

The Effect of Prenatal Drug Exposure and Caregiving Context on Children's Performance on a Task of Sustained Visual Attention

John P. Ackerman, PhD, Antolin M. Llorente, PhD, Maureen M. Black, PhD, Claire S. Ackerman, MA, Lacy A. Mayes, MA, Prasanna Nair, MD

ABSTRACT: *Objectives:* Three groups of children from low-income, urban environments were examined to determine the effects of prenatal drug exposure (PDE) and caregiving environment on sustained visual attention (SVA) at 7 years of age. *Methods:* Drug-exposed children remaining in maternal care ($n = 43$), drug-exposed children placed in nonmaternal care ($n = 45$), and community comparison (CC) children ($n = 56$) were administered a battery of neurocognitive tests, including the Conners' Continuous Performance Test (CPT). *Results:* PDE children remaining in maternal care displayed more omission errors than CC children. PDE children in nonmaternal care had intermediate scores that did not differ significantly from PDE children in maternal care or CC children. There were no group differences with respect to commission errors or reaction time. CPT errors of omission and commission were significantly correlated with parent-reported attention problems and academic achievement scores. *Conclusions:* PDE in the context of care provided by a maternal caregiver with persistent drug use patterns may contribute to problems in children's SVA at school-age. As parental drug abuse can interfere with the provision of early care, children raised in a drug-using context may be highly vulnerable to problems with self-regulation, including sustained attention. SVA problems may contribute to subsequent academic and behavioral problems as demands for concentration and sustained effort increase throughout childhood. Children who have been prenatally exposed to drugs or raised in a drug-using household may benefit from early intervention services to avoid problems in SVA that may interfere with subsequent neurocognitive functioning and academic performance.

(*J Dev Behav Pediatr* 29:467–474, 2008) **Index terms:** sustained visual attention, prenatal drug exposure, Conners' CPT.

Studies examining links between prenatal drug exposure (PDE) and children's neurocognitive development have produced mixed findings. In early childhood, difficulties have been documented in language development, arousal regulation, and attentional functioning after exposure to commonly abused drugs, such as cocaine and heroin.¹ By school-age, however, many of the associations between PDE and neurocognitive outcomes are attenuated when postnatal environmental factors are included in statistical models.^{2,3} These findings are not surprising given the critical role of caregiving in children's cognitive and behavioral development.⁴ Only recently have studies begun to emphasize the importance of environmental confounds, such as poverty, prenatal exposure to tobacco and alcohol, growth indicators, and variations in caregiving when evaluating how PDE influences children's neurocognitive functioning.^{5–8} Thus, PDE is a risk factor not only for neurocognitive problems, but also for environ-

mental challenges that can adversely affect children's development.⁹

There has also been a methodological shift away from a reliance on global assessments in favor of targeted neurocognitive outcomes. PDE-related differences in intelligence quotient and global development are often attenuated after accounting for environmental factors.^{6,7,10} The few studies that detected exposure-related differences beyond the preschool years examined specific neurocognitive domains such as language, attention, and self-regulation.^{11–15} Each of these domains is critical for learning and social interactions.

Among school-aged children exposed to cocaine or heroin in utero, a consistent finding is that PDE leads to suboptimal performance on tasks of sustained attention.^{5,13,14,16–19} Several mechanisms are thought to link deficits in attentional functioning to PDE. Cocaine and heroin cross the blood-brain barrier in the developing fetus.²⁰ Both substances can permanently alter dopaminergic and noradrenergic pathways in the developing brain,^{21–23} and these pathways play a role in regulating attention.²⁴ PDE may also impair neuronal migration and disrupt brain cell proliferation,²⁵ which can subsequently interfere with performance on frontally mediated neurocognitive tasks.²⁶ Distractibility and disrupted arousal regulation have been documented as early as infancy in children prenatally exposed to cocaine and heroin.^{21,27}

From the Division of Growth and Nutrition, Department of Pediatrics, University of Maryland School of Medicine, Baltimore, M.D.

Received March 2008; accepted September 2008.

This study was supported by the National Institute of Drug Abuse (R01-DA07432 and R01-DA021059).

Address for reprints: Maureen Black, Ph.D., 737 West Lombard St., Baltimore, MD; e-mail: mblack@peds.umaryland.edu.

Copyright © 2008 Lippincott Williams & Wilkins

Despite an accumulation of data from studies with young children indicating that PDE is associated with sustained visual attention (SVA) deficits, the precise nature of these deficits remains unclear. Several studies have found that PDE is associated with inhibitory control problems on various neurocognitive measures.^{7,13,14,18,28} Others have found that PDE is associated with slow and variable reaction time and an inattentive response style.^{5,12,19} Problems with SVA can adversely affect children's ability to concentrate, process information efficiently, and retain information for later recall, suggesting that such deficits are clinically meaningful.

PDE may contribute to neurocognitive deficits directly through teratogenic effects, but also indirectly through environmental conditions that disrupt the development of self-regulatory skills necessary for adequate attentional functioning.²¹ For example, drug-using mothers are more likely to present with comorbid mental health problems, less likely to provide sensitive care or supervision to their children, less likely to provide effective discipline, and are at higher risk to lose custody of their children than non-drug-using mothers.^{29,30} Co-occurring risk factors of PDE such as poverty, low caregiver intellectual functioning, and ongoing maternal alcohol, tobacco, and drug use also likely contribute to neurocognitive deficits in children with a history of PDE who continue to live in parental care.^{6,9} Although some studies have found that out-of-home placements contribute to difficulties with inhibitory control in young children,³¹⁻³³ children raised in nonparental care may experience a more sensitive and stimulating cognitive environment than children who remain in the care of a substance-abusing caregiver.

This study examines how PDE and postnatal environmental conditions are related to children's SVA at 7 years of age. We test three hypotheses: (1) Children with a history of PDE perform more poorly than nonexposed children on SVA, controlling for relevant demographic and environmental factors. (2) Among children with a history of PDE, those who remained in maternal care perform more poorly on SVA than children placed in nonmaternal care. (3) Performance on a computerized task of SVA is associated with academic achievement and caregiver-reported attentional skills.

METHOD

Study Design and Participants

Participants were part of a longitudinal randomized, controlled trial of a home-based intervention among drug-using women and their infants approved by the University's Institutional Review Board.³⁴ Women were recruited from a University Hospital serving a predominately inner-city, African-American population. Women were eligible for recruitment if they or their infants had a urine toxicology screen at birth that was positive for cocaine or heroin or a self-reported history of cocaine or heroin use during pregnancy ($\geq 2x/wk$). Eligibility criteria included gestational age ≥ 32 weeks, birth weight $\geq 1,750$ g, and no medical complications requiring admission to the neonatal intensive care unit.

Two-hundred and sixty-five mothers agreed to participate and kept a baseline 2-week appointment. At the baseline visit, perinatal data were collected from the infants' medical chart and the Addiction Severity Index (ASI)³⁵ was administered to determine the duration and frequency of drug use during pregnancy. Mothers were randomly assigned to a home intervention or control group. Families in the intervention group received developmentally oriented home visits for 1 year, based on the program used by the Infant Health and Development Program.³⁶ Families in the control group received monthly tracking visits. All families were assessed at 6-month intervals by trained research assistants blind to participant drug-exposure or intervention status. Families were also given information on drug treatment programs and were compensated for each evaluation.

At 7 years of age, 128 child/caregiver pairs of the original 265 were available for assessment. Causes of attrition were child deaths 8 (3%), foster care placement 37 (14%), moved out of state 6 (2%), and family could not be located or withdrew from the study 87 (33%). There were no attrition differences in neonatal characteristics, maternal drug use, urine toxicology, or other demographic variables.

Among the 128 children in the prenatal drug exposure (PDE) group, 32.8% had a positive infant or mother toxicology screen for cocaine only, 14.1% were positive for heroin only, 30.5% were positive for *both* cocaine and heroin, 10.1% did not have a positive toxicology screen, but self-reported frequent cocaine or heroin use during pregnancy ($>twice/wk$), and 12.5% were excluded from analyses, because mothers reported infrequent drug use during pregnancy and did not have a positive toxicology screen for cocaine or heroin ($n = 16$).

Seventy participants from a non-drug-exposed community cohort (CC) group served as a community standard for comparison. They were recruited from a primary care clinic serving the University Hospital when they were 5 years old. Medical records were reviewed to identify children delivered at the University Hospital at the same time as children from the PDE group, both the mother and infant had negative toxicology screens, and there was no history of drug use in the mother's medical records. Participants resided in the same community as participants in the PDE group and were matched for socioeconomic status, age of first pregnancy, gender, and race. At 7 years of age, 61 child/caregiver pairs from the CC group were available for assessment and all children in the CC group resided with their birth mother.

Child Measures

Conners' Continuous Performance Test

The Conners' Continuous Performance Test (CPT) assesses sustained visual attention (SVA) or the ability to discriminate between target and nontarget stimuli presented at variable intervals.³⁷ The CPT was completed on an IBM-compatible desktop computer in our clinical laboratory. Standard administration of the CPT consisted of 360 letters, which appeared on the computer screen, one at a time, for approximately 250 ms. Trials were pre-

sented in 18 blocks of 20 trials each. The 18 interstimulus interval (ISI) blocks consisted of a separate block-randomized ISI (1, 2, or 4 sec). Participants were required to press the spacebar when any letter except "X" appeared on the screen. The task took approximately 14 minutes to complete. The Conners' CPT was selected because of its potential sensitivity in detecting long-term neurobehavioral effects of PDE. Split-half reliability for all CPT-dependent variables range between 0.73 and 0.95 among healthy children. Test-retest reliabilities for a 3-month interval range between 0.55 and 0.84.

Dependent variables related to SVA included (1) number of omission errors, (2) number of commission errors, and (3) reaction time to correct responses. Omission errors occurred when subjects failed to press the spacebar on trials containing non-"X" letters. Commission errors occurred when subjects pressed the spacebar on trials when the letter "X" was presented. Reaction time to correct responses was a subject's mean response time to stimuli across all trials recorded in milliseconds. For CPT results to be valid, children must be somewhat motivated and understand task demands. If a child responded to less than 60% of the target letters (>3 SDs below the mean), his or her CPT protocol was deemed invalid.

Stanford-Binet Intelligence Scales: Fourth Edition

The Stanford-Binet Intelligence Scales—Fourth Edition (SB-IV) is a standardized test of intellectual functioning ($M = 100$, $SD = 16$) administered when children were 6 years of age to obtain an estimate of global intellectual functioning.³⁸ The SB-IV has excellent validity and reliability. Internal consistency coefficients for composite scores range from 0.95 to 0.99.

Wide Range Achievement Test-3

The Wide Range Achievement Test-3 (WRAT-3) was administered to children at 7 years of age and reliably measures basic skills in reading, arithmetic, and spelling.³⁹ Raw scores are converted into standard scores ($M = 100$, $SD = 15$).

Child Behavior Checklist

The Child Behavior Checklist (CBCL) was administered to caregivers when children were 7 years of age.⁴⁰ It is a 120-item caregiver-report form that assesses child behavior problems and has very good reliability and validity. In this investigation, we focused on the attention problems subscale to determine associations between SVA performance and caregiver-reported attention problems in the home environment.

Birth Weight for Gestational Age

The child's birth weight was recorded in the medical record at the University Hospital and standardized from the infant's gestational age and gender using Center for Disease Center norms.⁴¹ Birth weight for gestational age was used as an indicator of intrauterine physical growth and has been associated with several cognitive outcomes in childhood.⁴²

Nonmaternal Care

Changes in primary caregiver were recorded at each evaluation and defined as residing with a nonmaternal caregiver for at least one consecutive month. Number of

caregiver changes prior to 7 years of age was calculated. A dichotomous variable was also generated to indicate whether the child was in maternal or nonmaternal care at the time of each visit. On the basis of whether the child was in continuous maternal care (i.e., PDE in maternal care) or nonmaternal care (i.e., PDE in nonmaternal care) at the 7-year assessment, two subgroups were created. The majority of nonmaternal care placements (95%) were with kin caregivers (e.g., aunt, grandmother). Children who had a history of foster care but lived with a birth parent at the time of the evaluation were excluded from analyses because they did not fit criteria for either continuous maternal care or nonmaternal care ($n = 10$).

Caregiver Measures

Caregiver demographic data collected at the 7-year visit included caregiver age, education, marital status, relationship to the child, employment, and public cash assistance status. Additionally, the following measures of the caregiver environment were assessed.

Center for Epidemiological Studies—Depression

The Center for Epidemiological Studies—Depression (CES-D) is a valid 20-item assessment of depressive symptomatology.⁴³ This scale addresses multiple aspects of depression and requires respondents to rate their frequency of symptoms from 0 "rarely or never" to 3 "most or all the time," with higher summed scores indicating higher levels of depression.

Current Drug Use

Caregivers completed the ASI³⁵ and a drug use questionnaire to assess the frequency of current substance use. A dichotomous variable was generated reflecting self-reported cocaine and/or heroin use when children were 7 years old.

Kaufman Brief Intelligence Test

The Kaufman Brief Intelligence Test (K-BIT)⁴⁴ was used to measure caregiver intellectual ability. The K-BIT generates a composite score with a mean of 100 and a standard deviation of 15, comprised of verbal and non-verbal abilities. The validity and reliability of the K-BIT have been established among urban, African-American populations.⁴⁵

RESULTS

Preliminary Analyses

Based on our validity criteria for continuous performance test (CPT) responses (response to >60% of targets), 19 children were excluded resulting in a final sample of 144. Those with invalid CPT responses did not differ by drug exposure classification, nonmaternal care status, or other demographic variables; however, those with invalid CPT scores [mean Stanford-Binet Intelligence Scales—Fourth Edition (SB-IV) composite = 77.5] had significantly lower intelligence quotient (IQ) scores than those with valid CPT scores (mean SB-IV composite = 82.0), $t(183) = -4.61$, $p < .05$. Difficulty in understanding task instructions or following directions may have led to invalid response patterns.

Table 1. Sample Demographic Characteristics by Prenatal Drug Exposure and Caregiver Status

	Community Cohort (N = 56)	PDE in Maternal Care (N = 43)	PDE in Nonmaternal Care (N = 45)	ANOVA <i>p</i>
Baseline Assessment				
Birth weight (g)	3340 (592) ^{a,b}	2803 (402) ^a	2732 (397) ^b	<.001
Weight for gestational age (z-score)	-.09 (1.0) ^{a,b}	-.86 (.7) ^a	-.92 (.8) ^b	<.001
Head circumference (cm)	34.4 (1.7) ^{a,b}	32.8 (1.3) ^a	32.7 (1.5) ^b	<.001
Birth length (cm)	50.4 (2.5) ^{a,b}	48.1 (2.7) ^a	47.8 (2.4) ^b	<.001
Gender (% male)	45%	42%	42%	.95
Intervention status (%)	0 ^{a,b}	58 ^a	58 ^b	<.001
Prenatal tobacco exposure (%)	27 ^{a,b}	86 ^a	89 ^b	<.001
Prenatal alcohol exposure (%)	30	37	38	.68
Prenatal marijuana exposure (%)	0 ^{a,b}	37 ^a	22 ^b	<.001
Age 7 Assessment				
Caregiver age (at 7-yr assessment)	30.8 (5.4) ^{a,b}	36.8 (5.7) ^{a,c}	46.3 (10.7) ^{b,c}	<.001
Caregiver K-BIT standard score	82.0 (11.0)	82.8 (11.0)	79.8 (14.0)	.48
Caregiver education	11.6 (1.3)	11.5 (1.3)	11.3 (1.9)	.58
Caregiver married (%)	20	7	18	.19
Caregiver current drug use (%)	2% ^a	44 ^{a,c}	4 ^c	<.001
Caregiver unemployment (%)	9 ^{a,b}	28% ^a	40% ^b	<.001
Caregiver public assistance (%)	48	54	60	.50
Caregiver depressive symptoms	13.4 (10.8)	11.7 (10.1)	9.6 (8.0)	.15

Note: Post-hoc Tukey tests were run to determine significant between group differences on each outcome measure. ^aCC group differs significantly from the PDE maternal care group. ^bCC group differs significantly from the PDE non-maternal care group. ^cPDE maternal care group differs significantly from the PDE non-maternal care group. CC, community cohort; PDE, prenatal drug exposure; ANOVA, analysis of variance.

An initial comparison of children from the community cohort CC group ($n = 56$), the prenatal drug exposure (PDE) maternal care group ($n = 43$), and PDE nonmaternal care group ($n = 45$) revealed that both PDE groups differed from the CC group on indicators of neonatal growth and prenatal tobacco exposure (see Table 1). Caregivers of children in the CC group and PDE groups did not differ in age at first pregnancy, prenatal alcohol exposure, educational attainment, intellectual functioning, marital status, public assistance status, or depressive symptomatology at the time of the 7-year visit. Caregivers in the both PDE groups were significantly older and less likely to be employed than caregivers in the CC group. Among PDE children, those currently in nonmaternal care had caregivers who were older and less likely to be using illicit drugs than children living continuously in maternal care. As such, birth weight for gestational age, prenatal tobacco exposure, caregiver age, employment status, and current drug use were selected as covariates in primary analyses based on bivariate associations, whereas child gender, home-intervention status, prenatal alcohol exposure, and child IQ were selected as covariates based on a priori theoretical considerations.

Primary Analyses

To test the first two hypotheses that CPT performance differs by PDE history and caregiver group, we conducted two between-subjects univariate analyses of variance. First, we assessed whether PDE children performed more poorly than CC children on three CPT outcome variables

(i.e., omission errors, commission errors, and reaction time), controlling for potentially confounding variables identified in preliminary analyses. Univariate analysis of covariance (ANCOVA) indicated that PDE children had significantly more omission errors than nonexposed children, $F(1,134) = 4.37, p = .04, \eta^2 = .03$. Neither errors of commission nor mean reaction time were significantly associated with PDE history.

Caregiver group differences were examined (PDE in maternal care, PDE in nonmaternal care, and CC) on the same CPT outcome variables. ANCOVA revealed that errors of omission were significantly associated with PDE history and caregiver status, $F(2,133) = 3.22, p = .04, \eta^2 = .05$, controlling for potential confounding variables (see Table 2). Post-hoc Tukey tests revealed that PDE children in maternal care had significantly more omission errors than children in the CC group. PDE children in nonmaternal care had an intermediate number of omission scores that did not differ significantly from PDE children in maternal care or CC children. Neither errors of commission nor mean reaction time were significantly associated with PDE history and caregiver status, after controlling for potential confounding variables.

Finally, we tested the third hypothesis that sustained visual attention performance is associated with academic achievement and caregiver-reported attention problems. Associations between CPT performance and child intellectual, academic, and behavioral functioning at 7 years of age were assessed to establish convergent validity (see

Table 2. Child Cognitive and Behavioral Functioning at Age 7 years by Prenatal Drug Exposure and Caregiver Status

Sample Characteristics	Community Cohort (N = 56)	PDE in Maternal Care (N = 43)	PDE in Nonmaternal Care (N = 45)	ANCOVA <i>p</i>
Stanford-Binet composite (IQ)	83.9 (11.1)	83.0 (11.5)	79.0 (8.4)	.21
WRAT-3 reading achievement	97.9 (16.3)	94.1 (17.3)	94.7 (13.0)	.60
WRAT-3 spelling achievement	98.0 (18.7)	93.5 (15.9)	96.1 (12.0)	.51
WRAT-3 arithmetic achievement	92.6 (16.8)	89.2 (16.2)	90.0 (16.4)	.26
CBCL attention problems <i>t</i> score	48.6 (9.4)	49.0 (9.8)	52.2 (11.3)	.11
CPT total omission errors	34.1 (25.0) ^a	49.3 (32.9) ^a	37.3 (24.2)	.04
CPT total commission errors	22.1 (7.0)	21.8 (6.3)	23.4 (6.2)	.20
CPT mean reaction time (ms)	529 (95)	532 (82)	521 (105)	.42

Note: Group comparisons using ANCOVA were adjusted for child gender, birth weight for gestational age, prenatal tobacco exposure, prenatal alcohol exposure, intervention status, child IQ (except when IQ was the outcome), caregiver age, employment status, and caregiver current drug use. Post-hoc Tukey tests were run to determine significant between group differences on each outcome measure. ^aCC group differs significantly from the PDE maternal care group. CC, community cohort; PDE, prenatal drug exposure; ANCOVA, analysis of covariance; WRAT-3, Wide Range Achievement Test-3; CBCL, Child Behavior Checklist; CPT, continuous performance test.

Table 3). Number of CPT omission errors was negatively associated with child IQ as well as reading, spelling, and math achievement, and positively associated with caregiver-reported attention problems. Number of CPT commission errors was negatively associated with child IQ, reading achievement, spelling achievement, and marginally negatively associated with math achievement. There were no significant associations between CPT reaction time and child IQ, academic achievement, or caregiver-reported attention problems (*p*'s > .05). Thus, CPT performance, particularly the number of omission and commission errors, was associated with ecologically valid measures of intellectual, academic, and behavioral functioning.

DISCUSSION

We view the present findings from a biopsychosocial perspective that emphasizes the reciprocal influences of biological, psychological, and environmental factors on children's neurocognitive development.²¹ We found that children with a history of prenatal drug exposure (PDE) demonstrate greater difficulties with sustained visual attention (SVA) than demographically matched comparison children, even after statistically controlling for other economic, social, and environmental risk factors. This overall difference between PDE and community coherent children

indicates that exposure to cocaine and/or heroin in utero is associated with attentional problems that impair children's ability to attend to visual stimuli over time.

PDE poses the most significant risk for SVA deficits in the context of continuous maternal care, particularly in the presence of persistent drug use. In the present study, PDE children placed in nonmaternal care were typically removed from environments characterized by persistent maternal drug-use and were compared with children who remained in such an environment. Nearly half of the caregivers in the PDE maternal care group self-reported recent cocaine or heroin use. As caregivers in all groups were equivalent on most demographic, socioeconomic, and intellectual variables, the depressive symptoms that characterize drug using mothers^{29,30} and other aspects of a drug-using lifestyle may have interfered with the provision of sensitive and stimulating child care, thereby contributing to children's neurocognitive difficulties. Our results are consistent with evidence suggesting that children's neurocognitive functioning is vulnerable to non-optimal care in the first few years of life.^{3,4}

The present study extends findings of others who have reported that children with a history of PDE demonstrate difficulties on laboratory tasks of sustained attention.^{6,12-14,18,19} Studies that implemented the Conners' continuous performance test (CPT) found that PDE chil-

Table 3. Intercorrelations Among Child Cognitive and Behavioral Functioning Variables at Age 7 years

	1	2	3	4	5	6	7
CPT total omission errors	—						
CPT total commission errors	-.09	—					
CPT mean reaction time (ms)	.42*	-.39*	—				
SB-IV IQ standard score	-.16**	-.28***	-.05	—			
WRAT reading achievement	-.25***	-.18†	-.08	.54*	—		
WRAT spelling achievement	-.26***	-.22***	.01	.53*	.87*	—	
WRAT math achievement	-.22***	-.14**	-.10	.56*	.67*	.65*	—
CBCL Attention problem <i>t</i> -score	.19†	.08	-.01	-.28***	-.30*	-.29***	-.3*

Note: Children assessed at age 7 except for SB-IV, which was administered at age of 6 years. **p* < .001; ***p* < .01; ****p* < .001; †*p* < .05. CPT, continuous performance test; SB-IV IQ, Stanford-Binet Intelligence Scales—Fourth Edition; WRAT, Wide Range Achievement Test; CBCL, Child Behavior Checklist.

dren demonstrated more omission errors than nonexposed children at school-age.^{5,12,19} However, these studies did not specifically examine children's out-of-home placements because they related to CPT performance, but rather included a variable reflecting biological relatedness as a covariate in their analyses. Results from this study indicate that simply covarying out the effects of "nonmaternal care status" to reduce error variance does not sufficiently characterize a drug-exposed sample.

Drug-exposed infants and toddlers often display arousal modulation and selective attention problems.^{21,46-48} This initial vulnerability may present caregiving challenges that disrupt mother-child interactions, especially when occurring in the context of drug abuse. For example, previous findings from this cohort indicate that infants who remain in the care of drug-using mothers, even after the implementation of a developmentally oriented intervention, receive less responsive care and have worse cognitive outcomes than infants raised by non-drug-using mothers.^{34,49} The deficits displayed by children of drug-using mothers may result from factors associated with the mother's drug-using lifestyle such as low levels of sensitivity, structure, and limited emotional availability.^{34,39} Low levels of responsive care throughout early childhood can interfere with the development of basic self-regulatory skills such as sustained attention.⁵⁰ Overall, these findings reflect the importance of considering how children's caregiving context after PDE can affect the organization and efficiency of attentional networks.²¹

Children's performance on the Conners' CPT task was associated with academic achievement and caregiver-reported attention problems. CPT errors of omission and commission were predictive of low achievement and parent-reported attention problems. Although CPTs are not sufficient to diagnose ADHD, they are among the most frequently used tasks for detecting deficits underlying ADHD such as poor sustained attention and impulsivity.^{51,52} Children with attention problems exhibit poor performance on these measures, marked by frequent omissions of target stimuli, a highly variable and impulsive response style, or an abnormal reaction time.⁵³ Poor performance on these measures has been attributed to deficient regulatory functioning in the frontal lobes.⁵² The current study suggests that drug-exposed children, particularly those remaining in the care of mothers with persistent drug use, demonstrate less efficient SVA evidenced by increased omission errors and may be at increased risk for developmental problems. However, there remains unexplained variability in our models of children's SVA, suggesting that additional moderators likely need to be addressed in future studies.

This study suggests that children raised in low-income, drug-using environments are at risk for SVA problems. Children in the care of substance-using mothers may benefit from early intervention services that facilitate cognitive development, particularly those that focus on parent-child interactions to enhance children's developing self-regulatory skills.⁵⁴ Our results suggest that children raised by parents with a history of persistent drug use are

at risk for specific neurocognitive deficits and would benefit from targeted intervention services.

The current study has several limitations. The original sample was recruited to evaluate the effectiveness of an early developmental intervention. Data related to dosage of legal and illicit drugs were not available for the majority of subjects, limiting our ability to assess dose-related effects. Despite a lack of significant differences between children who remained enrolled in the study and children lost to follow-up, the high-attrition rate could have resulted in selection bias. We did not have adequate power to examine prenatal exposure effects pertaining to specific drugs, timing, or dosage, which can have important moderating effects.⁵⁵ It is difficult to determine the unique effects of specific substances on neurocognitive outcomes, because most women who use drugs during pregnancy engage in polysubstance use.⁵⁶ Caregiver's drug use at follow-up was assessed via self-report. It is likely that drug use was underreported, particularly among the biological mothers who may have been under less scrutiny from child protective services than nonmaternal caregivers. This study would have benefited from biological assays of ongoing caregiver substance use. Additionally, the inclusion of behavioral observations of caregiver-child interactions may have been useful in examining the potential mediating role of caregiver responsiveness.

The present study has several methodological strengths including the use of routine screening and enrollment of drug-exposed children at birth who were matched with a demographically similar group of comparison children. The inclusion of numerous potentially confounding variables in statistical analyses reduced the likelihood that associations between PDE and SVA could be attributed to co-occurring risk factors associated with maternal drug use. The use of academic achievement and intelligence quotient (IQ) data as well as caregiver report of attentional functioning to examine the convergent validity of computerized testing strengthen present findings. An examination of caregiving context within the group of PDE children contributed to a better understanding of the factors contributing to children's SVA.

In sum, PDE was associated with deficits in SVA in the context of continuous maternal care. Additionally, children in all groups displayed deficits in intellectual functioning and the ability to sustain concentration and focus on task goals over time relative to the entire population. Such findings confirm that poverty-related factors have an insidious effect on children's neurocognitive development, regardless of drug exposure.^{8,57} SVA deficits were correlated with problems in academic functioning and caregiver-reported attention problems suggesting that child attentional performance in a controlled testing environment is meaningfully associated with functioning in home and school environments. Although specific learning problems were not evident at the age of 7 years, poor SVA and low IQ may place children at risk for subsequent academic, behavioral, and social problems as environmental demands increase. Early intervention services, therefore, should be accessible not only to children who have been removed from parental care, but to those who

have not been identified by authorities. Interventions that focus on children's self-regulation of mood, motivation, and arousal are critical in providing a foundation for later academic success.⁵⁴

REFERENCES

- Kim J, Krall J. *Literature Review: Effects of Prenatal Substance Exposure on Infant and Early Childhood Outcomes*. Berkeley, CA: National Abandoned Infants Assistance Resource Center, University of California at Berkeley; 2006.
- Frank DA, Augustyn M, Knight WG, Pell T, Zuckerman B. Growth, development, and behavior in early childhood following prenatal cocaine exposure: a systematic review. *JAMA*. 2001;285:1613-1625.
- Brown JV, Bakeman R, Coles CD, Platzman KA, Lynch ME. Prenatal cocaine exposure: a comparison of 2-year-old children in parental and nonparental care. *Child Dev*. 2004;75:1282-1295.
- Shonkoff JP, Phillips DA. *From Neurons to Neighborhoods: The Science of Early Childhood Development*. Washington D. C.: National Academy Press; 2000.
- Accornero VH, Amado AJ, Morrow CE, Xue L, Anthony JC, Bandstra ES. Impact of prenatal cocaine exposure on attention and response inhibition as assessed by continuous performance tests. *J Dev Behav Pediatr*. 2007;28:195-205.
- Arendt R, Short E, Singer LT, et al. Children prenatally exposed to cocaine: developmental outcomes and environmental risks at seven years of age. *J Dev Behav Pediatr*. 2004;25:83-90.
- Hurt H, Brodsky NL, Roth H, Malmud E, Giannetta JM. School performance of children with gestational cocaine exposure. *Neurotoxicol Teratol*. 2005;27:203-211.
- Nair P, Black M, Ackerman J, Schuler M, Keane V. Children's cognitive-behavioral functioning at age 6 and 7: prenatal drug exposure and caregiving environment. *Ambul Pediatr*. 2008;8:154-162.
- Singer LT, Minnes S, Short E, et al. Cognitive outcomes of preschool children with prenatal cocaine exposure. *JAMA*. 2004;291:2448-2456.
- Messinger DS, Bauer CR, Das A, et al. The maternal lifestyle study: cognitive, motor, and behavioral outcomes of cocaine-exposed and opiate-exposed infants through three years of age. *Pediatrics*. 2004;113:1677-1685.
- Bada HS, Das A, Bauer CR, et al. Impact of prenatal cocaine exposure on child behavior problems through school age. *Pediatrics*. 2007;119:e348-e359.
- Bandstra ES, Morrow CE, Anthony JC, Accornero VH, Fried PA. Longitudinal investigation of task persistence and sustained attention in children with prenatal cocaine exposure. *Neurotoxicol Teratol*. 2001;23:545-559.
- Hickey JE, Suess PE, Newlin DB, Spurgeon L, Porges SW. Vagal tone regulation during sustained attention in boys exposed to opiates in utero. *Addict Behav*. 1995;20:43-59.
- Savage J, Brodsky NL, Malmud E, Giannetta JM, Hurt H. Attentional functioning and impulse control in cocaine-exposed and control children at age ten years. *J Dev Behav Pediatr*. 2005;26:42-47.
- Beeghly M, Martin B, Rose-Jacobs R, et al. Prenatal cocaine exposure and children's language functioning at 6 and 9.5 years: Moderating effects of child age, birthweight, and gender. *J Pediatr Psychol*. 2006;31:98-115.
- Heffelfinger A, Craft S, Shyken J. Visual attention in children with prenatal cocaine exposure. *J Int Neuropsychol Soc*. 1997;3:237-245.
- Mayes LC, Grillon C, Granger R, Schottenfeld R. Regulation of arousal and attention in preschool children exposed to cocaine prenatally. *Ann N Y Acad Sci*. 1998;846:126-143.
- Noland JS, Singer LT, Short EJ, et al. Prenatal drug exposure and selective attention in preschoolers. *Neurotoxicol Teratol*. 2005;27:429-438.
- Richardson GA, Conroy ML, Day NL. Prenatal cocaine exposure: Effects on the development of school-age children. *Neurotoxicol Teratol*. 1996;18:627-634.
- Malanga C, Kosofsky B. Mechanisms of action of drugs of abuse on the developing fetal brain. *Clin Perinatol*. 1999;26:17-37.
- Mayes LC. A behavioral teratogenic model of the impact of prenatal cocaine exposure on arousal regulatory systems. *Neurotoxicol Teratol*. 2002;24:385-395.
- Seidler FJ, Temple SW, McCook EC, Slotkin TA. Cocaine inhibits central noradrenergic and dopaminergic activity during the critical developmental period in which catecholamines influence cell development. *Dev Brain Res*. 1995;85:48-53.
- Stanwood GD, Washington RA, Shumsky JS, Levitt P. Prenatal cocaine exposure produces consistent developmental alterations in dopamine-rich regions of the cerebral cortex. *Neuroscience*. 2001;106:5-14.
- Glatt SJ, Bolanos CA, Trksak GH, Jackson D. Effects of prenatal cocaine exposure on dopamine system development: A meta-analysis. *Neurotoxicol Teratol*. 2000;22:617-629.
- Lidow MS, Song Z. Primates exposed to cocaine in utero display reduced density and number of cerebral cortical neurons. *J Comp Neurol*. 2001;435:263-275.
- Mayes LC, Molfese DL, Key APF, Hunter NC. Event-related potentials in cocaine-exposed children during a Stroop task. *Neurotoxicol Teratol*. 2005;27:797-813.
- Ornoy A, Segal J, Bar-Hamburger R, Greenbaum C. Developmental outcome of school-age children born to mothers with heroin dependency: importance of environmental factors. *Dev Med Child Neurol*. 2001;43:668-675.
- Bendersky M, Gambini G, Lastella A, Bennett D, Lewis M. Inhibitory motor control at five years as a function of prenatal cocaine exposure. *J Dev Behav Pediatr*. 2003;24:345-351.
- Minnes S, Singer LT, Arendt R, Satayathum S. Effects of prenatal cocaine/polydrug use on maternal-infant feeding interactions during the first year of life. *J Dev Behav Pediatr*. 2005;26:194-200.
- Black MM, Nair P, Kight C, Wachtel R, Roby P, Schuler M. Parenting and early development among children of drug-abusing women: effects of home intervention. *Pediatrics*. 1994;94:440-448.
- Barth RP, Brooks D. Outcomes for drug-exposed children eight years post-adoption. In: Barth RP, Brodzinsky D, Freundlich M, eds. *Adoption and Prenatal Drug Exposure: The Research, Policy and Practice Challenges*. Washington, DC: Child Welfare League of America; 2001:23-58.
- Landsverk J, Davis I, Ganger W, Newton R. Impact of child psychosocial functioning on reunification from out-of-home placement. *Child Youth Serv Rev*. 1996;18:447-462.
- Lewis E, Dozier M, Ackerman J, Sepulveda-Kozakowski S. The effect of placement instability on adopted children's inhibitory control abilities and oppositional behavior. *Dev Psych*. 2007;43:1415-1427.
- Schuler M, Nair P, Black M. Ongoing maternal drug use, parenting attitudes, and a home intervention: effects on mother-child interaction at 18 months. *J Dev Behav Pediatr*. 2002;23:87-94.
- McLellan A, Luborsky L, O'Brien C, Woody G. An improved diagnostic instrument for substance abuse patients: the addiction severity index. *J Nerv Ment Dis*. 1980;168:26-33.
- Enhancing the outcomes of low-birth-weight, premature infants. A multisite, randomized trial. The Infant Health and Development Program. *JAMA*. 1990;263:3035-3042.
- Conners CK. *The Conners Continuous Performance Test*. Toronto, Canada: Multi-Health Systems; 1994.
- Stanford-Binet Intelligence Scales. 4th ed. Chicago: The Riverside Publishing Company; 1987.
- Wilkinson G. Wide range achievement test-revision 3. In: Lutz, ed. FL: Psychological Assessment Resources; 2001.
- Achenbach TM. *Manual for Child Behavior Checklist and 1991 Profile*. Burlington: University of Vermont; 1991.
- Oken E, Kleinman K, Rich-Edwards J, Gillman M. A nearly continuous measure of birth weight for gestational age using a United States national reference. *BMC Pediatr*. 2003;3:6.
- Shenkin SD, Starr JM, Deary IJ. Birth weight and cognitive ability in childhood: a systematic review. *Psychol Bull*. 2004;130:989-1013.

43. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas*. 1977;1:385-401.
44. Kaufman A, Kaufman N. Kaufman brief intelligence test (K-BIT). In: Bloomington, MN, ed.: Pearson Assessments; 1990.
45. Grados JJ, Russo-Garcia KA. Comparison of the Kaufman Brief Intelligence Test and the Wechsler Intelligence Scale for Children - third edition in economically disadvantaged African American youth. *J Clin Psychol*. 1999;55:1063-1071.
46. Bendersky M, Lewis M. Prenatal cocaine exposure and impulse control at two years. *Ann N Y Acad Sci*. 1998;846:365-367.
47. Coles CD, Bard KA, Platzman KA, Lynch ME. Attentional response at eight weeks in prenatally drug-exposed and preterm infants. *Neurotoxicol Teratol*. 1999;21:527-537.
48. Karmel BZ, Gardner JM. Prenatal cocaine exposure effects on arousal-modulated attention during the neonatal period. *Dev Psychobiol*. 1996;29:463-480.
49. Schuler ME, Nair P, Black MM, Kettinger L. Mother-infant interaction: effects of a home intervention and ongoing maternal drug use. *J Clin Child Psychol*. 2000;29:424-431.
50. Landry SH, Miller-Loncar CL, Smith KE, Swank PR. The role of early parenting in children's development of executive processes. *Dev Neuropsychol*. 2002;21:15-41.
51. Llorente AM, Amado A, Voigt RG, et al. Internal consistency, temporal stability, and reproducibility of individual index scores of the Test of Variables of Attention (T.O.V.A.) in children with attention-deficit/hyperactivity disorder. *Arch Clin Neuropsychol*. 2001;16:535-546.
52. Barkley RA. *Attention-Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment*. 2nd ed. New York: Guilford; 1998.
53. Learth RA, Dupuy TR, Greenberg LM, Corman CL, Kindschi CL. *Test of Variables of Attention Professional Manual*. Los Alimitos, CA: Universal Attention Disorders; 1996.
54. Watson SMR, Westby CE. Strategies for addressing the executive function impairments of students prenatally exposed to alcohol and other drugs. *Commun Dis Q*. 2003;24: 194-204.
55. Delaney-Black V, Covington C, Nordstrom B, et al. Prenatal cocaine: Quantity of exposure and gender moderation. *J Dev Behav Pediatr*. 2004;25:245-63.
56. Lester BM, Elsohly M, Wright LL, et al. The Maternal Lifestyle Study: Drug use by meconium toxicology and maternal self-report. *Pediatr*. 2001;107:309-17.
57. NICHD. Duration and developmental timing of poverty and children's cognitive and social development from birth through third grade. *Child Dev*. 2005;76:795-810.

Book Review

Fine Motor Skills for Children with Down Syndrome: A Guide for Parents and Professionals, Second ed.

Maryanne Bruni, BScOT, Bethesda, MD, Woodbine House, 2006, 241 pp, \$19.95, softcover.

From her experience as both an occupational therapist and a parent of a child with Down syndrome, Maryanne Bruni seeks to share ideas for home—and school—based activities to promote the development of fine motor skills for children with Down syndrome from birth to 12 years of age. Her extensive book successfully communicates these ideas to her target audience of parents, teachers, and professionals.

The opening chapter describes Bruni's analogy of fine motor skills as a "house," of which the fundamental blocks are stability, bilateral coordination, and sensation, and the upper levels are dexterity and daily living skills (including school tasks, self-help skills, and household tasks/leisure activities). This theme drives the organization of the book, with individual chapters dedicated to each of these components. Other chapters focus on methods to motivate children to practice these skills, as well as overviews of fine motor and gross motor development in children with Down syndrome. Ms. Bruni ends the book with a chapter on

sensory processing in children with Down syndrome.

Bruni's text is succinct, and her numerous activities are easy to follow with her accompanying detailed step-by-step instructions and black-and-white photographs. The activities seem fun and generally do not require special or expensive tools (the appendix includes a list of common household items that can be converted to teaching tools). Each chapter is punctuated by a "Grandma's and Grandpa's list" of toys that help promote development of the particular skills described in the chapter, a list that many will likely find useful for gift-giving. Other gems in the book include a "handy basket" list for parents (a list of toys organized by age level), and a thorough chapter on school tasks, which includes sections on assessing readiness for and teaching printing skills, basics on hardware and software alternatives to printing, and examples of educational goals for children in inclusion settings (which may be particularly helpful for parents in developing their child's individualized

education program). The appendix includes several drawing worksheets and a list of resources for activity materials and various support organizations.

The "profiles" included in each chapter illustrate Bruni's recommendations in the form of a vignette, but were more distracting than informative. The evidence for some of the treatment approaches for sensory differences is only vaguely described, although Bruni does note when studies of protocols have not been performed specifically on children with Down syndrome.

Overall, Bruni's comprehensive book is highly practical and provides many activities for a wide age range of children. It should prove useful for anybody who is working with a child with Down syndrome to maximize his or her fine motor potential.

Patty Huang, MD
Division of Child Development
and Rehabilitation
Children's Hospital of Philadelphia
Philadelphia, PA