Responsivity to Offspring's Expression of Emotion among Childhood-Onset Depressed Mothers

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Abstract

This study examined responsivity of mothers with childhood-onset depression (COD) in relation to children's overt expression of positivity and distress. It was hypothesized that COD and control mothers would differ in contingent responsivity to their children's expression of both positivity and negativity or distress. Using observations and maternal reports of their own emotional reactions, COD mothers were found to be less responsive to children's expression of distress. A gender by group interaction was also found with respect to observed maternal responsiveness to child positivity, such that COD mothers were more responsive to girls' than boys' expression of positivity. The results are discussed in reference to transactional models of early child psychopathology.

Keywords: maternal depression, parenting, emotion regulation, childhood-onset depression, parent-child interaction
Parenting Risk Factors Among Offspring of Childhood-Onset Depressed Mothers

Research interest in the juvenile offspring of depressed adult probands is relatively recent. An early review of the literature suggested a high rate of impairment among children of such parents (Beardslee, Bemporad, Keller, & Klerman, 1983). More precise, diagnostically-oriented studies were prompted by data on depression in childhood, and were directed to exploring whether the offspring of probands with depressive illness were also at heightened risk for depressive illness. This research has focused on three questions: (1) Does the behavioral and psychological adjustment of offspring of adult depressives differ significantly from offspring of normals? (2) Is the age of onset of depression in the parent related to the timing and severity of the child’s risk for psychopathology? (3) Are there specific disorders for which offspring of depressed probands appear to be at heightened risk?

First, research on school-age to young adult offspring of depressed parents consistently has shown them to be at increased risk for major depression compared to the offspring of normal parents (Hammen et al., 1987; Weissman et al., 1984; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). These studies have been replicated despite variation in the methods of evaluating psychiatric status of the adult proband and the offspring.

Second, severity and age of onset of psychopathology among offspring consistently have been linked to the age of onset of depression in the affected parent (Bland, Newman, & Orn, 1986; Moldin, Reich, & Rice, 1991). That is, the children of parents for whom depression began prior to age 20 have demonstrated an increased risk of earlier and more severe forms of psychopathology, most notably childhood depression. In one study, offspring of early-onset parents had a 14-fold greater risk of having depression before age 13 compared to the risk in the children of older-onset cases (> age 20, Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1984).
Third, maternal depression appears to be associated with a host of maladaptive outcomes among school-age children, including, but not limited to, depression and its behavioral precursors (i.e., anxiety disorders) (see Downey & Coyne, 1990). Consistent associations have been found between maternal depression and child depression and dysthymia (Beardslee et al., 1983; Hammen et al., 1987; Radke-Yarrow, 1990), anxiety disorders (Fendrich, Warner, & Weissman, 1990), and externalizing problems (Radke-Yarrow, 1998; Zahn-Waxler et al., 1990). Although the majority of studies have found that living with a depressed parent is a risk factor for both childhood internalizing and externalizing difficulties, the most common diagnosis in offspring of depressed parents appears to be depression, especially among childhood-onset affected parents (Orvaschel, 1990).

Why would offspring of parents with a history of depression be at risk for depression themselves? One reason is that parents play a salient role in the development of children’s emotion regulation (ER). Parents influence ER through their overall caregiving style, such as being accessible versus ignoring, responsive and contingent versus insensitive, reciprocal versus unilateral, cooperative or helpful versus intrusive and controlling. They also are responsible for regulating the social environment in which arousal occurs for the child, particularly among young children. In addition, parents provide explicit training in how children should regulate their emotional expression via their direct responses to the child’s behavior and indirectly through modeling (Eisenberg, Fabes, & Murphy, 1996; Gottman, Katz, & Hooven, 1996).

The present investigation offers the opportunity to examine maternal responsivity to children’s positive and negative emotions among a group of adults who were identified with clinically-diagnosable depression prior to the age of 14, employing a unifying construct by which parenting could be compared among children of different developmental levels: emotion regulation (ER).
The term ER has been defined (Thompson, 1993) and continues to be defined in various ways (Campos, Frankel, & Camras, 2004; Cole, Martin & Dennis, 2004). For the purposes of the present study, emotion and ER are viewed as biologically-based reactions that coordinate biologically and psychologically adaptive responding to stimuli (Gross & Munoz, 1995; Thompson, 1994).

Importantly, the functionalist view of emotionality explicitly suggests that emotional experience is defined through transactions with the environment (Thompson, 1993). Once an emotion has been triggered, human beings display “variations in the intensity, persistence, modulation, onset and rise time, range, and lability of and recovery from emotion responses” (Thompson, 1994). The foregoing “dynamic” features are the response parameters that are affected by ER processes. Therefore, ER has been defined as involving “the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal feature, to accomplish one’s goals” and to facilitate functioning (Calkins, 1994; Saarni, 1999). Implicit in this definition is the likelihood of maintaining and inhibiting or subduing emotional arousal.

How do human beings acquire ER strategies and what are the sources of individual differences in this domain? Calkins (1994) has specified two domains of influence: (1) internal sources including innate neuroregulatory systems, behavioral traits, and cognitive components of the child; and (2) external sources including interactive caregiving styles of parents, regulation of the social environment by others in which arousal occurs, and explicit training received from caregivers. Early on, the development of emotion self-regulation is largely a function of the infant’s capacity to utilize regulatory strategies (e.g., gaze aversion) and the parent’s ability to recognize and meet the offspring’s regulatory needs (e.g., providing physical comfort when the infant is in distress). Both internal and external sources of the child’s ER are believed to influence one another (Calkins, 1994). Infant neuroregulatory or biologic systems and behavioral traits are influenced by caregiving styles and training, and caregiving styles are influenced by the child’s emotional reactivity to situations. With maturity, cognition as a factor in ER increasingly comes into play with development, as the
child acquires a schema about how people will behave under particular circumstances; however, skill acquisition continues to rely on the reciprocal interplay between internal and external factors (Calkins, 1994).

A focus on how children regulate their emotions has been used successfully to examine normative emotional development (Cole, 1986; Kagan, Reznick, & Snidman, 1988), but has less often been applied to clinical populations (with exceptions noted; Radke-Yarrow, 1998; Shipman & Zeman, 2001), or specifically as a proposed mechanism responsible for continuity in the intergenerational transmission of childhood-onset depression (COD). To begin to address some of these critical gaps in understanding, we examined differences in maternal responsivity to children's expressed emotion between two samples of families, one selected on the basis of a history of COD in the mother, and a comparison group in which no childhood psychiatric illness of the mother was evident (NCOD).

**Parenting and Emotion Regulation**

The notion that parents perform a vital function in the formation of children's regulation of affect is not new. Many theories of child development have emphasized the importance of parental input, yet relatively few have examined this issue empirically beyond the preschool period (Goodman & Gotlib, 1999; Saarni, 1999; Shipman & Zeman, 2001). Importantly, the nature of the role parents play in the process also changes with the developmental level of the child. For example, during the beginning of infancy, parents directly regulate distress by soothing with touch, changing their facial expression, altering the immediate environment, or gratifying the infant's needs (Kopp, 1989). However, with children's greater cognitive and emotional development, coupled with their increasing physical mobility, more diverse approaches become warranted (Shaw, Bell, & Giliom, 2000). Parents are required to set limits on the display of emotional dysregulation for fear of injury to the child, siblings, pets, or valuable and fragile objects. Similarly, increasing maturity demands for the
child may cause parents to restrict nondestructive displays of emotion (e.g., crying may be permitted at ages 2-3 but not at age 5 for the same incident).

We propose that one of the mechanisms whereby COD parents increase the risk of intergenerational transmission of depression in their offspring is by not being able to properly respond effectively to their children’s emotion experiences in response to provoking stimuli. COD mothers would be expected to be particularly likely to show impairments in responding to children’s expression of emotion, more so than mothers with a less chronic course or more recent onset of depression, because of earlier and more chronic disruptions in their own socialization process. Evidence suggests that compared to normal children, COD youth show maladaptive outcomes in multiple social domains, including higher rates of conflict with siblings and parents and more interpersonal problems with peers (Goodyer, Herbert, Secher, & Pearson, 1997; Goodman & Gotlib, 1999; Kovacs & Goldston, 1991). Thus, COD mothers’ inappropriate regulatory response to the emotionally aroused young offspring is presumed to reflect their own earlier developmental problems in multiple domains of ER and response styles (such as being critical, unresponsive) that evolved in conjunction with their history of depressive illness. For example, COD mothers own difficulties in utilizing social agents and cognitive processes in managing their own ER should be evident when faced with an offspring who is displaying emotional arousal such as distress to a fearful object. When the offspring becomes upset, the COD parent would be less likely to offer physical comfort, verbal reassurance, or helpful behavioral displays that could assist the child to reduce and contain the negative emotions. A similar lack of responsivity would also be expected in response to the child’s displays of positive affect. Thus, because of impairments in the development of social relationship skills that characterize COD mothers, we hypothesize that they would be less contingently responsive to their offspring’s expression of distress and positivity than NCOD mothers.

To further specify the linkages between COD status on parenting practices, we also examined the role of two other factors that might influence the relation between a history of COD and
caregiving: chronicity of maternal depression and child gender. First, regarding the COD status of mothers, its relations to child outcome may be due to the duration and chronicity of impairment. As there was variability in the frequency of life-time depressive episodes among COD probands following initial diagnosis of MDD or Dysthymia, we also examined their impact on responsivity to child emotions. If the magnitude of relations with parenting were found to be comparable, COD status may only represent a marker for chronicity of impairment. Alternatively, if COD status was found to be a better discriminator of maternal responsivity than chronicity, the results would suggest that COD status might be a unique risk factor for impairment in later parenting.

Second, there is also evidence to suggest that mothers in the COD group may respond differently to the negative and positive emotions expressed by sons versus daughters. For instance, several studies using normative samples indicate that parents may be more contingently responsive to the expression of both anger and sadness for girls rather than boys (Block, 1983; Eisenberg et al., 1998; Fuchs & Thelen, 1988) because of their heightened concern for girls to develop more relationship-oriented strategies for regulating emotions (Eisenberg et al., 1998; Hops, Biglan, Sherman, & Arthur, 1987; Nolen-Hoeksema & Girgus, 1994; Sheeber, Davis, & Hops, 2002). There is also the possibility that mothers may believe they have a less intuitive feel for responding to boys than girls because of the gender difference, a notion that was validated in Hetherington, Cox, and Cox’s (1979) study of post-divorce parent-child relationships. This gender difference in responding to emotions would be expected to be amplified among COD mothers because of their own deficits in ER, and who thus might be more sensitized to responding to boys’ expression of negative and positive emotions compared to mothers without a history of clinical depression.

In summary, we hypothesized that COD mothers would show less responsivity to children’s expression of distress and positivity than NCOD mothers. As an alternative explanation for relations that might emerge between COD status and parenting, we also investigated the potential role of chronicity of depression. Finally, we examined the potential moderating influence of child gender on
the relation between COD status and maternal responsivity, positing that COD mothers would be less likely to respond contingently to their son’s expression of emotion than they would be for daughters.

Methods

Subjects

In the present article, we report on 94 young adult female subjects and their 121 offspring. The overall sample includes: a) 38 adults with childhood-onset depression (COD), with a mean age of onset of first depression of 9.95 years (S.D.=2.71) and 65 COD offspring (52% male) with a mean age of 3.9 years. Nineteen (50%) of the COD probands participated with more than one offspring (12 with two children, six with three children, and one with four children); b) 56 normal controls (NCOD) and 56 offspring (52% male ) with a mean age of 3.8 years. For the 38 COD Proband mothers, early-onset diagnoses included major depressive disorder (MDD; n = 21, 56%), dysthymic disorder (DD; n = 9, 24%), and MDD with DD (n = 8, 21%). Through adolescence and adulthood, COD probands have also experienced a range of other disorders, including anxiety spectrum disorder (79%), conduct disorder (42%), and alcohol abuse or dependence (55%).

Program Project Subject Recruitment and Diagnostic Determination

The subjects for this study were enrolled in a larger Program Project examining the genetic, psychophysiological, and environmental risk factors for COD. Entry criteria for the Project included: having had childhood-onset mood disorder (probands), or no psychiatric illness (normal controls), no pre-existing major medical disorders, and no evidence of mental retardation. Childhood-onset mood disorder was operationally defined as a DSM-based psychiatric diagnosis of depression (major depressive and/or dysthymic disorder) by age 14. To be enrolled as a control, the subject had to have a lifetime-history free of major psychiatric disorder. Individuals with episodes of highly circumscribed conditions without functional impairment (e.g., brief period of marijuana use in college) were deemed eligible as controls.
The Psychiatric Evaluation Core of the Program Project, staffed by highly experienced and trained professional-level clinical evaluators, and independent best-estimate psychiatrists, was responsible for all psychiatric assessments and verified cases' life-time diagnostic status. Best-estimate consensus diagnostic procedures generally followed the steps outlined by Maziade et al (1992). All psychiatric assessments involved the use of semi-structured clinical interviews. Diagnoses were derived according to rules specified in the various versions of the DSM (DSM-III, DSM-IV; American Psychiatric Association, 1980; 1994).

Three main strategies were used to recruit adult subjects with childhood-onset mood disorder (CODs), namely: a) accessing individuals who had participated in clinical research studies during their juvenile years and had research records supporting the required diagnoses, b) accessing adult mental health clinics specializing in the treatment of mood disorders, and c) advertising via pamphlets, newspapers, or presentations directed at the general community or special interest groups.

Probands in the present sample, who had previously participated in research projects, include 14 (37%) cases that had been enrolled in a longitudinal naturalistic follow-up of childhood onset depression (Kovacs et al., 1984; 1997). These cases had multiple psychiatric assessments over the course of up to 20 years, using the semi-structured Interview Schedule for Children and Adolescents (ISCA) or its version for young adults (Sherrill & Kovacs, 2000). During childhood and young adulthood, a second informant (parent or partner) was also interviewed, whenever possible to corroborate the proband's report, and all relevant medical and related medical records were obtained. Cases were diagnosed via the DSM-III (American Psychiatric Association, 1980), and subjected to repeated consensus diagnostic reviews.

A further 19 (50%) of probands had been participants during their youth in other studies of shorter durations, including studies of juvenile anxiety disorder, adolescent depression and suicide, as well as conduct and associated disorders. Previous research diagnoses of these subjects,
obtained via the ISCA (Sherrill & Kovacs, 2000), some version of the K-SADS (Chambers et al., 1985), or the DISC (Costello et al., 1985), and corresponding ages of onset, were made available to the Project. However, owing to the length of time since their prior study participation, and to verify eligibility, potential probands were reassessed using the Structured Clinical Interview for DSM-IV Axis I modified to include some childhood-onset and selected Axis II disorders (SCID, First et al., 1995). SCID assessments also entailed an interview with a second informant about the proband. Two senior psychiatrists, blind to the subject's prior study classification, independently reviewed the SCID results and supporting records before arriving at the final consensus diagnoses.

The remaining five probands with childhood-onset mood disorder, recruited from clinics or the community, also were assessed via the SCID, using a direct interview, and an interview with a second informant. To verify childhood-onset of the diagnosis of interest, these cases were enrolled in the Project only if they had pediatric, medical, psychiatric, or related records of treatment seeking for emotional or behavioral problems, which specified the pertinent mood symptoms.

Controls were recruited via one of three ways, namely by: a) accessing cases who had participated in studies during their childhood or adolescence as normal controls ($n = 1$), b) using the Cole directory of households in neighborhoods comparable in socioeconomic status to the proband group and soliciting participation ($n = 18$), and c) advertising for volunteers in the general community or through special community programs led to the recruitment of the remaining ($n = 37$). The later source included a local Women, Infants, and Children (WIC) Center, providing nutritional services to income-eligible families of children age 5 and under. A pre-screening procedure was used to rule-out individuals unlikely to qualify (e.g. prior mental health treatment or hospitalization). Those who passed the screen received the SCID assessments and best-estimate consensus diagnostic procedure, the same as used for proband subjects described above.

*Intergenerational Study of Child Development Eligibility*

COD and NCOD subjects from the Program Project cohort with offspring between the
ages of 1 and 8 were asked to participate in the intergenerational study. In control families, only one child per family was selected; when multiple offspring were available, the one who came closest to matching the age and/or gender of COD offspring were selected. Efforts were made to ensure the NCOD group was similar to the COD on sociodemographic characteristics, child age, and gender. Chi-square tests failed to reveal significant group differences for child’s age (63.6% of COD age 1 to 4 versus 64.2% of NCOD offspring), gender (COD 53% male and NCOD 52% male), or child ethnicity (26% COD are African American, Hispanic, or biracial versus 34% of the NCOD group). However, NCOD mothers tended to be more highly educated than COD mothers (60% of the NCOD mothers had completed some college versus 42% of the COD mothers) than COD mothers, $X^2 (1, 121) = 7.96, (p = .093; two-tailed test).

**Procedure**

Qualifying families were seen around the time of their offspring's birthday annually during the course of 4 years. Criterion for seeing children around the time of their birthdays was more conservative for infants and toddlers (e.g., children age 1 were seen within 2 weeks of their birthdays). Mothers and target children were seen at the laboratory for 2 to 2.5 hours, with assessments for younger children taking less time than those for older children. Observational procedures were videotaped for later coding. All visits began with the child playing with toys on his/her own while the mothers completed questionnaires with the examiner in the same room. Following a brief clean-up task (the clean-up was not administered to 1 year olds), the ER tasks were administered, for which the mother and child were given instructions and reminder cards to ensure they proceeded from one task to the next in the appropriate order. These tasks were followed by a 10-minute break. During the second half of the assessment, age-specific observational tasks were administered (e.g., for preschoolers, cookie or waiting tasks), followed by having mothers complete questionnaires about child behavior, their own adjustment and
family support, and parenting, including the Emotions as a Child Questionnaire (ECQ). The current report focuses on observational data from the emotion regulation tasks and the ECQ.

**Measures**

*Interview Schedule for Children and Adolescents (ISCA, Sherrill & Kovacs, 2000).* The ISCA is a semi-structured interview, that was developed with the express purpose of making diagnoses according to DSM-III criteria. It was administered to the original cohort of COD probands and their parents separately by the same interviewer when probands were between 8 and 13 years old (see Kovacs et al., 1984) to establish presence of mood and other psychopathological disorders. Interviewers were Master’s or Ph.D. level mental health professionals who had received extensive training on the ISCA. Diagnoses were derived based on ISCA symptom ratings and assigned by consensus among the interviewers according to DSM-III criteria. Interrater reliabilities have been shown to be satisfactory for ISCA, with a mean intraclass correlation of .89 for psychopathologic symptoms; the reliability, construct availability and predictive validity of resultant diagnoses has also been established (Sherrill & Kovacs, 2000).

*Structured Clinical Interview for DSM-IV Patient Version (SCID).* The SCID (First et al., 1995) was used to assess lifetime psychiatric disorders among prospective self-referred COD probands and NCOD probands. The SCID is a semi-structured, clinician-administered diagnostic interview that includes modules corresponding to major DSM psychiatric classes. In order to establish the presence/absence of early-onset diagnoses in a manner that was comparable to that used with the original COD proband cohort, we expanded and adapted the SCID to include criteria for selected childhood diagnoses (separation anxiety, overanxious, attention deficit, and conduct disorder) and DSM-III (APA, 1980) current and lifetime criteria for affective disorders. The modified SCID was first administered to the prospective proband and then separately administered to a second informant, usually a parent or other close family
member, who provided collateral information on the potential proband. This procedure was also followed to rule out childhood-onset disorders among NCOD mothers. Ultimate study eligibility was based on final consensus diagnoses determined by two independent senior psychiatrists, who reviewed data from the first and second informant SCID administration and any extant treatment records.

**Emotion Regulation (ER) Tasks:** At ages 1-8, a series of 4-5 tasks were used to elicit both positive and negative emotion in the child. Each group of tasks was tailored to the developmental status of the child. For example, at age 1, the child and parent play for 1.5-2 minutes with the following stimuli: alligator boat, comeback wheel, curiosity box, tiger light that moves. At age 3, in which segments are increased to 2-3 minutes in length, ER tasks involve a dinosaur puzzle followed by an etch-a-sketch task, stack-n-pop and toss-a-cross games, and a wiggle ball. At age 5, the tasks include the game hungry hippo, marble works, a naming game (e.g., name things that fly), a shape sorter task, and a tractor treader. At ages six to eight, tasks used to elicit frustration included puzzles, mozaics, and naming tasks aimed at developmental levels slightly beyond the child abilities; tasks to elicit fear and anxiety included giving a wiggle ball (ages 3 and 6); and tasks designed to elicit positivity including having the mother and child play with a three-foot-tall tractor treader, a pop-n-catch game, and a big ball (three feet tall). While most tasks were designed to elicit positive affect in children, at least one of the tasks within each group was selected to provide a provocative or scary element (e.g., tiger light at age 1, wiggle ball that actually gyrates and moves at age 3). Negative emotion generally took the form of distress or fear in response to one of the more highly stimulating toys (e.g., wiggle ball).

To evaluate whether the tasks were successful in eliciting both positive and negative emotion, a series of paired sample tests were computed comparing the mean levels of positive or negative emotion demonstrated by children with ER tasks that were neutral in valence and not expected to elicit either positive or negative emotion at the same age (e.g., puzzles, naming
Maternal Depression and Parenting

For example, for negative emotion at age 3, a comparison was made between negative emotion shown during exposure to a vibrating wiggle ball (negative affect expected) and a dinosaur puzzle (low negative affect expected). Similarly, for positive affect at age 2, mean scores on positive affect were compared between a Tickle-Me-Elmo toy (positive) and a Bert and Ernie puzzle (neutral). For positive affect, differences were seen in the predicted direction at ages one through eight (i.e., for all tests \( p < .05 \), one-tailed tests). For negative affect, differences were found at only ages one through six, with nonsignificant trends in the expected direction at ages 7 and 8. For older children, decreased expression of negative emotion limited variability to detect such differences.

Maternal and child behavior were subsequently coded from videotapes based on the mother's and child's responses to the other's expression of positive or negative emotion (e.g., mother smiles after child laughs, mother offers help after child expresses frustration) within 10-second intervals. The following two interval-based codes were chosen to reflect contingent maternal responsivity to child expression of emotion: 1) responsivity to child distress and 2) responsivity to child positive affect. For both maternal responsivity to child distress and positivity, coders rated whether the maternal response was appropriate or not. For distress, appropriateness was considered to be behavior that soothed or downregulated the child's distress versus doing nothing or amplifying distress. For child positivity, an appropriate response was one that fueled the child's positivity, generally by reciprocating positive feedback. The respective Kappas for responsivity to distress and contingent positivity were .67 and .60. These levels are consistent with expectations for kappas established by Mitchell (1979) for observational codes, in the .5-.6 range. For both interval codes, the final code was a ratio of the total number of times the mother showed an appropriate response (i.e., numerator) divided by the total number of times the child expressed distressful or positive behavior. For example, if the mother reciprocated positivity in five of ten intervals in which the child displayed it, the score
would be .5. To supplement interval-based scores, four-point global ratings were also scored based on mother's contingent responsiveness to child distress and positivity, respectively. The global ratings were included to account for critical events that occurred across tasks, a limitation of the interval method. Weighted kappas for the global codes were .69 for contingent responsiveness to distress and .53 for contingent responsiveness to positivity. The correlation for the interval and global composite of maternal responsivity to child distress was .73 ($p < .001$) and for contingent positivity .77 ($p < .001$). To account for both interval and global ratings in the final score, a composite was generated by standardizing both scores, and then summing and averaging them.

As might be anticipated with such a wide age span, age of child was expected to be related to maternal parenting behavior. Pearson correlation coefficients were computed between child age and maternal variables to examine this issue. In fact, responsivity to child positivity ($r = - .36, p < .001$) was significantly related to child's age. Mothers were less likely to respond to child positivity as their children grew.

*Emotions as a Child Questionnaire* (ECQ, Magai, 1997; Klimes-Dougan, Brand, & Garside, 2001). This parent-report instrument was adapted from Magai's (1997) original child-report version of the instrument to assess maternal responses to children's expression of sadness, anger, and fear. Respondents are asked to rate how typical their responses to children's expression of emotion are on a 5-point likert scale, with 3 items for each type of response (i.e., three ‘sad’ items for ‘Neglect.’). Responses are specific for each emotion, but are grouped in terms of such responses as neglect (e.g., ‘usually didn't notice’, ‘I didn't respond’) or magnification (e.g., for afraid ‘told my child not to be a fraidy cat', for anger ‘got angry with my child’) of the child's expression of emotion. For the present analyses, three types of maternal responses were used in relation to child anger, sadness, and feeling afraid: Magnify, Neglect, and Reward. Reward includes parental behaviors that encourage the
expression of emotion (e.g., providing comfort, showing empathy). Cronbach alphas for the three response factors were .79 for Reward, .68 for Neglect, and .79 for Magnify. As the ECA variables represented composites of items from nominal scales, they were rescaled as zero-one (binomial) response variables to provide a more acceptable fit (i.e., increase reliability and accuracy) for the binomial likelihood function.

Results

Our central aim was to examine differences between COD and NCOD groups on observational measures and maternal reports of their own responses to children's expression of emotions. Even though the number of siblings in the study was unrelated to any of the dependent variables (all $p$-values exceeded .10), random effects were a concern because the COD group included more than one child per family. To account for the correlation among siblings in relation to maternal responsiveness (i.e., shared variance), family membership was included as a clustering variable in a random effects model. Thus, a series of one-way ANCOVAs were conducted using maternal diagnostic status as the grouping variable and shared variance among siblings in the COD group as a covariate. Analyses were completed with the statistical package R (R Development Core Team, 2003), which has the ability to test for random effects. Because mothers in the NCOD group had slightly higher levels of education, maternal education was also considered for use as a covariate. However, because it was subsequently found to be unrelated to any of the dependent variables, it was not included in any subsequent analyses.

Before examining group differences on maternal responsivity to children's expression of emotion, we explored whether children from the COD and NCOD groups displayed comparable levels of negative and positive affect during the ER tasks. As described above, in both cases one-way ANCOVAs were conducted using maternal diagnostic status as the grouping variable and shared variance among siblings in the COD group as a covariate. COD children were not
found to display more negative affect than NCOD children \( (F(2,107)=1.02, p > .10) \), but were found to show significantly less positive affect than NCOD children \( (F(2,107) = 3.82, p < .05) \). Although these data suggest that COD children express less positive affect than NCOD offspring during observational tasks, as the analyses below examine the proportion of parental responsivity to children’s individual expressions of positive emotion, they were not presumed to influence these contingently-based variables of parental responsivity.

Results for the ANCOVA analyses involving observations of maternal behavior and group status (and sibling effects as a covariate) are presented in Table 1. Significance was determined using p-values from one-tailed tests because of a priori hypotheses about the direction of effects. In the first comparison, maternal responsivity to child distress was found to be related to group status. Based on the composite of observed interval and global ratings, NCOD mothers were more likely to respond to child distress than COD mothers \( (p < .05)^1 \). However, maternal response to child positivity was not found to differ between groups.\(^2\) For the ECA variables, four of the nine comparisons were found to be significant, each in the expected direction. While COD and NCOD mothers were not found to differ in the amount they magnified children’s expression of sadness, fear, or anger, COD mothers were more likely to be neglectful in responding to offspring’s expression of sadness and fear \( (p < .05 \text{ for both}) \). Further, COD mothers were less likely than NCOD mothers to encourage the expression of their child’s sadness and fear \( (p < .05 \text{ for both}) \).

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Insert Table 1 about here

\(^1\)Raw scores were .59 (sd = .29) for COD versus .69 (sd = .33) for NCOD for the interval ratio variable of responsiveness to distress, and 3.13 (sd = .89) for COD versus 3.62 (sd = .57) for NCOD for the global rating of responsiveness to distress.

\(^2\)Raw scores were .50 (sd = .29) for COD versus .53 (sd = .25) for the interval ratio variables of responsiveness to positivity, and 2.43 (sd = .94) for COD versus 3.00 (sd = 1.00) for NCOd for the global rating of responsiveness to positivity.
To examine the impact of chronicity of depression on maternal responsiveness to children's emotion, the ER and ECA variables were regressed, in linear models, on the number of lifetime maternal MDD episodes (range: 1-4). Again, shared variance among siblings was entered as a covariate in each model. Linear regression was chosen over ANCOVAs for this analysis because the number of depressive episodes experienced by mothers within the COD group varied from one to four, allowing for a linear assessment of how maternal depressive episodes and the dependent variables relate. Results of the linear regressions revealed that neither one of the observed ER parenting variables or any of the nine ECA parenting variables were associated with number of episodes of MDD (all p-values exceeded .05).

A final goal was to examine the potential moderating role of child gender in relation to parental responsivity to children's expression of emotion. This was carried out by adding an interaction term for COD status and child gender to the ANCOVA models described earlier. As displayed in Table 2, for the ER variables, maternal responsivity to child positivity was found to be affected by an interaction between maternal diagnostic status and child gender ($p < .05$). COD mothers were more likely to respond to positive emotion from their daughters whereas NCOD mothers were more likely to demonstrate contingent positive affect with their sons. No such interaction effects were found for responsiveness to negative emotions, either for the observed ER variable or any of the ECA factors.

Discussion

This study sought to advance our understanding of how parents with childhood-onset depression may increase the risk of psychopathology in their offspring by the manner in which
they respond to their children's expression of negative and positive emotions. We began by examining mean levels of positive and negative emotionality between COD and NCOD children, which indicated higher levels of positivity for NCOD children but comparable levels of negative affect. We hypothesized that in response to their offspring's display of negative emotional arousal, COD mothers would be less likely to offer appropriate physical or verbal reassurance that could assist in modulating or reducing their child's negative emotions. We also anticipated that COD mothers would be less likely to reciprocate child's expression of positive affect. In both cases it was expected that these findings would be more pronounced for the parenting of boys rather than girls. The results were consistent with only some of these expectations.

Overall, in relation to maternal responses to boys' and girls' expression of emotion, the observational data indicated that COD mothers showed less responsivity to their child's distress, but did not differ from NCOD mothers in responding to their expressions of positive behavior. However, when child's gender was taken into consideration, COD mothers were more likely to respond contingently to daughter's but not to son's expression of positive affect, while in NCOD families mothers were more likely to respond to son's positive affect. When maternal self-report of their own emotional reactions were examined, COD mothers were found to have been more likely to endorse neglecting expressions of sadness or fear in their children, and provide less support for expressions of sadness and fear.

The pattern of results supports the notion that the parenting of COD mothers may play a salient role in the intergenerational continuity of psychopathology. Previous research on children of concurrently depressed mothers has highlighted differences between depressed and nondepressed parents on a number of parenting behaviors. For example, Radke-Yarrow (1998), Zahn-Waxler et al. (1990) and others (e.g., see reviews by Downey & Coyne, 1990; Goodman & Brumley, 1990; Goodman & Gotlib, 1999) have noted that depressed individuals are typically negative, critical, unresponsive, helpless, passive and less positive toward others,
including their offspring. In prior studies, such caregiving practices have been consistently linked to the development of behavior problems during both the preschool (Shaw et al., 1998) and school-age periods (Renken et al., 1989), including samples with depressed mothers (Goodman & Gotlib, 1999). That COD status was a more reliable predictor of maternal responsivity than chronicity of depression is also consistent with the notion that the timing of onset of depression plays a meaningful role in relation to later parenting, and perhaps continuity in the intergenerational transmission of psychopathology. Presumably, this is due to COD proband’s earlier and more chronic disruptions in their socialization process rather than number of depressive episodes experienced from late childhood through adulthood.

Many of the current results corroborate the findings of previous research on parenting in depressed mothers, noting their tendency to display less overall positivity and sociability and greater negativity towards the child. However, the focus on mother’s contingent responses to their children’s expression of positive and negative emotions in a sample at-risk for early psychopathology extends work on the development of ER in normative samples (Cole, 1986; Kagan, Reznick, & Snidman., 1988), and specifically offspring of COD probands (Radke-Yarrow, 1998). The group differences found in our observational ratings suggest that COD mothers may be less sensitive to or less knowledgeable about cues of offspring’s emotional distress, as indicated by their significantly lower responsivity to their children’s distressed behavior during observation and maternal reports of greater neglect in response to their children’s expression of sadness and fear. Theoretically, low levels of responsivity could fuel both internalizing and externalizing symptoms. From the perspective of attachment theory (Bowlby, 1980), children whose expression of sadness and fear are met with neglect would be expected to develop internal working models of mistrust in others, and eventually, low self-worth, leading to a pathway of self-doubt, anxiety, and in some cases, depression.
The gender difference regarding COD mother's less contingent responsivity to boys' expression of positive emotion is consistent with literature indicating that mothers may be more contingently responsive to the expression of emotion of girls (Block, 1983; Eisenberg et al., 1998; Hops et al., 1987; Nolen-Hoeksema et al., 1994). It is also consistent with the literature on boys' greater vulnerability to environmental stressors in early childhood, particularly regarding the effects of caregiving environments on early conduct problems (Keenan & Shaw, 1997; Shaw et al., 1998). As maternal depression has been consistently associated with child externalizing symptoms, particularly in boys, differences in maternal responsivity to boys' expression of positivity may be one mechanism by which cycles of negative coercion are more frequent among mother-son dyads (Patterson, 1982). Alternatively, as most past research in this area has focused on responding to children's expression of negative emotions (e.g., sadness), it is unclear how generalizable the current result is to more normative populations. Perhaps it is only within the context of COD mother's socialization experience that such a gender difference would be evident in responding to the positive emotions of offspring (Hetherington et al., 1979). It should also be noted that although this gender by diagnostic status interaction was identified, it was the only one among 11 tests computed. It should also be pointed out that the 10 nonsignificant interactions all examined negative rather than positive emotions. Caution is still warranted in evaluating the meaningfulness of this finding until it is replicated or disconfirmed in future studies.

The results also need to be interpreted in light of the significant differences between COD and NCOD children in rates of expressed positive emotion. Although our measurement of maternal responsivity should not have been heavily influenced by this difference based on the use of a ratio of maternal responsivity to child positivity, it is possible that over time such differences in expressed positivity would be associated with lower levels of maternal engagement and expressed positive affect for COD mothers. Alternatively, differences in levels
of positive affect among COD children could be related to living with a COD parent, who would be expected to show higher levels of negative affect and lower levels of positivity.

Overall the study provides data consistent with the notion that COD mothers differ from NCOD mothers in the way they respond to their children’s expression of emotion compared to NCOD parents. These differences may be involved in the increased risk of depression in children of depressed mothers. Other factors may also contribute to this risk. In particular genetic risk factors may also be transmitted to the children of depressed mothers. We are currently collecting genetic data on probands and their first-degree relatives to examine genetic influence in the development of COD and related disorders. In addition, we are examining associations between the parenting and parent’s psychophysiological reactivity (e.g., EEG right-frontal asymmetry) to link genetic risk with individual differences in psychophysiological reactivity and parent-child interaction.

However, the results need to be qualified by several methodological limitations. First, the sample size is relatively small, limiting our ability to detect effect sizes that are modest. Second, although differences in behavior were uncovered between COD and NCOD mothers, the results could be generic to other forms of parental psychopathology, including other forms of childhood-onset psychopathology (e.g., anxiety disorder). Future research needs to rule out this possibility before concluding that the current differences are specific or limited to COD populations. Third, the age span of the offspring was rather wide, ranging from infancy to middle childhood. While age effects of parenting were not evident, perhaps because coders were advised to take into consideration the age of the child when making their global ratings, different results may have been obtained in a more homogeneous age group (e.g., toddlers or preschoolers). Unfortunately, sample sizes for COD and NCOD groups were not sufficient to test relations in this manner. While we statistically controlled for the effects of having multiple siblings in the COD group, it would have been preferable to use only one child from each family...
in the COD group. Finally, although we were largely successful in eliciting both positive and negative emotions among children in our laboratory-based procedures, it was clearly more challenging to elicit negative emotion among older children. It would have been preferable to have had a greater number of procedures that elicited negative emotion for older children rather than only one per visit. Despite the limited variability in the expression of emotion for older children, we were still able to find between-group differences in mother’s response to offspring’s negative emotions.

If replicated, these results may have significant ramifications for understanding the development of psychopathology in early childhood, and attempts to provide primary or secondary prevention before symptomatology surpasses a clinical threshold. The differences COD mothers demonstrated in responding to their children’s expression of emotion may account for why their offspring are vulnerable to internalizing and/or externalizing problems. Efforts to prevent and treat the development of different types of childhood clinical disorders may have underestimated the similarity of antecedent conditions such diverse disorders share (e.g., conduct problems and depression). Specifically, parent’s inability to respond to their children’s emotions may lead to a wide range of maladaptive outcomes, moderated by child characteristics and the family’s ecological context. This notion is consistent with the concept of multifinality, which suggests the limited predictive validity of any one component of an organism or its environment (Cicchetti & Rogosch, 1996).

In summary, the findings offer initial evidence that parental responsiveness to children’s expression of emotions differs between COD and NCOD mothers, results that are consistent with models that emphasize emotion regulation. The results support the import of ER-related parenting factors and further research in this area, particularly studies that follow the effects of such practices on child adjustment over time and account for parenting responses to both negative and positive emotion.
References


Table 1: Means and standard deviations for Emotion Regulation (ER) and Emotions as a Child Questionnaire (ECQ) variables for COD and NCOD groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>COD</th>
<th>NCOD</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>(N = 65)</td>
<td>(N = 56)</td>
</tr>
<tr>
<td>ER: Contingent positive affect*</td>
<td>-.035 (1.08)</td>
<td>.269 (.965)</td>
</tr>
<tr>
<td>ER: Responsivity to distress*</td>
<td>-.168 (a) (.987)</td>
<td>.169 (a) (.960)</td>
</tr>
<tr>
<td>ECQ: Reward Sad</td>
<td>4.14 (b) (.879)</td>
<td>4.48 (b) (.770)</td>
</tr>
<tr>
<td>ECQ: Reward Angry</td>
<td>3.95 (1.844)</td>
<td>4.23 (.905)</td>
</tr>
<tr>
<td>ECQ: Reward Afraid</td>
<td>4.18 (c) (.819)</td>
<td>4.52 (c) (.678)</td>
</tr>
<tr>
<td>ECQ: Neglect Sad</td>
<td>1.80 (d) (.562)</td>
<td>1.63 (d) (.488)</td>
</tr>
<tr>
<td>ECQ: Neglect Angry</td>
<td>1.81 (.442)</td>
<td>1.71 (.481)</td>
</tr>
<tr>
<td>ECQ: Neglect Afraid</td>
<td>1.72 (e) (.449)</td>
<td>1.57 (e) (.437)</td>
</tr>
<tr>
<td>ECQ: Magnify Sad</td>
<td>1.95 (.787)</td>
<td>1.81 (.818)</td>
</tr>
<tr>
<td>ECQ: Magnify Angry</td>
<td>1.87 (.934)</td>
<td>1.60 (.655)</td>
</tr>
<tr>
<td>ECQ: Magnify Afraid</td>
<td>1.48 (.670)</td>
<td>1.35 (.618)</td>
</tr>
</tbody>
</table>

Note. The values presented in this table represent means and standard deviations for the ER and ECQ variables prior to their transformations and do not account for correlations between siblings. Means that have the same subscripts significantly differed at the \(p < .05\) level after accounting for sibling effects.

* Means for the observed ER variables represent composites of the standardized global and interval ratio scores.
Table 2: Interactive models for Emotion Regulation (ER) variables and gender

<table>
<thead>
<tr>
<th></th>
<th>Contingent Positive Affect</th>
<th></th>
<th>Responsivity to Distress</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>(SE)</td>
<td>T</td>
<td>Coefficient</td>
</tr>
<tr>
<td>Maternal Diagnostic Status</td>
<td>-.11</td>
<td>(.32)</td>
<td>.11</td>
<td>-.13</td>
</tr>
<tr>
<td>Child Gender</td>
<td>.22</td>
<td>(.26)</td>
<td>.67</td>
<td>.18</td>
</tr>
<tr>
<td>Diagnostic Status X Child Gender</td>
<td>-.78</td>
<td>(.40)</td>
<td>3.89**</td>
<td>-.38</td>
</tr>
<tr>
<td>Random Effect</td>
<td>.01</td>
<td>(.17)</td>
<td>.01</td>
<td>-.29</td>
</tr>
</tbody>
</table>

N = 121; * p < .05.