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Reactivity and Regulation in Children Prenatally Exposed to Cocaine

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Children prenatally exposed to cocaine may be at elevated risk for adjustment problems in early development because of greater reactivity and reduced regulation during challenging tasks. Few studies have examined whether cocaine-exposed children show such difficulties during the preschool years, a period marked by increased social and cognitive demands and by rapid changes in reactivity and regulation. The authors addressed this question by examining frustration reactivity and regulation of behavior during a problem-solving task in cocaine-exposed and -unexposed preschoolers. Participants were 174 4.5-year-olds (*M* age = 4.55 years, *SD* = 0.09). Frustration reactivity was measured as latency to show frustration and number of disruptive behaviors, whereas regulation was measured as latency to approach and attempt the problem-solving task and number of problem-solving behaviors. Results indicated that cocaine-exposed children took longer to attempt the problem-solving task but that cocaine-exposed boys showed the most difficulties: They were quicker to express frustration and were more disruptive. Effect sizes were relatively small, suggesting both resilience and vulnerabilities.

Keywords: reactivity, regulation, prenatal cocaine exposure

Regulation is the ability to initiate behavioral changes that meet goals and manage emotional and physiological reactivity and is among the most critical and rapidly developing capacities of early childhood. Individual differences in how children react to and regulate behavior during challenges and frustrations are an important aspect of child adjustment and temperament (Derryberry & Rothbart, 1997; Goldsmith et al., 1987; Rothbart & Bates, 1998; Rothbart & Derryberry, 1981; Thomas & Chess, 1977). Reactivity and regulation are closely related but distinct dimensions of behavior and emotion (Derryberry & Rothbart, 1997; Ramsay & Lewis, 2003), although there is continuing debate about their independence (Campos, Frankel, & Camras, 2004). Reactivity is marked by the dynamics of emotional responses, such as latency, intensity, and frequency of emotion; regulation is marked by adaptive attempts to cope with challenges (Cole, Martin, & Dennis, 2004; Thompson, 1994). Flexible and effective regulation characterizes mental health, and deficits in this core capacity are involved in a range of psychological disorders (Barkley, 1997; Saarni, 1999). Although regulation often refers to the regulation of affect, it also refers to effortful control, the intentional control and inhibition of behavior and attention (Derryberry & Rothbart, 1997; Eisenberg et al., 2005; Kochanska, 1997). In the present study, we observed reactivity and regulation during a frustrating problem-solving task. Reactivity was measured as emotional frustration, oppositionality, and aggression, whereas regulation was measured as the ability to approach the task and effectively solve the problem despite the frustration.

Over the past two decades, there has been growing concern that prenatal exposure to cocaine, through its actions on the central nervous system, increases risk for problems related to reactivity and regulation in infancy and childhood (Bendersky, Gambini, Lastella, Bennett, & Lewis, 2003; Bendersky & Lewis, 1998; Bennett, Bendersky, & Lewis, 2002; Chasnoff, Burns, Schnoll, & Burns, 1985; Chasnoff & Griffith, 1989; Delaney-Black et al., 2000, 2004; Dow-Edwards, Mayes, Spear, & Hurd, 1999; Harvey & Kosofsky, 1998; Napiorkowski & Lester, 1996). Cocaine-exposed newborns show impaired neurobehavioral functioning (Bingol, Fuchs, Diaz, Stone, & Gromisch, 1987; Griffith, Azuma, & Chasnoff, 1994; Lutiger, Graham, Einarson, & Koren, 1991; Martin, Barr, Martin, & Streissguth, 1996; Richardson, 1998; Singer et al., 2002; for a review, see Mayes, 1999a), and specific neural effects have been documented in brain systems associated with reactivity, regulation, and problem-solving capacities (e.g., the mesolimbic and midprefrontal cortices and the monoaminergic neurotransmitter system; Karmel, Gardner, & Freedland, 1996; Jones, Field, Davalos, & Hart, 2004; Mayes, 1994; Wang, Yeung, & Friedman, 1995; J. R. Woods, Plessinger, & Clark, 1987). Exposed toddlers and preschoolers show decreased reactivity modulation and inhibitory control (Bendersky et al., 2003; Bendersky & Lewis, 1998; Hawley, Halle, Drasin, & Thomas, 1995; Mayes, Bornstein, Chawarska, Haynes, & Granger, 1996), greater attentional impulsivity (Mayes, Grillon, Granger, & Schottenfeld, 1998), greater risk for specific cognitive impairments (Singer et al., 2004), and greater teacher-reported disruptive behavior (Delaney-Black et al., 2000, 2004). Older children prenatally exposed to cocaine may be slower to initiate the mental processes

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needed to solve a problem (Bandstra, Morrow, Anthony, Accornero, & Fried, 2001; Bandstra, Vogel, Morrow, Xue, & Anthony, 2004; Schroder, Snyder, Sielski, & Mayes, 2004; Singer et al., 2004).

However, other studies show significant resilience among cocaine-exposed children and fail to document vulnerabilities, such as neurotoxicity and withdrawal in infants (e.g., Eyler et al., 2001), child behavior problems (e.g., Accornero, Morrow, Bandstra, Johnson, & Anthony, 2002), and deficits in school readiness and IQ (e.g., Chasnoff et al., 1998; Pulsifer, Radonovich, Belcher, & Butz, 2004). In many of these studies, factors such as home environment and parenting characteristics rather than cocaine exposure predicted or mediated behavioral and cognitive outcomes.

Given these mixed results and given the difficulty of conducting prospective longitudinal studies, relatively little is known about the effect of cocaine exposure on reactivity and regulation during early childhood. Although some research on prenatal cocaine exposure has focused on social–emotional development (e.g., Accornero et al., 2002; Beeghly, Frank, Rose-Jacobs, Cabral, & Tronick, 2003; Bendersky, Alessandri, & Lewis, 1996; Jones et al., 2004), most studies have focused on cognitive and attentional development (e.g., Mayes et al., 1998; Pulsifer et al., 2004), and relatively few if any studies have specifically examined the effects of prenatal cocaine exposure on reactivity and regulation during frustrating problem-solving tasks. Such research is needed during the preschool years, which are marked by rapid maturation of the frontal lobes and associated improvements in executive control, reactivity modulation, and regulation of behavior (Bender, Word, DiClemente, & Crittenden, 1995; Diamond, 2002; Mayes, 1994). There is a need for research that uses observational assessment and seminaturalistic conditions rather than computer-based tasks and measures that are susceptible to reporter bias, such as maternal and teacher reports.

Prenatal cocaine exposure may have subtle but important effects on children that are not evident until more complex abilities are measured (Lester, 2000; Lester, Lagasse, & Seifer, 1998; Singer et al., 2004). For example, cocaine-exposed children may show small decrements in cognitive or emotional abilities that have increasingly disruptive effects as development proceeds (Cicchetti, Ganiban, & Barnett, 1991). Moreover, even in conjunction with significant signs of resilience, small problems with emotional reactivity and regulation, such as lower threshold for frustration and difficulty regulating behavior under emotional circumstances, may compromise a child's ability to negotiate challenging tasks (Leech, Richardson, Goldschmidt, & Day, 1999; Mayes et al., 1996). Children who are relatively more reactive to challenges and/or have difficulty regulating may be predisposed to greater stress, which could further exacerbate vulnerabilities (Mayes et al., 1998). Thus, reactivity and regulation during cognitive and emotional challenges are critically relevant to academic achievement and social adjustment during early childhood (Denham, 1998; Mayes et al., 1998; Richardson, 1998).

Various factors may interact with exposure in determining the effect of cocaine on the development of reactivity and regulation. Gender might be one such factor. Recent findings indicate that verbal reasoning and problem solving may be impaired in cocaine-exposed boys but not in cocaine-exposed girls (Bennett et al., 2002). Further, in studies of school-age children, teachers reported that boys but not girls exposed to cocaine were more likely to have clinically significant disruptive behavior problems, suggesting difficulties related to frustration reactivity and regulation, compared with cocaine-unexposed children (Delaney-Black et al., 2000, 2004). This is consistent with findings in animal research: Cocaine-exposed male rats have been found to perform more poorly than cocaine-exposed female rats on tasks assessing cognitive and motor development (Markowski, Cox, & Weiss, 1998; Spear, 1995).

To accurately evaluate the effect of prenatal exposure to cocaine on child reactivity and regulation, one must take potentially confounding factors into account, including neonatal risk factors such as prenatal exposure to other drugs, environmental risk factors, and intelligence. Women who use cocaine tend to drink more alcohol, smoke more cigarettes, and use more marijuana than those who do not use cocaine (Bendersky, Alessandri, Gilbert, & Lewis, 1996; N. S. Woods, Behnke, Eyler, Conlon, & Wobie, 1995). Prenatal exposure to these substances may have a unique impact on reactivity and regulation. Environmental risk factors such as poverty, high life stress, and maternal social isolation also are likely to have a negative impact on developmental outcomes and are generally more prevalent in children exposed to cocaine (Bendersky, Allesandri, Sullivan, & Lewis, 1995; Bendersky et al., 2003). Given the mixed support for effects of cocaine exposure on IQ, the effect of IQ was examined in this study to ensure that it did not interact with exposure in determining reactivity and regulation (Bennett et al., 2002; Pulsifer et al., 2004; Singer et al., 2004).

In summary, children, especially boys, prenatally exposed to cocaine may well be at risk for frustration reactivity and difficulties in regulating behavior during a frustrating and cognitively demanding problem-solving task. However, relatively little research has involved directly observing these effects during the preschool years, when the ability to modulate reactivity and regulate behavior is developing. Even fewer studies have assessed reactivity and regulation during problem solving, which is an important activity during early childhood (Melnick & Hinshaw, 2000). In this study, we assessed frustration reactivity and regulatory behaviors in cocaine-exposed versus -unexposed boys and girls during a frustrating problem-solving task when the children were 4.5 years old. Although we anticipated significant resiliency on the part of cocaine-exposed children, we hypothesized that prenatal exposure to cocaine would be associated with *greater frustration reactivity* (shorter latencies to show frustration, more numerous disruptive behaviors) and *greater difficulty regulating behavior* (longer latencies to approach and attempt the problem-solving task, fewer problem-solving behaviors), and, given previous findings on gender differences, we expected that these effects would be strongest for cocaine-exposed boys. The effects of potentially associated factors—including IQ; neonatal medical risk; environmental risk; and prenatal exposure to cigarettes, alcohol, and marijuana—were evaluated.

Method

Participants

Participants were 191 mothers and their children (95 boys, 88 girls), who were 4.5 years old ($M = 4.55$ years, $SD = 0.09$; ages ranged from 4.3 to 4.9 years), participating in a longitudinal study of developmental consequences of prenatal cocaine exposure. Children were predominantly African American (86%), with 11% of the children being European American and 3% Hispanic or Asian American. The racial composition did not differ between mothers who used or did not use cocaine during pregnancy.

Complete data were available for 174 children. Sixty-five were exposed to cocaine during pregnancy, and 109 were unexposed. Maternal education level ranged from 8th grade through post–high school training, with 54% being high school graduates. At the time of the study, 32% were receiving public assistance. Maternal education and public assistance did not differ between cocaine users and nonusers. Participation was voluntary, and incentives were provided in the form of vouchers for use at local stores.

Recruiters approached pregnant women attending participating hospital-based prenatal clinics and/or obstetric services in Trenton, New Jersey, or Philadelphia, Pennsylvania, between February 1993 and December 1995. Of these, 82% ($n = 384$) agreed to participate. 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 hours, exhibited congenital anomalies, were exposed to opiates or phencyclidine in utero, were born to mothers less than 15 years of age, or were infected with HIV ($n = 63$). Of these 321 children invited to participate, 258 children participated in the first lab visit at 4 months of age. Of the children lost to the study, 18 participants were placed in foster care on discharge and these families refused to participate, 27 families could not be contacted, and 18 chose not to continue.

At the 4.5-year visit, 191 mothers participated (60% of those who were invited to participate and 74% of those who participated in the first lab visit). Of the 67 families not seen at 4.5 years, 15 moved out of the area, 15 declined to participate at this age, 28 could not be contacted for this age point, 1 child and 2 mothers died, and 6 went to foster parents who refused to participate. There were no significant differences in the distributions of cocaine exposure, gender, perinatal medical risk, or environmental risk between participants who participated and those who refused to continue or were lost to the study from the neonatal period through 4.5 years of age. Among children participating in the study, 23 children were in foster care, 20 of whom had been prenatally exposed to cocaine. Foster care was not associated with any differences in risk or outcome variables, except for maternal use of alcohol during pregnancy, $p < .001$.[1]

Procedures and Measures

For the 4.5-year visit, mothers and children arrived at the research laboratory and were escorted into a playroom by a female experimenter. A set of toys was arranged on the floor to encourage child play. The experimenter described the study and obtained informed consent from the mother. Several tasks followed, including a frustration task. The tasks were administered to all participants in the same order. For the present study, only child behaviors during the frustration task (the impossible pulley task) were examined for measures of reactivity and regulation.

The impossible pulley task was a 3-min problem-solving task designed to elicit high levels of frustration. The task materials consisted of a basket and a pulley with a rope anchored with a knot tied to a hook in the wall. While the child was watching, the experimenter placed an attractive toy or food in the basket out of reach. The child was encouraged for 2 min to obtain the prize. This required figuring out that he or she had to untie the knot to release the rope from the hook in the wall and allow the basket to fall into reach. If the task was not solved after 1 min, the examiner drew the child's attention to the pulley (e.g., pointed to the pulley, the rope attached to it, and the hook in the wall), and the child was then allowed an additional 1 min to solve the problem.

To identify differences in reactivity and regulation, we coded frequency counts of the following six behaviors: (a) carrying out instrumental actions to obtain the prize without using force, such as untying the knot and reaching for the basket; (b) using items in the room to try to solve the problem, such as reaching for the basket with another toy; (c) making demands of adults, such as asking that the experimenter solve the task; (d) complaining to adults about the task; (e) showing aggression toward objects, such as banging the pulley or hitting the basket; and (f) showing aggression toward people, such as hitting or kicking the experimenter. An instrumental behavior composite score was created by summing frequency counts of instrumental actions and use of items in room. A disruptive behavior composite score also was created by summing counts of demands, complaints, and aggression toward objects and people. Proportion scores for the composites were calculated as the proportion of children showing instrumental behaviors (low or high levels were determined on the basis of a median split) and the proportion of children showing disruptive behaviors (proportions were for no, low, or high levels of disruptive behavior, and low or high levels were determined on the basis of a median split of the nonzero frequency counts).

Three latency scores were coded: *latency to first evidence of frustration* (including all behaviors composing the disruptive behavior composite score as well as signs of emotional collapse, such as crying), *latency to approach the task*, and *latency to first attempt to untie the knot*. Scores also were calculated for the proportion of children *approaching the task immediately* (approaching immediately vs. waiting) and *attempting the knot* (attempting vs. not attempting).

Composite scores, proportion scores, and latency scores were used in subsequent analyses as dependent variables indexing frustration reactivity and regulation. Frustration reactivity was measured as latency to first evidence of frustration and the disruptive behavior composite. Regulation was measured as the latency and proportion scores for approaching and attempting the task and the instrumental behavior composite.

Interrater agreement. A team of coders reviewed videotaped recordings of the sessions to generate data for analyses. Before coding, all coders achieved adequate interrater agreement. Interrater reliability for latency and frequency scores was calculated on the basis of 25% of the videotapes (45 tapes), which were randomly chosen. The average weighted Cohen's kappa coefficient for latency scores was .94 (ranging from .92 to .96) and for frequency scores was .91 (ranging from .80 to 1.00). Both coefficients reflected excellent agreement (Bartko, 1991; Fleiss, 1981). The average upper bound intraclass correlation for latency scores was .97 (ranging from .96 to .98) and for frequency scores was .95 (ranging from .89 to 1.00). Coders were blind to the exposure status of the participants and hypotheses of the study.

Neonatal medical risk score. On the basis of hospital records, prenatal and neonatal medical data were 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), respiratory complications (e.g., congenital pneumonia, apnea), metabolic disorders (e.g., hypoglycemia, failure to gain weight), cardiac problems (e.g., murmur, cardiac anomalies), and central nervous system (CNS) problems (e.g., CNS depression, seizures). Variables were weighted and summed to obtain the risk score, which ranged from 0 = *no risk* to 13 = *high risk*.

Environmental risk score. Demographic and lifestyle information was obtained through structured interviews administered to the mother during the 4.5-year laboratory visit. These interviews included questions about the mother's race, the mother's educational achievement, household composition, sources of income, maternal history of substance abuse, the number of caregivers, the regularity of the child's schedule, social support (determined by using the Norbeck Social Support Questionnaire; Norbeck, Lindsey, & Carrieri, 1981), and maternal life stressors (determined by using the Social Environment Inventory; Orr, James, & Casper, 1992). The variables were standardized, reverse coded if necessary so that higher values indicated greater risk, and summed. This cumulative risk score was then rescaled as a *t* score with a mean of 50 (see Bendersky & Lewis, 1998, Bendersky et al., 2003). Scores ranged from 24 to 81. Cumulative environmental risk measures have been found to explain more variance in children's outcomes than do single factors, including socioeconomic status (Hurt, Malmud, Betancourt, Brodsky, & Giannetta, 2001).

Prenatal substance exposure. Prenatal substance exposure information was obtained through a semistructured interview administered to the

[1] When analyses were rerun without children in foster care, patterns of effects remained the same.

mother by trained interviewers within 2 weeks of the infant's birth. The interview contained questions about the frequency, amount, and trimester of the mother's use of cocaine, alcohol, cigarettes, marijuana, opiates, PCP, tranquilizers, amphetamines, and barbiturates. Ratings were on an 8-point scale, from 0 = *no use* to 7 = *daily use*. For those children prenatally exposed to cocaine, average exposure was 0.60 g/day (*SD* = 0.91). Cocaine use was confirmed by results of analysis of newborns' meconium, which was 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. Children were considered exposed to cocaine if positive by maternal report or meconium assay. They were considered unexposed to cocaine if negative by both report and assay. For the current study, we also examined the effects of prenatal exposure to alcohol (number of drinks per day), cigarettes (number per day), and marijuana (number of joints per day).

IQ. When the children were 4 years old (*M* = 4.12, *SD* = 0.23), the Stanford–Binet Intelligence Scale, Fourth Edition (Thorndike, Hagen, & Sattler, 1986) was administered. The composite score was used. This measure has extensive standardization data and satisfactory reliability, including with African American children (Krohn & Lamp, 1999; Thorndike et al., 1986).

Results

Table 1 presents means and standard deviations for neonatal and environmental risk scores and amount of in utero exposure to alcohol, cigarettes, marijuana, and cocaine. Cocaine-exposed versus -unexposed children had significantly greater neonatal complications, $F(1, 169) = 9.14$, $p < .01$, and were exposed in utero to greater amounts of alcohol, $F(1, 165) = 32.31$, $p < .001$; cigarettes, $F(1, 165) = 54.10$, $p < .001$; and marijuana, $F(1, 164) = 4.96$, $p < .05$. However, environmental risk did not differ between cocaine-exposed and -unexposed children, and there were no gender or Gender × Exposure differences in the risk factors. These risk factors were not significantly correlated with the dependent variables (reactivity and regulation variables; correlations between −.13 and .14). We examined correlations between risk factors and outcomes separately for cocaine-exposed versus -unexposed children, and associations remained nonsignificant. Next, because child IQ might be associated with problem-solving ability, correlations between child IQ and outcome variables were examined (child IQ *M* = 84.14, *SD* = 11.47, range = 54–111). There were no significant correlations. Finally, all analyses of variance (ANOVAs) reported below were initially conducted using neonatal and environmental risk scores and child IQ as covariates, including their interactions with exposure group and gender. Results did not differ from those analyses without covariates. Thus, analyses without risk factors and IQ as covariates are reported below.

The dependent variables are located in the left-hand column of Table 2. Correlational analyses were used to evaluate the relations among these measures. The pattern of correlations was comparable across the four Exposure × Gender groups. Significant intercorrelations indicated that the greater the number of instrumental behaviors, the shorter the latency to approach the task, $r = -.29$, $p < .001$, but the longer the latency to attempt to untie the knot, $r = .34$, $p < .001$. This was perhaps because children using instrumental behaviors also used other problem-solving strategies before attempting the knot. The number of instrumental behaviors also was correlated positively with disruptive behaviors, $r = .15$,

Table 1
Descriptive Statistics for Neonatal and Environmental Risk Scores and Amount of In Utero Exposure to Alcohol, Cigarettes, Marijuana, and Cocaine

Risk variable	Total sample						No cocaine						Cocaine					
	Boys (*n* = 91)		Girls (*n* = 83)		Total (*n* = 174)		Boys (*n* = 62)		Girls (*n* = 47)		Total (*n* = 109)		Boys (*n* = 29)		Girls (*n* = 36)		Total (*n* = 65)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Neonatal risk (*n* = 170)	0.91	1.94	1.05	2.23	0.98	2.08	0.65	1.40	0.59	1.36	0.62	1.38	1.50	2.71	1.68	2.95	1.60	2.83
Environmental risk (*n* = 174)	50.54	9.84	50.53	10.38	50.54	10.07	49.89	9.58	49.82	10.44	49.86	9.91	51.92	10.40	51.46	10.37	51.66	10.30
No. of drinks/day (*n* = 166)	0.40	1.26	0.62	1.94	.50	1.61	0.03	0.16	0.02	0.7	0.03	0.13	1.29	2.08	1.48	2.84	1.40	2.50
No. of cigarettes/day (*n* = 166)	3.32	6.77	5.53	9.46	4.36	8.19	1.50	4.97	1.21	3.44	1.38	4.37	7.66	8.44	11.74	11.73	9.91	10.50
No. of marijuana joints/day (*n* = 165)	0.05	0.24	0.07	0.28	0.06	0.26	0.04	0.26	0.01	0.03	0.02	0.19	0.07	0.20	0.15	0.42	0.12	0.34
Average grams of cocaine/day (*n* = 65)	0.14	0.55	0.24	0.61	0.19	0.58							0.55	0.98	0.64	0.87	0.60	0.91

Note. The environmental risk score is a *t* score based on the sum of *z* scores from 10 risk variables.

Table 2
Descriptive Statistics for Outcome Variables

Dependent variable	Total sample									No cocaine									Cocaine								
	Boys (n = 91)			Girls (n = 83)			Total (n = 174)			Boys (n = 62)			Girls (n = 47)			Total (n = 109)			Boys (n = 29)			Girls (n = 36)			Total (n = 65)		
	M	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%
Latency to approach the task	10.07	17.69		14.13	30.01		12.01	24.37		8.85	12.63		16.98	35.34		12.35	25.27		12.69	25.45		10.40	21.05		11.42	22.96	
Approached immediately			41.1			33.8			37.6			37.7			26.1			32.7			48.3			44.1			46.0
Latency to first attempt to untie the knot	105.90	74.90		132.23	76.96		118.46	76.81		94.08	65.79		127.49	79.40		108.49	73.52		131.17	87.37		138.42	74.32		135.18	79.83	
Attempted knot			81.3			69.9			75.9			88.7			70.2			80.7			65.5			69.4			67.7
Latency to first evidence of frustration	133.57	105.21		153.29	105.34		142.98	105.43		151.08	99.64		149.47	105.48		150.39	101.72		96.14	108.72		158.28	106.45		130.55	111.07	
Showed frustration			54.9			43.4			49.4			50.0			46.8			48.6			65.5			38.9			50.8
No. of instrumental behaviors	11.62	9.10		15.12	11.60		13.29	10.48		11.56	8.79		16.32	13.11		13.61	11.07		11.72	9.89		13.56	9.21		12.74	9.49	
No. of disruptive behaviors	1.68	2.80		1.76	3.52		1.72	3.16		1.32	2.25		2.28	4.19		1.74	3.25		2.45	3.64		1.08	2.27		1.69	3.02	
Not disruptive			48.4			62.6			55.2			51.6			55.3			53.2			41.4			72.2			58.4
Low disruptive			36.3			18.1			27.6			37.1			21.3			30.3			34.5			13.9			23.1
High disruptive			15.3			19.3			17.2			11.3			23.4			16.5			24.1			13.9			18.5

Note. Values for latencies are in seconds.

$p < .05$, perhaps reflecting the general level of activity. Finally, as might be expected, the greater the number of disruptive behaviors, the shorter the latency to express frustration, $r = -.62$, $p < .001$.

These correlations suggest that reactivity and regulation scores were largely independent. However, to further explore the independence of these constructs, we examined whether children who expressed frustration relatively quickly or slowly (reactivity) differed in their problem-solving behavior during the task (regulation). Of the 174 children in this study, 88 children never showed frustration. For the remaining 86, we created two groups based on a median split (50th percentile = 19 s). These groups did not differ on our measures of regulation (latency to approach the task and latency to first attempt to untie the knot, number of instrumental behaviors). This further suggests that reactivity and regulation are independent dimensions.

Table 2 presents the means, standard deviations, and proportion scores for the dependent variables. To examine cocaine exposure and gender differences in child reactivity and regulation, we conducted a separate univariate ANOVA for each of the five dependent variables: latency to first evidence of frustration, number of disruptive behaviors, latency to approach the task, latency to first attempt to untie the knot, and number of instrumental behaviors. Substance exposure group and gender were the between-subjects factors. A high proportion of children did not receive scores for one or more of the behavioral codes. For example, 24.1% of children did not attempt the knot and 50.6% did not show frustration and thus did not have latency scores. Therefore, each ANOVA was followed by a chi-square analysis to examine cocaine exposure and gender group differences in the proportion of children who (a) showed frustration; (b) showed no, low, or high levels of disruptive behavior; (c) approached the task immediately; (d) attempted the knot; and (e) showed low versus high levels of instrumental behavior.

The ANOVA for latency to express frustration revealed a significant interaction between exposure and gender, $F(1, 173) = 3.76$, $p < .05$, $\eta^2 = 2\%$. Cocaine-exposed boys showed a significantly shorter latency to express frustration than did cocaine-unexposed boys, $t(89) = 2.38$, $p < .05$; cocaine-exposed girls, $t(63) = 2.32$, $p < .05$; and cocaine-unexposed girls, $t(74) = 2.12$, $p < .05$. There were no differences in frustration latency among cocaine-exposed girls and -unexposed girls and boys. Chi-square analyses further indicated that a greater proportion of cocaine-exposed boys versus cocaine-exposed girls showed frustration, $\chi^2(1, N = 174) = 4.56$, $p < .05$. Differences among cocaine-exposed boys, cocaine-unexposed boys, and cocaine-unexposed girls were not significant.

For the disruptive behaviors composite, the interaction between cocaine exposure and gender also was significant, $F(1, 173) = 5.50$, $p < .05$, $\eta^2 = 3\%$. Follow-up tests revealed trends for cocaine-exposed boys to evidence more disruptive behaviors than cocaine-unexposed boys, $t(89) = 1.81$, $p < .07$, and cocaine-exposed girls, $t(63) = 1.85$, $p < .07$, but they did not differ from cocaine-unexposed girls. Chi-square analyses confirmed these trends, revealing significant Exposure × Gender group differences in the proportion of children showing no, low, or high levels of disruptive behaviors, $\chi^2(2, N = 174) = 6.48$, $p < .05$. Follow-up chi square analyses for each level of disruptive behavior found that only the proportion of children showing no disruptive behavior differed significantly among groups, $\chi^2(1, N = 174) = 5.15$, $p < .05$. A smaller proportion of cocaine-exposed boys showed no disruptive behavior compared with cocaine-exposed girls, cocaine-unexposed boys, and cocaine-unexposed girls. The differences among cocaine-exposed girls, cocaine-unexposed boys, and cocaine-unexposed girls were not significant. Also, the differences between cocaine-exposed and -unexposed children and between boys and girls for low or high levels of disruptive behaviors were not significant.

Analyses of latency to approach the task did not reach significance, but analyses of latency to first attempt to untie the knot revealed a significant main effect of cocaine exposure. Cocaine-exposed children took longer to attempt to untie the knot than did cocaine-unexposed children, $F(1, 173) = 4.09$, $p < .05$, $\eta^2 = 3\%$. The interaction between cocaine exposure and gender did not reach significance. However, a chi-square analysis examining the proportion of children who attempted the knot yielded a significant Exposure × Gender interaction, $\chi^2(1, N = 174) = 4.44$, $p < .05$. A larger proportion of cocaine-unexposed boys attempted the knot compared with cocaine-unexposed girls, cocaine-exposed boys, and cocaine-exposed girls. The differences among cocaine-unexposed girls, cocaine-exposed boys, and cocaine-exposed girls for latency to first attempt to untie the knot were not significant, and group differences for latency to approach the task also were not significant.

The ANOVA for the instrumental behaviors composite yielded a main effect of gender, $F(1, 173) = 4.04$, $p < .05$, $\eta^2 = 3\%$, showing that girls evidenced more instrumental behaviors than did boys. Chi-square analyses for instrumental behavior did not reach significance.

Discussion

During the preschool years, children are often faced with problem-solving challenges that test their cognitive and emotional capacities. By maintaining emotional equilibrium and effectively exploring problems, children build a foundation for current and future success in academic and social domains. The results of the present study supported our hypothesis that prenatal exposure to cocaine increases the risk for problems related to frustration reactivity and regulation of problem-solving behavior in early childhood. However, evidence of significant resiliency also emerged, and effect sizes of these differences were relatively small. Indeed, prenatal cocaine exposure has been used as a model of the complex interplay between risk and vulnerability in young children (Mayes, 1999b). However, these early deviations in developmental trajectories may have an impact at later developmental stages. To our knowledge, this is the first study that observed reactivity and regulation in cocaine-exposed and -unexposed preschoolers during a challenging problem-solving task rather than relying on adult report of child behavior. Although these two constructs are interrelated, they also are distinct, as demonstrated by our results and as conceptualized by a rich literature on child temperament (e.g., Derryberry & Rothbart, 1997). Few studies of prenatal cocaine exposure have integrated these concepts from temperament research into the study of developmental trajectories of cocaine-exposed versus -unexposed children.

Overall, cocaine exposure appeared to have an effect on problem solving: Cocaine-exposed versus -unexposed children took longer to engage in the problem-solving task. Thus, it appeared that they were slower in defining and working on the problem at hand. Whether this was due to the cocaine-exposed children being

less engaged in the problem and/or less able to solve the problem remains unclear. Both possibilities are likely given that cocaine-exposed children are at risk for specific neurological deficits and problems associated with motivation, executive functions, reasoning, and problem solving (Bender et al., 1995; Davis et al., 1992; Mayes, 1994; Singer, Farkas, & Kliegman, 1992; Singer et al., 2004).

In this sample, prenatal exposure to alcohol, cigarettes, and marijuana differed between cocaine-exposed and -unexposed children but did not appear to be related to outcomes. Neonatal and environmental risk factors and IQ also failed to influence outcome measures beyond the effect of cocaine exposure. Thus, these findings strengthen the inference that prenatal exposure to cocaine was related specifically to the decrements in frustration reactivity and regulation detected in this study.

Consistent with previous research (Delaney-Black et al., 2000, 2004; Bendersky et al., 2003; Bendersky & Lewis, 1998; Bennett et al., 2002), we found that cocaine-exposed boys showed the most difficulties, as evidenced by shorter latencies to express frustration and a larger number of disruptive behaviors as compared with the cocaine-unexposed children and cocaine-exposed girls. This suggests that cocaine-exposed boys showed greater reactivity and less effective regulation. They appeared to have somewhat shorter fuses; that is, they became more quickly frustrated and more often acted out that frustration through complaints and aggression. However, Exposure $\times$ Gender differences were not found for all measures, and there were notable similarities between cocaine-exposed and -unexposed children. Cocaine-exposed boys were equally quick to approach the challenge and maintained the adaptive ability to generate instrumental and constructive problem-solving behaviors during the frustrating task. Moreover, cocaine-exposed girls showed almost none of the decrements evidenced by cocaine-exposed boys, and girls overall responded to the challenging task with significantly more instrumental behaviors than did boys. This resilience on the part of cocaine-exposed girls is intriguing and suggests the presence of gender-sensitive etiologies and trajectories in the development of psychopathology (Crick & Zahn-Waxler, 2003). These findings underscore the importance of acknowledging and seeking out examples of effective adjustment in cocaine-exposed children rather than emphasizing potential deficits. A number of studies document resilience in cocaine-exposed children, particularly those who receive adequate caregiving (Bennett et al., 2002; Brown, Bakeman, Coles, Platzman, & Lynch, 2004; Pulsifer et al., 2004; Singer et al., 2004).

Related to this, some children in our sample were living in foster care (23 children, 13%), 87% of whom were exposed to cocaine. Foster care was not associated with any differences in risk or outcome variables, except for greater maternal use of alcohol during pregnancy, $p < .001$. Yet, the quality of foster care is likely to have an impact on developmental outcomes. High-quality foster care could operate over time as a protective factor for cocaine-exposed children. However, foster care could be associated with a range of disruptions (e.g., loss of an attachment figure, instability due to multiple changes in foster care placement, little contact with biological caregiver or other kin) that could have a negative impact on adjustment. The complexity of this issue is not fully reflected in our environmental risk score. However, this score is based on assessment of the current caregiver (including foster caregiver) and does include factors such as stability of the environment. Future research would benefit from a careful assessment of foster care quality and stability.

Difficulties with the modulation of frustration and problem solving often have a reciprocal influence on each other (Dennis & Miller-Brotman, 2003; Derryberry & Rothbart, 1997; Rothbart & Bates, 1998). High levels of frustration likely have a negative impact on child persistence and problem solving during challenging tasks, whereas deficits in basic regulatory capacities, such as executive functions and cognitive control, are associated with increased frustration and aggression. These effects might have been compounded by cognitive decrements found in cocaine-exposed preschoolers, such as lower levels of visual-spatial skills, short-term memory, and general knowledge (Bennett et al., 2002; Singer et al., 2004).

This reciprocal influence between reactivity and regulation raises questions about their independence as constructs (Campos et al., 2004; Cole et al., 2004). It is difficult to distinguish behaviors associated with emotion from subsequent attempts to regulate emotion. For example, behaviors associated with frustration include increased persistence and working to overcome obstacles. These behaviors also serve to regulate frustration. Yet, there are important distinctions between emotional reactivity and regulation: Reactivity increases the probability of behavior but does not force the initiation of regulatory actions, and different regulation strategies have distinct implications for adjustment independent of emotional reactivity (e.g., Gross, 2002). Moreover, there is evidence that reactivity and regulation are distinct processes and that the balance between reactivity and regulation, rather than the strength of each alone, is a fundamental determinant of positive adjustment (Derryberry & Rothbart, 1997).

Although the results of this study were statistically significant, effect sizes were small (ranging from 2% to 3%). However, relatively small effects of cocaine exposure on reactivity and regulation may contribute to larger effects at older ages in this and other domains (Lester, 2000; Lester et al., 1998). This is consistent with an organizational perspective on development, which suggests that deficits in the negotiation of stage-salient tasks can have a strong impact on future development, even if initial problems are relatively small (Cicchetti et al., 1991). This occurs because the successful development of one competency increases the likelihood of successful development of subsequent competencies. Early capacities and abilities are thus carried forward, as are early vulnerabilities. This developmental principle may be particularly relevant to measures of reactivity and regulation. Reactivity and regulation can be thought of as gating mechanisms that direct and optimize attention, information processing, learning, and memory (Mayes et al., 1998). Moreover, chronically high reactivity or reduced regulation may be associated with increased activity of the stress response system, thus increasing child vulnerability for poor physical and mental health outcomes, particularly if children are exposed to stressful environments. Animal models also suggest that subtle behavioral and neuronal effects can have a powerful impact on the development of basic learning processes (e.g., Little & Teyler, 1996). For example, although cocaine-exposed rabbits react normally to standard learning stimuli, reducing the duration of stimuli by as little as several hundred milliseconds abolishes normal neuronal responding and impairs behavioral learning (Gabriel & Taylor, 1998).

Two aspects of this study may limit the generalizability of our findings. First, we examined frustration reactivity and regulation

during one task. It would have been optimal to assess children across multiple tasks during this assessment phase to obtain the most reliable measures of each construct. However, because the larger study focused on a broad array of functional skills, we only sampled this one behavior at 4.5 years. Replication using this and similar tasks would be necessary to confirm findings. Second, the retention rate was lower than desired by the 4.5-year assessment. Although this is a concern, participants did not significantly differ from those who refused to continue or who did not take part in this specific assessment in terms of cocaine exposure, environment and perinatal risk, or gender. This issue reflects the challenges inherent in working with families that experience multiple risk factors.

In addition, our measurements of cocaine exposure could have limited the specificity of our findings. Prenatal drug exposure information was collected retrospectively at the end of pregnancy and up to 2 weeks postpartum. A prospective design might have improved accuracy and reduced reporting biases. However, because poor, drug-using women often do not obtain early or consistent prenatal care, limiting the sample to women who had prenatal care throughout pregnancy would have resulted in an inadequate representation of the highest risk group. In addition, the cocaine-exposure variable was a dichotomous variable (used or did not use at any time during pregnancy). Details about cocaine exposure, such as the timing and amount of exposure, are absent from this variable. It is acknowledged to be very difficult at this juncture to obtain reliable information about timing and amount of exposure, as these variables are usually based on self-report and were entirely based on self-report in this study. Knowing if a woman used any cocaine during pregnancy from self-report and meconium analysis seems to be a more reliable although possibly less sensitive measure of cocaine use. Research conducted when more reliable methods of determining timing and amount of exposure become available should investigate whether such parameters of cocaine exposure influence patterns of effects.

The strengths of this prospective longitudinal study of prenatal cocaine exposure included observational assessment techniques, a focus on child competencies, and a theoretical framework rarely applied to studies of prenatal cocaine exposure: temperamental reactivity and regulation as they relate to the development of adjustment and maladjustment. Results illustrate remarkable resiliency but hint at the presence of vulnerability points, particularly in cocaine-exposed boys: greater frustration reactivity and greater difficulty in regulating behavior during problem solving. These results have implications for intervention and prevention, suggesting that treatment of children prenatally exposed to cocaine should anticipate specific problems related to reactivity, regulation, and problem solving while capitalizing on significant strengths and signs of positive adjustment. It is critical to conduct further longitudinal studies of prenatal cocaine exposure to delineate developmental trajectories toward risk and resilience.

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