

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

Daniel S. Shaw<sup>1</sup>, Joel Sherrill<sup>2</sup>, Drew Huffman<sup>1</sup>, Michael Schonberg<sup>3</sup>,  
Joella Lukon<sup>1</sup>, David Obrosky<sup>3</sup>, & Maria Kovacs<sup>3</sup>

Running head: Parenting risk factors of childhood-onset depression

<sup>1</sup>Department of Psychology, University of Pittsburgh

<sup>2</sup>National Institute of Mental Health

<sup>3</sup>Department of Psychiatry, University of Pittsburgh

Abstract

This study examined responsivity of mothers with childhood-onset depression (COD) in relation to children's overt expression of positive and negative emotion. It was hypothesized that COD and control mothers would differ in contingent responsivity to their children's expression of both positivity and different types of negative emotionality. 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

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

Research interest in the juvenile offspring of depressed adults 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). Offspring's elevated risk of psychopathology was repeatedly corroborated in subsequent work despite variability in the methods of evaluating psychiatric status of adult probands (Hammen et al., 1987; Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1984; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). More precise, diagnostically-oriented studies were then carried out to examine whether the offspring of adults with depressive illness were at heightened risk for depression and whether their risk status was affected by the timing and severity of the parent's depressive illness.

Regarding the specificity of psychopathology, maternal depression has been found to be associated with a host of maladaptive outcomes among school-age offspring, including, but not limited to, depression and its behavioral precursors (i.e., anxiety disorders) (see Downey & Coyne, 1990). Most of these studies have focused primarily on unipolar depression in the parent, with notable exceptions (Radke-Yarrow, 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 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).

In relation to the timing of parental depressive illness, 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). Using samples of older school-age children adolescents, offspring 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 et al., 1984).

Despite the increased risk of depressive illness for offspring of childhood-onset parents, relatively little is known about the underlying mechanisms associated with the intergenerational transmission of depression. In part, this is due to the practicality of prospectively following adults with childhood-onset depression (COD) from childhood to parenthood. Genetic and environmental influences (Kovacs & Devlin, 1998) and gene by environment interactions are likely implicated in the intergenerational transmission of depressive illness, but relatively little work has been conducted on the emergence of COD (Moffitt, Caspi, & Rutter, 2005). Among environmental contributors, parenting practices represent one possible mechanism by which genetic risk of depressive illness may be potentiated for offspring of COD parents. For example, for youth in the Dunedin longitudinal study, risk of severe antisocial behavior associated with the gene encoding the enzyme monoamine oxidase was only evident in the presence of parental maltreatment (Caspi et al., 2002). As caregiving practices have been demonstrated to be malleable, particularly in early childhood (Olds, 2002; Baydar, Reid, & Webster-Stratton et al., 2003), a focus on caregiving quality also has potential relevance for the prevention and treatment of COD.

How would parenting of COD adults potentially influence the course of depressive illness among their offspring? Parents play a salient role in the development of children's emotion regulation (ER) via socialization practices (Hill, Bush, & Rosa, 2003; Simons et al., 2002). Parents shape children's ER skills in many ways, through modeling, by communicating attitudes about the appropriateness of expression of specific emotions (e.g., anger, sadness), and by creating a context for the normative emotional climate of the home (Eisenberg, Cumberland, & Spinrad, 1998). In addition, recent work on socialization practices and children's development of ER skills has emphasized parent's immediate responses to children's expression of emotions (Denham, Bassett, & Wyatt, in press; Eisenberg, Fabes, & Murphy, 1996; Gottman, Katz, & Hooven, 1996). This line of research has examined contingent reactions to children's

expression of emotion primarily among normative samples (Denham et al., 2000; Denham & Kochanoff, 2002; Strayer & Roberts, 2004), with less work carried out among children at high risk for problem behavior (Sheeber, Allen, Davis, & Sorenson, 2000). The present study sought to extend our understanding of emotion socialization practices among a group of mothers who were identified with clinically-diagnosable depression prior to the age of 14. In conceptualizing potential group differences on caregiving practices between COD and control parents, we focused on how COD parents might respond differentially to offspring's expression of negative and positive emotions, and thereby influence the development of their offspring's ER skills.

### *Emotion Regulation*

The term emotion regulation (ER) has been defined in various ways (Campos, Frankel, & Camras, 2004; Cole, Martin & Dennis, 2004; Thompson, 1993). Controversy still exists in disentangling ER from related constructs, such as emotional reactivity and broader self-regulation (Derryberry & Rothbart, 1997; Forbes & Dahl, 2005), and discriminating factors involved in initiating versus regulating an emotion (e.g., Campos, Frankel, & Camras, 2004; Cole, Martin, & Dennis, 2004). However, for the purposes of the present study ER is viewed as a biologically-based reaction that coordinates 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, individuals 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. First, *internal sources* comprise innate neuroregulatory systems, behavioral traits, and cognitive components of the child. The second domain represents *external sources*, including 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 ER 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 distressed). 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 plays an increasingly more important role, 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). This framework also has been applied to clinical populations (Garber, Braafladt, & Weiss, 1995; Garber, Braafladt, & Zeman, 1991; Marshall & Fox, 2000; Radke-Yarrow, 1998; Sheeber et al., 2000; Shipman & Zeman, 2001), and specifically as a proposed mechanism responsible for continuity in the intergenerational transmission of COD. In fact, an increasing number of studies have shown that children's inability to regulate emotions adaptively is associated with internalizing symptoms among young children and with depressive symptoms and disorders among older children and adolescents (Eisenberg et al., 2001; Garber, Braafladt, & Weiss, 1995; Rubin, Coplan, Fox, & Calkins, 1995; Sheeber, Allen, Davis, & Sorensen, 2000; Silk, Steinberg, & Morris, 2003). To begin to address some of these critical gaps in understanding emotion regulation in children at risk for early-onset depression, we examined differences in maternal responsiveness

to children's expressed positive and negative emotions between two samples of families. The first sample was selected on the basis of a history of COD in the mother, with a comparison group in which no childhood psychiatric illness of the mother was evident (i.e., no childhood-onset depression - 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 socialization practices, yet relatively few have examined this issue beyond the preschool period (Goodman & Gotlib, 1999; Saarni, 1999; Sheeber et al., 2000; 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 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). With children's greater cognitive and emotional development, coupled with their increasing physical mobility, more diverse approaches become warranted (Shaw, Bell, & Gilliom, 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 7 in response to the same incident).

We propose that one of the mechanisms whereby COD parents increase the risk of intergenerational transmission of depression in their offspring would be not being able to properly respond 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 during childhood (Goodyer, Herbert, Secher, & Pearson, 1997; Kovacs & Goldston, 1991). 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 et al., 1997; Goodman & Gotlib, 1999; Kovacs & Goldston, 1991). COD mothers' inappropriate regulatory response to the emotionally aroused young offspring is hypothesized 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 (see Kovacs & Devlin, 1998 for review of literature linking deficits in ER to affective disorders in adults and children). Theoretically, COD mothers would more likely have a chronic history of difficulty in regulating their own emotions, especially within interpersonal contexts where they might have less control of utilizing strategies for downregulating expressions of emotion. Responding to offspring's expressions of emotion during episodes of depression and at times of less impairment would be hypothesized to be an ongoing challenge, particularly for mothers of young children, whose ER skills are less developed and more variable. As offspring's expressions of distress can often be emotionally charged, we would expect COD mothers to be less likely to offer physical comfort, verbal reassurance, or helpful behavioral displays that could assist the child to downregulate negative emotions. A similar lack of responsivity would also be expected in response to the child's displays of positive affect. Thus, because of hypothesized impairments in the development of social relationship skills that are thought to occur in COD mothers, we anticipated that COD mothers would be less contingently responsive to their offspring's expression of positive emotion than NCOD mothers.

To further specify the linkages between COD status on parenting practices, we also examined the role of three other factors that might influence the relation between a history of COD and caregiving: chronicity of maternal depression, current levels of maternal depressive symptoms, and child gender.

First, relationship between COD status and 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 mothers following initial diagnosis of Major Depressive Disorder (MDD) or Dysthymia, we also examined associations between chronicity of depression and responsivity to child emotions. If the

magnitude of relations with parenting was 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 responsiveness than chronicity, it would suggest that COD status might represent an independent risk factor for impairment in later parenting.

Second and relatedly, despite theory to suggest that having a history of *childhood-onset* depression would be associated with greater impairments in ER skills and less ability to respond contingently to offspring's expression of negative and positive emotions, it remains an empirical question as to whether such effects would be evident after accounting for mother's current level of depressive symptoms. For example, depressed mothers, with or without a COD onset, have been found to demonstrate deficits in ER (Bradley, 2000; Gross & Munoz, 1995). Thus, after computing initial analyses to examine associations between COD status and maternal responses to children's expressions of emotions, we accounted for the influence of mother's current depressive symptoms to see if effects of COD status on parenting, if present, would continue to be related to maternal responses of children's expressed emotion.

Third, 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 greater emphasis for girls to develop 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. These deficits were expected to sensitize COD mothers to responding to boys' expression of negative and positive emotions compared to mothers without a history of COD.

In summary, we hypothesized that COD mothers would show less responsivity to children's expression of distress and positivity than NCOD mothers. As alternative explanations for relations that might emerge between COD status and parenting, we also investigated the potential roles of chronicity of depression and mothers' current depressive symptoms. 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 versus their daughters expression of emotion.

## Methods

### *Recruitment and Diagnoses*

Children and their mothers were participants in a larger Program Project focusing on risk factors for childhood-onset mood disorder. Families with a history of COD were recruited into a larger program project through prior research studies or community advertisements. For adults with a history of COD, the two criteria for participation in the program project were the presence of a verifiable early-onset depressive disorder and willingness to participate in at least one of the component studies (genetics, psychophysiology, and parent-child interaction) in addition to diagnostic assessment. To participate in the current study, adult participants needed to be women enrolled in the parent-child interaction component of the program project, and have children in the age range of 1-9 years. Of the participants who completed the diagnostic assessment and met criteria for inclusion in the current study, all agreed to participate.

Families in the control group were recruited into the program project by accessing individuals who had participated in studies during their childhood or adolescence as "normal controls," soliciting participation using the Cole directory of households in neighborhoods comparable in socioeconomic status to the COD group (i.e, the Cole directory is a fee-based service that provides listings of individuals from different socioeconomic strata that have agreed to be contacted for potential participation in research studies), and advertising in the general community or through special community programs. For adults in

the NCOD group, criteria for participation in the program project included a lifetime history free of major psychiatric disorder. All adult participants were required to be free of major systemic medical disorders and without evidence of mental retardation.

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 and colleagues (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).

### *Participants*

In the present article, 94 young adult female subjects and their 121 offspring participated, including 38 COD mothers that met DSM criteria (DSM-III, DSM-IV; American Psychiatric Association, 1980; 1994) for major depressive and/or dysthymic disorder ( $n = 38$ ) by age 14. Informed consent was sought informally by the scheduling sub-corps at time of recruitment into the program project. During each assessment at our study, a trained research assistant obtained the mother's signature after reading over the consent and answering questions.

The COD mothers mean age of onset of first depression is 9.95 years ( $SD = 2.71$ ) and their 65 COD offspring (52% male) have a mean age of 3.9 years ( $SD = 2.21$ ). Nineteen (50%) of the COD mothers participated with more than one offspring (12 with two children, six with three children, and one with four children). These families were recruited through prior research studies or community advertisements. Fourteen (37%) cases had been enrolled in a longitudinal naturalistic follow-up of COD (Kovacs et al., 1984; Kovacs, Obrosky, Gatsonis, & Richards, 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). A further

19 (50%) COD mothers 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. The remaining five COD mothers were recruited from clinics or the community.

Because COD mothers differed greatly in the time they had spent involved as participants in the research study, we compared participants that had been part of the original Kovacs' study with other COD mothers on all 11 parenting variables. With the exception of one variable, no differences were found in maternal responsiveness to children's expressions of emotion. The one exception was for observed responsiveness to child distress, for which Kovacs' mothers showed significantly less responsiveness than COD mothers recruited more recently from other sources,  $t(1, 64) = -2.22, p < .05$ .

The normal control group (NCOD) consisted of 56 mothers and 56 offspring (52% male) with a mean age of 3.8 years ( $SD = 1.94$ ). Controls were recruited via one of three ways, other research studies ( $n = 19$ ), the general community ( $n=13$ ) or through special community programs ( $n = 24$ ). 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. Because NCODs differed in their recruiting source, we also compared the three groups on all 11 parenting variables. No differences emerged on any of the measures among those NCODs recruited from research studies, the Cole Directory, or through volunteers in the community (i.e., all  $p$  values  $> .05$ ).

#### *Intergenerational Study of Child Development Eligibility*

The current report includes data from families' initial assessment in the parent-child intergenerational study. Across groups, the mean age of children was 3.8 years ( $SD = 2.1$ , range = 1 to 8 years) and 52.5% of offspring were male. Fifty-seven per cent of children were European American with the remainder being African American (43%) or other ethnicity (4.5%), and the mean level of maternal education was completion of high school (range was from junior high to college degree). Efforts were made to ensure the NCOD group was similar to the COD group 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), child ethnicity (26% COD were African American, Hispanic, or biracial versus 34% of the NCOD group), or birth order (44.6% of COD versus 51.8% of NCOD were first born). There was a nonsignificant trend for NCOD mothers to be more highly educated than COD mothers (60% of the NCOD mothers had completed some college versus 42% of the 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. An additional informed consent was obtained from mothers prior to the beginning of initial laboratory assessments. Observational procedures were videotaped for later coding. For all children, the assessment began with a free-play episode for the child while 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 ER 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 mothers and their parents separately by the same interviewer when they were between 8 and 13 years old (see Kovacs et al., 1984) to establish presence of mood and other psychopathological disorders. 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 have 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. 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. In the current study, based on 50 cases, internal consistencies (i.e., kappas) for DSM-IV diagnosis of Major Depression and Dysthymia were .92 and .63, respectively.

*Beck Depression Inventory.* (BDI; Beck, Steer, & Garbin, 1988). The BDI is a well-established and widely used 21-item measure of depressive states that was administered to mothers during assessments to account for current depressive symptomatology. One score is generated for the BDI based on the sum of the most deviant responses endorsed for each item. Split-half reliability of the scale has been found to be high (.86 to .93).

*Emotion Regulation (ER) Tasks:* At ages 1- 8, a series of 4-5 tasks was selected based on their

probability to elicit positive (e.g., smiling, laughter) or negative (e.g., distress, fear, sadness) emotion in the child. Positive expressions of emotion included physical gestures (e.g., hugging or kissing mom), instances of smiling and laughing, and comments that were positive in both content and affect (e.g., “Good job, mom!”, “Cool!”, “This is fun!”, “I like this”, “Yeah!”, “Wow!”, “I did it!”). Negative emotion included expressed frustration with the task (e.g., “I can’t”, “I don’t know how”), asking the mother for help in a distressed or frustrated tone of voice, whining, nagging, complaining, or crying, and comments that were both negative in content and affect (e.g., “It’s scary”, “Stop it”, “This is boring”). 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 6 to 8, tasks included puzzles, mozaics, and naming tasks aimed at developmental levels slightly beyond the child abilities; tasks hypothesized 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 t-tests were computed comparing the mean levels of aggregates 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 tasks). 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 1 through 8 (i.e., for all tests  $p \leq .05$ , one-tailed tests). For negative affect, differences were found at only ages 1 through 6, 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 coding team was led by the lead author and included two doctoral students in clinical and developmental psychology, and two full-time staff members with undergraduate degrees in psychology. 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 was 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. In both cases appropriateness was based on maternal behavior that was expected to downregulate child's behavior rather than the child's actual response. In cases where mothers employed more than one strategy in responding to the child's expression of emotion, each successive strategy was treated and coded separately with respect to its ability to downregulate the child's affect. The respective Kappas for responsivity to distress and contingent positivity were .67 and .60. These levels are consistent with criterion established for Kappas 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. Interval-based scores were supplemented with four-point global scores of mother's contingent responsiveness to child distress and positivity so that coders could account for critical events that occurred across tasks, a limitation of the interval method. For purposes of the present study, 15% of tapes were coded to establish reliability. Weighted kappas for the global codes were .69 for contingent responsiveness to distress and .53 for contingent responsiveness to positivity. Means (and standard deviations) for interval scores were .21 ( $SD = .14$ ) for positivity and .07 ( $SD = .08$ ) for negativity, which represent the percentage of intervals in which positive or negative emotion was shown. 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. However, as child age did not differ between COD and NCOD groups, child's age was not included as a covariate in subsequent analyses.

*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 three items for each type of response (i.e., three 'sad' items for 'Neglect.'). Responses are specific for each emotion, but are grouped according to five strategies, three of which were chosen in the

present study: Magnify (e.g., ‘told my child not to be a fraidy cat’, for anger ‘got angry with my child’), Neglect (e.g., ‘usually didn’t notice’, ‘I didn’t respond’), and Reward (e.g., ‘helped my child deal with the issue,’ ‘comforted my child’). ‘Magnify’ refers to responses that amplify the child’s initial expression of sadness, anger, or fear; ‘Neglect’ involves parental unresponsiveness to these expressions of emotion; ‘Reward’ includes parental behaviors that encourage the expression of emotion (e.g., providing comfort, showing empathy). We included the ‘Neglect’ and ‘Magnify’ factors because of their theoretical link to upregulating negative emotion. Similarly, we included ‘Reward’ because such parental behaviors would be theoretically linked to downregulating of children’s expression of negative emotions. Cronbach alphas for the three response factors were .79 for Reward, .68 for Neglect, and .79 for Magnify. As the ECQ 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. In previous research using the ECQ, parent’s magnification and punitiveness of adolescent’s expression of emotion have been associated with teacher and youth reports of internalizing and externalizing problems (O’Neal and Magai, 2005).

### 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 maternal education 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,  $F(2,107) = 1.02, p > .10, d = .21$ , but were found to show significantly less positive affect than NCOD children,  $F(2,107) = 3.82, p < .05, d = .36$ . 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,  $F(1,73) = 5.66, p < .05, d = 1.9^1$ . However, maternal response to child positivity was not found to differ between groups.<sup>2</sup> 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,  $F(1,109) = 5.04, p < .05, d = .32$ , and fear,  $F(1,109) = 5.18, p < .05, d = .34$ . Further, COD mothers were less likely than NCOD mothers to encourage the expression of their child's sadness,  $F(1,109) = 3.79, p < .05, d = .41$ , and fear,  $F(1,109) = 5.24, p < .05, d = .45$ .

To examine the impact of chronicity of depression on maternal responsiveness to children's emotion, the ER and ECQ 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 ECQ parenting variables were associated with number of episodes of MDD (all  $p$ -values exceeded .05).

Next, to test whether maternal childhood depression status continued to be associated with parenting behaviors after accounting for the effects of current depressive symptoms, we re-computed analyses controlling for maternal reports on the BDI for the parenting variables that were found to differentiate between COD and NCOD mothers. After accounting for current maternal depressive symptoms, COD mothers were still found to report greater neglect in response to child displays of sadness,  $F(1,102) = 6.48, p < .05$ , and afraid,  $F(1,102) = 3.72, p < .05$ , and were observed to be less responsive to child displays of negative emotion than NCOD mothers,  $F(1,73) = 5.66, p < .05$ . However, after controlling for maternal BDI scores, group differences between COD and NCOD mothers in responsiveness to child displays of sadness and fear became nonsignificant (for sad,  $F(1,102) = 1.31, p$  changed from .03 to .13, and for afraid,  $F(1,102) = 1.44, p$  changed from .01 to .12 for afraid).

A final goal was to examine the potential moderating role of child gender in relation to parental responsiveness 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 responsiveness to child positivity was found to be affected by an interaction between maternal diagnostic status and child gender,  $F(1,103) = 3.86, p < .05$ , eta squared = .04. Inspection of the direction of the parameter value associated with the interactive effect and comparison of means for boys and girls between groups revealed that 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. This interaction remained significant when maternal BDI scores were added into the equation as a covariate,  $F(1,92) = 3.68, p < .05$ . No interaction effects were found for responsiveness to negative emotions, either for the observed maternal responses to child distress or any of the ECQ factors.

### Discussion

This study sought to advance our understanding of how parents with COD 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. Mean levels of positive and negative emotionality between COD and NCOD children indicated higher levels of positivity for NCOD children but comparable levels of negative affect. In accord with our expectations, COD mothers were observed to show less responsivity to their child's distress than NCOD mothers, but no group differences were found in responding to 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. Questionnaire data indicated that COD mothers were more likely to endorse neglecting expressions of sadness or fear in their children, and provide less support for expressions of sadness and fear. After accounting for current levels of maternal depressive symptoms, both observationally-based findings remained significant, as did maternal reports of higher rates of neglect in response to children's expression of fear and sadness. However, current depressive symptoms did significantly attenuate associations between COD status and maternal reports of responsivity to child sadness and fear.

One finding of interest was that COD children showed lower levels of positive affect than NCOD offspring. Research on the role of positive affect in the development of COD has received less attention than work on negative affect (Silk, Shaw, Skuban, Oland, & Kovacs, 2006). However, the current results are consistent with studies conducted with adults examining the use of cognitive processes to cultivate positive emotion. These studies suggest that the mobilization of positive emotion in the face of stress is

associated with lower depressive symptoms and greater psychological resilience, perhaps mediated by the physiological effects of positive emotion on the central and peripheral nervous system (Tugade & Fredrickson, 2004). At a broader level, the findings also are consistent with Fredrickson and Joiner's (2002) broaden-and-build theory of positive emotion. According to this theory, positive affect leads to a broadening of attention and cognition, facilitating creativity and flexibility in coping with problems. The experience of positive emotion thereby leads to an upward spiral that can have positive implications on an individual's ability to regulate negative emotion.

#### *Parenting and the Intergenerational Transmission of Depression*

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., 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 diagnostic status of the mother was a more reliable discriminator of responsivity than chronicity of maternal depression, and was still predictive of different forms of responsivity after accounting for current maternal depressive symptoms, is 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. Although such a conclusion is tentative at this date and would require a direct comparison of mothers who experienced depression beginning in late adolescence or adulthood, the current results are consistent with the notion that COD mother's earlier and more chronic disruptions in their socialization process rather than number of depressive episodes or the mother's current level of

depressive symptoms plays an important role in how they respond to their children's expression of emotion.

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 mothers (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. 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 girls' expression of emotion because of a greater press for girls to develop more relationship-oriented strategies for regulating emotions (Block, 1983; Eisenberg et al., 1998; Hops et al., 1987; Nolen-Hoeksema et al., 1994). The results also are consistent with research demonstrating boys' greater vulnerability to environmental stressors in early childhood, specifically 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 become frequent among mother-son dyads (Patterson, 1982). It should also be

noted that although a gender by COD 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 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 mothers 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.

The findings also 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, the study's cross-sectional design limits inferences about the directionality of findings. For example, over time differences in child's rates of expressed negative or positive emotions may influence maternal responsivity to child

behavior (Bell, 1968). Longitudinal data are needed to account for the effects of both parent and child effects on later child adjustment before drawing inferences about the potential effects of maternal responsivity on child psychopathology. Third, 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. Fourth, the age span of the offspring was rather wide, ranging from infancy to middle childhood; thus, 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. Fifth, the validity of the current findings may be limited to mothers with a childhood-onset of depression rather than mothers with depressive illness beginning after age 14. 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. For similar reasons, definitions of positive and negative emotion were necessarily broad because of the challenges in eliciting negative emotion within a laboratory setting. Ideally, we would have been able to focus on a more narrowly-defined subset of negative emotions. Despite the limited variability in the expression of emotion for older children and the range of negative and positive emotions included, we were still able to find between-group differences in mother's response to offspring's 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.

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## Footnotes

<sup>1</sup>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.

<sup>2</sup>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

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

	COD ( <i>N</i> = 65)	NCOD ( <i>N</i> = 56)
ER: Contingent positive affect*	-.035 (1.08)	.269 (.965)
ER: Responsivity to distress*	-.168 <sup>a</sup> (.987)	.169 <sup>a</sup> (.960)
ECQ: Reward Sad	4.14 <sup>b</sup> (.879)	4.48 <sup>b</sup> (.770)
ECQ: Reward Angry	3.95 (.844)	4.23 (.905)
ECQ: Reward Afraid	4.18 <sup>c</sup> (.819)	4.52 <sup>c</sup> (.678)
ECQ: Neglect Sad	1.80 <sup>d</sup> (.562)	1.63 <sup>d</sup> (.488)
ECQ: Neglect Angry	1.81 (.442)	1.71 (.481)
ECQ: Neglect Afraid	1.72 <sup>e</sup> (.449)	1.57 <sup>e</sup> (.437)
ECQ: Magnify Sad	1.95 (.787)	1.81 (.818)
ECQ: Magnify Angry	1.87 (.934)	1.60 (.655)
ECQ: Magnify Afraid	1.48 (.670)	1.35 (.618)

*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

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

$N = 121$ ; \*  $p < .05$

Names and addresses of potential future readers of the journal

Dr. Lauren Supplee, Department of Psychology, University of Pittsburgh, 210 S. Bouquet Street, 4103 Sennott Square, Pittsburgh, PA, 15260

Dr. Kristin Moilanen, Department of Psychology, University of Pittsburgh, 210 S. Bouquet Street, 4105 Sennott Square, Pittsburgh, PA, 15260

Dr. Xin Feng, Department of Psychology, University of Pittsburgh, 210 S. Bouquet Street, 4421 Sennott Square, Pittsburgh, PA, 15260

Dr. Rebecca Burwell, Department of Psychology, University of Pittsburgh, 210 S. Bouquet Street, 4107 Sennott Square, Pittsburgh, PA, 15260

Emily Skuban, Department of Psychology, University of Pittsburgh, 210 S. Bouquet Street, 4425 Sennott Square, Pittsburgh, PA, 15260

Ella Vanderbilt, Department of Psychology, University of Pittsburgh, 210 S. Bouquet Street, 4425 Sennott Square, Pittsburgh, PA, 15260