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Language Outcomes at 12 Years for Children Exposed Prenatally to Cocaine

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Abstract

Purpose—In this study, the authors aimed to examine the long-term effects of prenatal cocaine exposure (PCE) on the language development of 12-year-old children using a prospective design, controlling for confounding prenatal drug exposure and environmental factors.

Method—Children who were exposed to cocaine in utero (PCE; n = 183) and children who were not exposed to cocaine (i.e., no cocaine exposure [NCE]; n = 181) were followed prospectively from birth to 12 years of age and were compared on language subtests of the Test of Language Development—Intermediate, Third Edition (Hammill & Newcomer, 1997b), and phonological processing as measured by the Comprehensive Test of Phonological Processing (Wagner & Torgesen, 1999). The authors evaluated the relationship of PCE to language development through a multivariate analysis of covariance and regression analyses while controlling for confounders.

Results—Results show that PCE has small effects on specific aspects of language, including syntax and phonological processing. The caregiver variables of lower maternal vocabulary, more psychological symptoms, and a poorer home environment also had consistent effects on language and phonological processing scores.

Conclusions—These findings suggest that PCE continues to have small, subtle effects on specific aspects of language at age 12 years. Phonological processing skills were significantly related to the reading outcomes of letter–word identification, reading fluency, and reading comprehension, indicating that PCE also has small but lasting effects on the language skills that are related to later literacy skills.

Keywords

cocaine; phonological processing; reading outcomes; language outcomes; home environment; teratology; adolescents

Researchers have conducted studies examining the effects of prenatal cocaine exposure (PCE) on language skills in children for more than two decades, with some contradictory or at least equivocal results. In the early 1990s, case reports suggested that preschool children

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exposed to cocaine had specific language impairments that could potentially result in significant academic difficulties later at school age (Delaney-Black et al., 2000). Subsequent studies suggested that there were no significant effects of PCE on language (Hawley, Halle, Drasin, & Thomas, 1995; Hurt, Malmud, Betancourt, Brodsky, & Giannetta, 1997). Still other studies reported that the effects of PCE on language development were subtle, with other environmental factors—such as elevated lead levels—having more of an impact on language skills. Since that time, several large prospective cohort studies (Delaney-Black et al., 2000; Morrow et al., 2003; Singer et al., 2001) have documented language skills of children with PCE longitudinally. The findings have suggested that the effects of PCE appear to differ with age, largely due to changes in social, educational, and cognitive demands. These large cohorts are now entering adolescence, allowing for continued examination of long-term language outcomes. In this article, we review what is known about the language skills of children with PCE and present data from our large longitudinal cohort at 12 years of age.

Multiple Risk Factors Impacting Language

The examination of the language delays of children with PCE is complicated by multiple risk factors (biological, maternal, and environmental) that appear to impact language development. Studies have attempted to statistically control for these risk factors to tease out specific effects of PCE.

Biological factors

Children with PCE are typically exposed to multiple drugs in utero, including alcohol, tobacco, and marijuana. Researchers have examined the unique effects of cocaine by statistically controlling for other drug exposures. PCE has been shown to disrupt specific regions of the brain associated with dopamine responses, including anterior cingulate cortex, prefrontal cortex, and striatum (Harvey, 2004). Differences in brain function may influence higher cognitive processes such as sustained attention and auditory processing skills. Recent brain imaging studies of children with PCE have shown a decrease in white- and gray-matter volumes; it appears that this decrease is related to a decreased performance on neuropsychological tests (Dow-Edwards et al., 2006). In addition to drug effects, the perinatal factors of prematurity or low birth weight that required an extended hospital stay may also disrupt language development.

Maternal factors

A mother who uses drugs may not provide adequate language stimulation to her child, in part due to a chaotic, drug-seeking lifestyle (Mansoor et al., 2012; Minnes, Singer, Arendt, Farkas, & Kirchner, 2005; Siefer et al., 2004; Uhlhorn, Messinger, & Bauer, 2005). Also, other maternal factors such as education level, age, verbal skills, depression, psychosocial distress, and marital status appear to negatively affect the child’s language development. In some cases, placement in foster or adoptive care has been found to improve language outcomes for children with PCE compared with children who remained in the care of their biological mothers (Lewis et al., 2004). Children in foster or adoptive care had caregivers with higher vocabulary scores than those of the biological caregivers and attained IQ scores similar to those of children with no cocaine exposure (NCE; Singer et al., 2004). These results suggest that a cocaine-specific effect on language skills may be modified with an enriched environment.

Environmental factors

A variety of factors associated with poverty—including exposure to environmental toxins (such as lead), violence, and homelessness—may also negatively impact the child's language skills (Singer et al., 2008). In addition, studies of children with PCE have differed in the ages at which the children were assessed and the measures that were used for assessment. The effects of PCE on language development vary due to changing language demands from childhood to adolescence.

Review of Language Findings by Age at Assessment

Infant studies

Early studies of children with PCE suggested that children who were exposed and those who were not showed differences in auditory processing of information in the neonatal period. These studies suggested differences between neonates with PCE and those with NCE in their habituation to auditory stimuli. Infants with PCE were overreactive to a variety of stimuli, which, in part, may indicate that children with PCE may habituate more slowly to stimuli than do infants with NCE (Potter, Zelazo, Stack, & Papageorgiou, 2000). Additionally, researchers have found that infants with PCE startled more with auditory stimuli than did children with NCE, with cocaine exposure appearing to have a direct effect on sensorineural processing (Anday, Cohen, Kelley, & Leitner, 1989). Auditory evoked potentials from the brainstem and the cortex suggested some abnormalities in central auditory processing during the newborn period in children with PCE (Cone-Wesson, 2005; Tan-Laxa, Sison-Switala, Rintelman, & Ostrea, 2004). Thus, PCE appears to impair and decrease the speed of processing of auditory information. Singer and colleagues (Singer et al., 2001) reported lower auditory comprehension and total scores on the Preschool Language Scale—3 (PLS–3; Zimmerman, Steiner, & Pond, 1992) at 1 year of age for children with heavy PCE compared with infants with NCE. These auditory processing and comprehension deficits impact language skills and may affect subsequent academic achievements.

Early childhood studies

Studies of children with PCE in early childhood (ages 2–5 years) have reported mixed findings. Some researchers have found no differences in language skills between children with PCE and their peers (Bland-Stewart, Seymour, Beeghly, & Frank, 1998; Espy, Kaufmann, & Glisky, 1999; Hawley et al., 1995; Hurt et al., 1997), whereas others have indeed found differences (Bender et al., 1995; Nulman et al., 1994). The pattern of where differences have been found seems less consistent. For example, Bender and colleagues (Bender et al., 1995) found differences in receptive but not expressive language, whereas several research groups (Lewis et al., 2004; Morrow et al., 2004; Nulman et al., 1994; Pulsifer, Butz, Foran, & Belcher, 2008) found differences in expressive but not receptive language; still others reported both receptive and expressive language differences (Johnson, Seikel, & Madison, 1997). In a literature review, Lester and Lagasse (2010) reported that in eight studies of language in children with PCE, only one reported no negative effects, and one reported negative effects for boys only. All other studies found that PCE adversely impacted language skills.

Longitudinal analyses of language skills provide a clearer picture of PCE effects on language. In one longitudinal study of language across six time points—from 4 months to 3 years of age—researchers found stable effects of cocaine exposure, with children with PCE demonstrating overall lower language skills than children with NCE (Morrow et al., 2003). In addition, in our longitudinal study, we found stable negative effects of cocaine on language skills across 1, 2, 4, and 6 years of age (Lewis et al., 2007). Both groups—children with PCE and children with NCE—showed a decline in language performance over time.

Although global deficits have been demonstrated in longitudinal studies, specific domains of language must still be explored in an effort to understand discrepancy in cross-sectional findings. A few studies have examined specific language domains. Children with PCE demonstrated delays in semantics (Bland-Stewart et al., 1998); phonology (Madison, Johnson, Seikel, Arnold, & Schultheis, 1998); complexity of language in play (Malakoff, Mayes, Schottenfeld, & Howell, 1999); and discourse, pragmatics, and syntax (Mentis & Lundgren, 1995). Other researchers have found that children with PCE are more likely to fall into low-scoring language groups than children with NCE (Angelilli et al., 1994; Cone-Wesson, 2005; Delaney-Black et al., 2011; Lewis et al., 2004). Despite the inconsistent findings regarding language and PCE, the negative effects of PCE appear pervasive on many aspects of language.

Studies of PCE at school age

Because the demands on language skills increase during the school-age years—in large part due to academics, literacy acquisition, and changing social networks—a careful examination of PCE effects appears warranted. In several studies, researchers examined the effects of PCE at school age, with the results somewhat equivocal. Kilbride, Castor, and Fuger (2006) reported no differences at early school age (approximately 7 years) on the Clinical Evaluation of Language Fundamentals—Third Edition (CELF–3; Semel, Wiig, & Secord, 1995) between children with PCE and children with NCE. Conversely, other studies have reported negative effects of PCE on language at school age, with these effects varying as a function of amount of cocaine exposure (dose–response). The Miami Prenatal Cocaine study (Bandstra et al., 2011) reported a gradient (dose-dependent) relationship between PCE and receptive, expressive, and total language scores on the CELF–3 measured at 3, 5, and 12 years of age, with expressive language being most affected. Deficits in expressive language observed at age 3 years persisted at age 12 years. In another large longitudinal study, researchers examined language at 6.0 and 9.5 years of age and found language outcomes to be moderated by age, birth weight, and gender (Beeghly et al., 2006). Children with PCE showed poorer receptive language skills at age 6.0 years but not at age 9.5 years. Lower birth weight was associated with lower expressive and total language scores. In addition, researchers observed gender differences, with girls with PCE demonstrating greater deficits in expressive language than girls with NCE (Beeghly et al., 2006). In a review of 32 articles, representing 15 school-age cohorts of children with PCE, Ackerman, Riggins, and Black

(2010) concluded that PCE was related to deficits in sustained attention and behavioral self-regulation. Associations of other outcomes to PCE, including language, were small and influenced by environmental variables.

We obtained support for the relationship between phonological processing and cocaine exposure in our cohort at age 10 years (Lewis et al., 2011), with significant differences on the Phonological Awareness composite score of the Comprehensive Test of Phonological Processing (CTOPP; Wagner & Torgesen, 1999; $p = .01$) and on the Sentence Combining subtest of the Test of Language Development—Intermediate, Third Edition (TOLD–I:3; Hammill & Newcomer, 1997b; $p = .001$). The Phonological Awareness composite score consists of the Elision and Blending Words subtests. Although the Sentence Combining subtest is designed to examine syntax, it also requires the child to hold two or more sentences in memory while operating on them. Numerous studies have documented that phonological awareness skills are important for early literacy acquisition.

Human versus animal studies

The adverse effects of cocaine are not limited to human studies, with animal studies supporting the auditory processing differences after in-utero cocaine exposure. In animals, PCE accelerated maturation of the cochlea in rat pups, which appeared to cause auditory dysfunction by desynchronizing the development of the auditory pathway (Trigueiros-Cunha, Leão, Renard, Tavares, & Eybalin, 2006).

Adolescent studies

Adolescent follow-up studies of children with PCE are fewer and less well understood. A recent study of 13-year-old adolescents with PCE demonstrated that auditory processing deficits observed in infancy may persist into adolescence (Landi, Crowley, Wu, Bailey, & Mayes, 2011). In a paradigm using event-related potentials (ERPs), children with PCE demonstrated atypical responses to spoken language stimuli (nonwords) during low-level processing and later processing of speech. Structural imaging work demonstrated that adolescents with PCE showed reduced volume of the caudate[1] compared with control subjects (Avants et al., 2007).

Researchers examining language differences in adolescence have reported equivocal findings, in large part due to the different language domains that are assessed and to differential rates of attrition in the samples. A longitudinal study examining participants at 12.0, 14.5, and 17.0 years of age found no effects of PCE on results of the Peabody Picture Vocabulary Test—Revised (PPVT–R; Dunn & Dunn, 1997); these researchers report that language skills improved across time and that receptive vocabulary scores were related to the home environment (Betancourt et al., 2011). In a different longitudinal study, researchers found an association of PCE and lower scores on the expressive and total language scores on the CELF (Bandstra et al., 2011). Taken together, these studies argue for the further investigation of the long-term effects of PCE on language outcomes in adolescence.

[1]The *caudate* is associated with the dopaminergic system that regulates attention.

Cleveland Longitudinal Study of PCE

In the present article, we report on the 12-year outcomes of a large cohort of children with PCE who were followed prospectively from birth. This is a well-characterized unique cohort with a high retention rate at 12 years of age (90% of living participants). See online supplementary materials for a summary of the findings of this study thus far; participants were followed from birth to 10 years of age. Children with PCE differed from children with NCE on multiple cognitive and biological domains across the developmental trajectory, including language.

In designing the current longitudinal study, we were faced with the challenge of having limited time and resources to assess all of the domains in which we were interested. We determined that it was important to (a) assess children every year rather than on alternate years in order to keep the cohort intact, (b) limit the assessment to a single day so that children would not miss too much school and parents would not be too inconvenienced, and (c) observe the developmental trajectory of these children from pre-adolescence to adolescence. We wanted to assess both language and achievement; we did so on alternate years to minimize redundancy and to guarantee a measure of language (oral or written) for each year.

Children were assessed at 10, 11, and 12 years of age on multiple measures of cognitive ability, educational achievement, social skills, psychological profiles, medical history, executive functioning, and language. The TOLD–I:3 and the CTOPP were administered at ages 10 and 12 years. At the 10-year assessment of this cohort, we found PCE effects for specific language domains including syntax, semantics, and phonological processing (Lewis et al., 2011). In the present study, we aimed to investigate if the effects of PCE on language skills persisted at early adolescence (age 12 years) while controlling for multiple prenatal drug exposure and maternal variables such as age, education, psychological distress, vocabulary, and marital status. In response to out-of-home placements, we considered covariates including the current caregiver’s education and drug use and the quality of the current home environment. We also considered the child’s current IQ because it is known to influence language development. Twelve-year findings were related to literacy findings at the 11-year follow-up.

Method

Participants

We recruited 364 primarily African American children (183 of whom were positive for cocaine and 181 of whom were negative for cocaine) of low socioeconomic status (SES; Hollingshead, 1975) at birth from a large county teaching hospital in an urban location. Urine samples were obtained from the mother immediately before or after labor and delivery and were analyzed for cocaine metabolites, cannabinoids (tetrahydrocannabinol [THC]), opiates, phencyclidine (PCP), and amphetamines. Infant meconium drug analysis was also performed. Mothers were interviewed regarding their drug use. Birth, demographic, and medical characteristics were taken from hospital records with mother’s consent.

Mothers who used cocaine were older, had fewer prenatal visits, and had more children than mothers who did not use cocaine. In addition, mothers who used cocaine were more likely to use tobacco, alcohol, and marijuana in greater amounts during pregnancy than were mothers who did not use cocaine. Children exposed to cocaine were shorter in length, had smaller head circumference, and had lower weight at birth than children who were not exposed. Children were followed prospectively from birth at 1, 2, 4, 6, 9, 10, 11, and 12 years of age. The sample was drawn from a cohort recruited at birth (September 1994–June 1996) from a large county teaching hospital in an urban location; these individuals had participated in a longitudinal study of the sequelae of fetal drug exposure. We obtained institutional review board approval from University Hospitals of Cleveland and Metro-Health Medical Center for all participants, with informed consent obtained from parents and assent obtained from the child. Health Insurance Portability and Accountability Act of 1996 (HIPAA) was maintained. All participants were protected by a writ of confidentiality, which prevented the release of any participant information from the research, even under court order.

Women considered at high risk for drug use due to lack of prenatal care, behavior suggesting intoxication, a history of involvement with the Department of Human Services, or self-admitted use were administered drug toxicology screenings at the child's birth. Maternal and infant urine samples were obtained immediately before or after labor and delivery and were analyzed for the presence of cocaine metabolites (benzoylecgonine [BZE]), cannabinoids (THC), opiates, PCP, and amphetamines. Women who had urine drug screenings were approached by a research nurse for participation in the study. Upon agreement, infants had meconium drug analyses performed for cocaine and its metabolites (i.e., BZE, meta-hydroxybenzoylecgonine [M-OH-BZE], cocaethylene, cannabinoids [THC], opiates, PCP, amphetamines, and benzodiazepines). Screening assays were conducted through the use of polarization immunoassay reagents (fluorescence polarization immunoassay; U.S. Drug Testing Laboratories, Des Plaines, IL). Cutoff levels were as follows: cocaine and metabolites, opiates, 25 ng/g; amphetamines, 100 ng/g; PCP, 25 ng/g; and THC, 25 ng/g. Confirmatory assays were conducted. Specificity for both urine and meconium cutoffs was 99%.

Infants with PCE were identified on the basis of positive infant meconium, maternal urine, or maternal self-report to hospital or research staff, whereas control infants were negative on all three indicators. Women who used alcohol, marijuana, or tobacco during pregnancy were included in both groups. Of the 647 mothers identified, 54 were excluded (20 PCE and 34 NCE) from this study, with 15 not having meconium, two infants with Down syndrome, 16 having maternal psychiatric history, two being due to primary heroin use, five having human immunodeficiency virus status, one being due to an IQ < 70, one infant with fetal alcohol syndrome, two being due to maternal age under 19 years, three being due to a medical illness in the infant, four being due to chronic illness in the mother, and three for other reasons. Additionally, a total of 155 women (49 who used cocaine and 106 who did not use cocaine) refused to participate, and 23 (nine PCE and 14 NCE) did not come to the enrollment visit. The sample size of the original cohort was 415 (218 PCE and 197 NCE). By age 12 years, 12 of the children in the study group (nine PCE and three NCE; $\chi^2 = 3.0$; $p < .08$) had died. Of the 39 children not seen, the 26 children with PCE had higher alcohol

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exposure than the study participants and were more likely to have mothers with lower Picture Completion scores on the Wechsler Adult Intelligence Scale—Revised (WAIS–R; Wechsler, 1989); the 13 children with NCE had lower birth weights, shorter lengths, and more prenatal tobacco exposure, and their mothers were not married and had higher birth age compared with the study participants (see Tables 1 and 2).

Procedure

To assess prenatal drug exposure, we saw infants and their birth mothers immediately after birth, at which time the birth mother was interviewed regarding drug use. Birth mothers were asked to recall the frequency and amount of drug use for the month prior to pregnancy and each trimester of the pregnancy. Additionally, for tobacco, the number of cigarettes smoked per week was recorded; for marijuana, the number of joints smoked per week was recorded; for alcohol, the number of drinks of beer, wine, or hard liquor per week was computed (with each drink equivalent to 0.05 oz. of absolute alcohol); and for cocaine, the number of rocks consumed and amount of money spent per week were noted. For each drug, the frequency of use was recorded. We updated this drug assessment at each follow-up visit to provide a similar measure of current drug use and also administered the assessments to the foster or relative caregiver to provide a measure of caregiver postnatal environmental use.

Birth, demographic, and medical characteristics were taken from hospital records with the permission of the birth mother, and they included maternal race, age, and parity; number of prenatal care visits; type of medical insurance; and infant Apgar scores, birth weight, length, and head circumference. At enrollment in the study, maternal SES (Hollingshead, 1975) and educational level were calculated. Maternal vocabulary was measured through use of the PPVT–R. Two performance subtests of the WAIS–R—the Block Design and Picture Completion subtests—were administered, enabling an estimate of nonverbal intelligence. The Brief Symptom Inventory (BSI; Derogatis, 1992), which is a standardized self-report scale of severity of psychological distress, was administered at birth and at all subsequent visits. The General Severity Index, a summary score of the BSI, was used as an indicator of overall distress. The Hobel Neonatal Risk Index (Hobel, Hyvarinen, Okada, & Oh, 1973) was computed so that we could obtain a measure of neonatal medical complications. At the 10-year visit, the child’s placement (either [a] birth mother or relative or [b] foster or adoptive caregiver) was noted, and data on the current caregiver were updated. If the child had been placed with a new caregiver, intellectual measures of the caregiver were also updated. The Home Observation of the Environment (HOME; Caldwell & Bradley, 1984) was administered to the caregiver in an interview format as a measure of the quality of the caregiving environment at each visit. The HOME was administered in the laboratory as suggested by Jacobson and Jacobson (1995). The HOME total score at 10 years of age was used in our analyses.

Sample Characteristics

The women in both groups—those who used cocaine and the controls—were primarily African American, of low income, and not married (see Table 1). Women who used cocaine were older, had more children, and had fewer prenatal care visits than did controls. They also completed fewer years of education and had lower vocabulary scores on the PPVT–R. They used other drugs (i.e., alcohol and tobacco) more frequently and in higher amounts than did nonusers. Infants with PCE were more likely to be preterm and of lower birth weight, have smaller head circumference (used as a mediator of the cocaine effect), and have shorter birth length than infants with NCE (see Table 2). At birth, 49 (26%) infants with PCE were placed outside birth mother or relative care compared with only three (2%) infants with NCE ($p < .05$). By age 12 years, 41 (22.4%) children with PCE were in adoptive or foster care compared with eight (4.4%) children with NCE, $\chi^2(N = 364) = 25.3$, $p < .001$. Among the 141 children with PCE not in adoptive or foster care, 91 (25.0%) were with the biological mother, and 50 (13.7%) were in relative care. One child with PCE was in a residential facility. See Table 3 for a summary of current caregiver demographics.

Measures

Because of the extensive and lengthy assessments encompassing multiple social, emotional, and cognitive domains at 12 years of age, we did not perform a comprehensive speech and language assessment. Two language measures were individually administered—the TOLD–I:3 and the CTOPP—by examiners who were unaware of the children's cocaine status.

We selected the TOLD–I:3 because we wanted a comprehensive measure of language skills that assessed receptive and expressive language skills, as well as specific domains of syntax, and semantics that spanned ages 10, 12, and 14 years. The TOLD–I:3 allowed us to directly compare scores obtained at age 10 years on the TOLD–I:3 with the 12-year scores. In most of the previous studies of children with PCE, researchers reported only expressive, receptive, and total language scores and did not examine specific language domains. By examining subtests on the TOLD–I:3, we attempted to determine whether observed language differences were due to deficits of syntax, semantics, or both. We hypothesized that a deficit in syntactic skills would be present given our previous findings at age 10 years. We also hypothesized that because vocabulary skills tend to be more environmentally mediated, the PCE group should not differ from the NCE group, considering that both groups were low SES. Our considerations were supported by our previous findings and by the findings of Betancourt et al. (2011). In contrast, we hypothesized that syntactic skills might be more compromised in children with PCE because syntax may rely on hardwired neurological systems.

Further, we chose the TOLD–I:3 to assess language abilities because its authors reported studies showing the absence of cultural, gender, racial, and disability bias. Race and ethnicity were considered in the normative sample of the TOLD–I:3. The sample included African Americans (15%) and was 75% urban. Subgroups based on ethnicity and genders were considered in determining reliability and validity. Because our participants were primarily African American and urban, it was important to choose a test that included this population in its normative sample. In addition, we included a matched control group—which was recruited at birth and was followed longitudinally in the same manner as that used in studying the children with PCE—that was not exposed to cocaine.

The TOLD–I:3 is based on a linguistic model and is empirically validated so that researchers can assess both listening and speaking as well as syntax and semantics. The test's uses, as stated by Hammill and Newcomer (1997a), include (a) identifying children who are significantly below their peers in language proficiency, (b) determining children's specific strengths and weaknesses, and (c) measuring language in research studies. The current project sought to identify differences in strengths and weaknesses between children with PCE and children with NCE. The TOLD–I:3 assesses the understanding and meaningful use of spoken words, as well as different aspects of grammar. The test consists of six subtests, including Sentence Combining, Picture Vocabulary, Word Ordering, Identifying *Generals* (superordinate categories for words), Grammatical Comprehension, and Identifying *Malapropisms* (words that sound alike but have different meanings). Content validity is supported by linking the subtests to research, thus providing qualitative evidence (Hayward, Stewart, Phillips, Norris, & Lowell, 2008). The test manual states that content validity was demonstrated by the rationale for test items and format, the professional opinions of 180 experts, a classical item analysis, and a differential item functioning analysis (Hammill & Newcomer, 1997a). Age-standardized scores were computed for subtests. These subtest scores—rather than composite scores—were analyzed so that specific linguistic domains might be examined.

We chose the CTOPP to assess phonological processing abilities. This test is based on a model of phonological processing that includes phonological awareness, phonological memory, and rapid naming. These skills are essential to reading decoding and reading comprehension. The large normative sample included African American participants. Researchers use the test (a) to identify individuals whose phonological abilities are below those of their peers, (b) to document strengths and weaknesses in phonological processing, and (c) to measure phonological processing skills in research studies. In the current project, we sought to compare children with PCE to children with NCE on phonological processing skills and to relate these skills to reading decoding and reading comprehension. Subtests of the CTOPP designed to assess phonological awareness were the Elision and Blending Words subtests. To assess phonological memory, we used the Memory for Digits and the Nonword Repetition subtests from the CTOPP. Finally, rapid naming was assessed through two of the rapid naming subtests (Rapid Digit Naming and Rapid Letter Naming). Difficulties in one or more of these domains of phonological processing abilities may adversely affect an individual's ability to read. Age-standardized scores are available for each of the three composite scores (Phonological Awareness, Phonological Memory, and Rapid Naming) and six subtests (Elision, Blending Words, Memory for Digits, Nonword Repetition, Rapid Digit Naming, and Rapid Letter Naming) of the CTOPP.

Analyses

Baseline maternal characteristics, child characteristics, and prenatal drug exposure were summarized through the use of means and standard deviations for continuous variables and the use of frequencies and percentages for categorical variables. Comparisons between PCE and NCE groups were performed through the use of *t* tests, Wilcoxon rank sum, and Pearson chi-square (χ^2) tests. We transformed all positively skewed data, including drug self-report measures and General Severity Index, using the natural logarithm to achieve a distribution that approximated normality. We estimated Pearson correlations to examine relationships between (a) biological, maternal, and environmental covariates and (b) language outcomes.

We compared children with PCE who were either in foster or adoptive care or in biological maternal or relative care and children with NCE on environmental characteristics, caregiver characteristics, and the current caregiver's substance use using analysis of variance. We performed post hoc pairwise tests using the Tukey test in the event of significant group differences.

Linear regression, controlling for confounders, was used as a way of evaluating the relationship of cocaine exposure and language scores on the TOLD–I:3 and CTOPP. Cocaine exposure was entered first in the model and was retained throughout the modeling process. Covariates that correlated with the language outcome variable at $p \leq .20$ and that differed by group (cocaine exposure vs. no cocaine exposure) at $p \leq .20$ were entered into the regression model stepwise and were retained if, on entry, they were significant at $p < .10$ or caused substantial change (10%) in the cocaine-exposure coefficient. Environmental and prenatal factors were considered next, followed by demographic, prenatal and current drug exposure variables, and child IQ. We calculated adjusted mean language scores, controlling for confounding variables to compare PCE and NCE groups. In addition, we used linear regression to examine the relationship between reading skills that were assessed at the 11-year visit using the Letter-Word Identification, Reading Fluency, and Passage Comprehension subtests of the Woodcock–Johnson—III (Woodcock, McGraw, & Mather, 2001) and the significant 12-year language measures.

Results

Effects of Prenatal Drug Exposure on Language Outcomes at 12 Years of Age

Table 4 presents the unadjusted means for the language measures, and Table 5 presents the adjusted means. As shown in Table 5, we found that cocaine exposure had a negative effect on mean performance scores on the Phonological Awareness ($t = 2.38$, $p = .02$), Elision ($t = 2.51$, $p = .01$), and Blending Words ($t = 1.98$, $p = .05$) subtests of the CTOPP, after controlling for multiple confounders including home environment, alcohol exposure, cigarette use, maternal education, and IQ. An unexpected positive effect for cocaine exposure was observed for the Rapid Letter Naming subtest ($t = -2.68$, $p = .008$). Significant negative effects of cocaine exposure were found for the Sentence Combining subtest of the TOLD–I:3 ($t = 2.13$, $p = .03$). Effect sizes for these findings were small: Phonological Awareness, $\eta^2 = .03$; Elision, $\eta^2 = .04$, Blending Words, $\eta^2 = .02$, Rapid Letting Naming, $\eta^2 = .01$, and Sentence Combining, $\eta^2 = .03$.

Significant differences were not observed between the NCE group and the PCE group on the reading measures (Letter–Word Identification, Reading Fluency, and Reading Comprehension) administered at age 11 years. However, significant associations were found between (a) the Elision and Rapid Letter Naming subtests of the CTOPP and the Sentence Combining subtest of the TOLD–I:3 and (b) the Letter–Word Identification, Reading Fluency, and Passage Comprehension subtests of the Woodcock–Johnson—III. The Blending Words subtest of the CTOPP was significantly associated with the Letter–Word Identification and the Passage Comprehension subtests but not Reading Fluency (see Table 6).

Effects of Caregiver and Environmental Characteristics

Tables 7A and 7B display the correlation of the significant covariates (i.e., prenatal drug exposures, maternal characteristics, current caregiver characteristics, and child characteristics) with the language outcomes. All of the language measures were significantly related to the child's full-scale IQ score. The CTOPP and TOLD–I:3 subtests that were significantly different for children with PCE and for children with NCE showed the following significant correlations. The HOME score was significantly related to the Phonological Awareness composite score and the Elision, Blending Words, and rapid naming subtests of the CTOPP and the Sentence Combining subtest of the TOLD–I:3. The biological mother's vocabulary on the PPVT–R impacted language skills at age 12 years. Lower maternal PPVT–R scores were related to lower child scores on the Phonological Awareness composite score and the Elision and Blending Words subtests of the CTOPP and on the Sentence Combining subtest of the TOLD–I:3. Alcohol exposure during pregnancy was related to poorer language outcomes on the Phonological Awareness composite score and Elision subtest of the CTOPP and the Sentence Combining subtest of the TOLD–I:3. Current caregiver marijuana use was significantly related to the Blending Words subtest and the Rapid Naming composite score of the CTOPP.

Discussion

Our findings suggest that exposure to cocaine in utero continues to have small, primarily negative effects on language skills at early adolescence. Although we were unable to perform a comprehensive speech and language assessment due to time limitations, these differences remained even after we controlled for multiple biological and environmental factors related to language abilities. Significant differences between the group with PCE and those with NCE at adolescence, coupled with significant differences in language abilities across the developmental trajectory, suggests that PCE places an individual at risk for language deficits. Similar to our 10- year findings in this cohort (Lewis et al., 2011), specific language deficits were related to syntax and phonological awareness. During adolescence, greater demands are placed on language skills for academic achievement and social roles (Singer et al., 2008). Thus, the effects of PCE may present differently than at younger ages due to both biological and environmental changes (Ackerman et al., 2010).

Effects of PCE on Language

We found a small negative effect for PCE on the Sentence Combining subtest of the TOLD–I:3 after controlling for significant covariates of the home environment, maternal characteristics, and other prenatal drug exposures. This finding is consistent with our findings of significant cocaine effects on sentence combining at 10 years of age in this cohort (Lewis et al., 2011). Sentence combining exercises have long been advocated for both the assessment and instruction of syntactic maturity and proficiency in both spoken and written language (Scott & Nelson, 2009). On the Sentence Combining subtest of the TOLD–I:3, the examinee hears two to four simple sentences and creates a single sentence that uses attributive adjectives, phrasal coordination, adverbial elements, clausal coordination, a series construction, and relative clauses. The processing requirements of this task are great in that the individual has to hold as many as four simple sentences in working memory while generating a sentence. This subtest requires a more conscious level of structural awareness than does naturalistic language (Scott & Stokes, 1995). Although it is possible that working memory limitations of children with PCE influenced findings of the Sentence Combining subtest of the TOLD–I:3, by using IQ as a covariate, we attempted to control for working memory (digit span and letter–number sequencing) differences in the sample. Sentence combining tasks have been extended to written language as well, and syntactic abilities have been related to literacy acquisition. Complex sentences in written text may be challenging to readers with weak spoken language skills (Lewis, O'Donnell, Freebairn, & Taylor, 1998; Nelson & Van Meter, 2007). The poorer performance of the children with PCE may indicate less syntactic maturity when compared with their NCE peers. This may impact reading and writing as well as spoken language.

Our results are in agreement with earlier findings suggestive of syntax deficits in young children with PCE (Mentis & Lundgren, 1995). However, Mentis and Lundgren examined syntax in a language sample of five children with PCE (26–29 months old), whereas our sample was much older as well as larger, and we used a standardized measure. Similar to Betancourt et al. (2011), who found no deficit in the PPVT scores for children with PCE, we did not find vocabulary deficits on the Picture Vocabulary subtest of the TOLD–I:3 in children with PCE at age 12 years. These results highlight the need to examine specific language domains when studying the effects of PCE. Composite scores of language measures may not be sensitive to cocaine effects, especially at adolescence.

Effects of PCE on Phonological Processing

The Phonological Awareness composite score on the CTOPP showed a small significant negative PCE effect after we controlled for the home environment, maternal and current caregiver variables, prenatal alcohol exposure, and maternal and child IQs (see Table 5). Both of the subtests (Elision and Blending Words) showed consistent negative effects, with the core underlying skills appearing to be phonological processing skills. At the 10-year follow-up assessment, PCE also significantly affected the Phonological Awareness composite score and the Elision subtest but not the Blending Words subtest (Lewis et al., 2011). Phonological awareness skills are essential for efficient reading decoding in early readers (Catts, Fey, Zhang, & Tomblin, 1999; Nathan, Stackhouse, Goulandris, & Snowling, 2004; Scarborough, 2005). Lombardino, Riccio, Hynd, and Pinheiro (1997) found that the Elision task, which requires the child to delete sounds and syllables, is related to both reading decoding and comprehension. Thus, the fact that PCE affects these skills in an adverse way is concerning for future academic performance in reading.

An unexpected finding was that PCE had a significant positive effect on the Rapid Letter Naming subtest of the CTOPP. This may be due to the increased impulsivity observed in children with PCE (Ackerman et al., 2010). Faster processing speed, although assumed to be a desirable skill in academic performance, must be gauged against the backdrop of decreased accuracy. Students must adjust their speed to the ever-changing demands of the tasks at hand—that is, students must assess tasks and know which tasks they can perform quickly without sacrificing accuracy and which tasks they must perform more slowly to maintain accuracy.

Although the effect sizes for differences in both language and phonological processing were small, when we converted the effect sizes to standard units, we found that the PCE group differed from the NCE group by 4.48 standard unit differences on the Phonological Awareness composite score of the CTOPP, 1.18 units on the Elision subtest, 0.62 units on the Blending Words subtest, 0.61 units on the Rapid Letter Naming subtest, and 0.79 units on the Sentence Combining subtest (see online supplemental materials for the conversion calculations). This may be compared with the Miami Prenatal Cocaine study findings of a 3.0-unit difference on the Total Language score and Expressive Language score of the CELF–3. However, our findings are a more conservative estimate because we controlled for IQ, whereas the Miami study did not.

Lester, LaGasse, and Seifer (1998) demonstrated that even small effect sizes can result in a substantial number of children who require special services. In their meta-analysis, Lester et al. found that children with PCE have IQs that are 3.26 points lower than the IQs of children with NCE. This downward shift of the IQ distribution of children with PCE resulted in a 1.6% increase in the number of children with IQ scores less than 70, thus increasing the number of children requiring services by 80,550 children nationally. Lester et al. reported similar findings for language skills of children with PCE, with a 4.3% increase in children requiring clinical services.

Relationship of PCE to Literacy

Although we did not assess literacy at the 12-year visit due to the extensive time constraints of this longitudinal design, we did assess it at the 11-year visit. We examined the relationship of the 12-year language outcomes to the 11-year assessment of Letter–Word Identification, Reading Comprehension, and Reading Fluency measures. Phonological awareness, phonological memory, and rapid naming have positive relations to reading outcomes. However, it is not known whether differences in phonological processing skills between the children with PCE and the children with NCE will significantly impact literacy at and beyond 12 years of age. In our cohort, we did not find significant differences in scores on the Letter–Word Identification, Reading Fluency, and Passage Comprehension subtests at the earlier 11-year assessment. Lester and Lagasse (2010) reported that of seven studies that assessed academics at school age, only three showed that children with PCE performed more poorly than did controls as measured by standardized achievement tests and referrals for special services. Similar to language outcomes for children with PCE, standard measures such as the Woodcock–Johnson—III might not be sensitive enough to allow researchers to detect the subtle effects of PCE.

Caregiver and Environmental Influences on Language

Language outcomes for children with PCE are greatly impacted by both caregiver and environmental factors. The biological mother's vocabulary skill as measured by the PPVT was significantly associated with all of the subtests of the TOLD–I:3 and the Phonological Awareness subtest and Nonword Repetition subtest of the CTOPP. Maternal IQ, as estimated by the Block Design subtest of the WAIS–R, was also significantly correlated with language outcomes. These findings are consistent with earlier assessments of this cohort, which also found maternal vocabulary and IQ to be related to the child's language scores (Lewis et al., 2011). The influence of both genetics and environment on verbal skills is still strongly evident at 12 years of age. The current care-giver's education level and IQ, whether it be the biological mother or foster care mother, plays a role in language development. Environmental factors were highly influential on language skills. These positive effects of the caregiver's verbal skills and the home environment at early adolescence underscore not only the importance of a nurturing environment across the developmental trajectory but also the modifiability of language skills.

Strengths and Limitations of the Study

Strengths of this study include the use of a prospective design, inclusion of biological markers to determine cocaine exposure, excellent retention rate, ensuring that examiners were blind to cocaine status, and the assessment of specific language domains. Several limitations of this study should be noted.

The participants were limited to African American children in an impoverished environment. Both PCE and NCE groups were exposed to multiple drugs, including marijuana, alcohol, and nicotine. A study by Hoffman, Loeb, Brandel, and Gillam (2011) found that although there is substantial overlap in the measurement of oral language abilities between standardized language measures, each measure assesses some unique subsets of language abilities that are not accounted for by the other measures. Cocaine effects may impact language domains that were not assessed by either the TOLD–I:3 or the CTOPP. Use of decontextualized standardized measures may not have captured the breadth of language skills. More sensitive language measures may demonstrate greater effects of PCE. For example, social language skills were not assessed and appear important for adaptive social functioning in adolescence. Literacy was not assessed at the same age as the language measures due to the longitudinal design of the parent study; therefore, the relationship among phonological awareness skills, language, and literacy could not be adequately assessed. Twelve years of age is considered by some to be pre- or early adolescence, with the effects of PCE on later adolescence necessitating further follow-up.

Clinical Implications

Speech-language pathologists continue to see children with PCE on their caseloads. Interpreting the significance of this exposure on speech and language abilities is difficult due to the multiple biological and environmental factors impacting these children. In the current study, we found small but significant effects of PCE on standardized language measures. This large longitudinal study has documented subtle neurobehavioral deficits in multiple cognitive domains across the developmental trajectory that impact language and academic outcomes. Although initial concerns that children with PCE would have severe to profound deficits were not supported, small language and other cognitive deficits may have negative consequences on school achievement and vocational attainment over time.

Conclusions

The effects of PCE on language skills appear to persist into the school-age years, with these effects particularly relevant to phonological processing skills and syntactic maturity, which are thought to be highly related to literacy. Exposure to other drugs such as alcohol, tobacco, and marijuana may also contribute to poorer outcomes. In addition, the language learning environment may be influenced by the caregiver's psychological state, education, and IQ. These findings support earlier studies of language skills in the same cohort, including phonological processing difficulties and attentional deficits seen in infancy and early childhood, that may relate to these findings.

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References

Ackerman JP, Riggins T, Black MM. A review of the effects of prenatal cocaine exposure among school-aged children. Pediatrics. 2010; 125:554–565. [PubMed: 20142293]

Anday EK, Cohen ME, Kelley NE, Leitner DS. Effect of in utero cocaine exposure on startle and its modification. Developmental Pharmacology and Therapeutics. 1989; 12:137–145. [PubMed: 2721336]

Angelilli M, Fischer H, Delaney-Black V, Rubinstein M, Ager JW, Sokol RJ. History of in utero cocaine exposure in language-delayed children. Clinical Pediatrics. 1994; 33:514–516. [PubMed: 8001318]

Avants BB, Hurt H, Giannetta JM, Epstein CL, Shera DM, Rao H, Gee JC. Effects of heavy in utero cocaine exposure on adolescent caudate morphology. Pediatric Neuorology. 2007; 37:275–279.

Bandstra ES, Morrow CE, Accornero VH, Mansoor E, Xue L, Anthony JC. Estimated effects of in utero cocaine exposure on language development through early adolescence. Neurotoxicology and Teratology. 2011; 33:25–35. [PubMed: 21256422]

Beeghly M, Martin B, Rose-Jacobs R, Cabral H, Heeren T, Augustyn M, Frank DA. Prenatal cocaine exposure and children's language functioning at 6 and 9.5 years: Moderating effects of child age, birth weight and gender. Journal of Pediatric Psychology. 2006; 31:98–115. [PubMed: 15843502]

Bender SL, Word CO, DiClemente RJ, Crittenden MR, Persaud NA, Ponton LE. The developmental implications of prenatal and/or postnatal crack cocaine exposure in preschool children: A preliminary report. Journal of Developmental and Behavioral Pediatrics. 1995; 16:418–424. [PubMed: 8746551]

Betancourt LA, Yang W, Brodsky NL, Gallagher PR, Malmud EK, Giannetta JM, Hurt H. Adolescents with and without gestational cocaine exposure: Longitudinal analysis of inhibitory control, memory, and receptive language. Neurotoxicology and Teratology. 2011; 33:36–46. [PubMed: 21256423]

Bland-Stewart LM, Seymour HN, Beeghly M, Frank DA. Semantic development of African-American children prenatally exposed to cocaine. Seminars in Speech and Language Disorders. 1998; 19:167–187.

Caldwell, B.; Bradley, R. Home Observation for Measurement of the Environment. Little Rock: University of Arkansas Press; 1984. (Revised ed.)

Catts HW, Fey ME, Zhang X, Tomblin JB. Language basis of reading and reading disabilities: Evidence from a longitudinal investigation. Scientific Studies of Reading. 1999; 3:331–362.

Cone-Wesson B. Prenatal alcohol and cocaine exposure: Influences on cognition, speech, language, and hearing. Journal of Communication Disorders. 2005; 38:279–302. [PubMed: 15862811]

Delaney-Black V, Chiodo LM, Hannigan JH, Greenwald MK, Janisse J, Patterson G, Sokol RJ. Prenatal and postnatal cocaine exposure predict teen cocaine use. Neurotoxicology and Teratology. 2011; 33:110–119. [PubMed: 20609384]

Delaney-Black V, Covinton C, Templin T, Kershaw T, Nordstrom-Klee B, Ager J, Sokol RJ. Expressive language development of children exposed to cocaine prenatally: Literature review and report of a prospective cohort study. Journal of Communication Disorders. 2000; 33:463–481. [PubMed: 11141028]

Derogatis, L. The Brief Symptom Inventory: Administration, scoring, and procedures manual—II. Baltimore, MD: Clinical Psychometric Research; 1992.

Dow-Edwards DL, Benveniste H, Behnke M, Bandstra ES, Singer LT, Hurd YL, Stanford LR. Neuro-imaging of prenatal drug exposure. Neurotoxicology and Teratology. 2006; 28:386–402. [PubMed: 16832875]

Dunn, LM.; Dunn, DM. Peabody Picture Vocabulary Test—III. Circle Pines, MN: AGS; 1997.

Espy KA, Kaufmann PM, Glisky ML. Neuropsychologic function in toddlers exposed to cocaine in utero: A preliminary study. Developmental Neuropsychology. 1999; 15:447–460.

Hammill, DD.; Newcomer, PL. Examiner's manual: Test of Language Development—Intermediate. 3. Austin, TX: Pro-Ed; 1997a.

Hammill, DD.; Newcomer, PL. Test of Language Development—Intermediate. 3. Austin, TX: Pro-Ed; 1997b.

Harvey JA. Cocaine effects on the developing brain: Current status. Neuroscience Biobehavioral Review. 2004; 27:751–764.

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

Hayward, DV.; Stewart, GE.; Phillips, LM.; Norris, SP.; Lovell, MA. Language, phonological, and reading test directory. Edmonton, Alberta: Canadian Centre for Research on Literacy & Canadian Language and Literacy Network; 2008. Retrieved from www.ualberta.ca

Health Insurance Portability and Accountability Act of 1996, Pub. L. No. 104-191, § 110 Stat. 1936. (1996).

Hobel CJ, Hyvarinen MA, Okada DM, Oh W. Prenatal and intrapartum high-risk screening, I: Prediction of the high-rish neonate. American Journal of Obstetrics and Gynecology. 1973; 117:1–9. [PubMed: 4722373]

Hoffman LM, Loeb DF, Brandel J, Gillam RB. Concurrent and construct validity of oral language measures with school-age children with specific language impairment. Journal of Speech, Language, and Hearing Research. 2011; 53:1597–1608.

Hollingshead, AB. Four factor index of social status. Department of Social Work, Yale University; New Haven, CT: 1975. Unpublished manuscript

Hurt H, Malmud L, Betancourt NL, Brodsky NL, Giannetta JA. A prospective evaluation of early language development in children with in utero cocaine exposure in control subjects. Journal of Pediatrics. 1997; 130:310–312. [PubMed: 9042138]

Jacobson, JL.; Jacobson, SW. Strategies for detecting the effects of prenatal drug exposure: Lessons from research on alcohol. In: Lewis, M.; Bendersky, M., editors. Mothers, babies, and cocaine: The role of toxins in development. Hillsdale, NJ: Erlbaum; 1995. p. 111-128.

Johnson JM, Seikel A, Madison CL. Standardized test performance of children with a history of prenatal drug exposure to multiple drugs/cocaine. Journal of Communication Disorders. 1997; 31:231–244. [PubMed: 9621905]

Kilbride HW, Castor CA, Fuger KL. School-age outcome of children with prenatal cocaine exposure following early case management. Developmental and Behavioral Pediatrics. 2006; 27:181–187.

Landi N, Crowley MJ, Wu J, Bailey CA, Mayes LC. Deviant ERP response to spoken non-words among adolescents exposed to cocaine in utero. Brain and Language. 2011 Advance online publication. 10.1016/j.bandl.2011.09.002

Lester BM, Lagasse LL. Children of addicted women. Journal of Addictive Diseases. 2010; 29:259–276. [PubMed: 20407981]

Lester BM, LaGasse LL, Seifer R. Cocaine exposed children: The meaning of subtle effects. Science. 1998 Oct 23.282:633–634. [PubMed: 9841414]

Lewis BA, Kirchner HL, Short EJ, Minnes S, Weishampel P, Satayathum S, Singer LT. Prenatal cocaine and tobacco effects on children's language trajectories. Pediatrics. 2007; 120:e78–e85. [PubMed: 17606552]

Lewis BA, Minnes S, Short EJ, Weishampel P, Satayathum S, Min MO, Singer LT. The effects of prenatal cocaine exposure on language at 10 years of age. Neurotoxicology and Teratology. 2011; 33:17–24. [PubMed: 20600843]

Lewis BA, O'Donnell B, Freebairn L, Taylor HG. Spoken language and written expression: Interplay of delays. American Journal of Speech-Language Pathology. 1998; 7:66–73.

Lewis BA, Singer LT, Short EJ, Minnes S, Arendt R, Weishampel P, Min MO. Four-year language outcomes of children exposed to cocaine in utero. Neurotoxicology and Teratology. 2004; 26:617–627. [PubMed: 15315811]

Lombardino LJ, Riccio CA, Hynd GW, Pinheiro PB. Linguistic deficits in children with reading disabilities. American Journal of Speech-Language Pathology. 1997; 6:71–78.

Madison CL, Johnson JL, Seikel JA, Arnold M, Schultheis L. Comparative study of the phonology of preschool children prenatally exposed to cocaine and multiple drugs and nonexposed children. Journal of Communication Disorders. 1998; 31:231–243. [PubMed: 9621905]

Malakoff ME, Mayes LC, Schottenfeld RS, Howell S. Language production in 24-month-old inner-city children of cocaine-and-other-drug-using mothers. Journal of Applied Developmental Psychology. 1999; 20:159–180.

Mansoor E, Morrow CE, Accornero VH, Xue L, Johnson AL, Anthony JC, Bandstra ES. Longitudinal effects of prenatal cocaine use on mother-child interactions at 3 and 5 years. Journal of Developmental and Behavioral Pediatrics. 2012; 33:32–41. [PubMed: 22157442]

Mentis M, Lundgren K. Effects of prenatal exposure to cocaine and associated risk factors on language development. Journal of Speech and Hearing Research. 1995; 38:1303–1318. [PubMed: 8747823]

Minnes S, Singer LT, Arendt R, Farkas K, Kirchner HL. Effects of cocaine/poly drug use on maternal-infant feeding interaction over the first year of life. Journal of Developmental and Behavioral Pediatrics. 2005; 26:194–200. [PubMed: 15956868]

Morrow CE, Bandstra ES, Anthony JC, Ofir AY, Xue L, Reyes MB. Influence of prenatal cocaine exposure on early language development: Longitudinal findings from four months to three years of age. Developmental and Behavioral Pediatrics. 2003; 24:39–50.

Morrow CE, Vogel AL, Anthony JC, Ofir AY, Dausa AT, Bandstra ES. Expressive and receptive language functioning in preschool children with prenatal cocaine exposure. Journal of Pediatric Psychology. 2004; 29:543–554. [PubMed: 15347702]

Nathan L, Stackhouse J, Goulandris N, Snowling MJ. The development of early literacy skills among children with speech difficulties: A test of the "critical age" hypothesis. Journal of Speech, Language, and Hearing Research. 2004; 47:337–361.

Nelson NW, Van Meter A. Measuring written language abilities in narrative samples. Reading and Writing Quarterly. 2007; 23:287–309.

Nulman J, Rovet D, Altmann G, Bradley G, Einarson G, Koren G. Neurodevelopment of adopted children exposed in utero to cocaine. Canadian Medical Association Journal. 1994; 15:1591–1597. [PubMed: 7954158]

Potter SM, Zelazo PR, Stack DM, Papageorgiou AN. Adverse effects of fetal cocaine exposure on neonatal auditory information processing. Pediatrics. 2000; 105:e40. [PubMed: 10699142]

Pulsifer MB, Butz AM, Foran MO, Belcher HME. Prenatal drug exposure: Effects on cognitive functioning at 5 years of age. Clinical Pediatrics. 2008; 47:58–65. [PubMed: 17766581]

Scarborough, H. Developmental relationships between language and reading: Reconciling a beautiful hypothesis with some ugly facts. In: Catts, HW.; Kamhi, AG., editors. The connections between language and reading disabilities. Mahwah, NJ: Erlbaum; 2005. p. 3-24.

Scott CM, Nelson NW. Sentence combining: Assessment and intervention applications. Perspectives on Language Learning and Education. 2009; 16:14–20.

Scott CM, Stokes SL. Measures of syntax in school-age children and adolescents. Language, Speech, and Hearing Services in Schools. 1995; 26:309–319.

Semel, E.; Wiig, EH.; Secord, WA. Clinical Evaluation of Language Fundamentals. 3. Austin, TX: Psychological Corp; 1995.

Siefer R, LaGasse LL, Lester B, Bauer CR, Shankaran S, Bada HS, Liu J. Attachment status in children prenatally exposed to cocaine and other substances. Child Development. 2004; 75:850–868. [PubMed: 15144490]

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

Singer LT, Minnes S, Short E, Arendt R, Farkas K, Lewis B, Kirchner HL. Cognitive outcomes of preschool children with prenatal cocaine exposure. JAMA. 2004; 291:2448–2456. [PubMed: 15161895]

Singer LT, Nelson S, Short E, Min MO, Lewis B, Russ S, Minnes S. Prenatal cocaine exposure: Drug and environmental effects at 9 years. Journal of Pediatrics. 2008; 153:105–111. [PubMed: 18571546]

Tan-Laxa MA, Sison-Switala C, Rintelman W, Ostrea EM. Abnormal auditory brainstem response among infants with prenatal cocaine exposure. Pediatrics. 2004; 113:357–360. [PubMed: 14754949]

Trigueiros-Cunha N, Leão P, Renard N, Tavares MA, Eybalin M. Prenatal cocaine exposure accelerates morphological changes and transient expression of tyrosine hydroxylase in the cochlea of developing rats. Brain Research. 2006; 1086:55–64. [PubMed: 16626650]

Uhlhorn DS, Messinger DS, Bauer CR. Cocaine exposure and mother-toddler play. Infant Behavior and Development. 2005; 28:62–73.

Wagner, R.; Torgesen, J. Comprehensive Test of Phonological Processing. San Antonio, TX: Pro-Ed; 1999.

Wechsler, D. Wechsler Adult Intelligence Scale—Revised. San Antonio, TX: The Psychological Corporation; 1989.

Woodcock, RW.; McGraw, KS.; Mather, N. Woodcock–Johnson—III. Itasca, IL: Riverside; 2001.

Zimmerman, IL.; Steiner, VG.; Pond, RE. Preschool Language Scale—3. San Antonio, TX: The Psychological Corporation; 1992.

Table 1

Maternal characteristics for the prenatal cocaine exposure (PCE) and no cocaine exposure (NCE) groups.

	NCE (n = 181)				PCE (n = 183)						
Maternal demographic	***M***	***SD***	***n***	**%**	***M***	***SD***	***n***	**%**	***t***	***p***	χ^2
Mother's age at birth	25.45	4.67			29.70	5.00			−8.39	<.0001*	
Number of prenatal visits	8.72	4.89			5.20	4.57			7.10	<.0001*	
Parity	2.72	1.86			3.54	1.88			−4.17	<.0001*	
Education (yrs)	11.97	1.41			11.51	1.66			2.84	.005*	
PPVT–R standard score	78.03	14.74			73.21	14.25			3.12	.002*	
Block Design scale	7.20	2.08			6.84	2.10			1.63	.10	
Picture Completion scale	7.01	2.38			6.73	2.17			1.13	.26	
GSI	0.50	0.54			0.84	0.76			−5.16	<.0001*	
Average substance use											
Tobacco (cigarettes/day)	3.86	7.19			11.73	11.32			−7.75	<.0001*	
Alcohol (dose/wk)	1.39	4.61			10.10	17.75			−6.26	<.0001*	
Marijuana (dose/wk)	0.60	3.53			1.17	3.27			−1.57	.12	
Cocaine (units/wk)	0.00	—			22.92	38.38			−7.85	<.0001*	
Married			28	15.47			14	7.65		.02*	5.45
African American			146	80.66			151	82.51		.65	0.21
Employed			38	21.11			11	6.04		<.0001*	17.55
Low SES			177	97.79			178	97.80		.99	0.0001

Note. PPVT–R = Peabody Picture Vocabulary Test—Revised; GSI = Global Severity Index; SES = socioeconomic status.

* $p \leq .05$.

Table 2

Child characteristics for the PCE and NCE groups.

	NCE (n = 181)				PCE (n = 183)						
Child demographic	***M***	***SD***	***n***	**%**	***M***	***SD***	***n***	**%**	***t***	***p***	χ^2
1-min Apgar	7.93	1.67			8.00	1.43			−0.44	.66	
5-min Apgar	8.78	0.71			8.78	0.65			−0.06	.95	
Gestational age (wks)	38.49	2.86			37.91	2.82			1.94	.05*	
Hobel total	5.84	15.88			7.18	16.32			−0.79	.43	
Baby length (cm)	49.20	3.71			47.42	3.88			4.45	<.0001*	
Head circumference (cm)	33.49	2.38			32.37	2.08			4.82	<.0001*	
Birth weight (g)	3,110.53	700.70			2,734.48	636.56			6.09	<.0001*	
IQ at age 11 years	86.41	15.02			84.67	11.79			1.23	.22	
Male			87	48.07			82	44.81		.53	0.39
African American			145	80.11			150	81.97		.65	0.20
Microcephalic			8	4.47			27	15.00		.0008*	11.31
Small birth size			4	2.22			23	12.71		.0002*	14.34

* $p \leq .05$.

Table 3

Caregiver characteristics at the 12-year follow-up assessment.

	NCE (n = 181)		PCE: Biological/relative (n = 141)		PCE: Adopt/foster (n = 41)			
Characteristic	***M***	***SD***	***M***	***SD***	***M***	***SD***	***F***	***p***
Home score[a,b]	48.96	6.32	46.94	6.62	50.44	6.27	6.22	.002[*]
Years of education[a,b]	12.72	1.87	11.76	2.06	13.23	2.57	11.61	<.0001[*]
PPVT–R standard score[a,c]	79.78	15.74	76.51	13.49	90.88	14.55	12.23	<.0001[*]
Block Design scale	7.32	1.94	6.96	1.70	7.50	3.18	1.51	.22
Picture Completion scale	7.21	2.37	7.29	2.39	7.81	3.25	0.45	.64
GSI	0.37	0.49	0.40	0.49	0.22	0.21	1.78	.17
Current average substance use								
Tobacco (cigarettes/day)[a,b,c]	3.87	6.77	6.41	7.95	0.69	2.42	17.09	<.0001[*]
Alcohol (dose/wk)[a]	1.55	5.23	1.60	3.98	0.38	1.30	2.68	.07
Marijuana (dose/wk)	0.10	1.07	1.16	8.14	0	0	2.17	.12

Note. One child was in a residential facility at age 12 years.

[a]Biological/relative PCE differs from adopt/foster PCE ($p \leq .05$).

[b]Biological/relative PCE differs from NCE ($p \leq .05$).

[c]Adopt/foster PCE differs from NCE ($p \leq .05$).

[*]$p \leq .05$.

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Table 4

Unadjusted means of language outcomes.

Test	NCE (n = 181)		PCE (n = 183)			
	M	*SD*	*M*	*SD*	*t*	*p*
CTOPP Composite Scores						
Phonological Awareness	83.36	13.50	79.13	11.71	3.19	.002*
Elision	7.18	2.98	6.12	2.68	3.57	.0004*
Blending Words	7.28	2.19	6.93	2.05	1.56	.12
Phonological Memory	94.25	12.87	91.71	11.53	1.98	.05*
Memory for Digits	9.93	3.05	9.69	2.93	0.77	.44
Nonword Repetition	8.15	1.93	7.54	1.81	3.11	.002*
Rapid Naming	94.13	17.40	97.38	15.79	−1.86	.06
Rapid Digit Naming	8.95	2.82	9.40	2.67	−1.56	.11
Rapid Letter Naming	9.10	3.15	9.76	2.89	−2.08	.04*
TOLD–I:3 Composite Quotients						
Sentence Combining	6.88	2.32	6.13	2.13	3.23	.001*
Picture Vocabulary	7.64	2.56	7.46	2.18	0.73	.47
Word Ordering	6.48	3.17	6.24	2.75	0.76	.45
Identifying Generals	6.88	2.75	6.88	2.41	0.02	.99
Grammatical Comprehension	5.94	3.15	5.93	3.08	0.03	.97
Identifying Malapropisms	6.85	1.94	6.91	1.95	−0.33	.74
Woodcock–Johnson—III Reading						
Letter–Word Identification	93.20	15.63	92.45	12.73	0.50	.62
Reading Fluency	90.72	13.89	89.30	11.43	1.06	.29
Passage Comprehension	88.62	12.66	88.27	11.78	0.27	.79

Note. CTOPP = Comprehensive Test of Phonological Processing; TOLD–I:3 = Test of Language Development—Intermediate, Third Edition.

* $p \le .05$.

Table 5

Adjusted means of language outcome measures, by group.

Test	NCE (*n* = 181)		PCE (*n* = 183)			
	M	***SE***	***M***	***SE***	***t***	***p***
CTOPP Composite Scores						
Phonological Awareness[a]	82.81	0.86	79.57	0.90	2.38	.02*
Elision[b]	7.06	0.19	6.32	0.20	2.51	.01*
Blending Words[c]	7.26	0.16	6.70	0.19	1.98	.05*
Phonological Memory[d]	93.67	0.91	91.65	0.96	1.43	.15
Memory for Digits[e]	9.85	0.22	9.71	0.22	0.47	.64
Nonword Repetition[f]	8.04	0.14	7.64	0.15	1.87	.06
Rapid Naming[g]	94.28	1.35	97.44	1.39	−1.49	.13
Rapid Digit Naming[h]	8.95	0.21	9.37	0.22	−1.29	.20
Rapid Letter Naming[i]	9.01	0.23	9.95	0.24	−2.68	.008*
TOLD–I:3 Composite Quotients						
Sentence Combining[j]	6.74	0.15	6.20	0.18	2.13	.03*
Picture Vocabulary[k]	7.60	0.16	7.31	0.19	1.04	.30
Word Ordering[l]	6.26	0.19	6.05	0.22	0.66	.51
Identifying Generals[m]	6.88	0.17	6.72	0.20	0.53	.59
Grammatical Comprehension[n]	5.88	0.22	5.24	0.26	1.68	.09
Identifying Malapropisms[o]	6.85	0.14	6.81	0.15	0.17	.87

[a]Phonological Awareness: Adjusted for home score, mother's age at birth, maternal education, current caregiver education, maternal PPVT score, maternal block design scale, maternal alcohol exposure month prior, and IQ.

[b]Elision: Adjusted for home score, mother's age at birth, parity, current caregiver education, maternal marital status, maternal PPVT score, maternal block design scale, current caregiver average cigarette use, and IQ.

[c]Blending Words: Adjusted for home score, mother's age at birth, current caregiver education, maternal PPVT score, maternal block design scale, current caregiver block design scale, first trimester prenatal cigarette use, first trimester prenatal alcohol exposure, current caregiver average marijuana exposure, and IQ.

[d]Phonological Memory: Adjusted for current caregiver education, maternal PPVT score, second trimester prenatal alcohol exposure, first trimester marijuana exposure, and IQ.

[e]Memory for Digits: Adjusted for first trimester prenatal marijuana exposure and IQ.

[f]Nonword Repetition: Adjusted for home score, maternal education, current caregiver education, maternal PPVT score, third trimester prenatal alcohol exposure, and IQ.

[g]Rapid Naming: Adjusted for maternal GSI score, second trimester prenatal cigarette use, third trimester prenatal alcohol exposure, current caregiver average marijuana exposure, and IQ.

[h]Rapid Digital Naming: Adjusted for maternal GSI score, current caregiver average cigarette use, third trimester prenatal alcohol exposure, current caregiver average marijuana exposure, and IQ.

[i]Rapid Letter Naming: Adjusted for maternal GSI score, third trimester prenatal alcohol exposure, current caregiver average marijuana exposure, and IQ.

[j]Sentence Combining: Adjusted for home score, parity, maternal marital status, current caregiver education, maternal PPVT score, current caregiver block design scale, third trimester prenatal cigarette use, first trimester prenatal alcohol exposure, first trimester prenatal marijuana exposure, and IQ.

[k]Picture Vocabulary: Adjusted for home score, parity, maternal marital status, current caregiver education, maternal PPVT score, maternal block design scale, current caregiver block design scale, first trimester prenatal cigarette use, average prenatal alcohol exposure, first trimester prenatal marijuana exposure, and IQ.

[l]Word Ordering: Adjusted for home score, parity, maternal marital status, maternal education, current caregiver education, maternal PPVT score, maternal block design scale, current caregiver block design scale, average prenatal alcohol exposure, and IQ.

[m]Identifying Generals: Adjusted for home score, mother's age at birth, parity, maternal education, current caregiver education, maternal PPVT score, maternal block design scale, current caregiver block design scale, average prenatal cigarette use, third trimester prenatal alcohol exposure, first trimester prenatal marijuana exposure, and IQ.

[n]Grammatical Comprehension: Adjusted for home score, mother's age at birth, maternal education, current caregiver education, maternal PPVT score, maternal block design scale, current caregiver block design scale, prenatal cigarette use month prior, third trimester prenatal alcohol exposure, prenatal marijuana exposure month prior, and IQ.

[o]Identifying Malapropisms: Adjusted for home score, mother's age at birth, parity, maternal education, current caregiver education, maternal PPVT score, second trimester prenatal cigarette use, first trimester prenatal marijuana exposure, and IQ.

[*] $p \leq .05$.

Table 6

Association of reading outcomes at age 11 years with significant language findings at age 12 years.

Test	β	*b* (*SE*)	*t*	*p*
Letter–Word Identification (R^2 = .53)				
Elision	.34	1.66 (0.22)	7.57	<.0001
Blending Words	.12	0.81 (0.28)	2.89	.004
Sentence Combining	.33	2.06 (0.28)	7.45	<.0001
Rapid Letter Naming	.24	1.11 (0.17)	6.39	<.0001
Reading Fluency (R^2 = .49)				
Elision	.19	0.83 (0.20)	4.09	<.0001
Blending Words	−.03	−0.16 (0.26)	−0.61	.54
Sentence Combining	.41	2.29 (0.26)	8.93	<.0001
Rapid Letter Naming	.40	1.68 (0.16)	10.38	<.0001
Passage Comprehension (R^2 = .51)				
Elision	.26	1.10 (0.19)	5.72	<.0001
Blending Words	.08	0.48 (0.24)	1.97	.05
Sentence Combining	.41	2.22 (0.24)	9.20	<.0001
Rapid Letter Naming	.24	0.94 (0.15)	6.23	<.0001

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Table 7A

Correlation of covariates with language outcomes: Prenatal drug exposures and maternal characteristics.

	Prenatal drug exposures						Maternal characteristics									
	1st trimester cigarette exposure		Average prenatal cigarette exposure		3rd trimester alcohol exposure		Parity		Maternal marital status		Maternal PPVT		Mother's age at birth		Maternal Block Design	
Test	***r***	***p***	***r***	***p***	***r***	***p***	***r***	***p***	***r***	***p***	***r***	***p***	***r***	***p***	***r***	***p***
CTOPP Composite Scores																
Phonological Awareness	−.04	.44	−.05	.32	−.18	.0006*	−.13	.01*	.08	.11	.23	<.0001*	−.12	.02*	.24	<.0001*
Elision	−.12	.02*	−.14	.01*	−.20	.0002*	−.15	.003*	.09	.08	.22	<.0001*	−.11	.04*	.22	<.0001*
Blending Words	.08	.12	.08	.14	−.10	.06	−.05	.36	.04	.41	.16	.003*	−.09	.07	.18	.001*
Phonological Memory	−.02	.70	−.01	.82	−.09	.10	−.05	.36	−.005	.92	.08	.12	.005	.92	.08	.12
Memory for Digits	.008	.88	.006	.91	−.04	.50	−.02	.66	−.04	.42	.006	.91	.02	.74	.04	.47
Nonword Repetition	−.05	.31	−.04	.51	−.14	.01*	−.07	.20	.06	.28	.17	.001*	−.02	.74	.12	.02*
Rapid Naming	.03	.52	.04	.49	−.10	.07	.02	.65	−.03	.55	.02	.66	.008	.88	.002	.97
Rapid Digit Naming	.05	.39	.05	.32	−.08	.13	.04	.50	−.02	.76	−.002	.97	.002	.97	−.01	.81
Rapid Letter Naming	.03	.60	.02	.64	−.10	.06	.02	.67	−.04	.43	.05	.37	.02	.67	.01	.85
TOLD–I:3 Composite Quotients																
Sentence Combining	−.14	.009*	−.14	.008*	−.16	.003*	−.13	.01*	.09	.07	.20	.0002*	−.06	.23	.11	.04*
Picture Vocabulary	.09	.11	.08	.14	−.17	.002*	−.16	.002*	.06	.28	.31	<.0001*	.05	.34	.27	<.0001*
Word Ordering	−.01	.83	−.02	.76*	−.13	.01*	−.07	.19	.07	.17	.26	<.0001*	.05	.38	.15	.005*
Identifying Generals	.12	.02*	.12	.03*	−.14	.007*	−.14	.009*	−.04	.40	.32	<.0001*	.07	.21	.26	<.0001*
Grammatical Comprehension	.08	.12	.10	.05*	−.10	.05*	−.06	.26	−.03	.61	.21	<.0001*	.09	.07	.12	.02*
Identifying Malapropisms	.11	.04*	.10	.07	−.09	.09	−.08	.13	.008	.89	.24	<.0001*	.11	.04*	.20	.0002*

* $p \leq .05$.

Table 7B

Correlation of covariates with language outcomes: Current caregiver and child characteristics.

	Current caregiver characteristics								Child characteristic	
	Current caregiver education		Home score		Current caregiver Block Design		Current marijuana exposure		IQ	
Test	***r***	***p***	***r***	***p***	***r***	***p***	***r***	***p***	***r***	***p***
CTOPP Composite Scores										
Phonological Awareness	.16	.002*	.14	.007*	.21	<.0001*	−.06	.26	.58	<.001*
Elision	.17	.001*	.13	.02*	.15	.005*	−.01	.83	.57	<.0001*
Blending Words	.10	.07	.11	.03*	.22	<.0001*	−.10	.05*	.40	<.0001*
Phonological Memory	.03	.62	.09	.08	.04	.41	−.07	.19	.44	<.0001*
Memory for Digits	−.02	.64	.06	.29	.02	.77	−.07	.22	.36	<.0001*
Nonword Repetition	.10	.07	.11	.03*	.07	.18	−.05	.36	.37	<.0001*
Rapid Naming	.02	.76	.02	.73	.01	.81	.16	.002*	.35	<.0001*
Rapid Digit Naming	.006	.91	.03	.58	−.02	.77	.14	.008*	.33	<.0001*
Rapid Letter Naming	.02	.66	.007	.89	.03	.55	.16	.002*	.34	<.0001*
TOLD–I:3 Composite Quotients										
Sentence Combining	.11	.03*	.19	.0003*	.13	.02*	.02	.68	.62	<.0001*
Picture Vocabulary	.18	.0005*	.20	.0001*	.21	<.0001*	−.01	.85	.60	<.0001*
Word Ordering	.12	.02*	.18	.0006*	.17	.002*	.04	.48	.62	<.0001*
Identifying Generals	.19	.0002*	.17	.002*	.23	<.0001*	−.005	.93	.65	<.0001*
Grammatical Comprehension	.23	<.0001*	.13	.01*	.19	.0003*	−.06	.29	.45	<.0001*
Identifying Malapropisms	.15	.005*	.21	<.0001*	.14	.01*	.01	.84	.51	<.0001*

* $p \leq .05$.