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Risk-Taking Behavior among Adolescents with Prenatal Drug Exposure and Extrauterine Environmental Adversity

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Abstract

Objective—High-risk environments characterized by familial substance use, poverty, inadequate parental monitoring, and violence exposure are associated with an increased propensity for adolescents to engage in risk-taking behaviors (e.g., substance use, sexual behavior, and delinquency). However, additional factors such as drug exposure in utero and deficits in inhibitory control among drug-exposed youth may further influence the likelihood that adolescents in high-risk environments will engage in risk-taking behavior. This study examined the influence of prenatal substance exposure, inhibitory control, and sociodemographic/environmental risk factors on risk-taking behaviors in a large cohort of adolescents with and without prenatal cocaine exposure (PCE).

Method—Risk-taking behavior (delinquency, substance use, and sexual activity) was assessed in 963 adolescents (433 cocaine-exposed, 530 nonexposed) at 15 years of age.

Results—PCE predicted later arrests and early onset of sexual behavior in controlled analyses. Associations were partially mediated, however, by adolescent inhibitory control problems. PCE

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was not associated with substance use at this age. In addition, male gender, low parental involvement, and violence exposure were associated with greater odds of engaging in risk-taking behavior across the observed domains.

Conclusions—Study findings substantiate concern regarding the association between prenatal substance exposure and related risk factors and the long-term outcomes of exposed youth. Access to the appropriate social, educational, and medical services are essential in preventing and intervening with risk-taking behaviors and the potential consequences (e.g., adverse health outcomes, incarceration), especially among high-risk adolescent youth and their families.

Keywords

prenatal drug exposure; cocaine; adolescence; risk-taking behavior

In general, adolescence is a developmental period characterized by increased risk-taking behaviors. Risk-taking behaviors typically involve those that are potentially damaging to one's health, such as substance use, precocious or risky sexual behavior, stealing, fighting, and truancy (1). Increased willingness to engage in such behaviors among adolescents has also been associated with living in high-risk social and cultural environments (2–3) characterized by caregiver substance use, poverty, poor/inconsistent family management practices, association with delinquent peers, and exposure to domestic and/or community violence (4–9).

Involvement in delinquent behavior in adolescence is particularly concerning especially given that the consequences of such behavior may result in incarceration or serious damage to one's physical and/or mental health. Delinquent behaviors (e.g., aggression/fighting, disregard for safety precautions, crimes against people, gang involvement) have been observed in samples of pre-adolescents and adolescents born to substance abusing mothers, including those with prenatal cocaine exposure (PCE; 4, 7, 9–15). Such deviant behaviors are often most prevalent in males, both cocaine-exposed and nonexposed (9–11, 15). Follow-up studies of preadolescents and adolescents with PCE have also reported evidence that other factors—prenatal and postnatal nicotine exposure (13, 15), childhood exposure to violence (9, 12–13), ongoing caregiver substance use (4), and association with violent peers (9)—are associated with self-reported delinquent behavior. In addition, deficits in inhibitory control among children with PCE have been observed in recent research suggesting that exposed youth may be at increased risk of developing subsequent behavioral problems in part due to difficulties in regulating attention and impulsivity (16–18). These studies emphasize the potential mediating relationship adolescent inhibitory control has on later risk-taking behavior among cocaine-exposed youth.

Familial drug abuse and associated environmental factors have also been associated with a 2.4–4 fold factor increase in the development of a substance abuse disorder during adolescence (19–20). Thus, substantial concern exists regarding the potential implications of PCE and related environmental factors (e.g., poverty, ongoing caregiver substance use, lack of parental monitoring) that may increase risk for deviant social behavior and the initiation of substance use among exposed adolescents. Few studies have examined substance use in adolescents with PCE and results of the published studies are somewhat mixed. Among

preadolescent children (10–12 years of age), no direct association was observed between PCE and preadolescent drug use as determined via self-report (9) and hair analysis (21). However, Bennett et al. (2007) reported that preadolescent boys with PCE had a higher incidence of substance use as well as aggression and disregard for safety precautions compared to non-exposed controls. Studies of slightly older adolescents have yielded somewhat more consistent results. Delaney-Black et al. (2011) reported that both ongoing caregiver cocaine use and PCE predicted adolescent cocaine use at 14 years of age as assessed via self-report and drug testing. Over half of the biological mothers who used cocaine during pregnancy tested positive for current cocaine use at the 14-year child visit. Another study of 16-year-olds with PCE found an increased likelihood of both licit and illicit substance use in adolescents with the highest levels of PCE (12). These adolescents most commonly reported use of alcohol and marijuana. A recent report using the same cohort as the current study found evidence that neurobehavioral dysregulation (a construct assessing internalizing/externalizing behavior problems, psychopathology, and delinquent behaviors such as crimes against persons, theft, and truancy) was associated with initiation of substance use in youth with PCE between 8 and 16 years of age (14). In that study, PCE appeared to be a pathway through which subsequent neurobehavioral disinhibition influenced substance use among youth. Overall, these studies provide evidence supporting the transgenerational perpetuation of illicit substance use and/or abuse among adolescents with PCE.

Another area of concern among high-risk adolescents is the potential for risky or early onset of sexual behavior. Sexual activity among adolescents has been found to be significantly higher in urban versus suburban samples (22). In addition, several other factors have been associated with precocious or risky sexual behavior among adolescents such as parent divorce, inadequate parental monitoring or family involvement, male gender, and low SES (5–6; 23). Such factors may have significant implications on adolescent health and safety. For example, DiClemente et al. (5) reported that adolescents with less perceived parental monitoring were more likely to report lack of condom use or any form of contraception in their most recent sexual intercourse experiences and were 1.7 times more likely to have a lab-confirmed sexually transmitted disease (STD). In addition, parental substance abuse has been associated with early onset of sexual activity, particularly among males (23). To our knowledge, no published studies to date have examined the association between PCE and related sociodemographic/environmental risk factors with adolescent sexual behavior.

The purpose of the current study was to examine the influence of prenatal substance exposure and related risk factors on risk-taking behavior of adolescents with and without PCE in a large, multisite, predominantly urban sample. In addition, given observed deficits in inhibitory control among youth with PCE, difficulties in inhibitory control were explored as a potential mediator between PCE and subsequent risk-taking behavior. Dependent variables examined in this study included adolescent arrests, alcohol and drug use, and early onset of sexual activity. Specific delinquent behaviors were not examined as outcomes since they have been previously reported on (7, 14).

METHOD

Participants

Participants were enrolled at birth in the Maternal Lifestyle Study (MLS), a prospective, longitudinal investigation of the effects of prenatal cocaine and/or opiate exposure on short- and long-term child outcomes. Institutional review board approval and maternal consent for participation were obtained at each study site (Brown University, the University of Miami, the University of Tennessee-Memphis, and Wayne State University). See Bauer et al. (2005; 24) for detailed information regarding screening and participant enrollment. Exposure status was determined by maternal admission of drug use and/or a positive enzyme multiplied immunoassay technique (EMIT) screening for drug metabolites in meconium confirmed by gas chromatography/mass spectroscopy (GC/MS; 25). Nonexposure was determined by denial of cocaine and/opiate use during pregnancy and a negative meconium toxicology screen. Cocaine-exposed and comparison infants were individually matched on race, gender, and gestational age upon enrollment in this study in the hospital. A total of 1388 dyads (658 cocaine/opiate exposed and 730 nonexposed) attended the first clinic visit at 1 month of age. There were 22 visits scheduled between the 1-month and 15-year visits.

Of the original 1388 dyads enrolled in the study, 1041 attended the 15-year visit which was the primary inclusion criteria for current analyses. Adolescents were excluded if they had incomplete data ($n = 41$) or had prenatal opiate exposure only (no cocaine exposure; $n = 37$). The final sample consisted of 963 adolescents (433 with PCE and 530 with no cocaine exposure). Adolescents included in the analyses were more likely to have PCE, to be black, and to be female ($p < .05$) than adolescents lost to follow-up ($n = 425$). There were no group differences in prenatal alcohol, tobacco, or marijuana exposure, maternal education, or SES.

Measures

Prenatal Drug Exposure—Cocaine use during pregnancy was recorded dichotomously (yes/no) based on maternal self-report and meconium analysis of cocaine metabolites by GC/MS at birth. In supplemental analyses, PCE was defined in a dose-related manner when a significant association was found between the dichotomous PCE variable and specific adolescent risk-taking outcomes. The three-level PCE variable used in these analyses was defined as: 1) high PCE (≥ 3 times/week during the first trimester), some PCE (any other use during pregnancy), and no PCE. In our sample, 822 of the 963 participants had data for cocaine level (99 with high PCE [12%], 197 with some PCE [24%], and 526 with no PCE [64%]). Other drug exposures were included as control variables in analyses. Prenatal alcohol, tobacco, and marijuana were recorded dichotomously (yes/no) based on maternal self-report.

Sociodemographic and Environmental Risk Factors—Sociodemographic risk variables included: child gender, maternal race (black vs. other), maternal education at birth ($<$ high school vs. high school), and caregiver SES. Caregiver SES was the average Hollingshead Index of Social Position score (26) across all visits with higher scores indicating higher SES. Other aspects of the family environment were also assessed. Level of parental involvement was defined as the mean score of six items from the parent-report form

of the Child Health and Illness Profile-Adolescent Edition (27) administered at the 15-year visit. Items included how often adults in the home spent time with the adolescent (e.g., doing something fun, eating meals, talking and listening to adolescent). The scale has a Cronbach's alpha of 0.79 for this study sample. Caregiver abuse (yes/no) was determined by self-report of the caregiver's experience of physical or sexual violence. Child abuse (yes/no) was based on report of abuse/neglect to Child Protective Services at any visit between birth and 15 years. Total number of caretaker changes was based on information gathered at all visits. Caregiver arrest history was defined dichotomously (yes/no) as reported at the 4, 4 ½, and 11-year study visits. Caregiver postnatal cocaine intake (any use vs. no use) was assessed via caregiver-report from birth to 15 years. Other caregiver substance use variables were averaged based on caregiver report from birth to 15 years: Caregiver postnatal alcohol use (avg. ounces of absolute alcohol p/day), postnatal tobacco use (avg. # of cigarettes p/day), and postnatal marijuana use (avg. # of joints p/day).

Adolescent Arrests and Substance Use—Adolescent arrest history (yes/no) was assessed via self-report using the Police Contact questionnaire (28) while alcohol and drug use was assessed via adolescent self-report on the Things That You Have Done interview (TTYHD; 29–30). The TTYHD interview was administered using audio computer-assisted self-interviewing (ACASI) technology to facilitate more accurate reporting of behaviors that are highly sensitive/illegal (31). Substance use (yes/no) was based on the previous 12 months and categorized as 1) alcohol use, 2) tobacco use, and 3) marijuana use. Given the low occurrence of binge/problematic alcohol use and illicit drug use in this sample, adolescent alcohol abuse and use of other types of drug was not examined (e.g., opiates, methamphetamine, cocaine, hallucinogens, misuse of prescription medications). Adolescent-report of friends' substance use within the past 12 months was also measured using a parallel interview called Things that Your Friends Have Done (29).

Adolescent Sexual Behavior—Adolescent sexual behavior was assessed using the Adolescent Sexual Behavior Assessment (32) administered via ACASI. Self-reported sexual behaviors (yes/no) were categorized as 1) touching a partner's genitals; 2) engaging in oral sex; or 3) engaging in penetrative sex (vaginal/anal) within the adolescent's lifetime. Sexual experiences were considered for both opposite and same-sex partners. Initiation of adolescent sexual behaviors was categorized as having early onset versus average/no onset. Based on the data distribution of the current study sample, a 25th percentile cut-off was used to define early onset. Early onset was defined as initiation of sexual behavior occurring at age 13 or younger for touching and at age 14 or younger for oral or penetrative sex. In addition, adolescent condom use was also assessed and defined as having had sex without a condom during the adolescent's most recent sexual encounter (yes/no).

The Dating History Questionnaire (33) was also administered to adolescents by trained research examiners to assess adolescents' involvement in and perceptions of romantic experiences—whether they have ever had a boyfriend/girlfriend, have ever been in love, and view oneself as a “player”. The “player” item was included to help capture social desirability related to reporting of sexual behavior. These variables were coded dichotomously (yes/no).

Adolescent Inhibitory Control—Inhibitory control was examined as a potential mediator of adolescent risk-taking behavior given previous evidence of deficits in this area among youth with PCE. Inhibitory control was assessed via the attention problem subscale of the Child Behavior Checklist (CBCL; 34–35). Items on this subscale include features of inattention, hyperactivity, and impulsivity. The CBCL was administered verbally to caregivers by a trained research interviewer at the 13-year visit to assess recent adolescent behavior. T-scores were examined continuously in analyses.

Analyses

Chi-square tests were conducted comparing exposure groups (PCE vs. nonexposed) on risk-taking behaviors and demographics. Logistic regression models were then computed predicting each risk-taking behavior by PCE, accounting for all sociodemographic and environmental factors and study site. Where possible, the youth's report of whether his/her friends did the applicable behavior was also included in models. Models of early onset of sexual behaviors included the three aforementioned covariates regarding the adolescents' perceptions of their involvement in romantic relationships. Given previous literature supporting gender as a significant moderator of PCE on subsequent youth behavioral problems, the interaction between PCE and gender was tested for each model but only retained in the model if significant ($p < .05$). Lastly, mediation analyses were conducted if a significant association was found between PCE and a specific behavior in order to investigate the role of inhibitory control in potentially mediating this association. The effect of PCE on the behavior would be expected to decrease significantly after accounting for inhibitory control if a mediation relationship exists. Mediation was tested by computing a series of regression models to estimate direct and indirect effects and then calculated a Sobel test after accounting for dichotomous outcomes (36).

RESULTS

Sociodemographic characteristics, the distribution of reported behaviors, and mean inhibitory control scores between exposure groups are reported in Table 1. Groups were comparable on gender, age, and race. Adolescents with PCE had mothers with less education at birth and caregivers with higher SES, p 's $< .01$ consistent with previous findings in this study sample. PCE was also associated with increased likelihood of prenatal exposure to alcohol, tobacco, and marijuana, p 's $< .001$. In terms of substance use, there were no group differences for adolescent alcohol or tobacco use but there was a higher proportion of reported marijuana use among adolescents with PCE (13%; $p = .04$) compared to nonexposed adolescents (9%). Lastly, PCE was associated with an increased likelihood of adolescents having been arrested and engaging in sexual behaviors (touching, oral sex, penetrative sex), p 's $< .05$. There was no group difference in terms of having sex without a condom.

Adolescent Arrests

A logistic regression model was conducted predicting the association between PCE and related sociodemographic and environmental risk factors and adolescent police involvement resulting in arrest (see Table 2). In controlled analyses, PCE was associated with adolescent

arrests. Specifically, adolescents with PCE had increased odds of having been arrested by age 15 compared to nonexposed peers (OR, 2.21; 95% CI, 1.15–4.23), $p < .05$. Male gender and higher caregiver SES were also associated with increased odds of having been arrested. None of the other covariates (study site, prenatal alcohol, tobacco, and marijuana exposure, maternal education, race, adolescent age, parental involvement, caretaker or child abuse, community violence, caretaker changes, caregiver postnatal substance use, and caregiver arrest history) were associated with adolescent arrest history. Data was not available for friends' arrest history. There was also no significant interaction between PCE and gender on adolescent arrests.

In supplemental analyses, the three-level PCE variable was examined as a predictor of adolescent arrests controlling for the aforementioned covariates. Results of a logistic regression model indicated that there was no association between high PCE (versus no PCE) and adolescents arrests (OR, 1.33; 95% CI, 0.44–3.99; $p = .61$); however, there was a significant association between some PCE (versus no PCE) and adolescent arrests (OR, 3.13; 95% CI, 1.49–6.54; $p = .003$).

Adolescent Alcohol and Drug Use

Separate logistic regression analyses were conducted examining PCE and covariates as predictors of adolescent alcohol, tobacco, and marijuana use at 15 years of age. PCE was not associated with alcohol, tobacco, or marijuana use (see Table 2). However, higher adolescent age was associated with more likelihood of alcohol use, adolescent exposure to caretaker abuse (domestic violence) was associated with a decreased likelihood of alcohol use, and more caretaker changes were associated with a decreased likelihood of alcohol use. Black race and domestic violence were associated with a decreased likelihood of tobacco use. Less maternal education at birth was associated with increased adolescent marijuana use at 15 years of age. With regard to peer substance use, peer alcohol, tobacco, and marijuana use was significantly associated with adolescent's use of respective substances in the study sample with approximately 39–70 times greater odds for adolescents to engage in alcohol, tobacco, and marijuana use if their friends were as well (Table 2).

Adolescent Sexual Behavior

Table 3 illustrates results of separate logistic regression models predicting early onset of adolescent sexual behaviors as well as adolescents' use of a condom in their most recent sexual encounter. Adolescents with PCE had nearly two times greater odds of engaging in oral sex (OR, 1.90; CI, 1.17–3.06), $p < .05$. In addition, prenatal marijuana exposure was associated with early onset of penetrative sex. Adolescents who had mothers with lower education were more likely to engage in sex without a condom during their most recent sexual encounter. Lower levels of parental involvement were associated with early onset of touching, oral sex, and penetrative sex. Child abuse was associated with an increased likelihood of having engaged in oral sex by age 14 and having had sex without a condom during their most recent sexual encounter. Exposure to higher levels of community violence was also associated with increased odds of early onset of touching, oral sex, and penetrative sex. Compared to females, males were more likely to have early onset of sexual touching, oral sex, and penetrative sex. In addition, adolescents who reported having been in love,

having had a girlfriend or boyfriend, and considering oneself a “player” had 2.2 to 12.5 times greater odds of engaging in sexual behaviors, p 's < .05. Adolescents who considered themselves to be a “player” also had greater odds of having sex without a condom. Results of PCE×gender interaction analyses indicated that male adolescents with PCE had greater odds of having sex without a condom as well compared to males without PCE (See Table 3).

In supplemental analyses, the three-level PCE variable was examined as a predictor of early onset of oral sex among adolescents controlling for the aforementioned covariates. Results of a logistic regression model indicated that there was no association between high PCE (versus no PCE) and early onset of oral sex (OR, 1.39; 95% CI, 0.64–3.00; p = .41) but there was a significant association between some PCE (versus no PCE) and early onset of oral sex (OR, 1.92; 95% CI, 1.09–3.38; p = .02).

Inhibitory Control as a Mediator of PCE

Figure 1 presents the direct effects of PCE on arrests and early onset of oral sex, as well as the indirect effects of PCE via difficulties in inhibitory control. Results of Sobel tests of mediation indicated that the direct effect between PCE and these outcome variables decreased when including inhibitory control problems, supporting the role of inhibitory control problems as a partial mediator. It should be noted though that the mediation effect between PCE and early onset of oral sex was marginally significant (p = .05).

DISCUSSION

Rates of risk-taking behavior were relatively high in this adolescent sample as might be expected developmentally. However, characteristics of their high-risk social and cultural environment were associated with an increased likelihood of engaging in risky behavior (police involvement resulting in arrests, substance use, and risky or precocious sexual behavior) among adolescents in this sample.

Adolescent Arrests

Approximately 12% of adolescents with PCE reported having been arrested by age 15 approaching the national average (15.9% to 26.8%) in youth through age 18 (37). PCE was also a significant predictor of adolescent arrests in controlled analyses with adolescents with some PCE more likely to experience arrests than those with no PCE. These findings are consistent with evidence (14) suggesting that PCE is one pathway through which subsequent neurobehavioral dysregulation may emerge. Other notable sociodemographic factors (male gender, caregiver SES) were associated as well with increased odds of arrests. The emergence of externalizing problems (e.g., aggression, fighting) in males with PCE between early school-age and adolescence has been well-documented in the literature (9,–11, 15), with some arguing that males may be more vulnerable to prenatal drug exposures that influence the developing brain systems involved in emotional regulation and inhibitory control (38–39). It is also important to note, however, the potential limitation of assessing arrest history as a marker of delinquency among African American males. The actual occurrence of arrests in the current sample may in part reflect police behavior towards African American males, which has been historically bias, compared to females and other

ethnic groups. On the other hand, adolescent self-report data may also underrepresent undetected delinquent behaviors not resulting in arrest. Thus, these factors should be taken into account when interpreting study findings. Lastly, although it was not directly be assessed in this study, adolescents' affiliation with deviant peers may also be more likely for adolescents reared in urban environments characterized by poverty and increased violence, subsequently increasing risk for involvement in delinquent activities.

Adolescent Alcohol and Drug Use

PCE was not associated with adolescent substance use in the current study. The literature regarding substance use among youth with PCE is somewhat mixed; however, significant findings have been reported for youth age 14–16. Availability of biological confirmation of substance use would have substantially strengthened this study to more objectively assess substance use within this sample. When findings were compared to national estimates, patterns of adolescent substance use appeared similar to what might be expected. The national estimate for marijuana use among high school students is 20.8% with Black students estimated to use marijuana more often than their white and Hispanic peers (40). In this study, adolescents with PCE endorsed marijuana use (13%) more than nonexposed adolescents (9%) but these proportions remained lower than national averages. Further, Black adolescents were less likely to endorse tobacco use compared to adolescents of other races, mirroring national trends in which Hispanic and white adolescents report more frequent and regular tobacco use (40).

Beyond prenatal drug exposure, several peer and environmental factors were associated with adolescent substance use. In correspondence with national trends, older adolescents reported more alcohol use than younger teens (40). Interestingly though, adolescent exposure to domestic violence and more caretaker changes were associated with decreased likelihood of substance use. Lower maternal education at birth was also associated with greater odds of adolescent marijuana use. With regard to domestic violence, it is possible that older children may cope more actively either trying to prevent or intervene with the abuse or provide support to the victimized parent rather than coping through other means (e.g., substance use) (41). With regard to other family circumstances, Hawkins et al. (1992; 8) noted that availability of substances, the family's attitude towards substance use, and the family's use of alcohol and drugs were significant risk factors for the development of alcohol and drug problems among adolescents. It is possible that adolescents reared by caregivers with less knowledge about the impact of substance use or who use substances regularly are more likely to have access to these substances within the home and possibly find substance use to be a more acceptable behavior than peers who are not reared in such environments. However, if adolescents moved more often they may have been less likely to live with their biological parent who may have been involved in substance use, thus they may be less likely to have access to alcohol or other drugs in the home.

Despite the association of other factors on adolescent substance use, peer influence appeared to be the central factor associated with the likelihood of adolescent substance use above and beyond any other factor. This emphasizes the importance for parents in monitoring the peer relationships their children develop especially during adolescence.

Adolescent Sexual Behavior

In 2010, the Center for Disease Control and Prevention (CDC; 42) estimated that approximately 46% of high school students (9th–12th grade) have engaged in sexual intercourse. Among ninth graders in particular, prevalence of sexual intercourse was 31.6%. The prevalence of penetrative sex by age 14 among adolescents in our sample with PCE exceeded this national average for 9th graders. There was also a dose-related effect observed in this study in that adolescents with some PCE (vs. none) were more likely to engage in oral sex before age 14. To our knowledge, no other studies have reported associations between PCE and sexual behavior. Similar to other behavioral outcomes, findings may suggest that PCE contributes to some degree to behavioral disinhibition in exposed youth.

Similar to previous evidence (22), several risk factors (i.e., prenatal marijuana exposure, low maternal education, male gender, low parental involvement, child abuse, and exposure to community violence) were also associated with early onset of sexual behavior suggesting that within a sample of predominantly urban, high-risk adolescents parent/caregiver substance use and related environmental risks tend to increase the odds for early onset of adolescent sexual behaviors. It is likely that caregivers with a substance use history are less engaged in the daily activities of their children's lives and are more likely to live in economically deprived communities that tend to expose youth to higher levels of violence and tend to have limited access to community resources (e.g., after school programs/extracurricular activities for youth, sexual education/parenting resources, and mental health and substance abuse services). Low parental involvement/monitoring, in particular, has been found to be associated with adolescent sexual behavior as well as decreased likelihood of adolescents using any kind of contraception during sexual intercourse and increased likelihood of having a lab-confirmed STD (5). Lastly, although there was no significant association for race/ethnicity, findings mirror evidence suggesting that Black youth typically have their first sexual experiences earlier than white or Hispanic adolescents (43). Together, these findings raise concerns regarding the potential consequences of precocious or risky sexual behavior in at-risk youth.

Lastly, adolescents' perceptions of their romantic experiences were significantly associated with early onset of sexual behavior suggesting that how adolescents perceive their role or degree of involvement in romantic experiences has a considerable influence on their tendency to engage in sexual behavior. In this study, the odds of engaging in penetrative sex was nearly 13 times higher if the adolescent perceived him/herself as having dated or been in a committed relationship with at least one partner (i.e., having a girlfriend/boyfriend) clearly demonstrating the importance of dating and relationship formation in the onset of sexual behavior during adolescence. Harper et al. (2004; 44) further emphasized the roles of close friends in dating and sexual behaviors among inner-city African American adolescents, in particular, arguing that peer influence plays a significant role in how youth perceive their dating experiences and acquire new dating/sexual partners. It is essential to take such factors into account when approaching issues such as sex education with adolescents.

Inhibitory Control Problems as a Mediator

Problems with inhibitory control (attention, hyperactivity, and impulsivity) partially mediated the association between PCE and arrest history and early onset of oral sex. These findings mirror similar results using this study sample in which Fisher et al. (2011) reported an association between prenatal substance exposure (including PCE) and longitudinal growth in behavioral dysregulation (e.g., attention problems, internalizing/externalizing problems, and psychopathology) through age 14, controlling for environmental adversity (7). A follow-up to this report also found evidence that PCE contributed to neurobehavioral dysregulation among adolescents and that this dysregulation in turn was associated with initiation of substance use among exposed youth (14). These findings as well as others (45–46) suggest that there are multiple pathways through which prenatal substance exposure can increase risk for later behavior problems. While a direct association may exist between PCE and subsequent behavior problems, indirect pathways of influence also exist by which alterations in neurobehavioral regulation may affect later behavior expression such as difficulties in regulating attention or impulse control or displays of risky, precocious, or aggressive behavior.

Limitations

There were some noteworthy limitations of this study. First, given the elevated risk of this cohort (high prevalence of poverty, predominantly ethnic minority sample, and prenatal drug exposure), generalizability of study findings is limited to other high-risk populations. Limitations related to substance exposure involved lack of adequate lead exposure history to be used as a covariate as well as limited power when assessing the 3-level PCE variable due to having nearly twice as many participants in the some PCE group versus high PCE. In addition, although ACASI technology helps reduce potential underreporting/response bias among adolescents reporting sensitive or illegal behaviors, more objective drug screening methods such as urine or hair analysis would have substantially strengthened the current study. Lack of biologic confirmation was also a limitation in assessing prenatal tobacco and marijuana exposure in our sample. Further, caregiver substance use was assessed via interviewing with a trained researcher rather than using the ACASI computer-based system; therefore, underreporting of substance use was likely among caregivers despite assurance of confidentiality. Underreporting is an ongoing issue in research given the potential social/legal implications of drug use (47); therefore, future studies should utilize measurement approaches that facilitate increased accuracy in assessing substance use (e.g., drug screening). A similar issue with underreporting was also likely for adolescent and caregiver arrest history. Arrest history relied on self-report and arrest records were not obtained. Lastly, the measure of adolescent inhibitory control was limited to parent-report data on the CBCL rather than use of a more objective measure.

Public Health Implications

Several risk factors were identified as predictors of adolescent arrests, substance use, and early onset of sexual behavior. The potential implications of adolescents' involvement in risk-taking behavior can be serious at multiple levels. For instance, adolescents who engage in criminal/delinquent behavior may experience consequences such as adverse health

consequences (increased injuries), less academic success, increased occupational difficulties (unemployment, fewer employment options), increased economic difficulties (lower earnings), and increased interpersonal difficulties (37, 48–49). In addition, these outcomes influence families in terms of quality of relationships, increased family stress/conflict, and financial burden due to court-costs and living expenses of the offender. Lastly, burdens exist at a societal level with youth delinquency increasing crime rates, medical and social service costs, and costs of maintaining correctional facilities.

With regard to substance use, familial drug abuse, affiliation with drug-using peers, as well as other environmental factors clearly increase the opportunity for adolescents to experiment with drugs. Early experimentation with substances is associated with a substantially increased risk for the adolescent user to develop a substance abuse problem. Thus, serious concerns exist regarding the need for more community social and mental health services to help prevent and intervene with substance abuse issues in youth and families. The earlier families receive such services the more likely efforts can be made to effectively prevent later or persistent substance abuse difficulties.

Lastly, sexual risk behaviors among adolescents are a significant public health concern placing sexually active adolescents at increased risk for STDs, HIV infection, and unintended pregnancy. Knowledge of risk factors associated with increased sexual risk behaviors among adolescents should be used to design and implement youth prevention programs that promote sexual education, healthy attitudes about sex, as well as increasing knowledge of behaviors that will reduce the risks for HIV, STDs, and unintended pregnancy (41). Such programs could help reduce costs to society in terms of medical care and the increased need for social services among high-risk populations.

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REFERENCES

1. Igra, V.; Irwin, CE. Theories of adolescent risk-taking behavior. In: DiClemente, RJ.; Hansen, WB.; Ponton, LE., editors. *Handbook of Adolescent Health Risk Behavior*. New York: Plenum; 1997. p. 35-52.
2. Barnard M, McKeganey N. The impact of parental problem drug use on children: what is the problem and what can be done to help? *Addiction*. 2004; 99:552–559. [PubMed: 15078229]
3. Gibbons FX, Gerrard M, Lune LSV, et al. Context and cognitions: Environmental risk, social influence, and adolescent substance use. *Pers Soc Psychol Bull*. 2004; 30:1048–1061. [PubMed: 15257788]
4. Delaney-Black V, Chiodo LM, Hannigan JH, et al. Prenatal and postnatal cocaine exposure predict teen cocaine use. *Neurotoxicol Teratol*. 2011; 33:110–119. [PubMed: 20609384]
5. DiClemente RJ, Wingood GM, Crosby R, et al. Parental monitoring: Association with adolescents' risk behaviors. *Pediatrics*. 2001; 107:1363–1368. [PubMed: 11389258]
6. Ensminger ME. Sexual activity and problem behaviors among black, urban adolescents. *Child Dev*. 1990; 61:2032–2046. [PubMed: 2083510]
7. Fisher PA, Lester BM, DeGarmo DS, et al. The combined effects of prenatal drug exposure and early adversity on neurobehavioral disinhibition in childhood and adolescence. *Dev Psychopathol*. 2011; 23:777–788. [PubMed: 21756431]
8. Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychol Bull*. 1992; 12:64–105.
9. LaGasse LL, Hammond J, Liu J, et al. Violence and delinquency, early onset drug use, and psychopathology in drug-exposed youth at 11 years. *Ann NY Acad Sci*. 2006; 1094:313–318.
10. Bennett D, Bendersky M, Lewis M. Preadolescent health risk behavior as a function of prenatal cocaine exposure and gender. *J Dev Behav Pediatr*. 2007; 28:467–472. [PubMed: 18091092]
11. Bridgett DJ, Mayes LC. Development of inhibitory control among prenatally cocaine exposed and non-cocaine exposed youths from late childhood to early adolescence: The effects of gender and risk and subsequent aggressive behavior. *Neurotoxicol Teratol*. 2011; 33:47–60. [PubMed: 21256424]
12. Frank DA, Rose-Jacobs R, Crooks D, et al. Adolescent initiation of licit and illicit substance use: Impact of intrauterine exposures and post-natal exposure to violence. *Neurotoxicol Teratol*. 2010; 33:100–109. [PubMed: 20600847]
13. Gerteis J, Chartrand M, Martin B, et al. Are there effects of intrauterine cocaine exposure on delinquency during early adolescence? A Preliminary Report. *J Dev Behav Pediatr*. 2011; 32:393–401. [PubMed: 21558951]
14. Lester BM, Lin H, DeGarmo DS, et al. Neurobehavioral disinhibition predicts initiation of substance use in children with prenatal cocaine exposure. *Drug Alcohol Depend*. 2012; 126:80–86. [PubMed: 22608010]
15. Whitaker TM, Bada HS, Bann CM, et al. Serial pediatric symptom checklist screening in children with prenatal drug exposure. *J Dev Behav Pediatr*. 2011; 32:206–215. [PubMed: 21200328]

16. Eyler FD, Warner TD, Behnke M, et al. Executive functioning at ages 5 and 7 years in children with prenatal cocaine exposure. *Dev Neurosci*. 2009; 31:121–136. [PubMed: 19372693]
17. Rose-Jacobs R, Waber D, Beeghly M, et al. Intrauterine cocaine exposure and executive functioning in middle childhood. *Neurotoxicol Teratol*. 2009; 31:159–168. [PubMed: 19146950]
18. Savage J, Brodsky NL, Malmud E, et al. Attentional functioning and impulse control in cocaine-exposed and control children at age ten years. *J Dev Behav Pediatr*. 2005; 26:42–47. [PubMed: 15718883]
19. Biederman J, Faraone SV, Monuteaux MC, et al. Patterns of alcohol and drug use in adolescents can be predicted by parental substance use disorders. *Pediatrics*. 2000; 106:792–797. [PubMed: 11015524]
20. Kaplow JB, Curran PJ, Dodge KA. Child, parent, and peer predictors of early-onset substance use: A multisite longitudinal study. *J Abnorm Child Psych*. 2002; 30:199–216.
21. Warner TD, Behnke M, Eyler FD, et al. Early adolescent cocaine use as determined by hair analysis in a prenatal cocaine exposure cohort. *Neurotoxicol Teratol*. 2011; 33:88–99. [PubMed: 20647046]
22. Busen NH, Marcus MT, von Sternberg KL. What African-American middle school youth report about risk-taking behaviors. *J Pediatr Health Care*. 2006; 20:393–400. [PubMed: 17071370]
23. Resnick MD, Blum R. The association of consensual sexual intercourse during childhood with adolescent health risk and behaviors. *Pediatrics*. 1994; 94:907–913. [PubMed: 7971010]
24. Bauer CR, Langer JC, Shankaran S, et al. Acute neonatal effects of cocaine exposure during pregnancy. *Arch Pediatr Adolesc Med*. 2005; 159:824–834. [PubMed: 16143741]
25. Lester BM, ElSohly M, Wright LL, et al. The Maternal Lifestyle Study: Drug use by meconium toxicology and maternal self-report. *Pediatric*. 2001; 107:309–317.
26. Hollingshead, AB. Four factor index of social status. Yale University, New Haven, CT: Yale University. Unpublished manuscript; 1975.
27. Starfield, B.; Ensminger, M.; Riley, AW., et al. Child Health and Illness Profile-Adolescent Edition (CHIP-AE). Baltimore, MD: The Johns Hopkins University; 1994.
28. Huizinga, D. Denver Youth Survey: Youth Interview Schedule. Boulder, Colorado: The University of Colorado, Institute of Behavior Sciences; 1991.
29. Elliott, DS.; Huizinga, D.; Ageton, SS. Explaining delinquency and drug use. Beverly Hills: Sage Publications; 1985.
30. Elliott, DS.; Huizinga, D.; Menard, S. Multiple problem youth: Delinquency, substance abuse, and mental health problems. New York: Springer-Verlag; 1989.
31. Turner CF, Ku L, Rogers SM, et al. Adolescent sexual behavior, drug use, and violence: Increased reporting with computer survey technology. *Science*. 1998; 280:867–873. [PubMed: 9572724]
32. Dolezal, CMC.; Brackis-Cott, E.; Meyer-Bahlburg, H. Adolescent sexual behavior assessment. Unpublished manuscript; 2006.
33. Furman, W.; Wehner, EA. Dating History Questionnaire. University of Denver: Unpublished measure; 1992.
34. Achenbach, TM. Manual for Child Behavior Checklist/ 4–18 and 1991 Profile. Burlington, VT: University of Vermont, Dept. of Psychiatry; 1991.
35. Achenbach, TM. Manual for Child Behavior Checklist/ 2–3 and 1992 Profile. Burlington, VT: University of Vermont, Dept. of Psychiatry; 1992.
36. Kenny, DA. Mediation with Dichotomous Outcomes. 2008. Accessed April 4, 2012 from <http://davidakenny.net/doc/dichmed.doc>.
37. Brame R, Turner MG, Paternoster R, et al. Cumulative prevalence of arrest from ages 8 to 23 in a national sample. *Pediatrics*. 2012; 129:21–27. [PubMed: 22184650]
38. Bendersky M, Lewis M. Arousal modulation in cocaine-exposed infants. *Dev Psychol*. 1998a; 34:555–564. [PubMed: 9597364]
39. Bendersky M, Lewis M. Prenatal cocaine exposure and impulse control at two years. *Ann NY Acad Sci*. 1998b; 846:365–367. [PubMed: 9668426]
40. The National Center on Addiction and Substance Abuse. Adolescent substance use: America's #1 Public Health Problem. Columbia University; 2011.

41. Holt S, Buckley H, Whelan S. The impact of exposure to domestic violence on children and young people: A review of the literature. *Child Abuse Neglect*. 2008; 32:797–810. [PubMed: 18752848]
42. Center for Disease Control and Prevention. Youth Risk Behavior Surveillance-United States, 2009. US Department of Health and Human Services; 2010.
43. Upchurch DM, Levy-Storms L, Sucoff CA, Aneshensel CS. Gender and ethnic differences in the timing of first sexual intercourse. *Family Planning Perspectives*. 1998; 30:121–127. [PubMed: 9635260]
44. Harper GW, Gannon C, Watson SG, Catania JA, Dolcini MM. The role of close friends in African American adolescents' dating and sexual behavior. *J Sex Res*. 2004; 41:351–362. [PubMed: 15765275]
45. Lester BM, Bagner DM, Liu J, et al. Infant neurobehavioral dysregulation: Behavior problems in children with prenatal substance exposure. *Pediatrics*. 2009; 124:1355–1362. [PubMed: 19822596]
46. Lester BM, Padbury JF. Third pathophysiology of prenatal cocaine exposure. *Dev Neurosci*. 2009; 31:23–35. [PubMed: 19372684]
47. Ostrea EM Jr, Brady M, Gause S, et al. Drug screening of newborns by meconium analysis: A large-scale, prospective, epidemiologic study. *Pediatrics*. 1992; 89:107–113. [PubMed: 1727992]
48. Nagin DS, Farrington DP, Moffitt TE. Life-course trajectories of different types of offenders. *Criminology*. 1995; 33:111–139.
49. Wiesner M, Kim HK, Capaldi DM. History of juvenile arrests and vocational career outcomes for at-risk young men. *J Res Crime Delinq*. 2010; 47:91–117. [PubMed: 20448840]

Figure 1. Tests of Inhibitory Control Problems as a Mediator of the Effects of Prenatal Cocaine Exposure on Arrests and Early Onset of Oral Sex among Adolescents

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Values are regression coefficients (standard errors).

Sobel tests for mediation: Arrested ($Z(SE) = 2.04 (0.28)$, $p = 0.04$), and oral sex ($Z(SE) = 1.97 (0.02)$, $p = 0.05$).

Table 1

Demographics, Risk Factors, and Risk Taking Behaviors by Prenatal Cocaine Exposure

Characteristic	All (N = 963) N (%)	Prenatal Cocaine Exposure		
		Exposed (N = 433) N (%)	Not Exposed (N = 530) N (%)	<i>p</i>
Demographics				
Male	479 (50)	213 (49)	266 (50)	0.758
Race				
Black	772 (80)	346 (80)	426 (80)	0.856
White	119 (12)	54 (12)	65 (12)	0.923
Other	72 (7)	33 (8)	39 (7)	0.877
Maternal education at birth				
Less than high school	369 (38)	208 (48)	161 (30)	< 0.001
High school	393 (41)	153 (35)	240 (45)	0.002
More than high school	200 (21)	71 (16)	129 (24)	0.003
Age at 15-year visit, Mean (SD)	15 (0)	15 (0)	15 (0)	0.088
Caregiver SES (Hollingshead), <i>Mean (SD)</i>	4 (1)	4 (1)	3 (1)	< 0.001
Prenatal tobacco exposure	513 (53)	361 (83)	152 (29)	< 0.001
Prenatal alcohol exposure	593 (62)	330 (76)	263 (50)	< 0.001
Prenatal marijuana exposure	224 (23)	171 (39)	53 (10)	< 0.001
Inhibitory control problems at 13 years, Mean (SD)	57 (7)	57 (8)	56 (7)	0.004
Risk Taking Behaviors at 15 Years				
Arrested	81 (8)	50 (12)	31 (6)	0.002
Alcohol use	189 (20)	84 (20)	105 (20)	0.801
Tobacco use	52 (5)	28 (7)	24 (5)	0.198
Marijuana use	106 (11)	58 (13)	48 (9)	0.040
Touching (13 years)	253 (31)	132 (36)	121 (27)	0.008
Oral sex (14 years)	231 (26)	126 (31)	105 (22)	0.001
Penetrative sex (14 years)	291 (33)	147 (37)	144 (30)	0.022
Sex without condom (most recent encounter)	74 (9)	37 (9)	37 (8)	0.417

Note: The age range for adolescents attending the 15-year outcome visit was approximately 14 to 16 years.

Table 2

Logistic Regression Models of Arrests and Alcohol, Tobacco, and Marijuana Use at 15 Years

Variable	Arrested (N = 948)	Alcohol Use (N = 912)	Tobacco Use (N = 917)	Marijuana Use (N = 915)
	Adjusted OR (95% CI)	Adjusted OR (95% CI)	Adjusted OR (95% CI)	Adjusted OR (95% CI)
Clinic A	1.13 (0.45, 2.81)	0.80 (0.40, 1.61)	0.64 (0.23, 1.75)	0.76 (0.33, 1.77)
B	0.92 (0.34, 2.45)	1.06 (0.48, 2.31)	0.91 (0.29, 2.81)	1.77 (0.71, 4.45)
C	0.78 (0.25, 2.42)	0.82 (0.38, 1.76)	0.84 (0.24, 2.97)	0.30 (0.10, 0.87)*
Prenatal cocaine exposure	2.21 (1.15, 4.23)*	0.78 (0.44, 1.39)	1.37 (0.55, 3.42)	1.69 (0.84, 3.39)
Prenatal tobacco exposure	1.04 (0.53, 2.06)	1.38 (0.76, 2.50)	0.80 (0.30, 2.12)	0.76 (0.36, 1.59)
Prenatal alcohol exposure	1.00 (0.56, 1.78)	1.07 (0.67, 1.72)	0.99 (0.46, 2.13)	1.08 (0.61, 1.92)
Prenatal marijuana exposure	1.02 (0.56, 1.85)	1.47 (0.85, 2.52)	0.82 (0.33, 2.05)	0.94 (0.50, 1.78)
Maternal education at birth				
Less than high school	2.46 (0.93, 6.47)	1.28 (0.63, 2.57)	1.41 (0.46, 4.33)	2.50 (1.03, 6.03)*
High school	2.10 (0.82, 5.38)	1.48 (0.80, 2.73)	1.33 (0.48, 3.68)	2.39 (1.09, 5.23)*
Caregiver SES (Hollingshead)	1.76 (1.15, 2.68)**	0.85 (0.62, 1.16)	0.83 (0.50, 1.36)	1.11 (0.76, 1.63)
Male	3.00 (1.77, 5.09)***	0.82 (0.54, 1.25)	0.73 (0.37, 1.47)	1.59 (0.95, 2.69)
Black	0.78 (0.33, 1.83)	0.60 (0.30, 1.19)	0.19 (0.07, 0.52)**	0.57 (0.26, 1.28)
Other race	0.14 (0.02, 1.14)	1.11 (0.47, 2.64)	1.29 (0.37, 4.48)	1.05 (0.36, 3.02)
Age at 15 year visit	1.15 (0.63, 2.09)	1.99 (1.21, 3.29)**	0.91 (0.40, 2.07)	1.37 (0.76, 2.47)
Parental involvement	0.68 (0.41, 1.10)	0.70 (0.46, 1.06)	0.68 (0.32, 1.41)	0.73 (0.42, 1.26)
Caretaker abuse	0.74 (0.40, 1.34)	0.55 (0.32, 0.94)*	0.31 (0.11, 0.86)*	0.58 (0.31, 1.11)
Child abuse	1.55 (0.68, 3.50)	1.36 (0.65, 2.81)	1.86 (0.66, 5.26)	1.22 (0.53, 2.85)
Community violence	1.07 (1.00, 1.14)	1.01 (0.95, 1.08)	1.04 (0.93, 1.15)	1.03 (0.95, 1.11)
Caretaker changes	0.96 (0.83, 1.12)	0.85 (0.73, 0.99)*	1.21 (0.97, 1.52)	0.96 (0.81, 1.14)
Postnatal alcohol use	1.08 (0.66, 1.77)	0.99 (0.63, 1.53)	1.55 (0.81, 2.97)	0.67 (0.37, 1.21)
Postnatal marijuana use	0.43 (0.07, 2.59)	2.68 (0.82, 8.68)	2.77 (0.63, 12.14)	2.31 (0.80, 6.70)
Postnatal cocaine use	0.51 (0.24, 1.10)	1.84 (0.92, 3.72)	0.51 (0.15, 1.71)	0.79 (0.36, 1.73)
Caregiver history of arrest	1.22 (0.70, 2.12)	--	--	--
Reported friends did the behavior	--	38.56 (20.86, 71.28)***	69.82 (16.17, 301.47)***	64.16 (24.82, 165.88)***

Note:* $p < .05$;** $p < .001$; Odds ratios are adjusted for all predictor variables included in the model.

Table 3

Logistic Regression Models of Sexual Behaviors Outcomes

Variable	Touching (N = 758)	Oral Sex (N = 828)	Penetrative Sex (N = 821)	Sex without Condom (N = 799)
	Adjusted OR (95% CI)	Adjusted OR (95% CI)	Adjusted OR (95% CI)	Adjusted OR (95% CI)
Clinic A	0.59 (0.29, 1.23)	0.85 (0.44, 1.66)	1.11 (0.57, 2.15)	0.89 (0.32, 2.47)
B	0.79 (0.38, 1.64)	0.79 (0.39, 1.57)	1.27 (0.64, 2.54)	0.96 (0.33, 2.81)
C	0.55 (0.25, 1.18)	0.98 (0.48, 1.99)	1.23 (0.60, 2.49)	1.40 (0.48, 4.10)
Prenatal cocaine exposure	1.62 (0.96, 2.74)	1.90 (1.17, 3.06)**	1.50 (0.94, 2.39)	0.62 (0.24, 1.64)
Prenatal tobacco exposure	0.76 (0.44, 1.32)	0.83 (0.50, 1.37)	0.75 (0.46, 1.23)	0.54 (0.26, 1.14)
Prenatal alcohol exposure	0.95 (0.62, 1.47)	1.05 (0.70, 1.57)	0.98 (0.66, 1.46)	1.22 (0.65, 2.27)
Prenatal marijuana exposure	1.16 (0.70, 1.92)	0.88 (0.56, 1.40)	1.70 (1.08, 2.68)*	1.10 (0.55, 2.20)
Maternal education at birth				
Less than high school	1.44 (0.78, 2.66)	0.84 (0.47, 1.48)	1.46 (0.83, 2.54)	3.15 (1.04, 9.56)*
High school	1.01 (0.57, 1.79)	0.96 (0.57, 1.63)	0.93 (0.56, 1.57)	2.78 (0.97, 7.99)
Caregiver SES (Hollingshead)	0.99 (0.74, 1.34)	1.10 (0.83, 1.46)	1.12 (0.85, 1.48)	1.37 (0.88, 2.14)
Male	7.56 (4.91, 11.65)***	4.35 (2.95, 6.41)***	4.25 (2.93, 6.14)***	0.67 (0.31, 1.46)
Black	2.10 (0.97, 4.57)	0.88 (0.45, 1.70)	0.96 (0.49, 1.89)	1.11 (0.36, 3.41)
Other race	1.16 (0.44, 3.05)	0.40 (0.16, 1.01)	0.50 (0.20, 1.24)	1.63 (0.43, 6.10)
Age at 15-year visit	1.34 (0.84, 2.15)	1.10 (0.71, 1.71)	1.50 (0.98, 2.31)	1.17 (0.61, 2.27)
Parental involvement	0.61 (0.41, 0.90)*	0.62 (0.43, 0.89)**	0.68 (0.48, 0.96)*	0.86 (0.51, 1.46)
Caretaker abuse	1.02 (0.64, 1.63)	0.99 (0.64, 1.54)	1.06 (0.69, 1.63)	1.55 (0.77, 3.10)
Child abuse	2.03 (0.97, 4.25)	2.26 (1.17, 4.36)*	1.62 (0.82, 3.22)	4.30 (1.89, 9.81)***
Community violence	1.12 (1.05, 1.18)***	1.06 (1.01, 1.12)*	1.10 (1.04, 1.16)***	1.02 (0.94, 1.10)
Caretaker changes	1.10 (0.98, 1.24)	1.01 (0.90, 1.13)	0.98 (0.88, 1.10)	0.88 (0.73, 1.05)
Postnatal tobacco use	1.01 (0.97, 1.04)	1.03 (1.00, 1.06)	1.00 (0.97, 1.04)	1.01 (0.96, 1.06)
Postnatal alcohol use	1.50 (0.96, 2.34)	1.25 (0.81, 1.92)	1.22 (0.80, 1.85)	1.17 (0.65, 2.12)
Postnatal marijuana use	1.05 (0.40, 2.78)	1.26 (0.49, 3.22)	1.41 (0.50, 3.95)	1.40 (0.25, 7.84)
Postnatal cocaine use	0.65 (0.35, 1.22)	0.65 (0.37, 1.14)	0.69 (0.39, 1.23)	1.02 (0.44, 2.39)
Ever been in love	2.98 (2.00, 4.43)***	2.51 (1.73, 3.65)***	2.39 (1.67, 3.42)***	1.63 (0.93, 2.86)
Have had boyfriend/girlfriend	7.87 (2.66, 23.32)***	3.50 (1.51, 8.14)**	12.50 (3.78, 41.29)***	2.17 (0.71, 6.65)
"A player"	2.24 (1.10, 4.54)*	2.22 (1.15, 4.27)*	2.85 (1.50, 5.42)**	2.82 (1.30, 6.12)**
Gender × PCE				
Male: Exposed vs. not	--	--	--	3.31 (1.38, 7.91)**
Female: Exposed vs. not	--	--	--	0.63 (0.24, 1.64)

Note:* $p < .05$;** $p < .001$.

Overall test for gender by prenatal cocaine interaction for sex without condom: Wald $\chi^2(1)=8.43$, $p = 0.004$. Odds ratios are adjusted for all predictor variables included in the model.