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Children's Intellectual and Emotional–Behavioral Adjustment at 4 Years as a Function of Cocaine Exposure, Maternal Characteristics, and Environmental Risk

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Abstract

The authors examined 223 children at age 4 years for the effects of prenatal cocaine exposure, exposure to other substances, maternal and environmental risk factors, and neonatal medical problems on IQ, externalizing problems, and internalizing problems. Regression analyses showed that maternal verbal IQ and low environmental risk predicted child IQ. Cocaine exposure negatively predicted children's overall IQ and verbal reasoning scores, but only for boys. Cocaine exposure also predicted poorer short-term memory. Maternal harsh discipline, maternal depressive symptoms, and increased environmental risk predicted externalizing problems. In contrast, only maternal depressive symptoms predicted internalizing problems. These findings indicate that early exposure to substances is largely unrelated to subsequent IQ or adjustment, particularly for girls.

Two important attributes studied in preschool-age children are intelligence and emotional–behavioral adjustment. By age 4 years, IQ scores are relatively stable and predict academic success during the first few years of elementary school (Kaplan, 1996; Sameroff, Seifer, Baldwin, & Baldwin, 1993). Similarly, externalizing and internalizing problems also exhibit significant stability by age 4, and externalizing problems, in particular, predict later academic and social problems (Booth, Rose-Krasnor, McKinnon, & Rubin, 1994; Campbell, 1995; Campbell, Shaw, & Gilliom, 2000; Verhulst & van der Ende, 1992).

During the past 15 years, much concern has been raised about the potential effects of prenatal cocaine exposure on intellectual and emotional–behavioral development. Cocaine-exposed newborns are of younger gestational age and lower birth weight and have been found to have poorer neurobehavioral functioning, leading to concerns about later development (Bingol, Fuchs, Diaz, Stone, & Gromisch, 1987; Chasnoff, Griffith, MacGregor, Dirkes, & Burns, 1989; Lutiger, Graham, Einarson, & Koren, 1991). Although studies directly examining the effects of prenatal cocaine exposure on the human brain are lacking, cocaine is believed to impair brain development both directly through its effect on developing neurotransmitter systems critical to neuronal differentiation and brain structure formation and indirectly through its effect on blood flow to the developing fetal brain (Gawin & Ellinwood, 1988; Mayes &

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Bornstein, 1995). More specifically, cocaine has been found to block the reuptake of monoaminergic neurotransmitters, which may disrupt the development of neuronal circuitry in the fetus (Anderson-Brown, Slotkin, & Seidler, 1990; Lidow, 1995). Furthermore, vasoconstriction of cerebral blood vessels resulting from cocaine exposure may produce a hypoxic condition in the brain (Kurth et al., 1993; van de Bor, Walther, & Sims, 1990). Because of the inhibition of monoaminergic neurotransmitter systems, neurodevelopmental functioning in children prenatally exposed to cocaine might be expected to be compromised in areas related to reactivity, arousal modulation, and attentional regulation. In addition, reduction in placental and fetal blood flow might result in impaired information-processing and problem-solving ability (J. R. Woods, Plessinger, & Clark, 1987). These findings collectively suggest that prenatal cocaine exposure may be a risk factor for outcomes related to central nervous system (CNS) development, including intellectual functioning and behavioral regulation.

Studies assessing specific cognitive domains such as verbal reasoning, language skills, recognition memory, and information processing have found deficits related to prenatal cocaine exposure among children ranging from infancy to age 6 years (Griffith, Azuma, & Chasnoff, 1994; Jacobson, Jacobson, Sokol, Martier, & Chiodo, 1996; Nulman et al., 1994; Singer et al., 2001). In contrast, studies of global intellectual functioning generally have not found cocaine-related deficits (Azuma & Chasnoff, 1993; Frank, Augustyn, Knight, Pell, & Zuckerman, 2001; Griffith et al., 1994; Hawley, Halle, Drasin, & Thomas, 1995; Hurt et al., 1995; Hurt, Malmud, Betancourt, Brodsky, & Giannetta, 2001; Hurt et al., 1998; Kilbride, Castor, Hoffman, & Fuger, 2000; Nulman et al., 1994; Phelps & Cottone, 1999; Richardson, Conroy, & Day, 1996; Wasserman et al., 1998). Nonetheless, exceptions exist (Alessandri, Bendersky, & Lewis, 1998; Singer et al., 1997; van Baar & de Graaff, 1994). In addition, several methodological limitations exist in these earlier studies. Prior research has often been limited by modest samples of fewer than 30 cocaine-exposed children (Hawley et al., 1995; Nulman et al., 1994; Richardson et al., 1996; van Baar & de Graaff, 1994) and by low subject retention (Griffith et al., 1994; Wasserman et al., 1998). Furthermore, no study to date has reported examining Cocaine Exposure × Sex interactions in preschool or school-age children. Such Cocaine × Sex interactions are important given that the male fetus may be more susceptible to intrauterine factors affecting the CNS (Flannery & Liederman, 1994; Hynd & Semrud-Clikeman, 1989; Mathura, 1979; Montague, 1962; Weinberg, Zimmerberg, & Sonderegger, 1992).

Cocaine exposure also may affect emotional–behavioral adjustment. Although less research has examined such effects, several studies have found that cocaine-exposed infants and toddlers exhibit poorer emotional regulation and impulse control and increased externalizing problems (Bendersky & Lewis, 1998, 2001; Griffith et al., 1994; Hawley et al., 1995; Mayes, Bornstein, Chawarska, Haynes, & Granger, 1996).

Women who use cocaine during pregnancy are much more likely to prenatally use other substances, including alcohol, cigarettes, and marijuana (Bendersky, Alessandri, Gilbert, & Lewis, 1996; N. S. Woods, Behnke, Eyler, Conlon, & Wobie, 1995). Each of these substances may negatively affect development and needs to be controlled when researchers are examining cocaine's effects on children's intellectual and emotional–behavioral development. Women who use cocaine during pregnancy are also more likely to experience psychosocial risks (Bendersky et al., 1996; N. S. Woods et al., 1995). Environmental risk factors such as low socioeconomic status (SES), single parenthood, financial problems, and lack of support and stimulation in the home environment have been shown to be related to poorer intellectual functioning and increased emotional–behavioral problems in early childhood (Adams, Hillman, & Gaydos, 1994; Andersson, Sommerfelt, Sonnander, & Ahlsten, 1996; Brennan et al., 2000; Duncan, Brooks-Gunn, & Klebanov, 1994; Espy, Molfese, & DiLalla, 2001; Goldberg, Roghmann, McInerny, & Burke, 1984; Shaw, Owens, Giovannelli, & Winslow,

2001). Although such factors have increasingly been controlled in cocaine exposure research, the examination of important maternal characteristics that also may be more common among mothers with a history of substance use has been rare (Wasserman et al., 1998; N. S. Woods et al., 1995). Maternal characteristics such as low IQ, high levels of depressive symptoms, and the use of harsh discipline strategies (e.g., spanking and hitting) have generally been found to predict poorer intellectual functioning and greater emotional–behavioral problems among young children (Andersson et al., 1996; Brennan et al., 2000; Carter, Garrity-Rokous, Chazan-Cohen, Little, & Briggs-Gowan, 2001; Dodge, Bates, & Pettit, 1990; Downey & Coyne, 1990; Hammen, 1999; O'Leary, Smith Slep, & Reid, 1999; Wakschlag & Keenan, 2001). Thus, when researchers are examining the effects of prenatal cocaine exposure on children's development, the potential effects of other substances, environmental risk factors, and maternal characteristics should be taken into account. The present study is one of the first to examine the effects of cocaine exposure on children's intellectual and emotional–behavioral adjustment while observing the effects of other substance exposure, environmental risk, maternal intelligence, maternal depressive symptoms, and harsh discipline.

Environmental risk factors often covary with cocaine use (Bendersky et al., 1996) and, rather than cocaine use itself, are related to poor outcomes (Frank et al., 2001). For example, Hurt et al. (1995) concluded that low SES or minority status, but not cocaine exposure, had a substantial influence on cognitive functioning during infancy. Following their sample at 5 years, they found that cocaine-exposed children's lower scores on a developmental inventory could be explained by environmental factors in the home environment (Hurt et al., 2001).

We hypothesized (a) that prenatal cocaine exposure would predict increased child emotional–behavioral problems even after we controlled for the effects of prenatal exposure to other substances, environmental risk, and maternal characteristics and (b) that current psychosocial variables would predict child IQ and emotional–behavioral adjustment better than prenatal cocaine exposure. Furthermore, we sought to examine Cocaine Exposure × Sex interactions to test the hypothesis that boys exhibit greater impairment than girls in response to cocaine exposure.

Method

Subjects

The sample of 223 children (113 boys and 110 girls) and their mothers was recruited for a longitudinal study on the effects of prenatal exposure to cocaine on emotional development (see Bendersky & Lewis, 1998). Pregnant women attending participating hospital-based prenatal clinics and women who had just given birth in one of three hospitals in Trenton, New Jersey, or at the Medical College of Pennsylvania in Philadelphia were approached. Of these, 82% agreed to participate in the study. Informed consent was obtained at this time. Children were excluded from the study if they were born prior to 32 weeks of gestation, required special care or oxygen therapy for more than 24 hr, exhibited congenital anomalies, were exposed to opiates or PCP in utero, or were born to mothers infected with HIV. Mothers (N = 223) were predominantly African American (87%), with 9% Caucasian and 3% Hispanic. Mothers' median education level was 11th grade (SD = 1.6 years), and 63% of families received Aid for Dependent Children. Mothers ranged in age from 13.7 to 43.6 years (M = 25.6, SD = 6.1). Thirty-eight percent (n = 85) reported using cocaine during pregnancy. Among cocaine users, 61 also reported using alcohol, 73 reported smoking cigarettes, and 25 reported using marijuana. Among mothers who did not use cocaine (n = 138), 21 reported using alcohol, 29 reported smoking cigarettes, and 7 reported using marijuana. Participation was voluntary, and incentives were provided in the form of vouchers for use at local stores. Children's mean age was 4.1 years (range = 3.9 to 4.9 years; SD = 0.2) at the 4-year lab visit.

Procedure

Children were administered the Stanford-Binet Intelligence Scale: Fourth Edition (SB-IV; Thorndike, Hagen, & Sattler, 1986) by a trained female psychometrist. Mothers in an adjacent room were administered the Peabody Picture Vocabulary Test—Revised (PPVT–R; Dunn & Dunn, 1981), the Parent–Child Conflict Tactics Scale (PCCTS; Strauss, 1995), and the Beck Depression Inventory (BDI; Beck, 1978). The PCCTS was administered as an interview. Most mothers completed the BDI as a self-report; however, some mothers opted to have the BDI read to them in interview format by a female staff member.

Measures of Risk Factors

Environmental risk—The following maternal risk factors were assessed by interview at the 4-year assessment. The variables were standardized, reverse coded if necessary so that the higher the value the greater the risk, and summed to produce the environmental risk score (see Bendersky & Lewis, 1998). The score was a composite of maternal life stress (based on the Social Environment Inventory; Orr, James, & Casper, 1992), maternal social support network size (Norbeck Social Support Questionnaire; Norbeck, Lindsey, & Carrieri, 1981), number of regular caregivers (greater number = higher risk), regularity of child's schedule, stability of child's surroundings (Family Chaos Scale; A. Sameroff, personal communication, September 1993), single-parent household (living alone with children = higher risk), maternal race (non-White = higher risk), maternal education, and public assistance status (public assistance as main source of income = higher risk).

Maternal depressive symptoms—Mothers completed the 21-item BDI, which assesses affective, behavioral, cognitive, and motivational symptoms of depression. The BDI has high internal consistency, correlates highly with clinician ratings and with other validated self-report measures of depressive symptoms, and is able to discriminate anxiety from depression (Beck, Steer, & Garbin, 1988). The BDI has been used extensively with African Americans (Beck et al., 1988; Munford, 1994).

Maternal harsh discipline—Mothers also completed the 22-item PCCTS. The PCCTS contains subscales assessing the frequency of the following discipline styles during the past 12 months: nonviolent discipline, physical assault (e.g., spanked, slapped, hit), and psychological aggression (e.g., yelled, swore, threatened to spank but did not). For the present study, the physical assault and psychological aggression subscales were summed to form a harsh discipline composite. The included subscales have adequate reliability and validity and have been used with African Americans (Strauss, Hamby, Finkelhor, Moore, & Runyan, 1998).

Maternal verbal IQ—The PPVT–R was administered to mothers to provide an estimate of verbal IQ. The PPVT–R correlates highly with the verbal IQ scale of the Wechsler Adult Intelligence Scale (median r =.71; Dunn & Dunn, 1981), and the PPVT has previously been used as an estimate of maternal IQ (e.g., Longstreth et al., 1981). The PPVT has been used with African American adults and has been found to be a valid predictor of IQ for both African Americans and European Americans (Dunn & Dunn, 1981; Halpin, Simpson, & Martin, 1990).

Neonatal medical problems—Prenatal and neonatal medical data were abstracted by nurses who gained access to hospital records. This information was used to complete a neonatal medical risk scale consisting of 35 possible complications (Hobel, Hyvarinen, Okada, & Oh, 1973). Variables included general factors (e.g., low birth weight, fetal anomalies, and feeding problems), respiratory complications (e.g., congenital pneumonia, apnea, and meconium aspiration syndrome), metabolic disorders (e.g., hypoglycemia and failure to gain weight),

cardiac problems (e.g., murmur and cardiac anomalies), and CNS problems (e.g., CNS depression and seizures). Variables were weighted and summed to obtain the risk score, which ranged from 0 to 13.

Prenatal substance use—Substance use information was obtained through a semistructured interview. Interviews were conducted prenatally (65%), in the mother's room on the maternity ward if she had just delivered (30%), in our laboratories near the hospitals (3%), or in the mother's home within 2 weeks of the child's birth (2%). They were administered by trained interviewers and substance abuse counselors. The drug use interview contained questions about the frequency, amount, and trimester during which the participant used cocaine, alcohol, cigarettes, marijuana, opiates, PCP, tranquilizers, amphetamines, and barbiturates. Cocaine use was confirmed by results of analysis of newborns' meconium. The infants' meconium samples were screened with radioimmunoassay followed by confirmatory gas chromatography–mass spectrometry for the presence of benzoyl ecgonine (cocaine metabolite), cannabinoids, opiates, amphetamines, and PCP. Mothers showed no signs of PCP, heroin, or methadone use as determined by assay and by self-report in repeated interviews.

Child Measures

Intelligence—Children were administered the SB-IV. The SB-IV sub-scales of Abstract/Visual Reasoning, Quantitative Reasoning, Short-Term Memory, and Verbal Reasoning are summed to produce a composite IQ score. The SB-IV has extensive standardization data and satisfactory reliability, including with African American children (Krohn & Lamp, 1999; Thorndike et al., 1986).

Emotional–behavioral problems—Mothers completed the 112-item Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL includes factors assessing externalizing and internalizing problems. The CBCL has satisfactory reliability and validity (Achenbach, 1991) and has been used extensively with African Americans (e.g., Randolph, Koblinsky, Beemer, Roberts, & Letiecq, 2000). Mothers were given the option of reading the items themselves or having the items administered in interview format.

Results

Table 1 presents means and standard deviations, by cocaine exposure and sex, for each predictor and outcome variable. Of note, post hoc Scheffé analyses indicated that mothers of cocaine-exposed girls drank more alcohol during pregnancy than did mothers of nonexposed boys ($p < .01$) and girls ($p < .01$) and used more marijuana than did mothers of nonexposed boys ($p < .05$) and girls ($p < .05$). Mothers of cocaine-exposed children also smoked more cigarettes and used more cocaine during pregnancy than did mothers of nonexposed children ($p < .01$). In addition, boys who were exposed to cocaine had lower composite IQ scores than did nonexposed boys, exposed girls, and nonexposed girls ($p < .05$). Similarly, exposed boys had lower scores on the short-term memory subscale than did nonexposed boys ($p < .01$), exposed girls ($p < .05$), and nonexposed girls ($p < .01$). Cocaine-exposed boys also had lower abstract/visual reasoning scores than did nonexposed girls ($p < .05$). Table 2 presents correlations between each predictor and outcome variable.

We conducted hierarchical regressions to predict the two outcome domains: child IQ and emotional–behavioral problems. For these analyses, we entered cocaine exposure as a dichotomous variable.[1] First, we examined children's intelligence at 4 years. We entered neonatal medical problems in Step 1, followed by environmental risk in Step 2, maternal characteristics in Step 3, pre-natal substance exposure in Step 4, prenatal cocaine exposure in

Step 5, child sex in Step 6, and the interactions of prenatal cocaine use with sex, environmental risk, and each maternal characteristic in Step 7.

Neonatal health was first entered to rule out the possibility that any subsequent findings were due to early medical problems that may co-occur with other risk factors (e.g., environmental risk). We were also concerned by research that has found that mothers who are depressed, use harsh discipline, and have low IQs experience elevated levels of general environmental risk (Bacharach & Baumeister, 1998; Cicchetti, Rogosch, & Toth, 1998; Hecht & Hansen, 2001). Given our interest in examining the predictive ability of such maternal characteristics on child outcome independent of the effects of general environmental risk, we entered maternal characteristics in Step 3 after controlling for environmental risk in Step 2. Next, given that research on the effects of prenatal cocaine exposure needs to examine the unique variance from cocaine after considering the variance of covarying factors such as environmental risk, maternal characteristics, and exposure to other substances (Bendersky, Alessandri, Sullivan, & Lewis, 1995), we chose to enter cocaine exposure in Step 5 after entering exposure to other substances in Step 4.

Environmental, Maternal, and Perinatal Risk Factors as Predictors of Children's Intelligence

Table 3 presents the standardized regression coefficients (βs) at time of entry and for the final equation, change in R^2 for each block, and total model R^2 for the prediction of children's IQ scores. The total model significantly predicted children's composite IQ scores, explaining 19% of the variance ($p < .001$). Environmental risk accounted for significant variance in children's IQs. As we expected, children from high-risk environments had lower IQ scores. Maternal verbal IQ explained a significant amount of variance in children's IQs. Consistent with prior research, mothers with higher verbal IQs had children with higher IQs. Prenatal substance exposure failed to predict composite IQ. However, there was a significant interaction between cocaine exposure and sex after we controlled for environmental risk, maternal characteristics, and exposure to other substances. Boys who were prenatally exposed to cocaine had lower IQ scores ($M = 78.1$, $SD = 10.2$) than those who were not exposed ($M = 85.1$, $SD = 11.8$), $t(110) = 3.08$, $p < .01$. In contrast, the IQ scores of girls who were exposed to cocaine ($M = 86.7$, $SD = 11.8$) did not differ from those of unexposed girls ($M = 86.2$, $SD = 12.0$). Thus, in this sample, low IQ was related to being in a high-risk environment, having a mother with low verbal IQ, and being a boy exposed to cocaine.

We next examined each of the four Stanford-Binet subscales (see Table 3). As for the composite IQ, similar patterns of relations were found among these subscales. The Abstract/Visual Reasoning subscale was related to high maternal verbal IQ, low environmental risk, and female sex. The Quantitative Reasoning subscale was related only to substance exposure, because children exposed to alcohol actually had higher quantitative scores, whereas those exposed to cocaine had lower scores. Short-Term Memory sub-scale scores were related to high maternal verbal IQ, low environmental risk, a lack of cocaine exposure, and female sex. Finally, the Verbal Reasoning subscale was related to high maternal verbal IQ, low environmental risk, and an interaction between cocaine exposure and sex. This interaction was explained by a trend indicating that boys who were exposed to cocaine had lower verbal reasoning scores than boys who were not exposed ($M = 81.6$ vs. 85.8, respectively), $t(110) = 1.67$, $p < .10$, after we controlled for environmental risk, maternal characteristics, and exposure to other substances. In contrast, cocaine exposure was unrelated to verbal reasoning for girls, $t(104) = 1.14$, $p > .10$.

[1]We conducted a parallel series of hierarchical regressions using a three-level cocaine exposure variable (high exposure = mothers used at least twice per week vs. low exposure = mothers used less than twice per week vs. no exposure) rather than the dichotomous (exposure vs. no exposure) variable. Use of the three-level variable did not enhance model prediction for the composite IQ, externalizing problem, or internalizing problem scores, so the dichotomous variable was chosen to facilitate interpretation of interaction effects.

Environmental, Maternal, and Perinatal Risk Factors as Predictors of Children's Emotional–Behavioral Problems

As shown in Table 4, the same risk variables were entered as before in a hierarchical regression. We again entered neonatal medical problems in the first step, followed by environmental risk in the second step, maternal characteristics in the third step, prenatal substance exposure in the fourth step, prenatal cocaine exposure in the fifth step, sex in the sixth step, and interactions between prenatal cocaine exposure with sex, environmental risk, and maternal characteristics in the seventh step.

The total model predicted children's externalizing problems, explaining 20% of the variance ($p < .001$). Although harsh discipline, maternal depressive symptoms, and environmental risk were associated with more externalizing problems, substance exposure and the interactions showed no significant effect.

Internalizing problems were predicted by a somewhat different pattern. The total model explained 18% of the variance ($p < .001$). Environmental risk did not predict significant variance, whereas maternal characteristics (in particular, maternal depressive symptoms) were related to more internalizing problems. In contrast to externalizing problems, harsh discipline was not related to internalizing problems. Although exposure to substances other than cocaine collectively predicted internalizing problems in Step 4, none of the individual substances was a significant predictor of internalizing problems in Step 4. Cocaine exposure, sex, and the interaction terms also were unrelated to internalizing problems.

Discussion

In accord with many other recent reports, the impact of prenatal cocaine exposure on intellectual and behavioral outcomes was generally low or nonexistent (e.g., Frank et al., 2001; Gelman, 1998; Richardson et al., 1996). More current factors, such as present maternal characteristics, appear to have a greater impact on children's intellectual and emotional–behavioral adjustment. This pattern is consistent with prior research showing that current environmental factors are generally better predictors of developmental outcomes than are early biological risk factors such as perinatal medical problems (Adams et al., 1994; Escalona, 1982; Werner, Bierman, & French, 1971), low birth weight (Brooks-Gunn, Klebanov, & Duncan, 1996; Ornstein, Ohlsson, Edmonds, & Asztalos, 1991), prenatal substance exposure (Hurt et al., 1998, 2001), and other biological risk factors (e.g., Sameroff & Chandler, 1975). Such findings are consistent with the general notion that current context is more important than past events in influencing current behavior (Lewis, 1997).

Most studies have not found cocaine exposure to be related to preschool- or school-age children's IQ scores (Azuma & Chasnoff, 1993; Griffith et al., 1994; Hurt et al., 1998, 2001; Kilbride et al., 2000; Richardson et al., 1996; Wasserman et al., 1998). None of these studies, however, reported the results of Cocaine Exposure × Sex interactions, so it is unclear whether cocaine exposure may have differentially affected boys and girls in these earlier studies. The present findings indicate that cocaine exposure may be harmful to global intellectual functioning, but only for boys. The lower IQs of cocaine-exposed boys relative to unexposed boys are consistent with research showing males to have increased susceptibility to intrauterine factors affecting the CNS (Flannery & Liederman, 1994; Hynd & Semrud-Clikeman, 1989; Mathura, 1979; Montague, 1962; Weinberg et al., 1992). A second potential explanation for the discrepancy between the current and earlier findings concerns the age of examined children. To date, research on the cognitive effects of prenatal cocaine exposure has primarily examined children younger than 4 years, with a few notable exceptions (Hurt et al., 1998, 2001; Richardson et al., 1996; Wasserman et al., 1998). However, cocaine is believed to damage areas of the brain that may not exhibit functional impairment until school age, when increasing

cognitive demands, social expectations, and cumulative environmental risk may reveal previously unseen exposure effects (S. D. Dixon, 1989; Frank et al., 2001). It is also possible that negative cognitive effects of cocaine exposure may not be evident until age 4 years or later. In the present sample, for example, cocaine-exposed boys did not have mental development index scores lower than those of unexposed boys at age 8 months and 18 months (Alessandri et al., 1998). The presence of such a sleeper effect could explain why studies of infants and toddlers have generally failed to reveal links between cocaine exposure and global measures of intellectual functioning. Methodological differences also need to be considered, however, because cognitive assessments used with infants are, at best, modest predictors of later cognitive assessments, which may evaluate a somewhat distinct set of skills and include an emphasis on verbal skills (Bradley-Johnson, 2001; Columbo, 1993; Molfese & Acheson, 1997). Such differences in cognitive assessment may be particularly relevant to the present findings, given that cocaine-exposed boys were found to have lower verbal reasoning scores on the Stanford-Binet scale.

Cocaine exposure may affect specific domains of cognitive functioning that are obscured when only global IQ scores are reported. Short-term memory performance, for example, was significantly lower among cocaine-exposed children in the present sample. This finding is consistent with a growing literature reporting deficits in short-term or working memory among cocaine-exposed rats (Inman-Wood, Williams, Morford, & Vorhees, 2000) and nonhuman primates (Paule, Gillam, & Morris, 1998), with such deficits found to be specific to males in one study (Choi, Mazzio, & Soliman, 1998). Research with humans, however, has been inconsistent. Visual habituation, which measures short-term recognition memory, was found to be lower among cocaine-exposed infants in two studies (Jacobson et al., 1996; Struthers & Hansen, 1992) but not in three others, including an earlier report of the present sample (Alessandri et al., 1998; Karmel, Gardner, & Freedland, 1996; Mayes, Bornstein, Chawarska, & Granger, 1995). Several earlier studies examined 3- to 6-year-old children by using the Stanford-Binet scale but did not find differences in cocaine-exposed children and controls on the Short-Term Memory subscale (Griffith et al., 1994; Kilbride et al., 2000; Richardson et al., 1996), highlighting the need for replication of the present findings.

Cocaine exposure failed to predict increased externalizing problems, in contrast to some studies (Hawley et al., 1995; Yolton & Bolig, 1994) but consistent with others (Gelman, 1998; Richardson et al., 1996). Global measures of externalizing problems, however, may not be sufficiently sensitive to identify the particular type and context of aggressive behaviors exhibited by cocaine-exposed children. There is growing evidence that prenatal cocaine exposure does affect emotional regulation (Bendersky & Lewis, 1998, 2001; Mayes, 1999). Accordingly, we would expect cocaine-exposed children to exhibit increased externalizing problems in situations that most challenge their ability to regulate emotions, and not necessarily in other situations. Examination of the reactive–proactive aggression subtyping scheme may prove worthwhile in that emotional regulation is integral to the reactive subtype. Reactive aggression is defined as an angry, defensive response to frustration or perceived provocation, whereas proactive aggression is defined as an unprovoked, aversive means of influencing or coercing others (Dodge & Coie, 1987).

Overall, the present findings demonstrate some etiological specificity in the relation between maternal risk factors and child outcomes at 4 years. Consistent with prior research, maternal IQ was related to child IQ, whereas maternal depressive symptoms and the use of harsh discipline were related to child emotional–behavioral problems (Brennan et al., 2000; Dodge et al., 1990; Neisser et al., 1996; Wasserman et al., 1998). These findings held even after the negative effects of environmental risk were taken into account.

Maternal depressive symptoms, though unrelated to child IQ, were related to increased levels of externalizing and internalizing problems. The lack of any relation between depressive symptoms and child intellectual functioning is somewhat surprising given that maternal affect and depressive symptoms have been related to intellectual functioning during the preschool and early elementary school years (Cicchetti, Rogosch, & Toth, 2000; Estrada, Arsenio, Hess, & Holloway, 1987; Jennings & Connors, 1989; Kirsh, Crnic, & Greenberg, 1995). Maternal depressive symptoms, however, were related to higher levels of maternal ratings on both emotional–behavioral outcomes. Past research has raised the possibility that depressed mothers may be biased in evaluating their children and consequently overrate the presence of actual problems (Webster-Stratton & Hammond, 1988). Although the possibility of a rating bias needs to be considered and is a limitation of this study, there is ample research indicating that children of depressed mothers do exhibit more adjustment problems from studies that use raters other than mothers (Alpern & Lyons-Ruth, 1993; Fergusson & Lynskey, 1993; Querido, Eyberg, & Boggs, 2001). The process by which children of depressed mothers may exhibit increased emotional–behavioral problems appears to be multidetermined, involving genetic transmission (Sullivan, Neale, & Kendler, 2000) and parenting factors, as depressed mothers show increased negativity and disengagement toward their children (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). This negativity and disengagement may in turn lead to increased emotional or behavioral problems.

Maternal use of harsh discipline exhibited greater specificity as an emotional–behavioral risk factor, explaining significant variance in externalizing but not internalizing problems. The use of physical or otherwise negative parenting strategies has consistently been found to predict child externalizing problems (Dodge et al., 1990; Farrington & Loeber, 2000) but has less frequently been examined as a predictor of internalizing problems.

Although we examined some of the most relevant predictors of children's early intellectual and emotional–behavioral adjustment, additional predictors may be valuable to include in future research. Child temperament, for example, may predict future emotional–behavioral problems (Bates, Bayles, Bennett, Ridge, & Brown, 1991; Shaw, Owens, Vondra, Keenan, & Winslow, 1996) and intellectual functioning (W. E. Dixon & Smith, 2000). Father involvement among low-income, minority children also has been found to predict children's intellectual development (Coley, 2001). Supportive parenting in early childhood has been found to predict children's adjustment even after the negative effects of harsh discipline are controlled for (Pettit, Bates, & Dodge, 1997). Although we assessed current levels of maternal depressive symptoms, assessment of maternal symptoms across time may enhance the prediction of child adjustment, as children whose mothers report chronic depressive symptoms may have more externalizing problems than those whose mothers are only recently depressed (Fergusson & Lynskey, 1993). The timing of depressive symptoms may also be important, as children may be more susceptible to developing behavior problems at particular ages in response to maternal depression (Ghodsian, Zajicek, & Wolkind, 1984).

Multiple assessments over time also would be helpful in examining the potential effects of outcome variables on the predictor variables, particularly maternal depressive symptoms and parenting. Children with high rates of externalizing problems, for example, might elicit increased levels of maternal depressive symptoms and increased use of harsh discipline. Finally, future studies using multiple raters and behavioral measures of children's emotional–behavioral adjustment could examine whether the relation between maternal depressive symptoms and child adjustment extends beyond maternal ratings of child adjustment. Similarly, it would be important to document that maternal ratings of harsh discipline predict, for example, teachers' ratings of child adjustment and are not merely an artifact of mothers who frequently use harsh discipline also perceiving their child as having many problems.

The present findings suggest that interventions aimed at decreasing environmental risk factors (Aylward, 1997; Duncan & Brooks-Gunn, 2000) may have positive effects on children's intellectual development. Likewise, interventions that decrease the negative effects of environmental risk factors (Zigler, 1992) and maternal depressive symptoms (Sanders & McFarland, 2000) and that enhance parenting abilities (Serketich & Dumas, 1996) also may have positive effects on children's emotional–behavioral development. The challenge remains to identify children at risk from these factors at an early age and to overcome environmental and sociodemographic factors that serve as barriers in the effective implementation of such interventions (Kazdin & Wassell, 1999).

In conclusion, this study demonstrates that current environmental factors are more closely related to children's general intellectual and emotional–behavioral adjustment than are prenatal substance exposure or neonatal medical problems. The present findings provide further evidence that prenatal cocaine exposure is not a pervasive predictor of negative adjustment during early childhood. Nonetheless, the finding that boys who were exposed to cocaine did have significantly lower IQ scores than unexposed boys raises concern, as does the finding that cocaine-exposed children had poorer performance on short-term memory and quantitative reasoning tasks. Future studies of prenatal cocaine effects on intellectual functioning are needed to replicate these findings. As previously noted by Lester, LaGasse, and Seifer (1998), even the relatively small cocaine exposure effects that have been found on children's intellectual functioning in earlier research may actually lead to sizable increases in the number of children meeting criteria for special education services. Thus, further research is needed to examine the potential effects of cocaine exposure, environmental risk, and maternal characteristics on children as they enter the elementary school years.

References

Achenbach, T. (1991). *Manual for the Child Behavior Checklist /4–18 and 1991 Profile.* Burlington: University of Vermont, Department of Psychiatry.

Adams CD, Hillman N, Gaydos GR. Behavioral difficulties in toddlers: Impact of sociocultural and biological risk factors. Journal of Clinical Child Psychology 1994;23:373–381.

Alessandri SM, Bendersky M, Lewis M. Cognitive functioning in 8- to 18-month-old drug-exposed infants. Developmental Psychology 1998;34:565–573. [PubMed: 9597365]

Alpern L, Lyons-Ruth K. Preschool children at social risk: Chronicity and timing of maternal depressive symptoms and child behavior problems at school and at home. Development and Psychopathology 1993;5:371–387.

Anderson-Brown T, Slotkin TA, Seidler FJ. Cocaine acutely inhibits DNA synthesis in developing rat brain regions: Evidence for direct actions. Brain Research 1990;537:197–202. [PubMed: 2085773]

Andersson HW, Sommerfelt K, Sonnander K, Ahlsten G. Maternal child-rearing attitudes, IQ, and socioeconomic status as related to cognitive abilities of five-year-old children. Psychological Reports 1996;79:3–14. [PubMed: 8873781]

Aylward, G. (1997). Environmental influences: Considerations for early assessment and intervention. In S. M. C. Dollinger & L. F. DiLalla (Eds.), *Assessment and intervention issues across the life span* (pp. 9–34). Mahwah, NJ: Erlbaum.

Azuma SD, Chasnoff IJ. Outcome of children prenatally exposed to cocaine and other drugs: A path analysis of three-year data. Pediatrics 1993;92:396–402. [PubMed: 7689727]

Bacharach VR, Baumeister AA. Effects of maternal intelligence, marital status, income, and home environment on cognitive development of low birthweight infants. Journal of Pediatric Psychology 1998;23:197–205. [PubMed: 9640899]

Bates, J. E., Bayles, K., Bennett, D. S., Ridge, B., & Brown, M. M. (1991). Origins of externalizing behavior problems at eight years of age. In D. J. Pepler & K. H. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 93–120). Hillsdale, NJ: Erlbaum.

Beck, A. T. (1978). *Depression inventory.* Philadelphia: Center for Cognitive Therapy.

Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clinical Psychology Review 1988;8:77–100.

Bendersky M, Alessandri S, Gilbert P, Lewis M. Characteristics of pregnant substance abusers in two cities in the Northeast. American Journal of Drug and Alcohol Abuse 1996;22:349–362. [PubMed: 8841684]

Bendersky, M., Alessandri, S. M., Sullivan, M. W., & Lewis, M. (1995). Measuring the effects of prenatal cocaine exposure. In M. Lewis & M. Bendersky (Eds.), *Mothers, babies, and cocaine: The role of toxins in development* (pp. 163–178). Hillsdale, NJ: Erlbaum.

Bendersky M, Lewis M. Arousal modulation in cocaine-exposed infants. Developmental Psychology 1998;34:555–564. [PubMed: 9597364]

Bendersky, M., & Lewis, M. (2001, April). *Effects of prenatal cocaine exposure on arousal recovery at 30 months*. Poster session presented at the annual meeting of the Eastern Psychological Association, Washington, DC.

Bingol N, Fuchs M, Diaz V, Stone RK, Gromisch DS. Teratogenicity of cocaine in humans. Journal of Pediatrics 1987;110:93–96. [PubMed: 3794893]

Booth CL, Rose-Krasnor L, McKinnon J, Rubin KH. Predicting social adjustment in middle childhood: The role of preschool attachment security and maternal style. Social Development 1994;3:189–204.

Bradley-Johnson S. Cognitive assessment for the youngest children: A critical review of tests. Journal of Psychoeducational Assessment 2001;19:19–44.

Brennan PA, Hamman C, Andersen MJ, Bor W, Najman JM, Williams GM. Chronicity, severity, and timing of maternal depressive symptoms: Relationships with child outcomes at age 5. Developmental Psychology 2000;36:759–766. [PubMed: 11081699]

Brooks-Gunn J, Klebanov PK, Duncan GJ. Ethnic differences in children's intelligence test scores: Role of economic deprivation, home environment, and maternal characteristics. Child Development 1996;67:396–408. [PubMed: 8625720]

Campbell SB. Behavior problems in preschool children: A review of recent research. Journal of Child Psychology and Psychiatry 1995;36:113–149. [PubMed: 7714027]

Campbell SB, Shaw DS, Gilliom M. Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. Development and Psychopathology 2000;12:467–488. [PubMed: 11014748]

Carter AS, Garrity-Rokous FE, Chazan-Cohen R, Little C, Briggs-Gowan MJ. Maternal depression and comorbidity: Predicting early parenting, attachment security, and toddler social–emotional problems and competence. Journal of the American Academy of Child & Adolescent Psychiatry 2001;40:18–26. [PubMed: 11195555]

Chasnoff IJ, Griffith DR, MacGregor S, Dirkes K, Burns KA. Temporal patterns of cocaine use in pregnancy. Journal of the American Medical Association 1989;261:1741–1744. [PubMed: 2918671]

Choi S, Mazzio E, Soliman KFA. The effects of gestational cocaine exposure on pregnancy outcome, postnatal development, cognition, and locomotion in rats. Annals of the New York Academy of Sciences 1998;844:324–335. [PubMed: 9668690]

Cicchetti D, Rogosch FA, Toth SL. Maternal depressive disorder and contextual risk: Contributions to the development of attachment insecurity and behavior problems in toddlerhood. Development and Psychopathology 1998;10:283–300. [PubMed: 9635225]

Cicchetti D, Rogosch FA, Toth SL. The efficacy of toddler–parent psychotherapy for fostering cognitive development in offspring. Journal of Abnormal Child Psychology 2000;28:135–148. [PubMed: 10834766]

Coley RL. (In)visible men: Emerging research on low-income, unmarried, and minority fathers. American Psychologist 2001;56:743–753. [PubMed: 11558359]

Columbo, J. (1993). *Infant cognition: Predicting later intellectual functioning*. Newbury Park, CA: Sage.

Dixon SD. Effects of transplacental exposure to cocaine and methamphetamine on the neonate. Western Journal of Medicine 1989;150:436–442. [PubMed: 2735049]

Dixon WE, Smith PH. Links between early temperament and language acquisition. Merrill-Palmer Quarterly 2000;46:417–440.

Dodge KA, Bates JE, Pettit GS. December 21). Mechanisms in the cycle of violence. Science 1990;250:1678–1683. [PubMed: 2270481]

Dodge KA, Coie JD. Social-information-processing factors in reactive and proactive aggression in children's peer groups. Journal of Personality and Social Psychology 1987;53:1146–1158. [PubMed: 3694454]

Downey G, Coyne JC. Children of depressed parents: An integrative review. Psychological Bulletin 1990;108:50–76. [PubMed: 2200073]

Duncan GJ, Brooks-Gunn J. Family poverty, welfare reform, and child development. Child Development 2000;71:188–196. [PubMed: 10836573]

Duncan GJ, Brooks-Gunn J, Klebanov PK. Economic deprivation and early childhood development. Child Development 1994;65:296–318. [PubMed: 7516849]

Dunn, L. M., & Dunn, L. M. (1981). *Peabody Picture Vocabulary Test—Revised: Manual for Forms L and M.* Circle Pines, MN: American Guidance Service.

Escalona SK. Babies at double hazard: Early development of infants at biologic and social risk. Pediatrics 1982;70:670–676. [PubMed: 7133816]

Espy KA, Molfese VJ, DiLalla LF. Effects of environmental measures on intelligence in young children: Growth curve modeling of longitudinal data. Merrill-Palmer Quarterly 2001;47:42–73.

Estrada PE, Arsenio WF, Hess RD, Holloway SD. Affective quality of the mother–child relationship: Longitudinal consequences for children's school-relevant cognitive functioning. Developmental Psychology 1987;23:210–215.

Farrington DP, Loeber R. Epidemiology of juvenile violence. Child & Adolescent Psychiatric Clinics of North America 2000;9:733–748. [PubMed: 11005003]

Fergusson DM, Lynskey MT. The effects of maternal depression on child conduct disorder and attention deficit behaviours. Social Psychiatry and Psychiatric Epidemiology 1993;28:116–123. [PubMed: 8378806]

Flannery KA, Liederman J. A test of immunoreactive theory of the origin of neurodevelopmental disorders: Is there an antecedent brother effect? Developmental Neuropsychology 1994;10:481–492.

Frank DA, Augustyn M, Knight WG, Pell T, Zuckerman B. Growth, development, and behavior in early childhood following prenatal cocaine exposure: A systematic review. Journal of the American Medical Association 2001;285:1613–1625. [PubMed: 11268270]

Gawin FH, Ellinwood FH. Cocaine and other stimulants. New England Journal of Medicine 1988;318:1173–1182. [PubMed: 3283549]

Gelman, C. F. (1998). *Characteristics of a prenatally cocaine-exposed clinical population at school age.* Unpublished doctoral dissertation, Hofstra University.

Ghodsian M, Zajicek E, Wolkind S. A longitudinal study of maternal depression and child behaviour problems. Journal of Child Psychology and Psychiatry 1984;25:91–109. [PubMed: 6693528]

Goldberg ID, Roghmann KJ, McInerny TK, Burke JD. Mental health problems among children seen in pediatric practice: Prevalence and management. Pediatrics 1984;73:278–293. [PubMed: 6701051]

Griffith DR, Azuma SD, Chasnoff IJ. Three-year outcome of children exposed prenatally to drugs. Journal of the American Academy of Child & Adolescent Psychiatry 1994;33:20–27. [PubMed: 7511139]

Halpin G, Simpson RG, Martin SL. An investigation of racial bias in the Peabody Picture Vocabulary Test—Revised. Educational and Psychological Measurement 1990;50:183–189.

Hammen, C. (1999). Children of affectively ill parents. In H. C. Steinhausen & F. Verhulst (Eds.), *Risks and outcomes in developmental psychopathology* (pp. 38–53). Oxford, England: Oxford University Press.

Hawley TL, Halle TG, Drasin RE, Thomas NG. Children of addicted mothers: Effects of the "crack epidemic" on the caregiving environment and the development of preschoolers. American Journal of Orthopsychiatry 1995;65:364–379. [PubMed: 7485422]

Hecht DB, Hansen DJ. The environment of child maltreatment: Contextual factors and the development of psychopathology. Aggression and Violent Behavior 2001;6:433–457.

Hobel C, Hyvarinen M, Okada D, Oh W. Prenatal and intrapartum high-risk screening. American Journal of Obstetrics and Gynecology 1973;117:1–9. [PubMed: 4722373]

Hurt H, Brodsky NL, Betancourt L, Braitman LE, Malmud E, Giannetta J. Cocaine-exposed children: Follow-up through 30 months. Journal of Developmental & Behavioral Pediatrics 1995;16:29–35. [PubMed: 7730454]

Hurt H, Malmud E, Betancourt LM, Brodsky NL, Giannetta JM. A prospective comparison of developmental outcome of children with in utero cocaine exposure and controls using the Battelle Developmental Inventory. Journal of Developmental & Behavioral Pediatrics 2001;22:27–34. [PubMed: 11265920]

Hurt H, Malmud E, Braitman LE, Betancourt LM, Brodsky NL, Giannetta JM. Inner-city achievers: Who are they? Archives of Pediatric Adolescent Medicine 1998;152:993–997.

Hynd GW, Semrud-Clikeman M. Dyslexia and brain morphology. Psychological Bulletin 1989;106:447–482. [PubMed: 2682720]

Inman-Wood SL, Williams MT, Morford LL, Vorhees CV. Effects of prenatal cocaine on Morris and Barnes maze tests of spatial learning and memory in the offspring of C57BL/6J mice. Neurotoxicology and Teratology 2000;22:547–557. [PubMed: 10974593]

Jacobson SW, Jacobson JL, Sokol RJ, Martier SS, Chiodo LM. New evidence for neurobehavioral effects of in utero cocaine exposure. Journal of Pediatrics 1996;129:581–590. [PubMed: 8859266]

Jennings KD, Connors RE. Mothers' interactional style and children's competence at 3 years. International Journal of Behavioral Development 1989;12:155–175.

Kaplan C. Predictive validity of the WPPSI–R: A four year follow-up study. Psychology in the Schools 1996;33:211–220.

Karmel BZ, Gardner JM, Freedland RL. Arousal modulated attention at four months as a function of intrauterine cocaine exposure. Journal of Pediatric Psychology 1996;21:821–832. [PubMed: 8990726]

Kazdin AE, Wassell G. Barriers to treatment participation and therapeutic change among children referred for conduct disorder. Journal of Clinical Child Psychology 1999;28:160–172. [PubMed: 10353076]

Kilbride H, Castor C, Hoffman E, Fuger KL. Thirty-six month outcome of prenatal cocaine exposure for term or near-term infants: Impact of early case management. Journal of Developmental & Behavioral Pediatrics 2000;21:19–26. [PubMed: 10706345]

Kirsh SJ, Crnic KA, Greenberg MT. Relations between parent–child affect and synchrony and cognitive outcome at 5 years of age. Personal Relationships 1995;2:187–198.

Krohn EJ, Lamp RE. Stability of the SB:FE and K-ABC for young children from low-income families: A 5-year longitudinal study. Journal of School Psychology 1999;37:315–332.

Kurth CD, Monitto C, Albuquerque ML, Feuer P, Anday E, Shaw L. Cocaine and its metabolites constrict cerebral arterioles in newborn pigs. Journal of Pharmacology and Experimental Therapeutics 1993;265:587–591. [PubMed: 8496809]

Lester BM, LaGasse LL, Seifer R. October 23). Cocaine exposure and children: The meaning of subtle effects. Science 1998;282:633–634. [PubMed: 9841414]

Lewis, M. (1997). *Altering fate: Why the past does not predict the future.* New York: Guilford Press.

Lidow MS. Prenatal cocaine exposure adversely affects development of the primate cerebral cortex. Synapse 1995;21:332–341. [PubMed: 8869163]

Longstreth LE, Davis B, Carter L, Flint D, Owen J, Rickert M, et al. Separation of home intellectual environment and maternal IQ as determinants of child IQ. Developmental Psychology 1981;17:532–541.

Lovejoy MC, Graczyk PA, O'Hare E, Neuman G. Maternal depression and parenting behavior: A meta-analytic review. Clinical Psychology Review 2000;20:561–592. [PubMed: 10860167]

Lutiger B, Graham K, Einarson TR, Koren G. Relationship between gestational cocaine use and pregnancy outcome: A meta-analysis. Teratology 1991;44:405–414. [PubMed: 1835806]

Mathura CB. The vulnerability of the male CNS to early trauma: Implications for clinical neuropsychology. Clinical Neuropsychology 1979;1:34–35.

Mayes LC. Developing brain and in utero cocaine exposure: Effects on neural ontogeny. Development and Psychopathology 1999;11:685–714. [PubMed: 10624721]

Mayes, L. C., & Bornstein, M. H. (1995). Developmental dilemmas for cocaine abusing parents and their children. In M. Lewis & M. Bendersky (Eds.), *Mothers, babies, and cocaine: The role of toxins in development* (pp. 251–272). Hillsdale, NJ: Erlbaum.

Mayes LC, Bornstein MH, Chawarska K, Granger RH. Information processing and developmental assessments in three-month-old infants exposed prenatally to cocaine. Pediatrics 1995;95:539–545. [PubMed: 7700755]

Mayes L, Bornstein M, Chawarska K, Haynes OM, Granger R. Impaired regulation of arousal in 3-month-old infants exposed prenatally to cocaine and other drugs. Development and Psychopathology 1996;8:29–42.

Molfese VJ, Acheson S. Infant and preschool mental and verbal abilities: How are infant scores related to preschool scores? International Journal of Behavioral Development 1997;20:595–607.

Montague, M. (1962). *Prenatal influences.* Springfield, IL: Charles C Thomas.

Munford MB. Relationship of gender, self-esteem, social class, and racial identity to depression in Blacks. Journal of Black Psychology 1994;20:157–174.

Neisser U, Boodoo G, Bouchard TJ, Boykin AW, Brody N, Ceci SJ, et al. Intelligence: Knowns and unknowns. American Psychologist 1996;51:77–101.

Norbeck J, Lindsey A, Carrieri V. The development of an instrument to measure social support. Nursing Research 1981;30:264–269. [PubMed: 7027185]

Nulman I, Rovet J, Altmann D, Bradley C, Einarson T, Koren G. Neurodevelopment of adopted children exposed in utero to cocaine. Canadian Medical Association Journal 1994;151:1591–1597. [PubMed: 7954158]

O'Leary SG, Smith Slep AM, Reid MJ. A longitudinal study of mothers' overreactive discipline and toddlers' externalizing behavior. Journal of Abnormal Child Psychology 1999;27:331–341. [PubMed: 10582835]

Ornstein M, Ohlsson A, Edmonds J, Asztalos E. Neonatal follow-up of very low birthweight/extremely low birthweight infants to school age: A critical overview. Acta Paediatrica Scandinavica 1991;80:741–748. [PubMed: 1720269]

Orr S, James S, Casper R. Psychosocial stressors and low birth weight: Development of a questionnaire. Journal of Developmental & Behavioral Pediatrics 1992;13:343–347. [PubMed: 1401118]

Paule MG, Gillam MP, Morris P. The effects of cocaine on nonhuman primate brain function are age dependent. Annals of the New York Academy of Sciences 1998;844:178–182. [PubMed: 9668675]

Pettit GS, Bates JE, Dodge KA. Supportive parenting, ecological context, and children's adjustment: A seven-year longitudinal study. Child Development 1997;68:908–923.

Phelps L, Cottone JW. Long-term developmental outcomes of prenatal cocaine exposure. Journal of Psychoeducational Assessment 1999;17:343–353.

Querido JG, Eyberg SM, Boggs SR. Revisiting the accuracy hypothesis in families of young children with conduct problems. Journal of Clinical Child Psychology 2001;30:253–261. [PubMed: 11393925]

Randolph SM, Koblinsky SA, Beemer MA, Roberts DD, Letiecq BL. Behavior problems of African American boys and girls attending Head Start programs in violent neighborhoods. Early Education and Development 2000;11:339–356.

Richardson GA, Conroy ML, Day NL. Prenatal cocaine exposure: Effects on the development of school-age children. Neurotoxicology and Teratology 1996;18:627–634. [PubMed: 8947939]

Sameroff, A., & Chandler, M. J. (1975). Reproductive risk and the continuum of caretaking casualty. In F. D. Horowitz, M. Hetherington, S. Scarr-Salapatek, & G. Siegel (Eds.), *Review of child development research* (Vol. 4, pp. 187–244). Chicago: University of Chicago Press.

Sameroff AJ, Seifer R, Baldwin A, Baldwin C. Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. Child Development 1993;64:80–97. [PubMed: 8436039]

Sanders MR, McFarland M. Treatment of depressed mothers with disruptive children: A controlled evaluation of cognitive behavioral family intervention. Behavior Therapy 2000;31:89–112.

Serketich WJ, Dumas JE. The effectiveness of behavioral parent training to modify antisocial behavior in children: A meta-analysis. Behavior Therapy 1996;27:171–186.

Shaw DS, Owens EB, Giovannelli J, Winslow EB. Infant and toddler pathways leading to early externalizing disorders. Journal of the American Academy of Child & Adolescent Psychiatry 2001;40:36–43. [PubMed: 11195559]

Shaw DS, Owens E, Vondra J, Keenan K, Winslow E. Early risk factors and pathways in the development of early disruptive behavior problems. Development and Psychopathology 1996;8:679–700.

Singer L, Arendt R, Farkas K, Minnes S, Huang J, Yamashita T. Relationship of prenatal cocaine exposure and maternal postpartum psychological distress to child developmental outcome. Development and Psychopathology 1997;9:473–489. [PubMed: 9327234]

Singer LT, Arendt R, Minnes S, Salvator A, Siegel AC, Lewis BA. Developing language skills of cocaine-exposed infants. Pediatrics 2001;107:1057–1064. [PubMed: 11331686]

Strauss, M. A. (1995). *Manual for the Conflict Tactics Scales.* Durham: Family Research Laboratory, University of New Hampshire.

Strauss MA, Hamby SL, Finkelhor D, Moore DW, Runyan D. Identification of child maltreatment with the Parent–Child Conflict Tactics Scales: Development and psychometric data for a national sample of American parents. Child Abuse & Neglect 1998;22:249–270. [PubMed: 9589178]

Struthers JM, Hansen RL. Visual recognition memory in drug-exposed infants. Journal of Developmental & Behavioral Pediatrics 1992;13:108–111. [PubMed: 1577955]

Sullivan PF, Neale MC, Kendler KS. Genetic epidemiology of major depression: Review and meta-analysis. American Journal of Psychiatry 2000;157:1552–1562. [PubMed: 11007705]

Thorndike, R. L., Hagen, E. P., & Sattler, J. M. (1986). *The Stanford-Binet Intelligence Scale: Technical manual* (4th ed.). Chicago: Riverside.

van Baar A, de Graaff BMT. Cognitive development at preschool-age of infants of drug-dependent mothers. Developmental Medicine and Child Neurology 1994;36:1063–1075. [PubMed: 7958521]

van de Bor M, Walther FJ, Sims ME. Increased cerebral blood flow velocity in infants of mothers who abuse cocaine. Pediatrics 1990;85:733–736. [PubMed: 2184408]

Verhulst FC, van der Ende J. Six-year developmental course of internalizing and externalizing problem behaviors. Journal of the American Academy of Child & Adolescent Psychiatry 1992;31:924–931. [PubMed: 1400127]

Wakschlag LS, Keenan K. Clinical significance and correlates of disruptive behavior in environmentally at-risk preschoolers. Journal of Clinical Child Psychology 2001;30:262–275. [PubMed: 11393926]

Wasserman GA, Kline JK, Bateman DA, Chiriboga C, Lumey LH, Friedlander H, et al. Prenatal cocaine exposure and school-age intelligence. Drug and Alcohol Dependence 1998;50:203–210. [PubMed: 9649973]

Webster-Stratton C, Hammond M. Maternal depression and its relationship to life stress, perceptions of child behavior problems, parenting behaviors, and child conduct problems. Journal of Abnormal Child Psychology 1988;16:299–315. [PubMed: 3403812]

Weinberg, J., Zimmerberg, B., & Sonderegger, T. B. (1992). Gender-specific effects of perinatal exposure to alcohol and other drugs. In T. Sonderegger (Ed.), *Perinatal substance use: Research findings and clinical implications* (pp. 51–89). Baltimore: Johns Hopkins University Press.

Werner, E. E., Bierman, J. M., & French, F. E. (1971). *The children of Kauai.* Honolulu: University of Hawaii Press.

Woods JR, Plessinger MA, Clark K. Effect of cocaine on uterine blood flow and fetal oxygenation. Journal of the American Medical Association 1987;257:957–961. [PubMed: 3806879]

Woods, N. S., Behnke, M., Eyler, F. D., Conlon, M., & Wobie, K. (1995). Cocaine use among pregnant women: Socioeconomic, obstetrical, and psychological issues. In M. Lewis & M. Bendersky (Eds.), *Mothers, babies, and cocaine: The role of toxins in development* (pp. 305–332). Hillsdale, NJ: Erlbaum.

Yolton KA, Bolig R. Psychosocial, behavioral, and developmental characteristics of toddlers prenatally exposed to cocaine. Child Study Journal 1994;24:49–68.

Zigler E. Early childhood intervention: A promising preventative for juvenile delinquency. American Psychologist 1992;47:997–1006. [PubMed: 1510335]

Table 1

Means (and Standard Deviations) of Predictor and Outcome Variables

Variable	Cocaine-exposed		Unexposed		
	Boys (*n*=39)	Girls (*n*=46)	Boys (*n*= 74)	Girls (*n*= 64)	*F*(3, 214)
		Predictor variables			
Environmental risk	49.9_a (10.7)	51.4_a (10.5)	48.8_a (9.8)	49.4_a (9.8)	0.52
Maternal depressive symptoms	5.4_a (6.1)	6.9_a (7.6)	6.2_a (6.0)	5.4_a (6.8)	0.55
Maternal harsh discipline	16.9_a (9.5)	18.0_a (8.9)	16.9_a(8.9)	17.1_a (9.5)	0.14
Maternal verbal IQ	70.2_a (13.3)	71.7_a (11.7)	72.6_a (15.6)	73.2_a (15.4)	0.37
Neonatal medical problems	1.4_a (2.4)	1.4_a (2.7)	0.7_a (1.5)	0.5_a (1.2)	3.18^{a*}
Prenatal substance exposure					
Alcohol (drinks/day)	$1.0_{a,b}$(1.6)	1.9_a (3.8)	0.0_b (0.1)	0.0_b (0.1)	12.70^{**}
Cigarettes (per day)	7.4_a (7.7)	11.2_a (11.9)	1.3_b (4.5)	1.1_b (3.3)	25.50^{**}
Cocaine (grams/day)	0.5_a (0.7)	0.7_a (1.0)	0.0_b (0.0)	0.0_b (0.0)	23.56^{**}
Marijuana (cigarettes/day)	$0.1_{a,b}$(0.2)	0.5_a (1.9)	0.0_b (0.2)	0.0_b (0.0)	3.67^{*}
		Outcome variables			
Intelligence					
IQ, composite score	78.1_a (10.2)	86.7_b (11.8)	85.1_b (11.8)	86.2_b (12.0)	4.83^{**}
Abstract/visual reasoning	80.6_a (12.3)	$87.9_{a,b}$(11.7)	$86.4_{a,b}$ (13.0)	88.1_b (11.9)	3.36^{*}
Quantitative reasoning	86.3_a (10.6)	92.5_a (13.8)	90.0_a (11.2)	90.3_a (12.9)	1.44
Short-term memory	81.8_a (11.1)	88.7_b (9.5)	90.0_b (11.8)	92.4_b (10.8)	7.66^{**}
Verbal reasoning	81.6_a (11.1)	87.8_a (15.4)	85.8_a (13.3)	84.1_a (16.6)	1.38
Behavioral problems					
Externalizing problems	10.2_a (9.8)	11.0_a (8.1)	11.3_a (8.4)	9.5_a(6.8)	0.64
T score	49.7_a (12.1)	54.0_a (10.0)	51.8_a (10.8)	51.8_a (10.1)	1.12
Internalizing problems	2.5_a (2.8)	3.0_a (3.1)	4.1_a (4.1)	3.4_a (2.9)	2.04
T score	43.3_a (7.4)	43.6_a (7.9)	47.0_a (9.0)	44.9_a (7.6)	2.39

Note. Within rows, means with different subscripts differ significantly at $p < .05$ by Scheffé post hoc analyses.

[a] Although the overall *F* value for neonatal medical problems was statistically significant, Scheffé post hoc analyses did not indicate any significant group differences.

* $p < .05$.

** $p < .01$.

Table 2

Correlations Among Predictor and Outcome Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
Predictor variables													
1. Neonatal medical problems	—												
2. Environmental risk	.18*	—											
3. Maternal depressive symptoms	−.02	.32**	—										
4. Maternal harsh discipline	−.10	.17*	.26**	—									
5. Maternal verbal IQ	.00	−.28**	−.05	−.02	—								
6. Prenatal cocaine exposure	.23**	.06	.02	.04	−.04	—							
7. Prenatal alcohol exposure	.09	−.01	−.10	−.07	−.07	.62**	—						
8. Prenatal cigarette exposure	.14*	.17*	.27**	.11	−.09	.66**	.24**	—					
9. Prenatal marijuana exposure	.04	.06	.13	.18**	−.06	.34**	−.01	.10	—				
10. Child sex	−.05	.08	−.03	.05	.01	.08	.04	.09	.01	—			
Outcome variables													
11. Child IQ	−.04	−.23**	−.05	−.06	.34**	−.14	.00	−.04	−.07	.13	—		
12. Externalizing problems	−.03	.14	.27**	.32**	−.10	−.02	−.01	−.03	.18**	−.01	−.07	—	
13. Internalizing problems	−.04	.05	.31**	.17*	−.07	−.15*	−.12	−.04	.17*	.00	−.02	.68**	—

Note. Cocaine exposure was entered as a dichotomous variable (−1 = not exposed, 1 = exposed). Sex was coded as 1 = boys, 2 = girls. Correlations involving the dichotomous sex and cocaine exposure variables are based on Spearman's rho; the correlation between sex and cocaine exposure is based on a phi coefficient; all other correlations use Pearson correlation coefficients.

* $p \leq .05$ (two-tailed).

** $p \leq .01$

Table 3

Hierarchical Regressions Predicting Child Intelligence at 4 Years

Predictor	Composite IQ			Abstract/visual reasoning			Quantitative reasoning			Short-term memory			Verbal reasoning		
	Entry β	Final β	ΔR^2	Entry β	Final β	ΔR^2	Entry β	Final β	ΔR^2	Entry β	Final β	ΔR^2	Entry β	Final β	ΔR^2
1. Neonatal medical problems	−.04	−.02	.00	.02	.04	.00	.00	.00	.00	−.02	.05	.00	−.01	.00	.00
2. Environmental risk	−.20**	−.10	.04**	−.15*	−.06	.02*	−.07	−.03	.01	−.15*	−.08	.02*	−.25**	−.19*	.06**
3. Maternal characteristics			.08**			.07**			.02			.04*			.07**
Depressive symptoms	.00	−.02		−.02	−.05		.01	.00		−.06	−.10		.10	.13	
Harsh discipline	−.04	−.06		−.09	−.06		.06	.04		.02	.02		−.09	−.09	
Verbal IQ	.30**	.30**		.25**	.19*		.13	.16*		.20**	.16*		.26**	.28**	
4. Prenatal substance exposure			.00			.00			.05**			.02			.01
Alcohol	.02	.01		.03	.01		.20**	.23**		−.11	−.09		.05	.02	
Cigarettes	.01	.04		−.01	.00		.07	.13		−.07	.01		−.02	−.06	
Marijuana	−.04	−.05		−.04	−.06		.01	.02		.05	.04		−.09	−.11	
5. Prenatal cocaine exposure	−.13	−.12	.01	−.12	−.11	.01	−.16*	−.16	.02*	−.23**	−.22**	.04**	.04	.05	.00
6. Sex	.16*	.20**	.02*	.15*	.19**	.02*	.05	.07	.00	.18**	.21**	.03**	.05	.09	.00
7. Prenatal Cocaine Use			.03			.03			.01			.02			.03
× Sex	.15*	.15*		.12	.12		.05	.05		.11	.11		.14*	.14*	
× Environmental Risk	.10	.10		.06	.06		.09	.09		.03	.03		.05	.05	
× Maternal Depressive Symptoms	−.07	−.07		−.08	−.08		−.04	−.04		−.08	−.08		.02	.02	
× Maternal Harsh Discipline	−.02	−.02		.07	.07		−.01	−.01		.06	.06		−.06	−.06	
× Maternal Verbal IQ	.05	.05		−.08	−.08		.07	.07		−.01	−.01		.07	.07	
Total model R^2			.19**			.15**			.11			.17**			.18**

Note. Asterisks under each R^2 column indicate significance of change in R^2 for each block. Cocaine use was entered as a dichotomous variable (0 = no exposure, 1 = some exposure). Sex was coded as 1 = boys, 2 = girls.

* $p \leq .05$.

** $p \leq .01$.

Table 4

Hierarchical Regressions Predicting Children's Emotional–Behavioral Problems at 4 Years

Predictor	Externalizing problems			Internalizing problems		
	Entry β	Final β	ΔR^2	Entry β	Final β	ΔR^2
1. Neonatal medical problems	−.03	−.01	.00	−.04	.00	.00
2. Environmental risk	.14*	.02	.02*	.05	−.05	.00
3. Maternal characteristics			.13**			.11**
Depressive symptoms	.19**	.19**		.30**	.31**	
Harsh discipline	.27**	.23**		.11	.05	
Verbal IQ	−.08	−.04		−.07	−.01	
4. Prenatal substance exposure			.03			.03*
Alcohol	.06	.03		−.05	−.01	
Cigarettes	−.14*	−.09		−.12	−.05	
Marijuana	.11	.14		.12	.16*	
5. Prenatal cocaine exposure	.04	.02	.00	−.14	−.14	.01
6. Sex	−.06	−.05	.00	−.05	−.04	.00
7. Prenatal Cocaine Exposure			.03			.02
× Sex	.05	.05		.04	.04	
× Environmental Risk	−.03	−.03		.08	.08	
× Maternal Depressive Symptoms	−.09	−.09		.01	.01	
× Maternal Harsh Discipline	−.05	−.05		−.08	−.08	
× Maternal Verbal IQ	.08	.08		.13	.13	
Total model R^2			.20**			.18**

Note. Asterisks under each R^2 column indicate significance of change in R^2 for each block. Cocaine use was entered as a dichotomous variable (0 = no exposure, 1 = some exposure). Sex was coded as 1 = boys, 2 = girls.

* $p \leq .05$.

** $p \leq .01$.