet the assumptions of the statistical analyses. Two variables, the second laboratory cortisol sample and the bedtime cortisol sample, were still skewed after this transformation. For these variables, outliers greater than 2.5 standard deviations above the mean were pulled in to the highest value. Three outliers were identified for each variable.

Before examining the relation between maternal depression and child salivary cortisol levels, the potential confounding influences of child health, child sleep quality, and child stress levels were examined. Children from different maternal depression groups did not differ significantly in terms of health or quality of sleep prior to home saliva sample collection, stress level on the day of laboratory saliva sample collection, medication usage, or caffeine consumption. A 4 (time) x 4 (maternal depression group) x 2 (child sex) repeated-measures ANOVA with natural log-transformed child salivary cortisol as the dependent variable revealed a significant main effect of time (F (3, 252) = 33.03, $p < .001$, $\eta_p^2 = .26$), reflecting the well-known circadian rhythm of cortisol. The Greenhouse-Geisser correction was used for this analysis.

Relations Between Child Psychophysiology and Behavior

Pearson's correlations were used to examine the relations between child psychophysiology and behavior at age 6 ½ years. In order to reduce the number of variables analyzed, psychophysiological measures were averaged across conditions (with the exception of child salivary cortisol which has a known circadian rhythm) before testing correlations with parent and teacher-reported child behavior constructs. Table 8 presents the results of these analyses. Higher parent-reported internalizing symptoms were associated with reduced right frontal brain activation (higher EEG alpha power). Higher parent-reported externalizing-aggressive behaviors were associated with reduced right frontal brain activation, lower average heart rate, higher average vagal tone, and higher evening salivary cortisol levels. Higher parent-reported externalizing-ADHD behaviors were associated with lower average heart rate and higher average vagal tone. Higher parent-reported social competence was associated with lower laboratory post-stressor cortisol levels. Higher teacher-reported social competence was associated with higher left parietal EEG power (lower activation). There were no relations between teacher-reported internalizing or externalizing problems and child psychophysiology.

Phase 4: Child Frontal Brain Activity and Contextual Risk Factors as Mediators of the Relation between Maternal Depression and Child Behavior

The final goal of the study was to examine the combined influence of maternal depression, child psychophysiology, and contextual risk factors on child behavior at age 6 ½ years. Of the psychophysiological measures used in this study, only child frontal brain activity was selected as a potential mediator of the relation between maternal depression and child behavior for several reasons. The rationale was threefold. First, of the

psychophysiological measures assessed in this study, only child frontal brain activity and RSA reactivity demonstrated a significant association with maternal depression thereby meeting a critical condition for testing mediation (Baron & Kenny, 1986). Second, child frontal brain activity was found to partially mediate the relation between maternal depression and child behavior problems at an earlier time point in this study (Dawson et al., in press). Third, previous research suggests patterns of frontal brain activity may index individual differences in emotion and behavior regulation (Dawson, Panagiotides et al., 1992).

Zero-order correlations among the independent and dependent variables were examined. An aggregate measure of child frontal brain activity was created by averaging left and right frontal brain activity across conditions. Maternal depression was moderately correlated with both child frontal brain activity ($r = .30$, $p = .003$) and contextual risk factors ($r = .46$, $p < .001$). Frontal brain activity and contextual risk were also moderately correlated ($r = .29$, $p = .004$). Zero-order correlations between independent variables and child behavior outcomes are presented in Table 9. Only parent-reported child behavior outcomes met preliminary conditions for mediation, including correlations with both maternal depression and at least one potential mediator.

Path analysis, conducted using Bentler's EQS program, was used to investigate whether contextual risk factors and/or child frontal brain activity mediated the relation between maternal depression and parent-reported child behavior outcomes. Contextual risk factors and child frontal brain activity were hypothesized to partially mediate the relation between maternal depression and parent-reported child behavior outcomes. It was expected that there would be a significant reduction in the direct relation between

maternal depression and child outcome when the mediators were included in the model. A separate path analysis was tested for each child outcome. Analyses were conducted using the covariance matrix and maximum likelihood estimation with robust standard errors.

Figures 8a – 8d present the standardized path estimates and R^2s for the models predicting parent-reported internalizing, externalizing-aggressive, externalizing-ADHD, and social competence respectively. As shown in Figure 8, for all four child behavior outcome variables, the direct relation between maternal depression and child behavior outcome was reduced to non-significant when the mediating paths were added. As expected, contextual risk factors were found to mediate the relation between maternal depression and child behavior outcomes. Maternal depression was associated with higher levels of contextual risk which was in turn associated with increased child behavior problems and decreased social competence. Child frontal brain activity was not found to be a significant mediator; the paths between child frontal brain activity and child behavior were not significant.

Based on a formula derived by Sobel (1982), tests for mediation were computed to determine whether contextual risk factors significantly mediated the relation between maternal depression and child outcomes. The mediated effect was calculated by multiplying the unstandardized path coefficients for the paths from the independent variable (maternal depression) to the mediator (contextual risk) and from the mediator to the dependent variable (child outcome). The standard error of the mediated effects were computed using Sobel's formula (Sobel, 1982). A significance test was calculated by dividing the mediated effect by the associated standard error and comparing the value to

values of normal distributions. Results indicated that contextual risk factors significantly mediated the relations between maternal depression and child internalizing behaviors (z = 2.53, $p < .05$), externalizing-aggressive behaviors ($z = 2.14$, $p < .05$), externalizing-ADHD behaviors ($z = 2.92$, $p < .05$), and social competence ($z = -2.83$, $p < .05$).

Table 1

Fit statistics and mean parameter estimates (standard errors in parentheses) for linear and quadratic growth curve models

Parameter	Linear Model	Quadratic Model
Mean intercept	4.117 (0.457)***	5.491 (0.616)***
Mean slope	-0.370 (0.096)***	-1.466 (0.332)***
Mean quadratic	--	0.160 (0.044)***
Intercept variance	19.263 (3.447)***	35.290 (6.167)***
Slope variance	0.600 (0.153)***	7.107 (1.825)***
Quadratic variance	--	0.095 (0.034)***
Log likelihood	-2451.447	-2432.502
BIC	4961.579	4943.249

*$p < .05$ **$p < .01$ ***$p < .001$

Table 2

Fit statistics for two-, three-, and four-class growth mixture models

Model	Log Likelihood	df	BIC	Entropy
One-class	-2432.502	16	4943.249	--
Two-class	-2406.207	20	4910.220	.945
Three-class	-2377.211	24	4871.791	.955
Four-class	-2360.068	28	4857.066	.939

BIC = Bayesian Information Criteria

Table 3

Mean parameter estimates (standard errors in parentheses) for final three-class growth mixture model

Parameter	Decreasing N = 40	Chronic N = 11	Stable Low N = 82
Mean intercept	14.926 (0.657) ***	12.252 (1.174) ***	1.008 (0.373) **
Mean slope	-6.874 (0.402) ***	-0.079 (0.644)	0.313 (0.237)
Mean quadratic	0.800 (0.062) ***	-0.072 (0.102)	-0.039 (0.038)
Intercept variance	4.363 (1.982) *	4.363 (1.982) *	4.363 (1.982) *
Slope variance	0.730 (0.830)	0.730 (0.830)	0.730 (0.830)
Quadratic variance	0.014 (0.019)	0.014 (0.019)	0.014 (0.019)

* $p < .05$ ** $p < .01$ *** $p < .001$

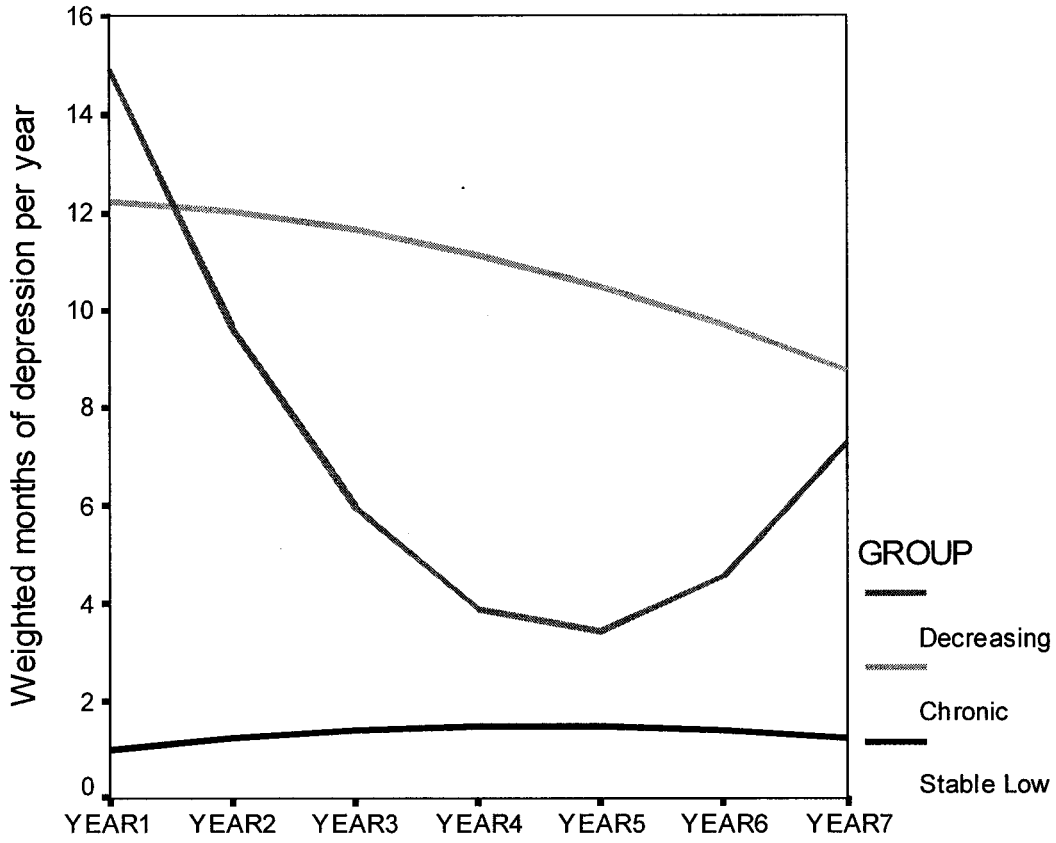


Figure 1. Average predicted trajectories for latent classes of depressed mothers

Table 4

Mean (and standard deviation) of CES-D scores by maternal depression group

Measure	Chronic	Decreasing	Stable Low	Non-depressed
Child age 12 months***	24 (12.3)	24 (7.2)	12 (10.8)	3 (1.9)
Child age 24 months***	20 (11.8)	16 (11.1)	7 (6.2)	5 (4.2)
Child age 3 ½ years***	21 (11.4)	14 (9.8)	10 (8.8)	4 (3.0)
Child age 4 ½ years***	18 (14.8)	16 (9.2)	9 (6.9)	3 (3.3)
Child age 6 ½ years***	16 (8.4)	13 (8.6)	8 (6.6)	3 (2.7)

*$p < .05$ **$p < .01$ ***$p < .001$

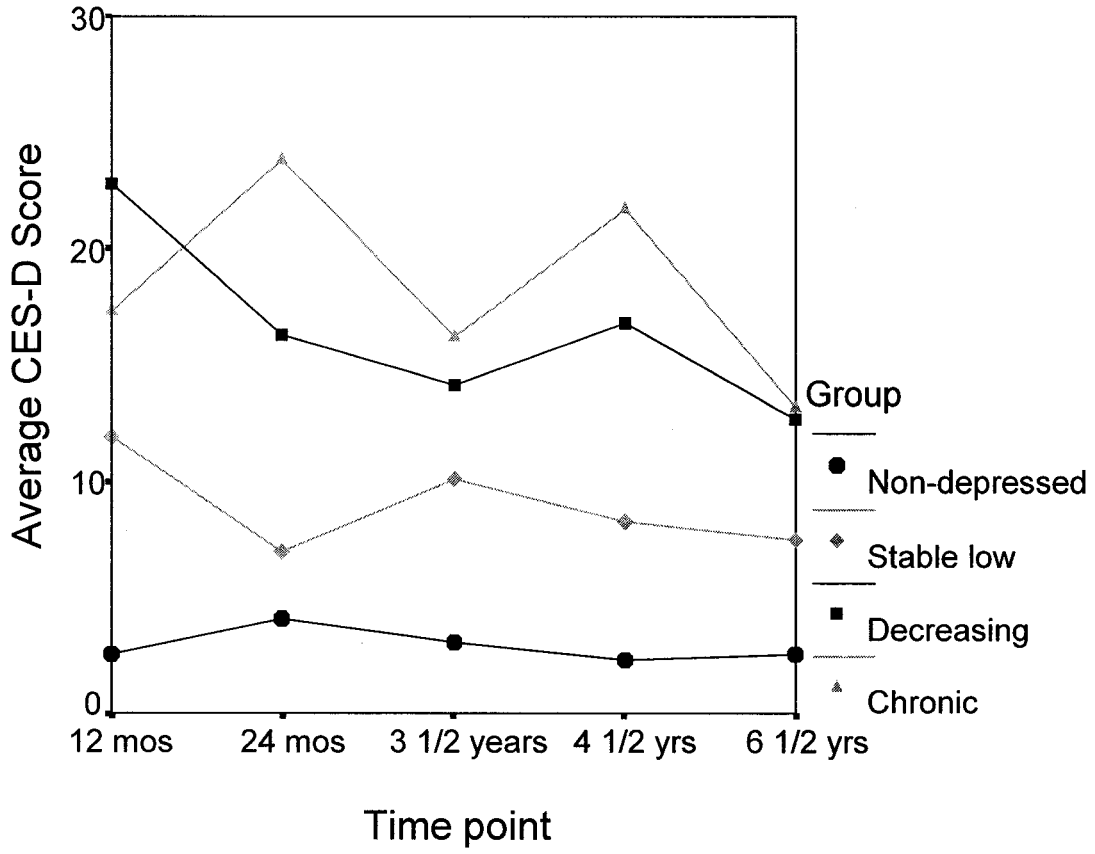


Figure 2. Average CES-D scores by maternal depression group

Table 5

Current SCID diagnosis counts (and percentages) by maternal depression group

Measure	Chronic	Decreasing	Stable Low	Non-depressed
Child age 12 months***				
No diagnosis	2 (18.2%)	3 (7.5%)	65 (79.3%)	26 (100%)
Subthreshold	4 (36.4%)	25 (62.5%)	7 (8.5%)	0
Major Depression	5 (45.5%)	12 (30.0%)	10 (12.2%)	0
Child age 24 months***				
No diagnosis	2 (20.0%)	23 (67.6%)	61 (92.4%)	23 (100%)
Subthreshold	1 (10.0%)	5 (14.7%)	5 (7.6%)	0
Major Depression	7 (70.0%)	6 (17.6%)	0	0
Child age 3 ½ years***				
No diagnosis	2 (25.0%)	23 (76.7%)	61 (91.0%)	20 (100%)
Subthreshold	1 (12.5%)	6 (20.0%)	5 (7.5%)	0
Major Depression	5 (62.5%)	1 (3.3%)	1 (1.5%)	0
Child age 4 ½ years***				
No diagnosis	4 (36.4%)	23 (67.6%)	68 (93.2%)	23 (100%)
Subthreshold	5 (45.5%)	9 (26.5%)	0	0
Major Depression	2 (18.2%)	2 (5.9%)	5 (6.8%)	0
Child age 6 ½ years*				
No diagnosis	6 (60.0%)	27 (87.1%)	67 (91.8%)	23 (100%)
Subthreshold	0	0	1 (1.4%)	0
Major Depression	4 (40.0%)	4 (12.9%)	5 (6.8%)	0

*$p < .05$ **$p < .01$ ***$p < .001$

Table 6

Internal consistency and factor loadings for child behavior constructs

	Cronbach's alpha		Item Loading	
Construct	Parent	Teacher	Parent	Teacher
Internalizing Behavior	.84	.87		
CABI Internalizing-Isolated			.67	.73
CABI Internalizing-Anxious			.79	.77
CBCL Withdrawn Scale			.78	.87
CBCL Anxious / Depressed Scale			.76	.80
Externalizing-Aggressive Behavior	.88	.83		
CABI Externalizing-Aggressive			.90	.85
CBCL Delinquent Behavior Scale			.74	.60
CBCL Aggressive Behavior Scale			.86	.92
Externalizing-ADHD Behavior	.86	.92		
CABI Externalizing-Hyperactive			.94	.91
CBCL Attention Problems Scale			.81	.83
ADHD Rating Scale, Total Score			--	.91
Social Competence	N/A	.87		
CABI Social Competence—Parent report			--	--
CABI Social Competence—Teacher report			--	--
CBS, Prosocial with Peers—Teacher report			--	--

Note: Standardized coefficients are presented.

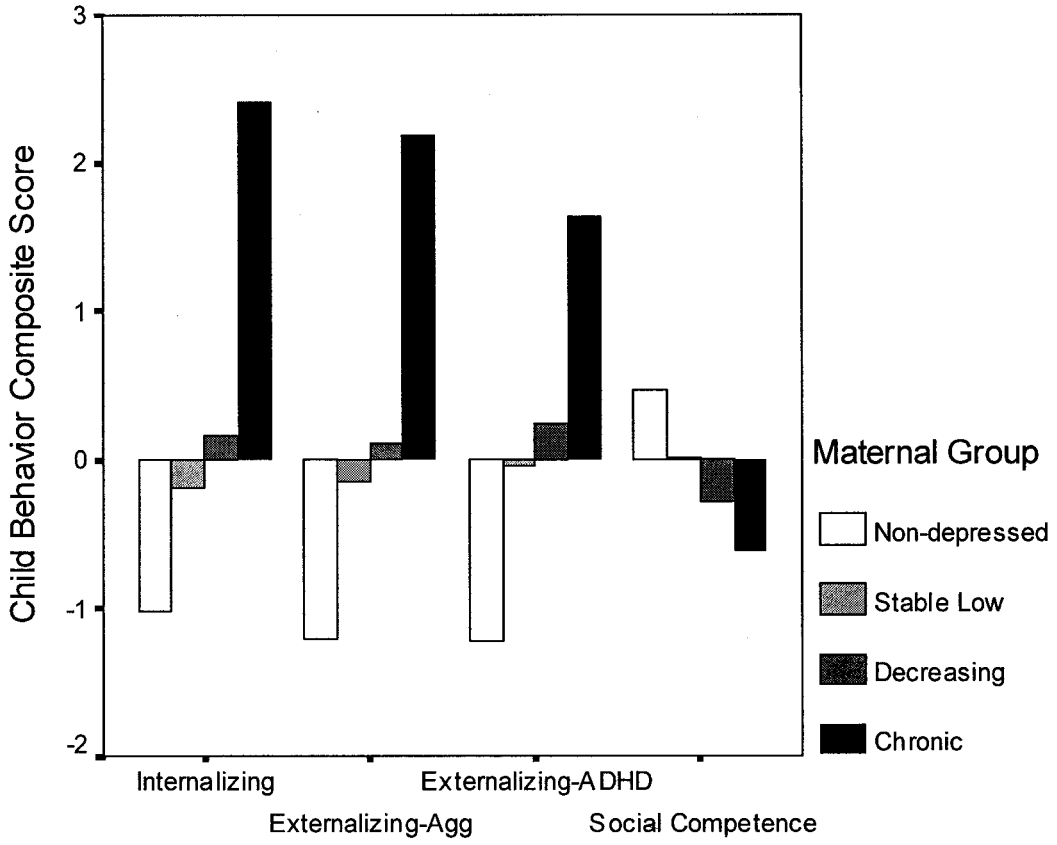


Figure 3. Parent-reported child behavior composite scores by maternal depression group

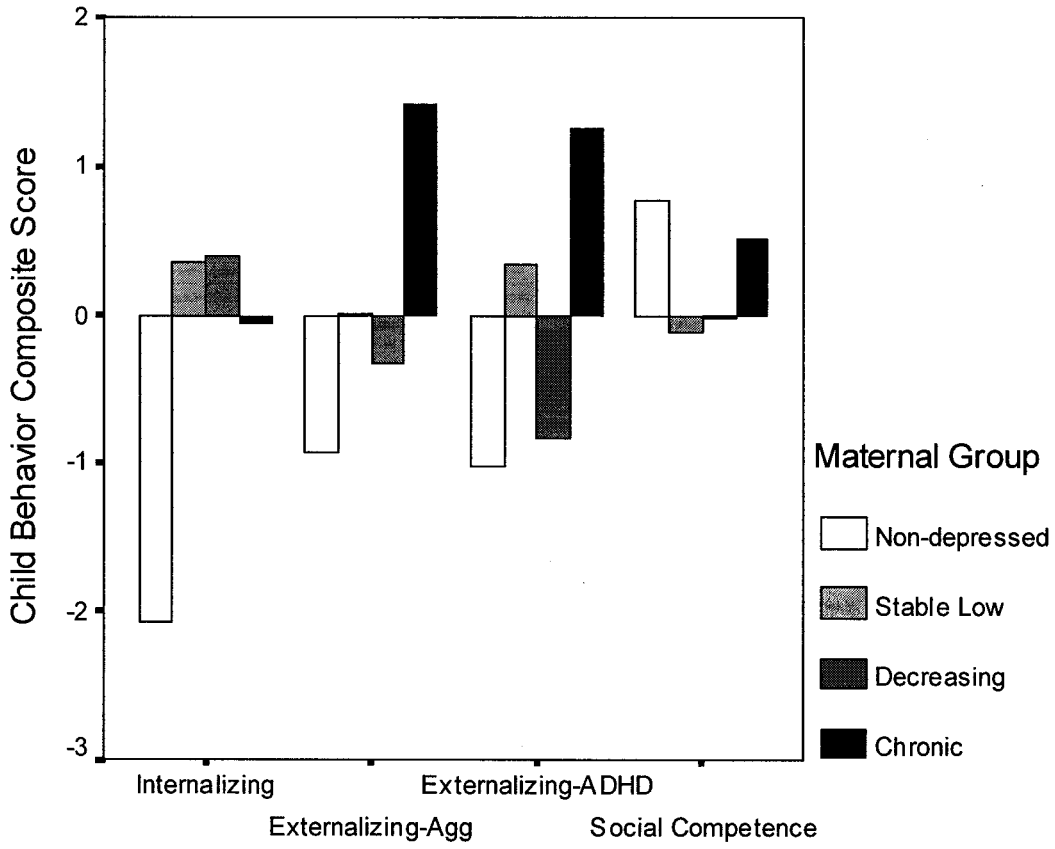


Figure 4. Teacher-reported child behavior composite scores by maternal depression group

Table 7

Frequency of DISC diagnosis by maternal depression group

Diagnosis	ND	SL	D	C	Total
Anxiety Disorders					
Specific Phobia	1 (4.3%)	13 (17.8%)	9 (30%)	2 (20%)	26 (19.0%)
Separation Anxiety	0	4 (5.6%)	1 (3.3%)	2 (20%)	7 (5.1%)
Social Phobia	0	1 (1.4%)	1 (3.3%)	1 (10%)	3 (2.2%)
Agoraphobia	2 (8.7%)	0	0	0	2 (1.5%)
Posttraumatic Stress	0	2 (2.7%)	0	0	2 (1.5%)
Behavioral Disorders					
Oppositional Defiant	3 (13%)	13 (17.8%)	8 (26.7%)	4 (40%)	29 (21.2%)
Attention Deficit	0	6 (8.3%)	5 (16.7%)	2 (20%)	13 (9.6%)
Conduct Disorder	0	0	1 (3.3%)	1 (10%)	2 (1.5%)

Note: ND = non-depressed, SL = stable low, D = decreasing, and C = chronic

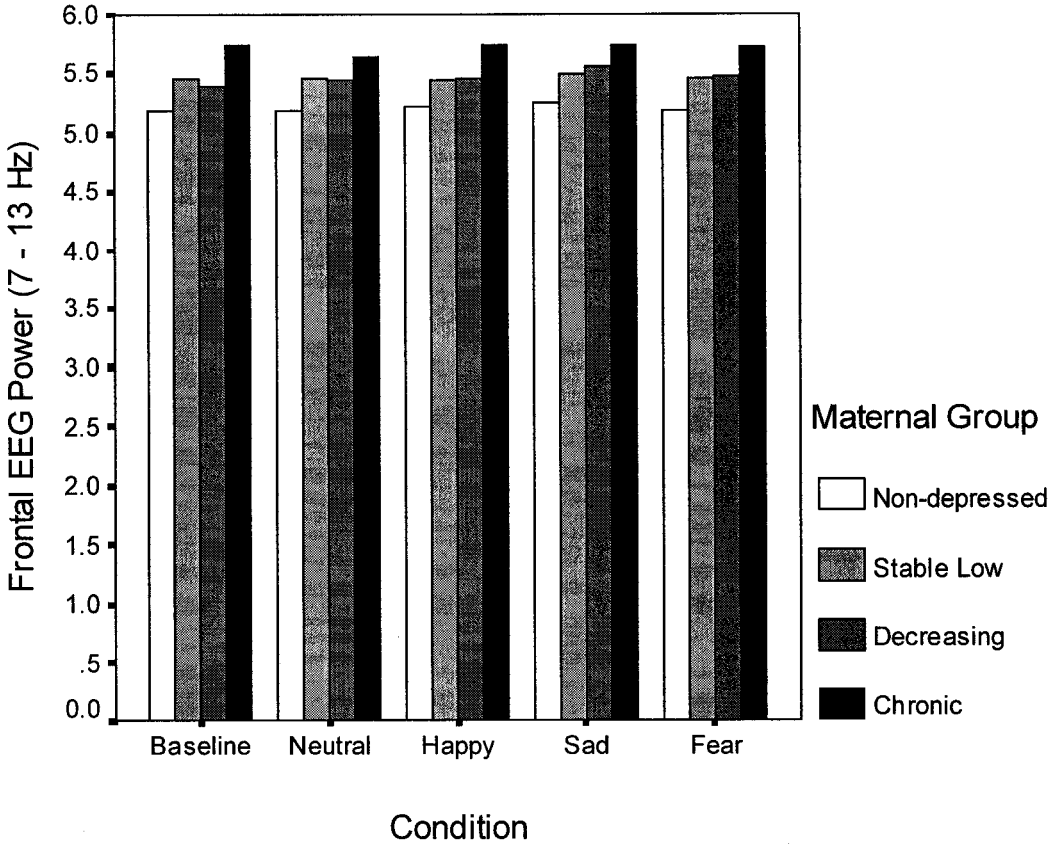


Figure 5. Frontal EEG power by maternal depression group

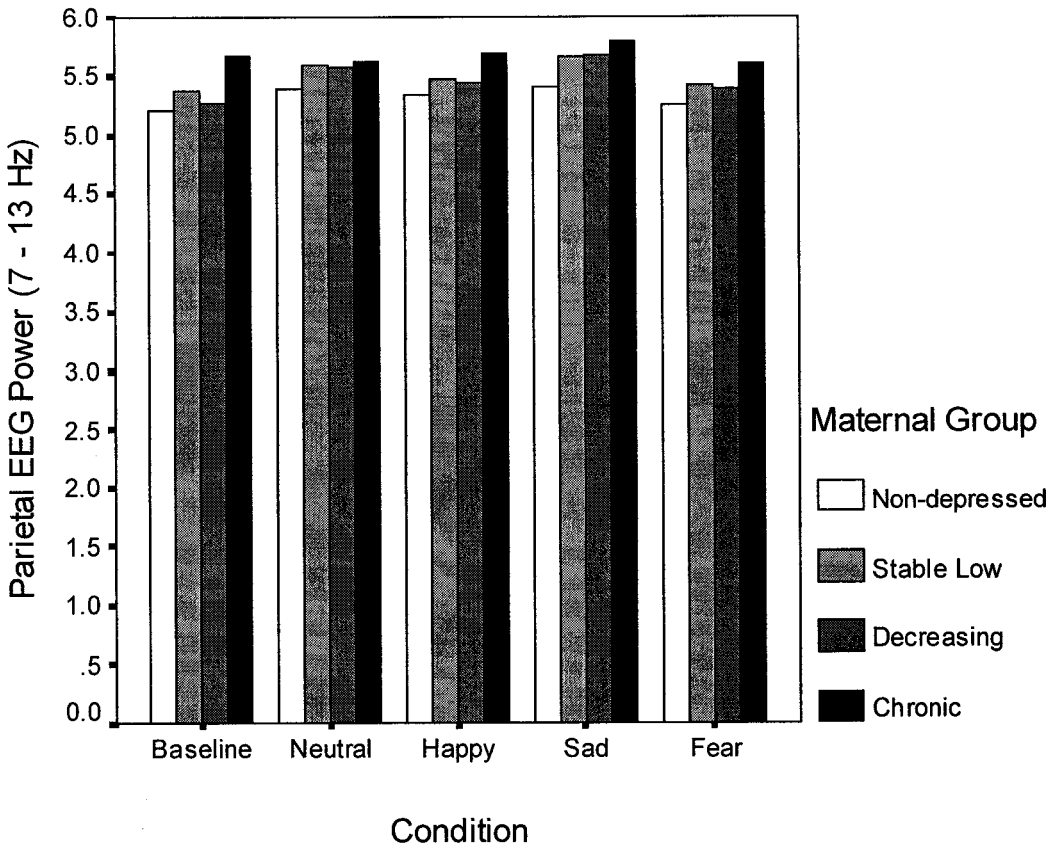


Figure 6. Parietal EEG power by maternal depression group

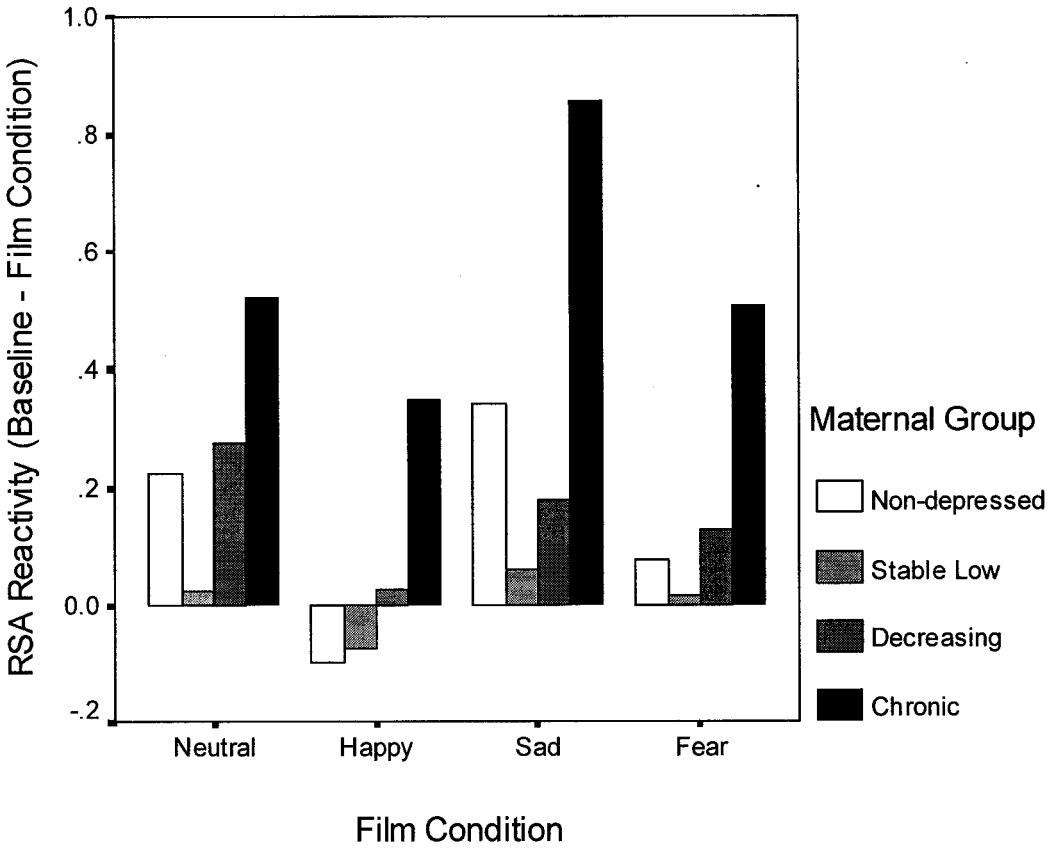


Figure 7. RSA reactivity by maternal depression group

Table 8

Pearson's correlations between child psychophysiology and child behavior constructs ($^{*}p < .05$, $^{**}p < .01$)

	Parent Report				Teacher Report			
	Internalizing	Aggressive	ADHD	Social	Internalizing	Aggressive	ADHD	Social
Left Frontal EEG	.17	.14	.18	-.04	.03	.01	-.00	.18
Right Frontal EEG	.21*	.20*	.18	-.07	.10	.06	.02	.13
Left Parietal EEG	-.03	-.01	-.01	.02	-.07	-.06	-.07	.21*
Right Parietal EEG	-.02	-.05	-.05	.00	-.01	-.03	-.05	.20
Mean Heart Rate	-.03	-.25*	-.27**	.19	.03	-.14	-.13	.11
Mean Vagal Tone	.15	.30**	.31**	-.18	-.04	.16	.10	-.03
Baseline Cortisol	.02	.10	.04	-.11	.05	.12	.18	-.19
Post-stressor Cortisol	.02	.11	.10	-.21*	-.03	.02	-.06	-.17
Home1 Cortisol	.07	.06	.09	-.09	-.01	.08	-.10	-.06
Bedtime Cortisol	.08	.25**	.11	-.12	.10	.17	.14	-.20

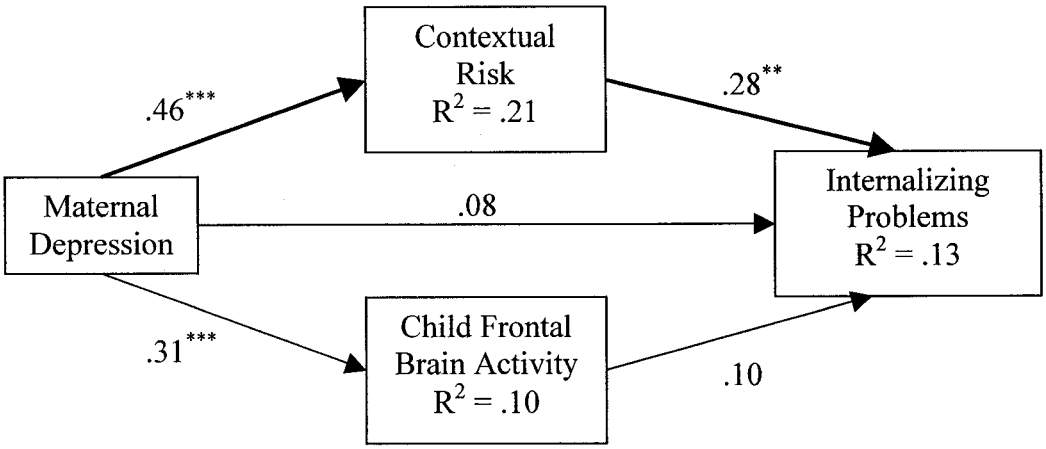
Table 9

Zero-order correlations between predictor variables and child behavior outcomes

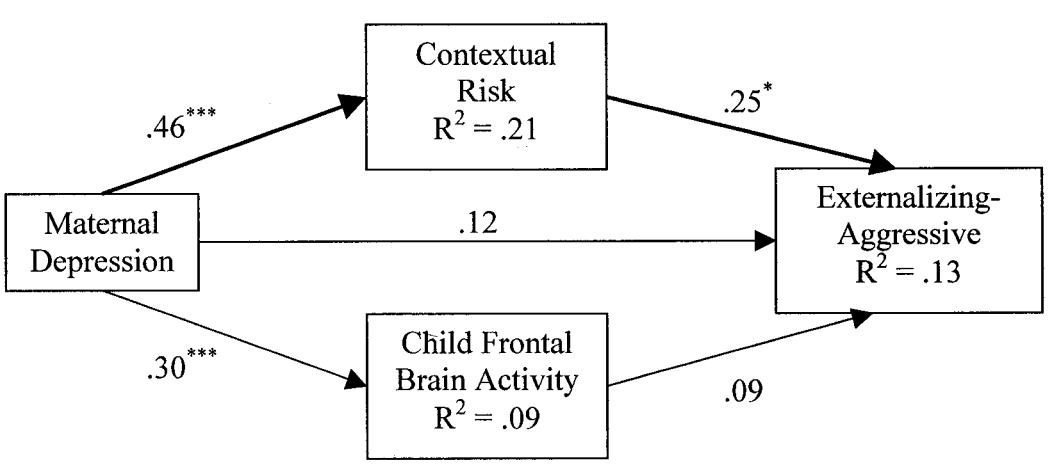
	Maternal Depression	Contextual Risk	Child Frontal Brain Activity
Parent-report			
Internalizing	.24**	.30***	.20*
Externalizing-Aggressive	.24**	.28**	.20*
Externalizing-ADHD	.24**	.32***	.20*
Social Competence	-.21*	-.33***	-.07
Teacher-report			
Internalizing	.08	.24**	.11
Externalizing-Aggressive	.09	.29**	.04
Externalizing-ADHD	.00	.16	.01
Social Competence	.06	-.20*	.15

* $p \leq .05$ ** $p < .01$ *** $p < .001$

(a)



(b)



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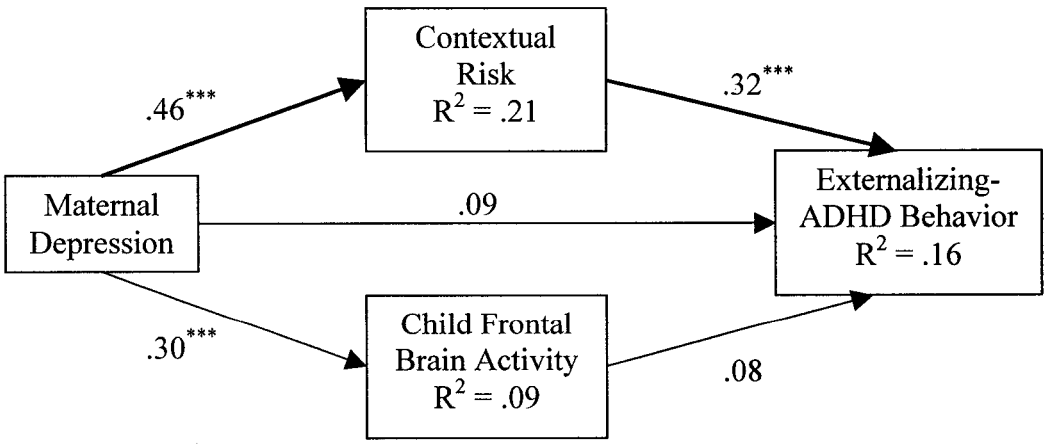

(continued)

(d)

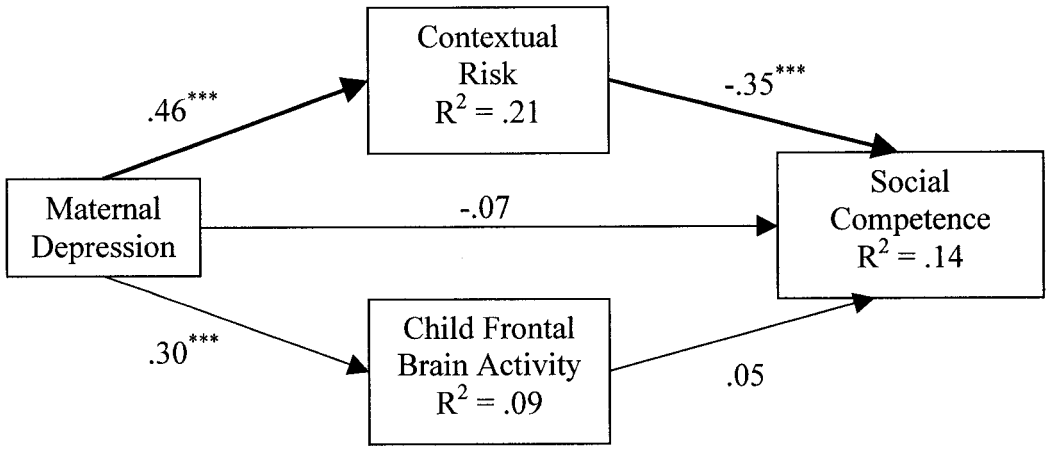

Figure 8a-d. Path models examining children's frontal brain activity and contextual risk factors as mediators of the relation between maternal depression and parent-reported child behavior outcomes. Standardized path coefficients are presented. Bold lines indicate significant mediation effects. (a) Model for internalizing behavior. Comparative Fit Index (CFI) = .96. (b) Model for externalizing-aggressive behavior. CFI = .94. (c) Model for externalizing-ADHD behavior. CFI = .97. (d) Model for social competence. CFI = .95. $^{*}p \leq .05$ $^{**}p < .01$ $^{***}p < .001$

Discussion

This study investigated the associations among the longitudinal course of maternal depression, co-occurring risk factors in the family environment, and school age children's psychophysiology and behavior. Using a sample of mostly middle class, Caucasian mothers and their children, this study examined data that chronicled the mother's depression over seven years, from the child's birth through age 6 ½ years, and related differences in the longitudinal course of maternal depression to differences in child psychophysiology and behavior at age 6 ½ years. This investigation advanced existing research on children of depressed mothers in three important ways. First, latent growth mixture modeling was used to identify trajectories of maternal depression over a seven year period. Most previous longitudinal studies have failed to evaluate maternal depression during the intervals between assessments, and thus have not accounted for variability in the course of depression. Second, extending previous research with infants and preschool children, this study examined the relation between maternal depression and child psychophysiology in school age children. Finally, this study evaluated whether child psychophysiology and contextual risk factors mediate the relation between maternal depression and child behavioral outcomes.

Latent growth mixture models identified three latent classes of depressed mothers based on information about the mother's depression from her child's birth through age 7 years. Other measures of depression provided preliminary validation of these groupings. Three groups of mothers, those with chronic, decreasing, or stable low depression, emerged from the data. The rate of chronic depression (8% of depressed mothers) in this sample is similar to the prevalence rates observed in epidemiological studies. For

example, Keller and colleagues (Keller et al., 1992) found approximately 11.5% of their sample experienced chronic depression over a 5 year follow-up period. Notably, patients in the Keller study did not meet full criteria for major depression during the entire follow-up period, which is similar to the mothers with chronic depression in this sample who, although chronically symptomatic, also experienced periods of subthreshold depression. Mothers with decreasing depression did not completely recover during the study period. These mothers continued to experience less frequent, low to moderate levels of depression. This finding is not surprising given the recurrent nature of depressive disorders (Judd, 1997) and the tendency of subthreshold episodes to precede or follow episodes of major depression (Angst & Merikangas, 1997). The identification of these different groups highlights the importance of assessing depression during the interval between assessments and assessing for subthreshold depression.

Maternal Depression and Child Behavior Outcomes

Consistent with previous studies of the chronicity and severity of maternal depression (Alpern & Lyons-Ruth, 1993; Brennan et al., 2000; Campbell et al., 1995; NICHD Early Child Care Research Network, 1999), child behavioral outcomes varied as a function of the longitudinal course of maternal depression. In general, children of chronically depressed mothers had the highest level of externalizing behavior problems, the highest rate of behavior disorder diagnoses, and the lowest social competence, corroborating research that has found depression chronicity and severity to predict child behavior outcomes (Brennan et al., 2000; Campbell et al., 1995; Luoma et al., 2001; NICHD Early Child Care Research Network, 1999). Notably, this finding held for both parent and teacher-reported externalizing behavior problems. Many previous studies

have relied exclusively on mother reports of child behavior problems (Brennan et al., 2000; Munson et al., 2001). The addition of father and teacher reports in this study reduces concerns about potential reporter bias and increases the validity of the results.

In contrast to some previous studies, this study did not find a relation between maternal depression group and parent or teacher report of child internalizing behavior outcomes. In the final model, however, months of maternal depression displayed a significant, although modest correlation with parent-reported internalizing problems. The inconsistency of this finding suggests a weaker association between maternal depression and school age children's internalizing behaviors, which may not have been detectable in the group analysis because of the small sample size in the chronic depression group. Some previous studies of young children of depressed mothers have used a composite measure of child behavior problems rather than examining the differential effects of maternal depression on children's internalizing and externalizing behaviors (Brennan et al., 2000; NICHD Early Child Care Research Network, 1999; Seifer et al., 2001). The studies that have examined internalizing and externalizing problems separately have had inconsistent results with respect to internalizing problems. Some have found associations between maternal depression and child internalizing symptoms (Alpern & Lyons-Ruth, 1993; Essex et al., 2001; Zahn Waxler et al., 1990), while others have not (Cicchetti et al., 1998; Luoma et al., 2001). This inconsistency may reflect developmental differences in the expression of behavior problems. Clinical levels of depressive symptoms are rare in young children. Associations between maternal depression and child internalizing symptoms are more consistently found for older children and adolescents (Anderson & Hammen, 1993; Hammen et al., 1991; Lee & Gotlib, 1991). Furthermore, parents and

teachers may be less aware of young children's internalizing symptoms. Some research has shown parents report fewer internalizing symptoms in their children compared to the children themselves. This explanation is plausible given that children of chronically depressed mothers were more likely to endorse elevated symptoms of loneliness and social dissatisfaction. Finally, internalizing and externalizing behavior problems often co-occur. In this sample, 68.6% of children with elevated parent-reported externalizing-aggressive behaviors (greater than .5 SD above the mean) and 69.4% of children with elevated parent-reported externalizing-ADHD behaviors (greater than .5 SD above the mean) also had elevated parent-reported internalizing behaviors (greater than .5 SD above the mean). Therefore, the stronger relation between maternal depression and child externalizing behavior at this age may partially relate to the high incidence of comorbidity in this sample.

Children of mothers with decreasing depression had higher parent-reported externalizing-ADHD behaviors and lower parent-reported social competence compared to children of non-depressed mothers. Children of mothers with stable low depression also had higher parent-reported externalizing-ADHD behaviors compared to children of non-depressed mothers. Other studies have also found associations between non-chronic maternal depression and adverse child outcomes. Alpern and Lyons-Ruth (1993) found that depressive symptoms when children were 18-months old predicted children's anxious and withdrawn behavior at age 5-years. In the same study, concurrent, but non-chronic maternal depression was associated with mother and teacher reports of attention problems and hyperactive behaviors. Essex and colleagues (Essex et al., 2001) found that maternal depression during infancy was associated with increased child internalizing

problems during kindergarten, while maternal depression during the toddler / preschool period only was associated with increased externalizing behavior problems for girls. Taken together, these findings suggest there are enduring effects of non-chronic exposure to maternal depression. The nature of these effects, however, remains unclear. As in the Alpern & Lyons-Ruth study (Alpern & Lyons-Ruth, 1993), in this sample, non-chronic maternal depression was associated with increased externalizing behaviors (e.g. hyperactivity and attention problems) that did not reach more serious, deviant levels characterized by aggressive behaviors. This study, however, failed to find an association between earlier, non-chronic maternal depression and child internalizing problems. This difference in findings may reflect the nature of the samples studies. The Alpern & Lyons-Ruth study (Alpern & Lyons-Ruth, 1993) was based on a sample of low-income families, whereas this sample included mostly middle-class mothers. Although the Essex study (Essex et al., 2001) included higher income families, their sample was much larger (421 children) and more variable in terms of family socioeconomic status. Family income has been found to predict children's depressive symptoms above and beyond the effects of maternal depressive symptoms (Graham & Easterbrooks, 2000).

The effects sizes for the relations between maternal depression and child behavior were small to medium in magnitude. This may reflect the fact that this study was based on a relatively homogenous, middle-class sample of depressed and non-depressed mothers. In addition, increased attrition of lower income, unmarried, depressed mothers in this sample may have led to a more conservative estimate of the association between maternal depression and child behavior outcomes.

Maternal Depression and Child Psychophysiology

Extending earlier research on infants and preschool children of depressed mothers, this study found an association between maternal depression and school age children's brain activity. Consistent with findings in this same sample at the preschool age (Dawson et al., in press), school age children of mothers with chronic depression were found to have decreased generalized brain activation (higher EEG alpha power) across all brain regions assessed. Reduced generalized brain activation (higher EEG alpha power) may reflect reduced attentiveness or engagement with the external environment. When frontal and parietal regions were analyzed separately, however, the finding held for the frontal, but not the parietal region. The specificity of this finding to the frontal region corroborates previous research that has found associations between maternal depression and child frontal brain activity (Dawson et al., 1997; Dawson, Frey, Panagiotides et al., 1999; Field, Fox et al., 1995; Jones et al., 2000) and may reflect the well-established role of the prefrontal cortex in emotion regulation and expression (Davidson, 2000; Marshall & Fox, 2000).

Whereas during infancy and in one prior study of preschool children, children of depressed mothers were found to have relative right frontal EEG asymmetry compared to children of non-depressed mothers (Dawson et al., 1997; Jones et al., 2000; Jones, Field, Fox, Davalos, & Gomez, 2001; Jones et al., 1998; Jones, Field, Fox, Lundy et al., 1997), in this study, school age children of depressed mothers were found to have reduced generalized frontal brain activation. Differences in findings may relate to differences in methodology, developmental differences, and / or differences in the populations studied. In their study of preschool children of depressed mothers, Jones and colleagues (Jones et

al., 2000) examined resting EEG while children sat quietly with their eyes open, while this study used cascading bubbles as a baseline followed by emotion-eliciting film conditions. In addition ,the studies of Jones and colleagues (Jones et al., 2000; Jones et al., 2001; Jones et al., 1998; Jones, Field, Fox, Lundy et al., 1997) were based on samples of low SES, mostly ethnic minority mothers, whereas the current study used a sample of mostly Caucasian, middle class mothers. Subtle EEG asymmetries may be absent or less detectable in preschool and school age children of middle-class depressed mothers. Resources and protective factors in the lives of higher SES families may attenuate the impact of maternal depression on child psychophysiology and behavior. For example, higher SES depressed mothers may have access to better treatment options and alternate caretakers for their children. There is some evidence that affluence moderates the impact of maternal depression on parenting behavior (Lovejoy et al., 2000); therefore, children of more affluent depressed mothers may have less exposure to disrupted mother-child interactions, which have been hypothesized to compound the effects of maternal depression on child psychophysiology (Field, 2000). To date, no other study has compared frontal brain activity in school age children of depressed and non-depressed mothers. Because so few studies have examined frontal brain activity in children of depressed mothers beyond infancy, the implications of this finding remain unclear. Additional research is necessary to replicate and explain this finding. In particular, research on the psychophysiology of children of depressed mothers that uses a more economically and ethnically diverse sample could examine the potential moderating influences of SES and ethnicity. Research that directly examines the possible moderating role of mother-child interactions would also help elucidate the circumstances under

which children of depressed mothers are most likely to develop atypical psychophysiology.

With respect to autonomic activity and stress hormone levels, children of chronically depressed mothers were found to have higher RSA reactivity compared to children of mothers with stable low depression. Previous studies of infants and preschool children have also found associations between maternal depression and measures of parasympathetic activity (Field et al., 1996; Field, Pickens et al., 1995; Jones et al., 1998). These studies found that maternal depression is related to lower vagal tone in infants and preschool children. Although this study did not find differences in baseline vagal tone, children of chronically depressed mothers were found to have greater parasympathetic withdrawal in response to the emotion-eliciting film clips. Tonic differences in vagal tone observed during infancy and preschool may indicate differences in temperament or emotionality, whereas differences in vagal reactivity may reflect differences in attention, emotional regulation, or mood (Beauchaine, 2001). For all children, RSA reductions and heart rate acceleration were observed during the neutral and negative emotion-eliciting film conditions, suggesting vagal withdrawal. Children of mothers with chronic depression, however, exhibited a more pronounced reduction in RSA across all conditions, including the happy film condition when increases in RSA would be expected. Although RSA suppression in response to challenge has been found to relate to lower rates of behavior problems and more adaptive emotion regulation in preschool children, and sustained attention in school-age children (Calkins & Fox, 2002), too much suppression may mark a propensity toward fight or flight responding and has been associated with anxiety disorders and impulsive aggression (Beauchaine, 2001).

Both anxiety disorders and aggressive behaviors have been observed in children of chronically depressed mothers. Thus, high RSA reactivity in children of the most chronically and severely depressed mothers may indicate general dysregulation and a vulnerability to psychopathology.

In contrast to previous studies of infants (Dawson et al., 2001; Lundy et al., 1999), this study did not find an association between maternal depression and child heart rate or salivary cortisol levels. Differences in heart rate may be less detectable because of the dual influence of sympathetic and parasympathetic branches of the autonomic nervous system on heart rate (Berntson, Cacioppo, Quigley, & Fabro, 1994). Elevated stress hormone levels have been associated with major depression in adults (Heuser, 1998; Nemeroff, 1988); however, differences in stress hormone levels are less reliably found in depressed children (Goodyer et al., 1991; Kaufman et al., 1997). Given that none of the children in this study met diagnostic criteria for major depression, it is not surprising that the children of depressed mothers who are at risk for depression did not exhibit elevated salivary cortisol levels. In a separate study of a subgroup of this same sample when the children were 7-years-old, children of depressed mothers who also had clinically significant levels of internalizing problems were found to have elevated salivary cortisol levels (Ashman et al., 2002); however the experimental paradigm was different and time of day of sample collection was better controlled in this later study.

Child Psychophysiology and Behavior

As expected, associations were found between child psychophysiology and behavior; however, the magnitudes of the correlations were small and the direction of some of the findings was surprising. Higher parent-reported child externalizing-

aggressive behaviors were associated with reduced right frontal brain activation (higher EEG alpha power), lower heart rate, higher vagal tone, and higher evening salivary cortisol levels. Higher parent-reported child externalizing-ADHD behaviors were associated with lower heart rate and higher vagal tone. Higher parent-reported internalizing symptoms were associated with reduced right frontal brain activation. Higher parent-reported social competence was associated with lower laboratory post-stressor cortisol levels. Higher teacher-reported social competence was associated with higher left parietal EEG power (lower activation). Given the contemporaneous nature of these associations, it is unclear whether alterations in psychophysiology precede, follow, or simply co-occur with the development of behavior problems. Future studies should examine the temporal association between atypical psychophysiology and child behavior problems in order to address the question of whether underlying biological vulnerabilities increase risk for developing emotional and behavior problems later in life.

Frontal Brain Activity

Higher internalizing and aggressive behaviors were found to be associated with reduced right frontal brain activation. Activation of the right frontal region has been associated with a bias toward negative affect, shyness, withdrawal-type emotion regulation strategies, and behavioral inhibition (Davidson, 2000; Davidson et al., 1990; Davidson & Fox, 1989; Fox, 1991). The association between reduced right frontal activation and aggression may reflect disinhibition associated with aggressive behaviors, including angry affect and impulsivity. Children with externalizing behavior problems have been found to be more prone to anger, impulsivity, and low regulation (Eisenberg et al., 2001). Brain imaging studies of violent adults have found reductions in frontal brain

activation, and antisocial children have been found to have impairments in executive functions typically associated with the frontal region (see Raine, 2002 for a review). The positive relation between internalizing symptoms and right frontal EEG power was unexpected given the well-established association between right frontal brain activation and depression and withdrawn behaviors. Thus, gender differences were analyzed to further understand this finding.

When boys and girls were examined separately, the relation between frontal EEG and behavior problems was stronger for girls. In fact, for girls, the relation between higher internalizing behavior and higher frontal EEG power (lower activation) was significant for both the left (r = .29, p = .03) and right (r = .34, p = .01) frontal regions, whereas for boys, the association between internalizing problems and frontal EEG was not significant for either hemisphere (r = -.02 and -.00, p = ns for the left and right frontal regions respectively). The relation between reduced right frontal EEG activation and higher aggressive behaviors had similar magnitudes for girls and boys; however, for girls, the relation was significant (r = .27, p = .04), while for boys, the relation was only a trend (r = .26, p = .08). The reduced generalized frontal brain activation in girls with higher internalizing problems may reflect reduced engagement with the external environment. Other studies have found gender differences in frontal brain activity (Baving, Laucht, & Schmidt, 2003; Miller et al., 2002). In a recent study, Baving, Laucht, and Schmidt (2003) found gender differences in the relation between externalizing behavior problems and frontal EEG activity. Specifically, 11-year-old girls with externalizing behaviors displayed relative right frontal EEG activation and boys with externalizing behaviors displayed the opposite pattern.

Autonomic Activity

Higher parent-reported externalizing behavior problems were found to be associated with lower heart rate and higher vagal tone. Associations between aggressive or antisocial behavior and low heart rate have been frequently observed (Raine, 2002; Raine et al., 1997), suggesting an association between underarousal of the autonomic nervous system and aggressive behaviors. Low heart rate and aggressive behavior are not invariably associated, however, especially in young children (van Hulle, Corley, Zahn Waxler, Kagan, & Hewitt, 2000); therefore, further research is still needed to elucidate the conditions under which relations between low heart and aggressive behavior are found. The positive association between externalizing behaviors and RSA stands in contrast to previous studies that have found the opposite relation. Other research has consistently found associations between lower RSA and higher externalizing behavior in children at risk for externalizing disorders, especially boys (Beauchaine, Katkin, Strassberg, & Snarr, 2001; Calkins & Dedmon, 2000; Mezzacappa et al., 1997; Pine et al., 1998). For example, Pine and colleagues (Pine et al., 1998) reported lower heart period variability in 11-year-old boys with externalizing problems. High comorbidity of internalizing and externalizing behaviors in this sample may partially explain this finding. At least one previous study found higher heart rate variability among preadolescent boys with comorbid depression and conduct problems in comparison to boys with conduct problems alone (Beauchaine, Gartner, & Hagen, 2000). Furthermore, high vagal tone is not invariably related to lower behavior problems and better emotion regulation. One study of school age children found the opposite relation for girls (Eisenberg et al., 1995). Eisenberg and colleagues (Eisenberg et al., 1995) found a relation between high vagal

tone and low social competence in school age girls. Girls with high vagal tone were also higher on nonconstructive coping and negative emotionality. Children with higher vagal tone may also be less inhibited, a characteristic which, in excess, may be perceived more negatively by parents and teachers. Future research that includes direct observations of child inhibition would be useful in examining this hypothesis. Although this explanation is plausible, it cannot be ignored that findings on the relation between RSA and externalizing behavior in this sample do not corroborate most previous research. Additional research is necessary to replicate and explain this finding.

HPA Axis Activity

The negative association between parent-reported social competence and laboratory post-stressor cortisol levels suggests that children with higher social competence had an attenuated response to the laboratory stressor. For the group overall, however, the laboratory stressor did not produce cortisol elevations. In fact, on average, "post-stressor" cortisol levels were significantly lower than laboratory baseline cortisol levels ($t(113) = 3.86$, $p < .001$), suggesting that the task was not stress inducing for most children. The laboratory situation itself is a novel situation for children and may have inflated baseline cortisol levels for all children. Children with higher social competence may become more comfortable with the new environment over time compared to children with lower social competence. This finding requires replication, but may suggest that children with higher social competence recover more rapidly from the mild stress of novel situations.

The positive association between parent-reported aggressive behaviors and evening cortisol levels was not expected. Previous studies of cortisol and aggressive

behavior have been inconsistent, but those studies that have found associations between salivary cortisol and aggressive behaviors in children have found the opposite relation (McBurnett et al., 2000; Smider et al., 2002). In these studies, low cortisol levels were related to higher rates of aggressive and disruptive behaviors. Interestingly, in both these studies, the relation was specific to boys with disruptive behavior problems. In this sample, when boys and girls were analyzed separately, the significant relation between higher evening cortisol levels and aggressive behaviors held for girls ($r = .28$, $p = .03$), but not boys ($r = .19$, $p =$ ns). Whether this reflects a true gender difference is unclear given reduced sample sizes when the sample is split and the fact that the correlation is in the same direction for both boys and girls. More likely, the finding of higher evening cortisol in children with aggressive behaviors reflects the high level of comorbidity between internalizing and externalizing behaviors in this sample (noted above). Higher baseline cortisol and cortisol reactivity has been found to be associated with comorbidity between disruptive behavior disorders and anxiety (McBurnett et al., 1991; van Goozen et al., 2000).

It is not surprising that relations between child behavior and salivary cortisol levels were not found for other measures of cortisol collected in this study given the limitations in the manner in which salivary cortisol samples were collected. Specifically, with the exception of evening cortisol samples, which were collected at a similar time across participants, time of day was not controlled in this study. This variation in time of sample collection was not optimal for cortisol data collection given the known circadian rhythm of cortisol; however, the nature of the sample did not permit controlling for time of day of laboratory sessions. Session scheduling had to be coordinated with mothers'

work schedules and children's school schedules. Furthermore, more recent research suggests that single measures of cortisol may reflect reactivity to recent stressors rather than true baseline cortisol levels. A more reliable method for assessment of baseline cortisol levels involves collecting samples at the same time of day across several days and averaging those samples (Smider et al., 2002). Unfortunately, funding restrictions and demands on families for participation in other aspects of the study did not permit saliva collection on multiple days. Future research should endeavor to control for time of day effects and measure cortisol across multiple days.

Tests of Mediation

Consistent with previous research, contextual risk factors, such as low marital satisfaction and high family conflict, were found to mediate the relation between maternal depression and child behavior outcomes (Cicchetti et al., 1998; Dawson et al., in press; Fergusson et al., 1995; Hammen, 2002). Higher levels of maternal depression were associated with increased contextual risks which were in turn associated with more negative child behavioral outcomes. These results suggest maternal depression may influence child behavior through its association with a more stressful social context. The contextual risks assessed in this study may have been particularly potent because of their stability over time, suggesting repeated or chronic exposure to stressful life circumstances. Although this analysis implies a directional effect, it is important to note the correlational nature of the data cannot detect causality. As implied in this analysis and as Hammen and colleagues have suggested (Hammen, 2002), depressed mothers may generate interpersonal difficulties and stressful life events because they have impaired coping and interpersonal problem-solving skills. There is some evidence that depression

contributes to the occurrence of stressful life events (Hammen, 1996). Alternatively, maternal depression may result from a more stressful environment or the two risk factors co-occur. More research examining the temporal association between stressful life events and episodes of maternal depression is needed to investigate the mechanism through which contextual risk factors affect the relation between maternal depression and child behavioral outcomes.

Limitations and Future Directions

Although this study advances research on maternal depression in important ways, there are still several limitations. First, because of sample size limitations, more complex models examining mechanisms of risk transmission from mother to child were not able to be examined. Sample size limitations may have also reduced power for the detection of interaction effects and more subtle effects, such as the expected differences in EEG asymmetry. In addition, limitations of the sample did not allow for separating out the effects of depression chronicity and severity as has been accomplished in a previous study with a much larger sample (Brennan et al., 2000). In this sample, mothers with chronic depression were also more severely depressed over the course of the study, and therefore, these two dimensions of depression are confounded. Future research with larger samples should investigate more complex models of multiple mechanisms of risk and their potential interactions.

Second, the population studied was mostly middle class, Caucasian mothers and their children; therefore, results from this study may not generalize to other populations and may not be directly comparable to other studies of more diverse, low income populations of depressed mothers. The homogenous nature of this sample and the

attrition of some of the more affected mothers may have decreased the magnitude of some effects. Future research with a more ethnically and socioeconomically diverse sample of mothers and children will allow for the examination of the potential moderating influences of SES and ethnicity.

Summary

In summary, this study provides evidence that maternal depression, especially chronic and severe maternal depression is associated with less optimal behavior and psychophysiology in school age children. Children of chronically depressed mothers had higher levels of parent and teacher-reported externalizing problems, reduced generalized frontal brain activation, and higher RSA reactivity to emotion eliciting conditions. Contextual risk factors, such as family conflict and life stress, were found to partially account for the relation between maternal depression and child behavior. Child psychophysiology was found to relate to child behavior, especially for girls; however, the magnitudes of these associations were small and should be interpreted with caution. Psychophysiology and behavior may be more strongly related in children with more severe psychopathology, such as conduct disorder or major depression. These diagnoses were rare or nonexistent in this sample. The results of this study contribute to our understanding of the mechanisms by which maternal depression places children at risk for emotional and behavioral difficulties. Early identification of children at risk may inform early intervention and prevention research and intervention programs.

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monitoring program using the longitudinal interval follow-up evaluation. Journal of Psychiatric Research, 35(5), 297-305.

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Weissman, M. M., Prusoff, B. A., Gammon, G. D., Merikangas, K. R., Leckman, J. F., & Kidd, K. K. (1984). Psychopathology in the children (ages 6-18) of depressed and normal parents. Journal of the American Academy of Child Psychiatry, 23, 78-84.

Weissman, M. M., Warner, V., Wickramaratne, P., Moreau, D., & Olfson, M. (1997). Offspring of depressed parents. Archives of General Psychiatry, 54, 932-940.

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Wheeler, R. E., Davidson, R. J., & Tomarken, A. J. (1993). Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. Psychophysiology, 30, 82-89.

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EDUCATION

University of Washington, Seattle, WA
Candidate for Ph.D. in Child Clinical Psychology — *April 2002*
Master of Science, Child Clinical Psychology — *Summer 1999*
Brown University, Providence, RI
Bachelor of Arts with honors, Educational Studies — *May 1995*

HONORS AND AWARDS

Gatzert Child Welfare Fellowship, 2003
National Institutes of Health Intramural Research Training Award (IRTA), 1995 - 1996
Phi Beta Kappa, 1995
MacGraw Fellowship for thesis research, 1994 - 1995

RESEARCH EXPERIENCE

Graduate Research Assistant, Maternal Depression Project *Fall 1997 – May 2003*
Department of Psychology, University of Washington
Supervisor: Geraldine Dawson, Ph.D.
Research assistant for NIMH-funded, longitudinal investigation of the effects of maternal depression on children's psychophysiological and behavioral development. Conducted diagnostic interviews with parents and children. Administered neuropsychological testing battery to children. Collected psychophysiological data. Directed videotaped mother-child interaction sessions. Conducted statistical analyses. Developed and maintained longitudinal databases. Prepared research protocols. Supervised undergraduate lab assistants. Prepared, submitted, and published manuscripts.

Graduate Research Assistant, Autism Research Project *Sum. 2000 – Sum. 2001*
Department of Psychology, University of Washington
Supervisors: Geraldine Dawson, Ph.D., Annette Estes, Ph.D., and Jeff Munson, Ph.D.
Research assistant for NICHD-funded, multi-site investigation of the early development and neurobiology and genetics of autism. Conducted structured diagnostic observations of children with autism and developmental disabilities (DD). Administered cognitive, language, and neuropsychological assessments to children with autism and DD, and typically developing siblings. Conducted structured diagnostic interviews with parents. Interviewed parents about psychiatric and family mental health history. Conducted cognitive, memory and neuropsychological assessments with parents. Wrote psychological reports. Conducted statistical analyses. Assisted with data management.

IRTA Fellow, Bipolar Disorder Research Project *Sum. 1995 – Spring 1997*
Clinical Psychobiology Branch, NIMH (Bethesda, MD)
Supervisors: Ellen Leibenluft, M.D. and Thomas Wehr, M.D.
Research assistant for clinical investigations of biological / social rhythms and rapid cycling bipolar illness. Initiated and independently conducted study of social rhythms. Recruited, screened, and trained volunteers. Coordinated weekly outpatient clinic for bipolar patients. Conducted data entry and statistical analysis. Developed and maintained longitudinal databases. Designed graphic presentations of data for oral and written presentations. Assisted manuscript preparation.

Research Apprentice, Sleep Research Laboratory *Fall 1994 - Spring 1995*
Department of Psychology, Brown University (Providence, RI)
Supervisor: Mary Carskadon, Ph.D.
Research assistant for a sleep deprivation study. Trained extensively in research protocol and polysomnographic electrode placement techniques (EEG, EOG, and EMG). Administered performance tests, surveys, and questionnaires. Conducted telephone interviews. Administered Sleep Habits Survey to high school students. Entered data.

CLINICAL EXPERIENCE

Psychology Resident – Child Clinical Track *Summer 2003 – Spring 2004*
Department of Psychiatry and Behavioral Sciences, University of Washington
Internship Director: Joan Romano, Ph.D.

- Consultation and Liaison Service, Children's Hospital and Regional Medical Center
  Supervisors: Stan Whitsett, Ph.D. and Kathleen Myers, M.D.
  Three-month full-time rotation: Conducted psychiatric assessments and short-term behavioral interventions (e.g. relaxation, cognitive behavioral therapy) for children and adolescents in inpatient pediatric units, emergency room, and outpatient clinics. Primary consultations included children experiencing psychological concerns in the context of chronic illness, eating disorders, conversion disorders, pain management, and behavioral management for treatment noncompliance. Consulted with physicians, nursing staff, social workers, and child life specialists.
- Inpatient Child Psychiatry, Children's Hospital and Regional Medical Center
  Supervisors: Rose Calderon, Ph.D. and Christopher Varley, M.D.
  Three-month full-time rotation: Provided intensive case management for children and adolescents with severe mental health problems, including those enrolled in a specialized Eating Disorders Program. Conducted in-depth assessments, individual and family therapy, and behavior plan management. Coordinated care with a multidisciplinary team including psychiatrists, social workers, nursing staff, and pediatric mental health specialists. Consulted with outpatient providers and school. Wrote comprehensive discharge summaries.
- Neuropsychology Consultation Service, CHRMC
  Supervisor: David Breiger, Ph.D.
  Six-month half-time rotation: Conducted neuropsychological assessments with children and adolescents with a variety of medical, neurodevelopmental, learning, and

behavioral problems. Conducted parent interviews. Consulted with schools. Wrote comprehensive neuropsychological reports. Participated in feedback sessions with families.

- Outpatient Psychiatry Clinic, Children's Hospital and Regional Medical Center
  Supervisor: Matt Speltz, Ph.D.
  Six-month half-time rotation: Conducted comprehensive assessments and treatment for children and adolescents with a wide variety of mental health concerns. Conducted individual and family therapy, and parent consultation. Coordinated intervention plans with school teachers and counselors. Participated in multiple weekly consultation meetings for ethical and legal issues, psychotropic medication information, and Dialectical Behavior Therapy (DBT) training. Co-led DBT skills group for adolescents.

Clinic Intake Staff / Teaching Assistant *Fall 2001 – Fall 2002*
Psychological Services and Training Center, University of Washington
Supervisor: Corey Fagan, Ph.D.
Conducted clinical phone interviews with prospective clients. Provided crisis intervention services and referral resources for callers. Served as emergency clinic backup. Assisted clinic director in teaching Clinical Methods: Interviewing and Ethics. Assisted supervision of less advanced clinical students. Co-led group supervision for beginning clinical students.

Practicum Student, Eating Disorders Clinic *Summer 2002 – May 2003*
Children's Hospital and Regional Medical Center
Supervisor: Rosemary Calderon, Ph.D.
Conducted individual outpatient therapy with adolescents with eating disorders. Co-led weekly inpatient eating disorders process group. Co-conducted detailed intake interviews and information sessions for adolescents and their families. Participated in weekly case consultation. Completed clinical documentation.

Staff Therapist, Psychological Services and Training Center *Win. 1998 – May 2003*
Department of Psychology, University of Washington
Supervisors: Corey Fagan, Ph.D. and various community practitioners
Provided individual adult therapy (CBT, motivational interviewing), individual child and adolescent therapy (CBT, developmental, child-directed play therapy), parent consultations, substance use evaluations, and immigration court evaluations in a university-based training clinic. Received hour-for-hour supervision for each client.

Practicum Student, Parenting Clinic *Fall 2000 – Spring 2001*
University of Washington
Supervisors: Caroyln Webster-Stratton, Ph.D. and Jamila Reid, Ph.D.
Co-led school-based social skills groups for Kindergarten students. Extensively trained in empirically-supported social skills curriculum. Collaborated with teachers to develop specific behavior plans for individual students.

Practicum Student, Prime Time Project *Fall 2000 – Spring 2001*
Odessa Brown Children's Clinic
Supervisor: David Stewart, Ph.D.
Provided individual counseling for a high-risk African-American adolescent as part of a multisystemic team of service providers. Observed counseling sessions and evaluations with high risk, juvenile offenders.

Practicum Student, Pediatric Psychology Service *Fall 1999 – Spring 2000*
Mary Bridge Children's Hospital and Health Center (Tacoma, WA)
Supervisor: Karen Pavlidis, Ph.D.
Conducted comprehensive psychological assessments, including IQ, academic, learning / memory, executive function, social-emotional, and personality assessments, for children and adolescents in an outpatient hospital setting. Interviewed parents. Wrote comprehensive psychological reports for each assessment.

Counselor, DC Rape Crisis Center *Fall 1995 – Winter 1997*
Washington, DC
Staffed rape crisis hotline for DC Metropolitan area. Supported and counseled sexual assault survivors. Accompanied survivors through hospital procedures. Trained in sexual assault legislation, rape trauma syndrome, crisis counseling and advocacy.

Counselor, Ramapo Anchorage Camp *Summer 1994*
Rhinebeck, NY
Supervised recreational and artistic activities for 4-year-old mostly minority, emotionally disturbed and / or learning disabled children during a 4-week overnight camp experience. Provided emotional support in a family-like environment. Taught basic life skills. Developed and implemented individualized behavior plans.

TEACHING EXPERIENCE

Graduate Teaching Assistant, Intelligence Assessment *Spring 2003*
Department of Psychology, University of Washington
Supervisor: David Breiger, Ph.D.
Co-supervised graduate students in conducting standardized cognitive assessments with young adults and children, and in writing psychological reports.

Writing / Rhetoric Fellow, Rose Writing Fellows Program *Fall 1993 - Spring 1995*
Brown University, Providence, RI
Supervisor: Rhoda Flaxman
Selected to teach writing process and rhetorical skills to fellow students. Assisted faculty in integrating writing and speaking into their courses. Critically analyzed and commented on students' oral and written compositions. Conferenced with students about oral and written assignments. Trained extensively in writing theory and peer tutoring strategies.

Fellow for Students with Alternate Learning Styles *Fall 1993 - Spring 1995*
Brown University, Providence, RI
Assisted individual college students with learning disabilities in improving writing skills and developing a writing process. Worked intensively with one student each semester, helping him/her with all his/her writing assignments.

CLINICAL CASE PRESENTATIONS AND GUEST LECTURES

Clinical case presentation of a 9-year-old boy and his mother being seen for individual child therapy and parent training (February, 2002). Presented at the clinical psychology graduate student colloquium.

Conducting clinical intake interviews with children, adolescents and their parents. (October, 2001 & 2002). Lecture delivered to psychology graduate students in Clinical Methods class.

Effects of maternal depression on early developing biological systems related to risk for affective disorder. (October 1999). Guest lecture delivered to a graduate class on Neurobiology and Behavior at Seattle Pacific University.

SPECIALIZED CLINICAL TRAINING

Introduction to Acceptance and Committment Therapy *Spring 2004*
Instructor: Steven Hayes, Ph.D., Department of Psychology, University of Nevada
One-day workshop covering ACT processes and techniques, particularly acceptance, cognitive defusion, and behavioral commitment strategies.

Cognitive Behavioral Analysis System of Psychotherapy *Autumn 2003*
Instructor: Jack Carr, Ph.D., University of Washington, Department of Psychiatry and Behavioral Sciences
Weekly didactic including theory and practice in the treatment of chronic depression in adults.

Interpersonal Psychotherapy *Autumn 2003*
Instructor: David Lischner, M.D., University of Washington, Department of Psychiatry and Behavioral Sciences
Weekly didactic concerning interpersonal therapy theory and techniques in the treatment of adults.

Meal Support Training *Autumn 2003*
Instructor: Dave Hall, PMHS, Children's Hospital and Regional Medical Center
Five-week seminar teaching the fundamentals of meal support training for children and adolescents with eating disorders.

Skills Training in Dialectical Behavior Therapy: The Essentials *Winter 2003*
Instructors: Linda Dimeff, Ph.D. and Shireen Rizvi, M.S., Behavioral Technology Transfer Group
Two-day workshop covering the fundamentals of DBT skills training; learned the content of the four skills training modules, how to apply DBT strategies in skills training, and how to organize and structure DBT skills training classes for clients.

Cross-cultural Counseling Consultation Group *Winter 1999 – Spring 2001*
Instructors: Kimberly Barrett, Ed.D. and William George, Ph.D., University of Washington Department of Psychology
Weekly didactic and case consultation group concerning cultural and clinical issues related to diverse clients; read and discussed relevant historical and current materials.

Anxiety Disorders Treatment Group *Winter 2000*
Instructor: David Kosins, Ph.D., University of Washington Department of Psychology
Weekly didactic and case consultation group on the assessment and treatment of anxiety disorders, with emphasis on manualized, empirically-supported treatments for social phobia, generalized anxiety disorder, and panic disorder.

Assessment and Treatment of Suicidal Behavior *Summer 1999*
Instructor: Marsha Linehan, Ph.D., University of Washington Department of Psychology
Twenty-hour workshop on standards of care, crisis management, and long-term treatment strategies for acutely and chronically suicidal clients.

Assessment and Treatment of Addictive Behaviors *Autumn 1998 - Spring 1999*
Instructors: G. Alan Marlatt Ph.D. and Jason Kilmer, Ph.D., University of Washington Department of Psychology
Three-quarter weekly didactic seminar and case consultation group including training in models of addiction, assessment and treatment of addictive behaviors, with an emphasis on motivational interviewing and relapse prevention techniques; completion of a supervised substance use evaluation.

MEMBERSHIP IN PROFESSIONAL SOCIETIES

American Psychological Association (student affiliate), 1996-present
Society for Research in Child Development (student affiliate), 1998-present

ORGANIZATIONS AND COMMUNITY ACTIVITIES

Graduate Psychology Action Committee, 1998 – 2002
Clinical Case Presentation Requirement Committee (student representative), 2002

POSTER PRESENTATIONS

Clark, C. H., Shocket, L. S., Turner, E. H., Ashman, S. B., and Rosenthal, N. E. (1996). Efficacy of the light visor in maintaining antidepressant response in SAD patients. Poster presented at the 8th annual meeting of the Society for Light Treatment and Biological Rhythms, Bethesda, MD.

PUBLICATIONS

Dawson, G., Ashman, S. B., Panagiotides, H., Hessl, D., Self, J., Yamada, E., & Embry, L. (2003). Preschool outcomes of children of depressed mothers: Role of maternal behavior, contextual risk, and children's brain activity. Child Development, 74, 1158-1175.

Ashman, S. B., Dawson, G., Panagiotides, H., Yamada, E., and Wilkinson, C. W. (2002). Stress hormone levels of children of depressed mothers. Development and Psychopathology, 14, 333-349.

Ashman, S. B., & Dawson, G. (2002). Maternal depression, infant's psychobiological development, and risk for depression. In S. H. Goodman & I. H. Gotlib (Eds). Children of Depressed Parents: Mechanisms of Risk and Implications for Treatment. Washington, DC: American Psychological Association.

Dawson, G., Ashman, S. B., Hessl, D., Spieker, S., Frey, K., Panagiotides, H., & Embry, L. (2001). Autonomic and brain electrical activity in securely- and insecurely-attached infants of depressed mothers. Infant Behavior and Development, 24, 135-149.

Dawson, G., Ashman, S. B., & Carver, L. J. (2000). The role of early experience in shaping behavioral and brain development and its implications for social policy. Development and Psychopathology, 12, 695-712.

Dawson, G. & Ashman, S. B. (2000) On the origins of a vulnerability to depression: The influence of the early social environment on the development of psychobiological systems related to risk for affective disorder. In C. A. Nelson (Ed). The Effects of Adversity on Neurobehavioral Development, Minnesota Symposia on Child Psychology, Volume 31. New Jersey: Lawrence Erlbaum Associates, Inc.

Leibenluft E., Ashman S.B., Feldman-Naim S., & Yonkers K.A. (1999). Lack of relationship between menstrual cycle phase and mood in a sample of women with rapid cycling bipolar disorder. Biological Psychiatry, 46(4), 577-580.

Ashman, S. B., Monk, T. H., Kupfer, D. J., Clark, C. H., Myers, F. S., Frank, E. & Leibenluft, E. (1999). The relationship between social rhythms and mood in patients with rapid cycling bipolar disorder. Psychiatry Research, 86(1), 1-8.

Leibenluft, E., Schmidt, P. J., Turner, E. H., Danaceau, M. A., Ashman, S. B., Wehr, T. A. & Rubinow, D. A. (1997). Effects of leuprolide-induced hypogonadism and testosterone replacement on sleep, melatonin, and prolactin secretion in men. Journal of Clinical Endocrinology and Metabolism, 82(10), 3203-3207.