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Inhibitory Motor Control at Five Years as a Function of Prenatal Cocaine Exposure

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Abstract

This study examined children's (n = 140, age 5 years) ability to inhibit a motor response as a function of prenatal cocaine exposure. We hypothesized that cocaine-exposed children would perform worse than unexposed children on the Contrary Tapping task. Results indicated that cocaine exposure, high environmental risk, male gender, and low child IQ each were related to poorer inhibitory control. An interaction indicated that cocaine effects were specific to children who lived in relatively low-risk environments. Cocaine-exposed children made an error sooner than unexposed children if they lived in low-risk environments but not if they lived in high-risk environments. Potential underlying mechanisms and the importance of examining cocaine exposure effects in the context of children's existing environment are discussed.

Index terms

cocaine exposure; inhibitory control; impulsivity

The effects of cocaine on human development have received much attention in the last 20 years since "crack" became available and large numbers of children have been exposed to it during gestation.[1] There is accumulating evidence that the development of emotional and regulatory functions is likely to be affected by prenatal cocaine exposure.[2–8] This is consistent with the mechanism of effect of cocaine on the developing central nervous system. Prenatal exposure to cocaine is likely to affect regulatory control through its action on the monoaminergic neurotransmitter systems, in particular the dopamine (DA) system in the mesolimbic and mid-prefrontal cortices.[9–13] These brain regions are believed to provide the neuronal substrates

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This study explores whether prenatal cocaine exposure alters one aspect of behavioral control— the ability to inhibit a response under changing circumstances, part of the array of abilities under executive functioning. It is refreshing to see a report investigating mental functioning in this group of youngsters that goes beyond standard measures of global intelligence. The authors have highlighted the contribution of the postnatal environment to a child's abilities in this area, something that comes as no surprise to anyone working with a population in which drug use is a factor in children's lives. Children in low-risk environments do better than children who remain in high-risk circumstances. The analytic model is innovative but solid, according to our statistical reviewers, taking into account the interactional nature and non-normally distributed feature of these data. The effect size of these factors is small, so let the reader exercise caution in generalizing the results. —Editor

for inhibiting prepotent responses, shifting attention to less salient stimuli, and making complex decisions requiring planning.[10,14–18] Several studies have found that children prenatally exposed to cocaine show greater difficulty inhibiting a voluntary response. Bendersky and Lewis[4] found that at 2 years of age cocaine-exposed children were less able to inhibit reaching for and taking a cookie when told not to. Similarly, Mayes and Grillon[7] found that cocaine-exposed children showed greater impulsivity in a continuous performance task at 4½ and 5½ years of age.

In examining whether prenatal exposure to cocaine impairs children's later functioning, confounding factors, such as prenatal exposure to other drugs, have to be controlled. Women who use cocaine also tend to drink more alcohol, smoke more cigarettes, and use more marijuana than those who do not use cocaine.[14,15] Prenatal exposure to these substances may impact inhibitory control in their own right. Effects of environmental risk and gender also must be considered. Environmental variables such as poverty, high life stress, and maternal social isolation are likely to have a negative impact on developmental outcomes and are generally more prevalent in children exposed to cocaine.[16] In addition, gender seems to play an important role in determining the effects of cocaine exposure. For example, male rats gestationally exposed to cocaine have been found to perform more poorly in a reversal acquisition task[17] and on a motor task[18] than exposed female rats. In a study of 6-year-old children, boys exposed to cocaine, but not girls, were reported by their teachers to be more likely to have clinically significant externalizing and delinquent behavior problems than unexposed children.[5]

In this study we examined whether prenatal exposure to cocaine resulted in poorer inhibitory control at 5 years of age using Contrary Tapping, a motor control task. For this task, subjects must tap twice if the experimenter taps once and tap once if the experimenter taps twice. Luria[19] found adults with frontal lobe damage to exhibit impaired performance on this task, which has since been widely used in neurological assessments of patients with frontal lobe damage.[20] Among children, Diamond and Taylor[20] documented that performance on the tapping task improves between age 3 and 6 years, and suggest that this improvement is related to important changes within the developing frontal cortex. Hence, Contrary Tapping may assess inhibitory control in frontal brain regions believed to be damaged by prenatal cocaine exposure.

The specific questions addressed in this study included the following: (1) Do cocaine-exposed children have greater difficulty inhibiting the prepotent tendency to mimic the experimenter's action? (2) Are environmental risk and general child cognitive functioning (i.e., IQ) important factors related to children's inhibitory control capacity? (3) Are boys at heightened risk for poor inhibitory control? (4) Do gender, environmental risk, and child IQ moderate the effects of cocaine in this inhibitory control task?

METHOD

Participants

One hundred-forty children were studied at 5 years of age. Participants (70 boys; 70 girls) were a mean age of 5.1 years (SD = 0.1) at the time of the study. Ninety-two were unexposed to cocaine during pregnancy, and 48 were exposed to cocaine. Children were predominantly African-American (86%), with 11% European-American and 3% Hispanic- or Asian-American. Pregnant women attending hospital-based prenatal clinics and who had just delivered at hospitals in low socioeconomic status areas of Trenton and Philadelphia were recruited for a longitudinal study on the effects of prenatal exposure to cocaine. Of these, 82% (n = 384) agreed to participate. Children were excluded from the study if they were born before 32 weeks of gestation, required special care or oxygen therapy for more than 24 hours, exhibited congenital anomalies, were exposed to opiates or PCP in utero, or were born to mothers infected with HIV (n = 63). An additional 18 subjects were lost to the study because they 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.

Of 258 children who participated in the first laboratory visit at 4 months of age, 188 (73%) participated in the 5-year laboratory visit. Of the 70 families not seen at 5 years, 15 moved out of the area, 28 declined to participate, 18 could not be contacted, 1 child and 2 mothers died, and 6 subjects went to foster parents who declined to participate. There were no significant differences in the distributions of cocaine exposure, gender, perinatal medical risk, or environmental risk between subjects who participated and those who refused to continue or were lost to the study from the neonatal period through 5 years of age. Three subjects, however, had missing data (e.g., did not complete the laboratory visit). In addition, 33 of the remaining 185 subjects failed to successfully complete a practice trial and hence were not administered the Contrary Tapping task. Cocaine exposure was not related to whether children completed a practice trial successfully ($\chi^2[1] = 0.01$, $p > .10$). Of the remaining 152 children, 12 had missing data regarding their level of cocaine exposure because (1) their biological mother could not be reached for interview and foster parents or alternate caregivers were unaware of their exposure level (n = 8), (2) the child tested positive for cocaine in the meconium screen but his or her mother denied cocaine use during the initial interview (n = 3), or (3) the biological mother agreed to participate in the study but refused to complete the perinatal substance use interview (n = 1).

Procedure

This study examined the ability to remember a rule and inhibit a prepotent response using the Contrary Tapping task.[20] In this task, immediately after the experimenter tapped once with a wooden dowel, the child was to tap twice with the dowel; when the experimenter tapped twice the child was to tap once. The experimenter explained the rules to the subjects and had them practice each rule immediately after the instructions. Then the experimenter administered two practice trials of each condition in random order to be sure the child understood the task. Testing began if the child passed this pretest. If not, another attempt was made to teach the task, and again the pretest was administered. As noted previously, 33 subjects failed to pass the pretest the second time and were excluded from further analyses. Each session consisted of a series of 16 trials using a randomly generated sequence of single- and double-tap trials. All subjects received the same order of trials. In each trial the experimenter tapped and then immediately gave the stick to the child for the response. The procedure was videotaped.

Measures

Inhibitory Control—The trial in which the child made the first error and the number of correct responses given by the child on the 16 trials were coded as measures of motor inhibitory control capacity. These two measures were highly correlated ($r = .71$, $p < .001$), suggesting that children who made the first error earlier also tended to have a generally poor performance. Because the trial of first error had greater variability, it was used as the measure of inhibitory control capacity. The results were similar using the number of correct responses.

Prenatal Substance Exposure—Prenatal substance exposure information was obtained through a semi-structured interview administered to the mother by trained interviewers (substance abuse counselors or study personnel trained in substance use interview techniques) within 2 weeks of the infant's birth. Interviews were conducted in an examination room at the prenatal clinic, in the mother's room in the maternity ward if she had just delivered, in our laboratories near the hospital, or in the woman's home. The drug use interview contained questions about the frequency, amount, and trimester of the mother's use of cocaine; the form of cocaine used; the frequency of the mother's use of prescription and nonprescription medications, as well as other substances (8-point scale, from 0 = "no use" to 7 = "daily use");

the disruptiveness of substance abuse to her life; and the history of her substance abuse. Substance interview information was confirmed by analysis of the newborn's meconium using radioimmunoassay followed by confirmatory gas chromatography-mass spectrometry.

Environmental Risk Score—Demographic and lifestyle information were obtained through structured interviews administered to the mother when the subjects were 4½ years of age. These interviews included questions about the mother's race, educational achievement, single-parent household, sources of income, maternal history of substance abuse, number of children in the household, the number of caregivers, regularity of the child's schedule (i.e., 15 items assessing whether routine behaviors such as eating breakfast, taking a bath, and going to bed occur "the same time every day" [1], "same time every day except for weekends" [2], "varies day-to-day within a 3-hour time period" [3], or "varies day-to-day more than 3 hours" [4]), stability of surroundings (i.e., number of changes in the "bed child sleeps in," "room child plays in," and so forth, during the past 6 months), social support measured with the Norbeck Social Support Questionnaire,[21] and maternal life stressors based on the Social Environment Inventory.[22] Table 1 contains descriptive statistics of each environmental risk variable.

In general, there were no significant differences between cocaine-exposed and unexposed children on the individual variables. As seen in Table 1, however, mothers of cocaine-exposed children were less likely to be residing with a mate (χ^2 [1] = 7.56, $p < .01$), were more likely to be receiving public assistance (χ^2 [1] = 7.51, $p < .01$), and reported higher levels of social support (t[55.9] = 2.15, $p < .05$). The variables were standardized into z scores, reverse coded if necessary so that the higher the value the greater the risk, and summed to produce a cumulative risk score. This cumulative risk score was then rescaled as a t score (mean = 48.42, SD = 8.92, range = 26.78–77.37).[3] Cumulative environmental risk measures have been found to explain more variance in children's outcomes than single factors, including socioeconomic status.[23,24]

General Cognitive Functioning (IQ)—At 4 years, children were administered the Stanford-Binet Intelligence Scale, Fourth Edition (SB-IV).[25] The SB-IV subscales of abstract and visual reasoning, quantitative reasoning, short-term memory, and verbal reasoning were standardized and summed to produce a composite IQ score. The SB-IV has extensive standardization data and satisfactory psychometric properties, including with African-American children.[25,26] The SB-IV also has high 2-year test-retest reliability for 4-year-old children[26] and thus was not repeated at the 5-year laboratory visit.

RESULTS

Because children's outcomes may be related to level of exposure,[4,27] the cocaine-exposed children were divided further into those whose mothers reported using cocaine less than twice per week on average (*lightly exposed*, n = 22) and those whose mothers used cocaine at least twice per week (*heavily exposed*, n = 26). Exposure is described in terms of the number of days per week that cocaine was used because the purity and dosage of street drugs is so variable. The definitions of heavy and light exposure have been used in prior studies.[3,28,29] Women who used cocaine frequently during pregnancy also consumed a larger number of daily alcoholic drinks than the other groups (1 drink = 1 oz liquor, 4 oz wine, or 12 oz beer; unexposed, mean = 0.03, SD = 0.14; lightly exposed, mean = 0.56, SD = 1.31; heavily exposed, mean = 2.72, SD = 4.34; F[2, 137] = 19.80, $p < .001$; post hoc $p < .05$, Duncan multiple range test). Both groups of cocaine users smoked significantly more cigarettes than women who did not use cocaine but did not differ from each other (unexposed, mean = 1.59, SD = 4.72; lightly exposed, mean = 8.50, SD = 9.22; heavily exposed, mean = 10.25, SD = 8.43; F[2, 137] = 24.32, $p < .001$; post hoc $p < .05$, Duncan multiple range test). There also was a trend for a

difference in the number of marijuana joints smoked per day ($F[2, 137] = 2.84$, $p = .06$). Women who used cocaine frequently during pregnancy used more marijuana than did those in the other groups (unexposed, mean = 0.03, SD = 0.21; lightly exposed, mean = 0.04, SD = 0.13; heavily exposed, mean = 0.54, SD = 2.28; post hoc $p < .05$, Duncan multiple range test).

A generalized linear model (GLM) was used to evaluate the effects of cocaine exposure, gender, environmental risk, child IQ, and their interactions on Contrary Tapping performance. GLM, unlike multiple linear regression, allows models to befit to data that follow probability distributions other than the normal distribution.[30] The Poisson distribution with log link was used in the analysis. It was considered more appropriate than the normal distribution because the trial at which the first error occurred is a count measure. Significance levels were determined using a χ^2 distribution. Terms were entered sequentially into the GLM, as shown in Table 2. Exposure to alcohol, cigarettes, and marijuana during pregnancy each were not significantly related to the trial of first error and thus were not used as covariates.

Environmental risk was entered in the first step, followed by gender, prenatal cocaine exposure, and child IQ. This ordering tested whether prenatal cocaine exposure was related to the trial of first error over and above the effects of environmental factors and gender. Child IQ was entered in the fourth step to examine the contribution of general cognitive functioning to Contrary Tapping performance. Because cocaine exposure may negatively affect children's cognitive functioning,[31–33] we entered cocaine exposure before child IQ to examine exposure effects on Contrary Tapping independently of IQ. In the fifth step we examined 2-way interactions between predictor variables to test for moderator effects. Prior research, for example, has found gender to interact with cocaine exposure in predicting child IQ such that exposed boys, but not girls, were found to have lower IQs.[31]

Main Effects: Environmental Risk, Gender, Cocaine Exposure, and IQ

Environmental Risk—As can be seen in Table 2, environmental risk was significantly associated with the trial of first error. Children from high-risk environments made their first error sooner than those living in low-risk environments. Environmental risk scores were quartiled to determine whether this was a linear effect. The means from least to highest risk were $mean_{Q1} = 6.83$, $mean_{Q2} = 7.53$, $mean_{Q3} = 7.36$, and $mean_{Q4} = 6.10$, indicating that the highest risk group made errors faster than the other three groups. GLM analysis confirmed that this high-risk group was significantly different from quartiles 2 and 3 (ps $< .05$), but not from quartile 1. Environmental risk was dichotomized in subsequent analyses examining interactions such that high risk indicates the highest 25% of the sample.

Gender—As can be seen in Table 2, there was a significant main effect for gender. Examination of the mean trial of first error indicated that boys made errors sooner than girls ($mean_{Boys} = 6.16$, SD = 5.28; $mean_{Girls} = 7.83$, SD = 5.99, $p < .001$).

Cocaine Exposure—Cocaine showed a significant effect such that exposed children made errors sooner than unexposed children ($mean_{Exp} = 6.42$, SD = 5.30; $mean_{NExp} = 7.29$, SD = 5.89, $p < .05$). Examination of the different levels of exposure revealed no differences between lightly versus heavily exposed children.

Child IQ—After controlling for the effects of environmental risk, gender, and cocaine exposure, child IQ also showed a significant effect (Table 2). A subsequent median split found children with lower IQ scores made errors sooner on the Contrary Tapping task ($mean_{LIQ} = 5.85$, SD = 5.54; $mean_{HIQ} = 8.18$, SD = 5.81, $p < .05$).

Two-Way Interactions

As shown in Table 2, the block of two-way interactions contributed significant variance to the prediction of Contrary Tapping performance. Environmental risk was a moderator of cocaine exposure such that cocaine-exposed children made an error sooner only if they lived in low-risk environments ($mean_{ExpLRsk}$ = 6.47, SD=5.33; $mean_{NExpLRsk}$ = 7.63, SD = 6.02; $p < .05$). No difference was found between exposed and unexposed children living in higher risk environments ($mean_{ExpHRsk}$ = 6.29, SD = 5.41; $mean_{NExpHRsk}$ = 6.10, SD = 5.36). Unexposed children living in low-risk environments made their first errors significantly later than each of the other three groups (*p*s < .05).

IQ also moderated the effects of cocaine exposure. Although there was no main effect for light versus heavy cocaine exposure on Contrary Tapping performance, level of exposure did interact with IQ. Among children who were unexposed or only lightly exposed to cocaine, low IQ was associated with making an error sooner ($mean_{N/LExpLIQ}$ = 5.75, SD = 5.56; $mean_{N/LExpHIQ}$ = 8.41, SD = 5.96; $p < .05$). However, for children with high levels of exposure, IQ was unrelated to when the first error occurred ($mean_{HExpLIQ}$ = 6.19, SD = 5.64; $mean_{HExpHIQ}$ = 6.50, SD = 4.50). Of note, children with IQ scores below the median were disproportionately likely to have been cocaine exposed ($\chi^2[1] = 9.17$, $p < .01$).

IQ and gender also interacted. This seems to be explained by a trend for boys with low IQs to make errors sooner than girls with low IQs ($mean_{B\text{-}LIQ}$ = 4.86, SD = 4.78; $mean_{G\text{-}LIQ}$ = 7.03, SD = 6.20, $p = .10$). However, gender did not differentiate the trial of first error among children with high IQs ($mean_{B\text{-}HIQ}$ = 7.62, SD = 5.73; $mean_{G\text{-}HIQ}$ = 8.62, SD = 5.91).

Finally, a trend was found for cocaine exposure and gender to interact; exposed boys tended to make errors sooner than exposed girls ($mean_{ExpB}$ = 4.95, SD = 4.11; $mean_{ExpG}$ = 7.46, SD = 5.85, $p = .09$). However, gender did not differentiate trial of first error among unexposed children ($mean_{NExpB}$ = 6.64, SD = 5.64; $mean_{NExpG}$ = 8.07, SD = 6.14).

DISCUSSION

In the current study, we compared the performance of 5-year-old children who were prenatally exposed to cocaine with that of unexposed children on a conflict inhibitory control procedure (i.e., a task in which the object was not only to withhold an impulsive response but also to provide an incompatible motor response). We found that cocaine-exposed children had greater difficulty inhibiting the prepotent response of imitation of the experimenter's action because they succeeded in fewer trials before making the first error on the Contrary Tapping task. This is consistent with findings of poorer impulse control in prenatally exposed children from the same cohort at 2 years of age.[4] In that study, cocaine-exposed children were quicker than unexposed children to reach for, take, and eat a cookie when they were told not to.

The relation between exposure to cocaine and inhibitory control has not been investigated extensively in preschool-and school-aged children. Espy and colleagues[34] found cocaine-exposed toddlers to exhibit less inhibition and poorer emotional regulation. In addition, 6-year-old cocaine-exposed children were reported to have lower rates of sustained attention, which is believed to be related to inhibitory control,[35] than unexposed children in a continuous performance task.[8] Faster responding with an increased number of commission errors, indicating an impulsive response style, also has been reported for cocaine-exposed children in continuous performance tasks at 4½ and 5½ years.[7] Together with the findings from the current study, this body of work supports the hypothesis of an association between in utero cocaine exposure and increased difficulty inhibiting prepotent or salient responses in older children.

Such inhibitory control deficits may be related to memory deficits. Short-term memory, and in particular working memory, deficits have been related to poor inhibitory control.[35] Cocaine exposure has been found to produce short-term memory deficits in rats.[36] Furthermore, children exposed to cocaine have been found to show short-term memory deficits. Singer and colleagues,[37] for example, found exposed infants to exhibit deficits on a visual recognition memory task, whereas cocaine-exposed children in the present sample exhibited lower scores on the short-term memory scale of the Stanford-Binet Intelligence Scale, Fourth Edition (SB-IV) at age 4 years.[31] A relation between inhibitory control and working memory is not unexpected, especially because both depend on development of the prefrontal cortex.[38]

There are further reasons to suspect that development of inhibitory control and cocaine exposure may be related. Both animal and human studies have shown that prenatal exposure to cocaine particularly affects the development of monoaminergically innervated regions, such as the mesolimbic and mid-prefrontal cortices.[9,10,12] Inhibitory control capacity has been argued to be the hallmark of frontal lobe function,[19,39] and deficits have been found both in adults with severe damage in the frontal cortex[40] and children born with phenylketonuria, a disorder that alters the levels of dopamine (DA) in the frontal lobe.[41] The mesolimbic area also has been found to support inhibition control processes. The DA neurons have terminals in the medial prefrontal cortex and the anterior cingulate (AC) nucleus. The AC has a close relationship with the basal ganglia, which provide DA innervation from the ventral tegmental area and cortical outflow of the limbic system, resulting in close integration with emotion systems.[40,42,43] The “limbic cortex” consists of the medial orbitofrontal cortex and the AC. These structures are implicated in inhibitory or effortful control requiring suppression of a dominant response to perform a subdominant one and are believed to be the neuronal substrates of emotional regulation and modulation.[44,45] The interaction between subcortical DA systems and prefrontal cortex plays a key role in inhibiting prepotent responses, shifting attention to less salient stimuli, and making complex decisions requiring planning.[10,41,46–49]

Inhibitory control is also related to factors such as gender and environmental risk. Delaney-Black and colleagues,[5] for example, found that prenatal exposure, gender, and postnatal environmental risk factors all were related to teacher-assessed externalizing behaviors in school-aged children. Postnatal environmental factors in particular were found to predict attention and externalizing behavior problems. These findings indicate that both cocaine exposure and environmental risk factors may be related to child functioning and raise the question of whether exposure and environmental risk might interact to predict functioning.

Our results confirm the existence of environmental risk effects, because children living in the highest risk environments made the first error sooner than those living in low-risk environments. We also found an interaction between environmental risk and cocaine exposure. As expected, unexposed children in the lowest risk environments had the best performance on the task, because they successfully completed more trials before making an error. Cocaine-exposed children living in low-risk environments were faster in making the first error than unexposed children living in the same environment. Unexpectedly, however, cocaine exposure was not related to Contrary Tapping performance among children in high-risk environments, suggesting that environmental conditions may play a more important role than cocaine exposure for children in such high-risk environments.

In the current study a trend also was found for gender to moderate the effect of cocaine exposure on inhibitory control. Although boys in general made errors sooner than girls, cocaine-exposed boys tended to perform the worst. The main effect for gender is consistent with the traditional view of boys as more impulsive and with other studies on the development of inhibitory control.[39,50] The gender-specific effect of cocaine exposure also is consistent with previous

research[5,17] indicating that boys exposed to cocaine may be more at risk of poor development of inhibitory control than girls, although the reasons are still unclear.

Child IQ also moderated the effects of cocaine exposure. Low IQ was associated with poorer Contrary Tapping performance only for children who were unexposed or lightly exposed to cocaine and not for those who were heavily exposed to cocaine. Given that this finding cannot be attributed to children with a high IQ receiving lower exposure to cocaine, the finding suggests that if a child's general cognitive functioning is unaffected by cocaine exposure then the child's Contrary Tapping performance is also unaffected by cocaine exposure. However, given that child I Q was a significant predictor of Contrary Tapping performance in the present sample, and that a higher proportion of cocaine-exposed children were below the median on IQ, these findings should not be interpreted as indicating that cocaine has no effect on Contrary Tapping performance.

The examination of cocaine exposure effects on children's inhibitory control is still in its early stages. The present study has several strengths, because it controlled for environmental risk and examined potential moderators of cocaine exposure, namely, environmental risk, gender, and child IQ. Nonetheless, several limitations deserve mention. First, our findings are specific to Contrary Tapping and need to be extended using other assessments of inhibitory control because such measures tend to be only moderately intercorrelated.[51] Second, at present it is unclear whether such cocaine effects on inhibitory control improve or worsen with age, which would be important to document given that the prefrontal cortex continues to develop through adolescence.[38,52] Hence, our findings do not necessarily generalize to older children or adults who are prenatally exposed to cocaine. More research integrating biological, socialization, and developmental processes on the development of inhibitory control is needed, particularly because difficulties inhibiting prepotent responses are likely to manifest in later problems in social regulation,[50,53] impulsivity, high-risk behavior, and aggression. Third, the self-report measure of gestational cocaine use was collected retrospectively at the end of the pregnancy. This may have led to some unreliable reports of level of cocaine use early in the pregnancy as the result of memory failures. However, exposure to cocaine was confirmed by assay of the newborn's meconium, and both meconium and report had to be negative for an infant to be classified as unexposed. If a woman denied use, but the meconium assay was positive, the subject was not used in this study. Therefore, we are very confident that the measure of whether or not the subject was exposed to cocaine during gestation is reliable. Finally, the analyses had sufficient power to detect large differences; however, the data indicate relatively small effect sizes. Thus, future research must verify these findings using relatively large samples.

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Table 1

Descriptive Statistics for Environmental Risk and Contrary Tapping Variables

Environmental risk	Cocaine-Exposed		Unexposed		Total Sample		
	Mean/%	SD/n	Mean/%	SD/n	Mean/%	SD/n	*p*
Life stressors (number endorsed)	6.46	4.30	6.36	4.49	6.39	4.42	
Minority race (% yes)	96	46	87	80	90	126	
Maternal education (yr)	11.98	1.30	11.60	1.52	11.72	1.46	
Number of children in household	3.51	2.00	3.11	1.77	3.24	1.85	
Number of regular caregivers	1.66	0.48	1.84	0.48	1.79	0.49	
Regularity of child's schedule	1.88	0.56	1.71	0.56	1.76	0.56	
Resides with mate (% yes)	23	11	47	48	39	54	*
Public assistance (% yes)	48	23	25	23	33	46	*
Social support[a]	16.14	8.91	12.74	6.24	13.82	7.33	*
Stability of child's surroundings	1.05	1.76	1.14	1.82	1.11	1.79	
Composite (*t* score)[b]	47.87	9.23	49.49	8.29	48.42	8.92	
Child IQ	84.00	11.81	88.03	10.74	86.74	11.21	*
Contrary tapping							
Number correct (of 16)	11.10	3.83	11.68	3.81	11.49	3.81	
Trial of first error	6.42	5.30	7.29	5.89	6.99	5.69	*

[a] Social support is computed as the number of "significant people in your life" multiplied by the mean supportiveness rating (1 = "not at all" to 5 = "a great deal" supportive).

[b] The composite score is a *t*-score based on the sum of z-scores from the 10 environmental risk variables.

* $p \leq .05$.

Table 2

Generalized Linear Model to Predict Trial of First Error During Contrary Tapping Task

	^{2}(Deviance)	*p*	R^2
1. Environmental risk	5.93	.01	.04
2. Gender	14.14	.00	.14
3. Cocaine exposure	5.31	.02	.17
4. Child IQ	9.23	.00	.22
5. Two-way interactions	(20.52)	(.00)	.33
Cocaine × environmental risk	5.60	.02	
Cocaine × gender	3.34	.07	
Cocaine × IQ	9.94	.00	
IQ × gender	6.82	.01	
IQ × environmental risk	0.01	.91	