The Relationship of Prenatal Alcohol Exposure and the Postnatal Environment to Child Depressive Symptoms

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Objective This study examined the association between prenatal alcohol exposure and child depressive symptoms, and the mediating effects of maternal and child characteristics. **Methods** Participants were 42 children aged 4–5 years and their biological mothers. Prenatal alcohol consumption was assessed by self-report of maximum drinks per drinking occasion. The Pictorial Depression Scale (PDS) measured child depressive symptoms. Mother–child interactions were assessed using the family interaction puzzle task. **Results** Structural equation modeling indicated that prenatal alcohol exposure was associated with more negative child affect. In turn, mothers of more negative children were less emotionally connected to their children, and those children had higher levels of depressive symptomatology. Results could not be explained by current maternal drinking patterns or maternal depression. **Conclusions** Study findings highlight the importance of examining prenatal alcohol exposure as a risk factor in the prediction of childhood-onset depression and the environmental mechanisms that may mediate that relationship.

Key words prenatal alcohol exposure, child depression

Prenatal alcohol exposure has been implicated as a significant neurodevelopmental teratogen resulting in a host of neurocognitive deficits including developmental delays and problems with learning, memory, attention, inhibition, and state regulation (for reviews, see Coles & Platzman, 1993; Mattson & Riley, 1998; Streissguth & O'Malley, 2000). Moreover, subtle effects on learning and behavior have been found at relatively low levels of exposure (Jacobson & Jacobson, 1994; Jacobson, Chiodo, Sokol, & Jacobson, 2002). Despite the vast literature on the poor neurodevelopmental outcomes of prenatally exposed individuals, there has been comparatively little research on their psychosocial outcomes. However, an emerging literature has begun to document the associations between prenatal alcohol exposure and a number of behavioral, emotional, and social problems in children, including externalizing (e.g., hyperactivity, antisocial behavior) and internalizing (e.g., depression) problems, as well as somatic complaints, psychiatric disorders, and poor interpersonal skills (Brown et al., 1991; Carmichael Olson et al., 1997; O'Connor et al., 2002; Roebuck, Mattson, & Riley, 1999; Steinhausen & Spohr, 1998; Thomas, Kelly, Mattson, & Riley, 1998). Moreover, there is evidence that such problems may emerge as early as infancy and the preschool years (O'Connor, 2001) and often persist into adolescence (Steinhausen & Spohr, 1998) and adulthood (Famy, Streissguth, Unis, 1998; Streissguth, Barr, Kogan, & Bookstein, 1996).

Many of the studies examining the psychosocial outcomes of prenatally exposed individuals have focused on the emergence of externalizing behavior problems, documenting higher rates of inattentive, hyperactive, and aggressive behavior in alcohol-exposed children compared to nonalcohol exposed children (Mattson & Riley, 2000; Nanson & Hiscock, 1990). Increasingly, however, researchers are investigating the potential link between prenatal alcohol exposure and internalizing problems, specifically depressive symptoms. Childhood depression has been increasingly recognized as a serious disorder associated with significant impairment, chronicity, and

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Journal of Pediatric Psychology 31(1) pp. 50–64, 2006
doi:10.1093/jpepsy/jsj021
Advance Access publication March 31, 2005

risk for relapse (Luby, 2000), and major depressive disorders have been diagnosed in children as young as preschool age (Luby et al., 2003). Clinicians and researchers have noted the importance of identifying and intervening with depressed children as early as possible, as children do not appear to simply outgrow this disorder (Luby et al., 2003). Thus, examining prenatal alcohol exposure as one possible risk factor for the emergence of depressive symptoms in young children appears to be an important focus of research.

Roebuck et al. (1999) found that in a sample of 3- to 16-year-old children, those with heavy prenatal alcohol exposure were reported by their parents to have higher rates of depressive symptoms than control children. It is possible that these children may have also been at higher risk for depressive symptoms because many were mentally retarded, as previous research suggests that mental retardation is associated with an increased risk of psychiatric illness (King, State, Shah, Davanzo, & Dykens, 1997). However, prenatal alcohol exposure has been linked to depression in children (O'Connor et al., 2002) and adults (Famy et al., 1998) with normal intelligence as well.

Examining the role of prenatal alcohol exposure in child depressive symptoms would complement other separate, but related lines of research. A number of studies have suggested that children of alcoholics are at higher risk for a variety of emotional and behavioral problems (Das Eiden, Leonard, & Morrisey, 2001; Fitzgerald, Davies, & Zucker, 2002; Loukas, Zucker, Fitzgerald, & Krull, 2003), including depressive symptomatology (Chassin, Pitts, DeLucia, & Todd, 1999; Christensen & Bilenberg, 2000; Hammen, Rudolph, Weisz, Rao, & Burge, 1999; Sher, 1997).

Notably, some studies have suggested that maternal alcoholism may uniquely contribute to the prediction of children's internalizing behavior (Chassin et al., 1999; Chassin, Rogosch, & Barrerra, 1991; Steinhausen, Gobel, & Nestler, 1984). Although such findings may suggest that maternal alcoholism confers some genetic risk for depression, it is also possible that prenatal alcohol exposure may be contributing to the increased risk for depression among children whose mothers are alcoholics. Additionally, there is evidence for assortative mating among adults with alcohol problems (Abel, 1992; Hall, Hesselbrock, & Stabenau, 1983; Russell, 1990) and thus alcoholic fathers may be partnered with mothers who consumed alcohol during their pregnancies. Previous research has shown that women partnered with alcoholic men are twice as likely to be alcoholic themselves as compared to women without alcoholic partners (Windle, Windle, Scheidt, & Miller, 1995). Thus, even among those studies that have found increased risk for depression among children of alcoholic fathers (Christensen & Bilenberg, 2000; Wall, Garcia-Andrade, Wong, Lau, & Ehlers, 2000), it remains possible that prenatal alcohol exposure may account for some of the linkages between paternal alcohol use and child depression.

Recently, researchers have highlighted the limitations of perspectives that focus exclusively on either the teratogenic effects of prenatal exposure or on the impact of current parental alcohol use on children's development (Carmichael Olson, O'Connor, & Fitzgerald, 2001). Moreover, they have emphasized the importance of examining the contributions of both the prenatal and postnatal environments in shaping children's developmental trajectories (Carta et al., 2001), including the development of psychopathology among children (Carmichael Olson et al., 2001; Carmichael Olson, Morse, & Huffine, 1998; O'Connor & Kasari, 2000).

Mother–child transactions are one key aspect of the postnatal environment that would be important to consider when exploring how prenatal alcohol exposure may potentiate the risk for adverse developmental outcomes. Carmichael Olson et al. (1998) have suggested that children with Fetal Alcohol Syndrome (FAS) and related conditions may be more likely to have negative caregiving experiences than children with other disabilities, including parents who may not be able to interact in supportive ways with their children and/or who are currently abusing alcohol or other substances. This hypothesis was borne out in a study by O'Connor, Sigman, and Kasari. (1992,1993), who examined maternal behavior in a sample of primarily Caucasian middle class women and their firstborn 1-year-old infants. Mothers who drank at higher levels during pregnancy exhibited less elaboration of their children's self-initiated behavior and provided less cognitive stimulation to their infant children than mothers who drank at lower levels or abstained during pregnancy.

There also may be qualities of the prenatally exposed infant that may make it more challenging for the mother to provide warm, supporting caregiving. Previous studies suggest that prenatally exposed infants may be predisposed to exhibit more irritability and negative affect (Coles & Platzman, 1993; O'Connor et al., 1992), and such irritability and negative affect may contribute to a less harmonious mother–child relationship. Indeed, a number of developmental studies have demonstrated that infant temperamental variations impact mothers' behavior toward their infants (Belsky, Rovine, & Taylor,

1984; van den Boom., 1991). For example, van den Boom (1991) demonstrated that mothers of children who were irritable in the first few days of life were more likely to show declining involvement with the infant during the first 6 months than were mothers of less irritable infants. Thus, the transactional contributions of both mother and child would be important to consider when exploring how prenatal alcohol exposure may increase a child's vulnerability to depression.

A related line of research has found associations between parental alcohol use and disturbances in family relationships, including impaired parent–child interactions (Famularo, Kinscherff, & Fenton, 1992; Jacob, Krahn, & Leonard, 1991; Weinberg, 1997; Whipple, Fitzgerald, & Zucker, 1995), and some researchers have speculated that the quality of maternal caregiving may mediate or moderate the effects of parental alcohol use on child outcomes (Curran & Chassin, 1996). Although many of these studies have involved families with alcoholic fathers, those studies that have focused on mothers with alcohol abuse problems have characterized these mothers as unresponsive and uncaring in their interactions with their children (Giglio & Kaufman, 1990; Steinhausen et al., 1984). Thus, it would be important to also consider active parental alcohol use when exploring the linkages among prenatal alcohol exposure, disturbed parent–child relationships, and child psychopathology.

Children with prenatal alcohol exposure may be at further risk for depression by virtue of their mother's own genetic liability. A number of studies have found higher rates of depression in the mothers of depressed children than in the mothers of children with other psychiatric disorders (Kovacs, Devlin, Pollock, Richards, & Mukerji, 1997; Mitchell, McCauley, Burke, Calderon, & Schloredt, 1989; Puig-Antich et al., 1989). Furthermore, there are strong links between depression and alcohol abuse problems among women (Alcohol and Health, 1997; Gomberg, 1993, 1994), with some studies documenting the strongest associations among African-American women (Grant & Harford, 1995). Thus, it is not surprising that higher rates of alcoholism have also been found among mothers of depressed children as compared to mothers of psychiatric controls (Kovacs et al., 1997). Additionally, there is evidence that depressive symptomatology is associated with a greater likelihood of maternal alcohol consumption during pregnancy (Zuckerman, Amaro, Bauchner, & Cabral, 1989). Moreover, parent–child interactions among prenatally exposed children and their mothers may be further compromised if a mother is experiencing depressive symptoms herself, as depressed mothers appear to have more negative and less positive interactions with their children than nondepressed mothers (Hamilton, Jones, & Hammen, 1993; Jacob & Johnson, 1997). Thus, a mother who is both depressed and currently using alcohol may find it particularly challenging to respond to her child in a positive manner, particularly a child with prenatal alcohol exposure who already may be predisposed to exhibit higher levels of negative affect. The present investigation extends previous findings by examining the ways in which prenatal alcohol exposure may act in concert with other risk factors in setting the stage for the development of child depressive symptoms in a sample of mothers and children from primarily ethnic minority, lower socioeconomic backgrounds (SES). Although such families are often the focus of studies on the impact of prenatal exposure to alcohol and other substances, we know relatively less about how postnatal environments (i.e., compromised mother–child transactions) may confer further vulnerability upon children who may be at increased risk for less than optimal developmental trajectories for other reasons (i.e., socioeconomic disadvantage).

Using structural equation modeling, we proposed a model on the basis of the assumption that prenatal alcohol exposure would be associated with child negative affect, which would relate to the mother's emotional supportiveness of the child. Guided by the literature, we hypothesized that children with higher levels of prenatal alcohol exposure would display more negative affect in interaction with their mothers. We further hypothesized that the mothers of children with higher levels of exposure would demonstrate less optimal emotional support of their preschoolers and that these children would endorse more depressive symptoms than children with less prenatal alcohol exposure. The direct associations of mothers' prenatal, postnatal, and current drinking behavior with child depressive symptoms were investigated. Finally, we examined maternal report of current drinking, depressive symptoms, and emotional connectedness in interaction with the child.

Method

Participants

All participants were recruited from the general population of patients seen at a large university medical center and from affiliated health clinics serving the surrounding community. The medical center is located in an area that

serves a predominately minority and economically disadvantaged community. It is estimated that 46% of the surrounding community live below the poverty level. Prior to participant recruitment, approval from the IRB of the medical center was obtained.

Potential participants were informed of the study by flyers posted at the medical center and in the community. Using this recruitment procedure, we were able to recruit women who used alcohol during pregnancy in varying amounts, from abstinent to heavy use so that a full spectrum of exposure was obtained. However, as recommended by Jacobson and Jacobson (1996), we oversampled women who used greater amounts of alcohol to obtain an adequate number of more heavily exposed children.

Women who called requesting information about the study were told of the purpose of the research and study instruments were described over the phone. Women wishing to participate in the study were asked basic demographic information and given a brief alcohol and drug screen. Only mothers and children who spoke English as their primary language were eligible for the study. In addition, all mothers had to be 21 years of age or older and all children had to have been living with their biological mothers since birth. Children with medical conditions, major sensory or motor deficits were excluded from study. Children were selected because of their primary exposure to alcohol, although mothers who used some marijuana and/or some cocaine during pregnancy were not excluded. Of the sixty-eight mothers who were screened, 8 dyads (12%) were eliminated because of the mother's primary and daily use of cocaine or other illegal drugs and 18 mothers (26%) failed to keep their appointments. Demographic data collected during screening on the 18 mothers who failed to keep their appointments revealed no significant differences between these mothers and study participants on prenatal alcohol consumption, ethnicity, maternal age, marital status, education, or income.

Table I summarizes the demographics of the sample. Participants were 42 English-speaking mother–child dyads. The sample consisted of predominantly African American (83%) women between the ages of 21 and 44 (M = 31.57, SD = 6.92). Most of the women were single parents (86%), with incomes of less than $10,000 per year (65%). The Hollingshead ratings mean score was 23.02 (SD = 9.97) suggesting that most women would be classified in the social strata of semiskilled workers (Hollingshead, 1975). Seventy-six percent of the women were receiving aid to families with dependent children (AFDC). On average, sample mothers had received 12.43 (SD = 1.58) years of education. Twenty-six percent of mothers had not graduated from high school, 38% were high school graduates, 31% had some college, and 5% were college graduates. Mean IQ of mothers was 84.52 (SD = 12.97) on the Kaufman Brief Intelligence Test (K-BIT; Kaufman & Kaufman, 1990).

Table I. Demographic Characteristics of Sample Mothers and Children

Demographic	*n*	%
Ethnicity		
African American	35	83
Hispanic	3	7
Other	4	10
Age (Years)		
21–30	20	48
31–44	22	52
Father in home		
Married/partner	6	14
Single parent	36	86
Education		
Nonhigh-school graduate	11	26
High-school graduate	16	38
Some college	13	31
College graduate	2	5
Yearly Income		
<$5,000	2	5
$5,000–$10,000	25	60
$10,000–$25,000	15	35

Sample children included 27 boys and 15 girls with a mean age of 4 years, 9 months (SD = 9.56). Mean child IQ was 89.60 (SD = 11.44) on the K-BIT. The average height of sample children was 108.78cm (SD = 6.50 cm), weight was 18.94 kg (SD = 3.84 kg), and head circumference was 50.86 cm (SD = 1.70 cm).

Procedure

Study measures were collected during two sessions with the child and mother. The first session took place at a research laboratory at the medical center and the second session occurred in the child's home. During Session 1, after reading and signing the consent forms, the mother was interviewed regarding her alcohol consumption patterns during pregnancy, following the birth of her child, and currently. The mother also completed developmental and medical history questionnaires and the Beck Depression Inventory—II (BDI; Beck, 1996), and was administered the K-BIT (Kaufman & Kaufman, 1990). Each child was given a physical examination by the study pediatrician. The child was then administered

the K-BIT and the Pictorial Depression Scale (PDS; O'Connor & Kasari, 2000). During the Session 2 home visit, the mother was asked to play with her child for 10 min using a structured parent–child interaction paradigm developed by Chase-Lansdale, Brooks-Gunn, and Zamsky (1994). This task was videotaped for later scoring.

Measures

Alcohol, caffeine, smoking, and other drug use

Study mothers were queried retrospectively about their prenatal alcohol consumption using a detailed interview adapted from a measure developed by Day and Robles (Day & Robles, 1989).[1] The measure used for this study, maximum drinks per drinking occasion, was selected based upon previous work demonstrating that it was a valid predictor of prenatal alcohol exposure effects (O'Connor & Kasari, 2000; O'Connor et al., 1993; O'Connor, 2001; O'Connor, Kogan, & Findlay, 2002). This measure consists of a simple count of the maximum number of drinks the mother reports consuming per drinking occasion. Postnatal (birth to current) and current alcohol intake was estimated in the same manner. Container size and amount of absolute alcohol were considered when estimating alcohol consumption levels. One drink was considered to be .60 oz of absolute alcohol, thus, one 12 oz can of beer containing .05% absolute alcohol was considered one drink, whereas one 16 oz can of .08% malt liquor was considered two drinks.

Caffeine ingested during pregnancy and currently was calculated according to the procedure of Jacobson, Fein, Jacobson, Schwartz, and Dowler (1984). No postnatal caffeine measures were obtained. The number of cups of coffee and tea was calculated along with the number of caffeinated soft drinks. Coffee was assigned a weight of 3, whereas caffeinated tea and soft drinks were assigned a weight of 1. The sum of these weighted scores was used as the measure of caffeine consumption. Using this score, the average amount of caffeine consumed per day was calculated using the frequency weighting from the Jessor, Graves, Hanson, and Jessor (1968) system.

Estimates of cigarette smoking and other drug use during pregnancy were also collected. Amount of smoking was estimated by a simple count of the number of cigarettes the mother reported smoking each day. Of the mothers who smoked, all reported smoking brands high in nicotine. Drug use was estimated from each mother's report of the number and kinds of drugs she consumed throughout pregnancy. The number of prescription versus nonprescription drugs taken during pregnancy was calculated separately for data analysis. Marijuana and cocaine use during pregnancy were also examined. Mothers were asked about the number of marijuana cigarettes they smoked per week. Cocaine use was coded on a scale from 0 to 2 with 0 representing no cocaine use, 1 representing use 2 or fewer times a week, and 2 representing use of cocaine 3 or more times a week, but less than daily. Estimates of current use of cigarettes, prescription and nonprescription drugs, marijuana and cocaine were coded using the same system as that used to calculate pregnancy levels. These estimates were not obtained for the postnatal period.

Child report of depressive symptoms

The PDS, a 27-item self-rating scale based on the Children's Depression Inventory (CDI; Kovacs, 1992), and adapted by O'Connor and Kasari (2000) for use with children 4–6 years of age was used to assess child depressive symptoms. The PDS consists of pictures of two side-by-side identical children with neutral facial expressions (girl figures were used for girls and boy figures for boys). The items from the PDS are worded in such a way that the child can respond to one of two statements, one of which reflects depressive symptomatology, by pointing to one of the two figures. The questions are read to the child and the child is required to point to the figure that is most like him/her. For example, one set of statements that the child could respond to is (1) "This child feels sad" or (2) "This child does not feel sad." The order of presentation of negative versus positive statements is counterbalanced to eliminate response bias. The sum of the responses reflecting depressive symptomatology constitutes the total score for the measure. This measure demonstrated good concurrent validity (r = .85) when compared to the CDI in a sample of older children. Furthermore, using a cut off score of >10, the PDS was found to distinguish between depression and non-depression (based on DSM-IV criteria) in 87% of a sample of child inpatients (O'Connor, 2000).

[1]Although there is some debate over using retrospective accounts of prenatal alcohol consumption, some researchers have suggested that women may underreport drinking levels if asked during pregnancy and that more valid measures may be obtained postnatally (Ernhart et al., 1988). Indeed, Ernhart et al. (1988) found that retrospective reports of drinking collected 5 years after pregnancy were more highly related to scores on a measure of alcohol-related problems and to craniofacial anomalies in children than were reports collected during pregnancy. Furthermore, when questioned 5 years after the birth of their children, a large proportion of the women provided retrospective reports that were appreciably higher than those reports given while they were pregnant.

Maternal report of depressive symptoms

Previous work suggested that the level of mother's current depressive symptoms would be associated with the child's depressive symptoms (O'Connor & Kasari, 2000). For this reason, the mother's depression was measured using the BDI-II (Beck, 1996). This inventory is a 21-item self-report measure of depressive symptomatology that has strong internal consistency (r = .93) and adequate test retest reliability (r = .68) and validity (r = .63). Scores of 0–9 are considered to be in the minimal range, 10–16 in the mildly depressed range, 17–29 in the moderately depressed range, and 30–63 in the severely depressed range. The BDI-I and BDI-II have been used to study depression in mothers of prenatally exposed children from low SES backgrounds (Jacobson et al., 2002; O'Connor & Kasari, 2000).

Family Interaction Puzzle Task

During the structured 10-minute family–child interaction task, the child was presented with four puzzles of increasing difficulty. Mothers were instructed to "let your child work on the puzzle by him/herself at first and then give him/her any help that you think he/she needs." During the task, the child typically experiences gradual, mild frustration and needs parental assistance (Chase-Lansdale et al., 1994).

Using the Puzzle Task Coding Manual for Parent–Child Interactions in Young African-American Multigenerational Families (Chase-Lansdale, Zamsky, & Brooks-Gunn, 1989), two subscales of the Puzzle Task Coding Scales were examined: maternal *Emotional Connectedness* and child *Negative Affect*. Maternal *Emotional Connectedness* represents the emotional support and overall quality of positive expressions and responses directed toward the child by the mother. Child *Negative Affect* measured the intensity and frequency of the child's degree of unhappiness, sadness, and hurt expressed toward the parent while working on the puzzle task. These subscales were coded on a 5-point Likert scale. Two coders, blind to maternal alcohol consumption levels, were trained to code the interaction videotapes. Interobserver reliability was calculated for 14 participants and *kappas* equaled .68 and .77, for maternal *Emotional Connectedness* and child *Negative Affect*, respectively. Once reliability was established, one coder coded the mother's behavior while the second coder coded the child's behavior.

Physical Examination

All physical examinations were conducted by the study pediatrician who had extensive experience in evaluating children with FAS and related conditions. This pediatrician was trained to reliability by Dr Kenneth Lyon Jones to conduct physical examinations and dysmorphology assessments for the Western Cape, South Africa Fetal Alcohol Syndrome Study (May et al., 2000). For the current investigation, the study pediatrician examined the child using a dysmorphology checklist emphasizing physical anomalies associated with prenatal alcohol exposure. The pediatrician was unaware of the mother's reported alcohol and other teratogen consumption patterns. A total score indicating the total number of physical anomalies exhibited by the child was calculated. In addition, the child's height, weight, and head circumference (OFC) were measured.

Results

Prenatal, Postnatal, and Current Alcohol and Other Substance Use

For the entire sample, the mean maximum drinks per occasion during pregnancy was 4.55 drinks (SD = 6.11). Regarding prenatal use of other substances, the weighted daily average amount of caffeinated drinks consumed during pregnancy was 4.05 cups (SD = 4.90). Twelve (29%) mothers reported smoking 10 or more cigarettes a day during pregnancy, and twenty-nine (69%) took prescription or over-the-counter medications during pregnancy. Eight (20%) of the mothers used marijuana at least once a day. Three (7%) of the mothers reported cocaine use 1–2 times per week during pregnancy, whereas cocaine use ≥ 3 times a week but less than daily was reported by six (14%) of the mothers.

Alcohol consumption during pregnancy was significantly correlated with smoking, r = .49, p <.001, and with cocaine use, r = .55, p < .001. However, alcohol consumption was not significantly correlated with caffeine ingestion, marijuana use, or the use of prescription or other nonprescription drugs during pregnancy.

The mean score for postnatal alcohol consumption for the sample was 6.93 maximum drinks per occasion (SD = 9.41). No data were available for postnatal smoking, caffeine, or other medication or drug use.

Regarding current maternal alcohol consumption, the mean maximum drinks averaged 1.86 drinks (SD = 3.17) for the entire sample. Many of the heavier drinking women in the sample had participated in alcohol treatment programs (31% of the sample or 13 women) and had reduced or stopped drinking by the time their children were between 4 and 5 years of age. Average weighted caffeinated drinks currently was 3.57 cups (SD = 3.65). Twelve (28%) mothers reported smoking 10 or more cigarettes a day currently. Twenty-nine (69%) women reported using

prescription or over-the-counter drugs currently. Five mothers reported current marijuana use, and one mother reported current cocaine use (1–2 times per week).

Current alcohol consumption was associated with current marijuana use, $r = .35$, $p < .05$, but was not correlated with current caffeine ingestion, smoking, cocaine use, or the use of prescription or other nonprescription drugs.

Relation of Prenatal, Postnatal, and Current Maternal Alcohol Consumption to Child Height, Weight, Head Circumference and Physical Dysmorphology

Correlational analyses were run to examine the associations of alcohol with child physical features. Prenatal, postnatal, and current alcohol consumption scores were used in these analyses. Current maternal use of alcohol was included in these analyses as some researchers (Jacobson et al., 2002) have suggested that current drinking levels may influence retrospective recall, making it difficult to ascertain effects in studies that collect data on pregnancy alcohol use retrospectively. Thus, examining the associations of prenatal, postnatal, and current maternal use of alcohol with child physical features known to be associated with prenatal alcohol exposure would shed some light on the validity of the use of retrospective reporting in this study. Results revealed that prenatal alcohol exposure was related to child head circumference ($r = -.44$, $p < .01$) and the number of physical anomalies noted on physical examination ($r = .39$, $p < .01$). However, head circumference was not related to postnatal or current alcohol use ($r = -.08$, *ns*; $r = .21$, ns, respectively), nor was the number of physical anomalies related to postnatal or current alcohol consumption ($r = .08$, *ns*; $r = -.14$, *ns*, respectively). Stepwise regression analyses using prenatal, postnatal, and current alcohol scores revealed that after controlling for postnatal and current alcohol use, prenatal exposure continued to be significantly related to head circumference and dysmorphology ($\beta = -.52$, $p < .01$ and $\beta = .47$, $p < .01$, respectively). Data were also available on the use of other teratogens during the prenatal and current assessment periods. Neither prenatal nor current use of other teratogens was related to either head circumference or to number of physical abnormalities.

Relation of Prenatal, Postnatal, and Current Maternal Alcohol Consumption to Child Negative Affect

It was hypothesized that exposure to alcohol prenatally would be related to the child's expression of negative affect. In testing this hypothesis, it was important to also examine the possible relationships of child negative affect to mother's postnatal and current alcohol use, as well as to other substances, including caffeine, smoking, marijuana, cocaine, prescription and nonprescription drug use. Results revealed that child negative affect was related to both prenatal ($r = .56$, $p < .001$) and postnatal ($r = .46$, $p < .001$) alcohol use, but not current alcohol use. Additionally, child negative affect was significantly correlated with prenatal cocaine use ($r = .47$, $p < .001$), but not with prenatal or current smoking, caffeine, marijuana, prescription or nonprescription drugs.

To further examine the above relationships, maximum number of drinks per occasion for the prenatal, postnatal, and current time periods were entered into a multiple regression analysis with child negative affect as the outcome variable. Additionally, because prenatal cocaine use was related to both prenatal alcohol use and child negative affect, and prenatal smoking was related to prenatal alcohol use, these variables were also entered into the equation. After these variables were entered into the regression, only prenatal alcohol exposure remained a statistically significant predictor of negative affect ($\beta = .36$, $p < .05$).

Relation of Current Maternal Alcohol Consumption to Maternal Connectedness

Although it was proposed in the model that prenatal alcohol consumption would relate to child negative affect which in turn would relate to maternal connectedness, it was also important to consider the possibility that maternal connectedness might be directly linked to her current alcohol consumption. Thus we examined the association maternal report of current alcohol use with maternal connectedness. There was no association of maternal current alcohol use with maternal connectedness ($r = -.11$, *ns*).

Relation of Prenatal, Postnatal, and Current Maternal Alcohol Consumption to Child Depressive Symptoms

It was hypothesized that prenatal exposure to alcohol would be related to child depressive symptoms. Indeed, higher levels of prenatal alcohol exposure were associated with higher levels of depressive symptomatology on the Pictorial Depression Scale ($r = .35$, $p < .05$). To further examine this association, participants were divided into two groups. An abstinent-light group ($n = 22$) was composed of children whose mothers reported drinking one or fewer drinks per drinking occasion prenatally ($M = 0.5$, $SD = 0.21$), and a moderate-heavy group ($n = 20$)

was composed of those children whose mothers reported drinking two or more drinks per occasion prenatally (M = 9.50, SD = 5.58). The cut point of two or more drinks was based upon data that suggests that behavioral effects are found even at these low levels of exposure (Carmichael Olson et al., 1997; O'Connor & Kasari, 2000). Using a criterion score on the PDS of > 10 to represent a significant level of depressive symptoms, 9% (2/22) of children in the abstinent-light group met criterion. In contrast, 40% (8/20) of children in the moderate-high group endorsed a significant number of depressive symptoms (Fisher's Exact Test, $p < .05$).

It was further speculated that the mother's postnatal and current drinking levels might also be related to child depressive symptoms, but these associations were not statistically significant (r = .08, *ns* for postnatal; r = .17, *ns* for current). Regarding other teratogens, child depressive symptoms were not significantly related to prenatal caffeine (r = –.11, *ns*), smoking (r = .05, *ns*), marijuana (r = –.02, *ns*), or cocaine use (r = – .04, *ns*), or to current caffeine (r = – .06, *ns*), smoking (r = – .03, *ns*), or marijuana (r = .12, *ns*) use. Child depressive symptoms were marginally related to current cocaine use (r = .28, *ns*); however, it is important to note that this association was entirely accounted for by the child of only one mother in the sample who reported currently using cocaine. When this one child was excluded from the analyses, there was no relationship between children depressive symptoms and mother's current cocaine use.

Relation of Maternal Depressive Symptoms to Maternal Use of Alcohol and Other Teratogens, Mother–Child Interaction, and Child Depressive Symptoms

Sample mothers reported a mean level of depressive symptoms of 15.71 (SD = 9.59) on the BDI-II. The majority (59.5%) of women were classified as minimally (scores of 0–9) or mildly (10–16) depressed, 31% were moderately depressed (17–29), and 9.5% were severely depressed (30+). Contrary to predictions, maternal depressive symptoms were not associated with prenatal (r = –.09, *ns*), postnatal (r = .16, *ns*), or current alcohol (r = .18, *ns*) use. Furthermore, maternal depressive symptoms were not significantly related to prenatal caffeine (r = .23, *ns*), smoking (r = .22, *ns*), marijuana (r = .25, *ns*), or cocaine use (r = .25, *ns*), or to current caffeine (r = .11, *ns*), marijuana (r = .00, *ns*), or cocaine (r = –.08, *ns*) use. Maternal depressive symptoms were associated with current maternal smoking (r = .41, $p < .01$). There was no association between mother's behavior during interaction with the child (*Emotional Connectedness*) and her report of depressive symptoms (r = –.03, *ns*), suggesting that these two variables were measuring unique aspects of the mother's functioning. Finally, examination of the mother's total score on the BDI-II in relation to child depressive symptoms reported on the PDS revealed no statistical association in this sample (r = .06, *ns*).

Other Possible Covariates

In assessing possible associations among prenatal alcohol exposure, child negative affect, mother–child interaction, and depressive symptoms, it was important to consider that these relations could be attributed to other factors. Before model testing, possible covariates (child IQ, gender and ethnicity, number of siblings in the home, and maternal IQ, age, socioeconomic status, income, marital status, and education) were selected for analysis on the basis of literature suggesting that they might be associated with the variables under study. Jacobson and Jacobson (1996) have suggested including covariates that correlate with study outcome measures at a probability level of $p < .10$. However, chi-square and zero-order correlations revealed that none of these variables was significantly associated with child negative affect, maternal emotional connectedness, or child depressive symptoms in the present sample.

Prenatal Alcohol Exposure, Child Negative Affect, Mother Emotional Connectedness, and Child Depressive Symptoms: Model Testing

To examine the complex relations among study variables, path analysis was employed using the EQS program for structural equation modeling (Bentler, 1995). Goodness-of-Fit of the models was assessed using the Robust Comparative Fit Index (RCFI) on the basis of Satorra-Bentler χ^2 statistic (Bentler & Dudgeon, 1996). These indices perform better with small samples. The RCFI ranges between 0 and 1 and compares the improvement of fit of the hypothesized model to a model of independence among the measured variables, while adjusting for sample size. Values greater than .95 are desirable and indicate that 95% or more of the covariation in the data is reproduced by the hypothesized model (Hu & Bentler, 1999). Root mean square errors of approximation (RMSEAs) are also reported to indicate the size of the residuals. Values less than .06 indicate a relatively good fit between the hypothesized model and the observed data (Hu & Bentler, 1999). For structural equation modeling, Bentler (1995) suggests that the ratio of sample size to number of free parameter estimates can be as low as 5 : 1 under normal theory.

Prior to model testing, maximum drinks per drinking occasion during pregnancy was recomputed adjusting for the effects of both prenatal smoking and cocaine use because there were significant correlations between the predictor variable of prenatal alcohol consumption and variables of prenatal smoking and cocaine use and between prenatal cocaine use and child negative affect. Further, data were analyzed for univariate and multivariate kurtosis to evaluate normality of variable distributions. Univariate kurtosis ranged from –1.17 to 2.20. Mardia's normalized estimate was 0.47 with a kappa of 0.05, so data were not transformed. Table II is the covariance matrix of study variables; included in the table are zero-order correlations.

Figure 1 shows the proposed model for assessing the associations among prenatal alcohol exposure, child negative affect, maternal emotional connectedness and child depressive symptoms. The model is shown as a path diagram using the conventional method of placing measured variables in rectangles. One-way arrows represent regression paths. The variances of errors in measured variables were estimated but are not included in the figure. Variables with one-way arrows pointing toward them represent hypothesized influences of other variables and are considered dependent variables. All estimates were standardized, thus, regression coefficients can be interpreted as beta coefficients. In this model, child depressive symptomatology is the dependent variable predicted by the indirect effects of prenatal alcohol, negative child affect, and maternal emotional connectedness. In addition, a direct path from prenatal alcohol to child depressive symptoms was also predicted. Because current maternal alcohol use was not related to the child variables or to maternal emotional connectedness, this variable was not included in the model. Results revealed that the proposed model was a good fit, S-B χ^2 (2, N = 42) = 1.97, p = .37, RCFI = 1.00, RMSEA = 0.00).

Table II. Covariance Matrix

Variable	1	2	3	4
1.Prenatal alcohol exposure[a]	24.33			
	(1.00)[b]			
2. Child negative affect	1.10	0.31		
	(0.40)**	(1.00)		
3. Mother emotional	–0.42	–0.16	0.56	
connectedness	(–0.11)	(–0.34)*	(1.00)	
4. Child depressive symptoms	0.73	0.03	–0.12	0.19
	0.35*	0.11	(–0.32)*	(1.00)

[a]Prenatal alcohol exposure standardized residuals after partialling out the effects of smoking and cocaine.
[b]Numbers in parentheses are correlations (r).
*$p < .05$.
**$p < .01$.

Discussion

Results from this study extend previous research findings suggesting that prenatal alcohol exposure represents a significant risk factor for the emergence of depressive symptomatology in young children. Children whose mothers reported moderate to heavy alcohol consumption during pregnancy reported significantly higher levels of depressive symptoms than children whose mothers reported abstinence or light alcohol consumption during pregnancy. Although many studies of children with prenatal alcohol exposure have focused on externalizing problems, these findings add to the mounting body of evidence indicating that such children should also be monitored for the possible onset of depression as they mature.

This investigation also focused on examining other prenatal factors as well as aspects of the postnatal environment that might act in concert with prenatal alcohol exposure in increasing children's risk for depressive symptoms. Data analysis indicated a good fit for a model in which it was hypothesized that (1) prenatal alcohol exposure would relate to higher levels of negative affect in the child; (2) negative affect in the child would be associated lower levels of maternal emotional connectedness during mother–child interactions; and (3) lower levels of maternal emotional connectedness would relate to higher levels of child depressive symptoms. Thus, it appeared that the mother–child transaction might have

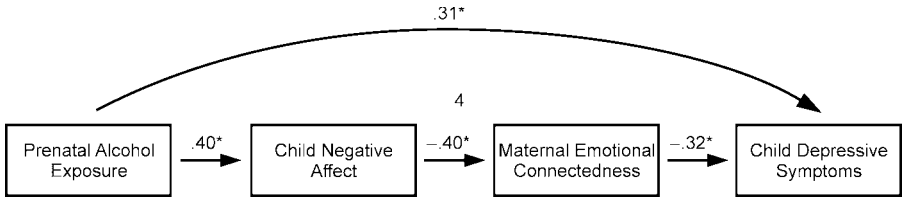

Figure 1. Relation between prenatal alcohol exposure, child negative affect, maternal emotional connectedness, and child depressive symptoms. Regression coefficients (represented as one-way arrows) are standardized ($^*p < .05$).

mediated the effects of prenatal alcohol exposure on depressive symptomatology in the child.

Although this model provided a good fit for the hypothesized relationships, it is important to be cautious in drawing any definitive conclusions regarding causality using correlational data. Previous research suggests that children can influence parenting behavior (Belsky et al., 1984; van den Boom, 1991), and it seems possible that the children who exhibit higher levels of depressive symptoms are less likely to elicit emotional support and expressions of positive affect from their mothers. Although lower levels of maternal emotional support and expressions of positive affect may contribute to the emergence of child depressive symptoms, it is also possible that higher levels of child depressive symptomatology may incline mothers to be less supportive and positive with their children. Family systems researchers have long noted the mutuality of parent–child relationships (for reviews, see Cox & Paley, 1997; Minuchin, 1985; Sameroff & MacKenzie, 2003), and it seems likely that a prenatally exposed child and his/her caregiver(s) would mutually influence one another over the course of the child's development. Sameroff and MacKenzie (2003) have noted that "the development of the child is a product of the continuous dynamic interactions of the child and the experience provided by his or her family and social context" (p. 614). Thus, continuing efforts to understand the developmental trajectories of at-risk children (in this case, children with prenatal alcohol exposure) must consider the contributions of both parent and child.

Importantly, a direct relation between prenatal alcohol exposure and child depressive symptoms was also found. This result, in combination with the finding that sample children with prenatal alcohol exposure had smaller head circumferences, is consistent with neuroanatomical findings suggesting that prenatal exposure is associated with smaller brain size and with possible damage to the basal ganglia, a collection of frontosubcortical nuclei implicated in the regulation of mood (Roebuck, Mattson, & Riley, 1998; Soares & Mann, 1997). Although we have no information regarding brain damage in the children for this study, given that individuals with prenatal alcohol exposure do appear to be at increased risk for mood disturbances, the exploration of possible underlying brain mechanisms would seem to be an important area for further inquiry.

A number of other prenatal and postnatal factors were examined for their contributions to child depressive symptoms. Although prenatal cocaine exposure was related to child negative affect (and thus was controlled for during model testing), prenatal exposure to cocaine and other potential teratogens (marijuana, nicotine, caffeine, prescription and nonprescription drugs) was not associated with child depressive symptoms. Similarly, current maternal use of other substances was also unrelated to depressive symptoms in the child. It is possible that current maternal alcohol use in this sample was not at a high enough level so as to significantly contribute to child depressive symptoms.

Surprisingly, report of maternal depressive symptoms was also unrelated to child depressive symptoms. The assessment of both maternal and child depressive symptomatology merits some discussion. It is important to note that neither the BDI-II or the PDS are intended as measures of adult or child clinical depression, respectively, which is best diagnosed through structured interview and observation by a skilled clinician. The relatively low number of severely depressed women in the sample may account for the absence of an association between maternal depression and maternal emotional connnectedness, and it is quite possible that the ability of mothers to provide support to their children would have been affected in a sample of more highly impaired mothers. Given the strong association between alcohol abuse and depression among women (Alcohol and Health, 1997; Gomberg, 1993, 1994; Grant & Hartford, 1995), a focus on clinically depressed mothers of children with prenatal exposure would shed further light on the ways in which maternal factors may compound the risks for prenatally exposed children.

Limitations and Future Directions

The reliance on mothers' retrospective accounts of prenatal alcohol use 4 to 5 years after the birth of their child potentially raises questions as to the accuracy of such retrospective accounts. This issue has been examined, and there is some evidence supporting the validity of retrospective reports (Ernhart, Morrow-Tlucak, Sokol, & Martier, 1988). Researchers examining maternal recall of prenatal alcohol consumption have suggested that women may underreport drinking levels if asked during pregnancy and that more valid measures may be obtained postnatally (Ernhart et al., 1988). Of particular relevance for this study, Ernhart et al. (1988) found that retrospective reports of drinking collected 5 years after pregnancy were more highly related to scores on a measure of alcohol-related problems and to craniofacial anomalies in children than were reports collected during pregnancy. Moreover, when questioned 5 years after the birth of their children, a large proportion of the women provided retrospective reports that were appreciably

higher than those reports given while they were pregnant. However, other research suggests that antenatal accounts of prenatal drinking may be more valid than retrospective accounts (Jacobson et al., 2002). In a careful analysis, Jacobson and associates (2002) found reports of alcohol consumption taken during the antenatal period to be more highly associated with multiple measures of infant behavior at 13 months than retrospective reports of pregnancy drinking levels. Like Ernhart and associates (1988), they also found that retrospective recall yielded reports of higher levels of drinking than antenatal reports and that there was a statistically significant correlation between postpartum or current drinking and retrospective reports of consumption during pregnancy. They noted that these findings might suggest that current drinking levels may influence retrospective recall, making it difficult to ascertain effects in studies that collect data on pregnancy alcohol use retrospectively. Given this legitimate concern, we examined the correlation between maternal report of prenatal drinking levels and current levels and found no correlation. Furthermore, measures of child head circumference and physical dysmorphology, while yielding significant correlations with maternal recall of prenatal consumption, were unrelated to reports of postnatal and current drinking levels. These findings suggest that although mothers may not remember precise levels, the levels that they do recall may reflect their relative standing in relation to other women. That is, women who report higher levels of alcohol consumption may be relatively heavier drinkers than women who report lower levels. This being said, although in this study we found that there is value in obtaining mothers' retrospective reports of alcohol consumption during pregnancy, these reports should not be presumed to reflect accurate estimates when examining teratogenic thresholds.

There is some evidence that maternal alcohol problems may make a stronger contribution to children's internalizing problems than paternal alcohol problems (Chassin et al., 1991; Chassin et al., 1999). Thus the present investigation was focused primarily on the ways in which maternal factors (e.g., maternal emotional connectedness, maternal depression and maternal current drinking) might act in concert with prenatal alcohol exposure in predicting adverse developmental trajectories for children. However, there is also a strong literature implicating paternal alcoholism in increasing children's risk for behavioral and emotional problems (Das Eiden et al., 2001; Loukas et al., 2003). Thus, an additional limitation of this study to be noted is the absence of father data. Most of the mothers in the sample identified themselves as single parents (86%). However, previous research (Johnson, 2001) suggests that even in households headed by single mothers, fathers (or other male figures) may assume a strong parenting role with the child even if they do not reside in the child's home. Ideally, future studies would examine both maternal *and* paternal contributions when investigating the role of prenatal alcohol exposure in the emergence of child depressive symptoms. Such investigations would be ideally suited to begin disentangling the contributions of prenatal alcohol exposure, and maternal and paternal psychopathology, drinking, and parenting in increasing children's risk for negative outcomes.

A final limitation of the study is the possible sampling bias introduced by relying on a relatively small self-selected sample. Notably, a significant number (31%) of women in the study had received treatment for alcohol use problems, and thus may not be representative of the larger population of women who consume alcohol either prenatally or postnatally. However, it is important to note that we were able to recruit women who used alcohol during pregnancy in varying amounts, from abstinence to heavy use so that the full spectrum of exposure was obtained.

Despite these limitations, this study represents an important step in continuing efforts to investigate the relation of prenatal alcohol exposure and several aspects of the postnatal environment. Although differentiating the contributions of prenatal and postnatal factors remains a challenging task, the current findings add to the understanding of the ways in which prenatal alcohol exposure may act in concert with other risk factors in conferring increased vulnerability to the onset of childhood depression. Identifying how these processes may operate early in development is critical to efforts to design effective interventions for high-risk children.

Acknowledgement

We thank Dr. Richard Findlay and Dr. Nina Kogan for their assistance on this study. This research was funded by NIAAA Center Grant U24 (AA11899) and by the Office for Research on Minority Health awarded to the Charles R. Drew University of Medicine and Science Collaborative Alcohol Research Center.

Received March 1, 2005; accepted March 1, 2005.

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