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Pathways to adolescent sexual risk behaviors: Effects of prenatal cocaine exposure

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ABSTRACT

Background: To assess the impact of prenatal cocaine exposure (PCE) on adolescent sexual risk behaviors. Externalizing behavior, teen substance use, and early sexual intercourse were examined as pathways mediating the effects of PCE on sexual risk behaviors.
Methods: Adolescents ($N = 364$; 185 PCE, 179 non-cocaine exposure (NCE); 205 girls, 159 boys), primarily African–American and of low socioeconomic status, were prospectively enrolled in a longitudinal study at birth. Risky sexual behaviors were assessed at ages 15 and 17. Externalizing behavior at 12 years was assessed with the Youth Self-Report. Substance use, via self-report and biologic assays, and early (before age 15) sexual intercourse were assessed at age 15. Path analyses with the weighted least squares estimator with mean and variance adjustments were performed.
Results: The final structural equation model-based path model, $\chi^2 = 31.97$ (df = 27), $p = .23$, CFI = .99, TLI = .99, RMSEA = .021, WRMR = .695, indicated a direct effect of PCE on sexual risk behavior ($\beta = .16$, $p = .02$). Although PCE was related to greater externalizing behavior ($\beta = .14$, $p = .009$), which in turn, predicted early sexual intercourse ($\beta = .16$, $p = .03$), leading to sexual risk behavior ($\beta = .44$, $p < .001$), bootstrapping indicated a non-significant indirect effect ($\beta = .01$, $p > .10$). Substance use was correlated with early sexual intercourse ($r = .60$, $p < .001$) and predicted sexual risk behavior by age 17 ($\beta = .31$, $p = .01$).
Conclusions: Prenatal cocaine exposure was related to more engagement in sexual risk behaviors, suggesting the importance of reducing substance use among pregnant women as a means of prevention of offspring substance use and sexual risk behavior.

1. Introduction

Sexual risk behaviors, including multiple sexual partners, infrequent condom and contraceptive use, and having sex under the influence of alcohol or drugs, contribute to unintended pregnancy and sexually transmitted infections (STIs) including HIV. Data from the 2013 Youth Risk Behavior Survey indicate that only 59% of sexually active high school (grades 9–12) students reported condom use during the last time they had sexual intercourse (Kann et al., 2014). Although sexually active teens and young adults (ages 15–24) represent only 25% of the sexually active population, they account for nearly half of new STI cases (Centers for Disease Control and Prevention (CDC), 2014) and 26% of all new HIV cases in the US (CDC, 2012). The 2003–2004 National Health and Nutrition Examination Survey (NHANES) indicated 38% of sexually experienced female adolescents aged 14–19 had laboratory evidence of STI (Forhan et al., 2009).

Prenatal cocaine exposure (PCE) may increase the vulnerability for adolescent sexual risk behavior (De Genna et al., 2014; Lambert et al., 2013; Min et al., 2015). Approximately 214,000 infants are exposed in utero to illicit drugs, including cocaine, each year in the United States (Substance Abuse and Mental Health Services Administration (SAMHSA), 2014); however, the effects of PCE on adolescent sexual behavior have not been well-established, with only three published studies from three prospective birth cohorts to date (De Genna et al., 2014; Lambert et al., 2013; Min et al., 2015). Although these studies indicated that, compared to non-cocaine exposed (NCE) adolescents, adolescents with PCE initiated sexual intercourse earlier (De Genna et al., 2014) and were more likely

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to have sexual intercourse (Min et al., 2015) or oral sex before age 15 (Lambert et al., 2013), they mainly focused on the age at first sexual intercourse. Only one study (Lambert et al., 2013) examined PCE effects on other sexual risk behaviors, reporting significant PCE effects on engaging in sex without a condom in boys only.

Multiple mechanisms may account for the link between PCE and sexual risk behavior. Maternal use of cocaine during pregnancy directly impacts the developing fetal brain by altering/disrupting the monoaminergic neurotransmitter system involving dopamine, serotonin, and norepinephrine in the prefrontal cortex (Kosofsky et al., 1994; McCarthy et al., 2014). Disruption in the prefrontal cortex has been implicated in problems of inhibitory control, attention, increased risk taking behaviors, and executive function (Thompson et al., 2009), all of which likely contribute to sexual risk behaviors (Goldenberg et al., 2013; Khurana et al., 2012). In addition, PCE may indirectly increase vulnerability to sexual risk behavior through biological and environmental confounders, including prenatal exposure to other substances such as alcohol (Larkby et al., 2011), tobacco (Maughan et al., 2004), and marijuana (Goldschmidt et al., 2000), ongoing parental substance abuse (Elkington et al., 2011) and psychological distress (Minnes et al., 2010), and elevated lead ($\geq$10 μg/dL) levels (Lane et al., 2008; Min et al., 2009; Singer et al., 2008). Further, poor quality of the home environment (Singer et al., 2008) including poor attachment to caregiver (Warner et al., 2011) and inadequate parental monitoring (Min et al., 2014a,b), sexual victimization (De Genna et al., 2014), violence exposure (Frank et al., 2011), and adoptive/foster care placement (Singer et al., 2004) may obscure the effects of PCE. Prospective, longitudinal birth cohort studies demonstrated effects of PCE on externalizing behavior (Ackerman et al., 2010; Buckingham-Howes et al., 2013; Min et al., 2014a,b; Minnes et al., 2010) and substance use (Frank et al., 2011; Minnes et al., 2014; Richardson et al., 2013), well-known precursors to sexual risk behaviors (Ramrakha et al., 2007; Wu et al., 2010).

The present study examined the impact of PCE on sexual risk behaviors assessed at ages 15 and 17 using path analyses. We previously reported more externalizing behavior problems at age 12 (Min et al., 2014b) and greater alcohol, tobacco and marijuana use (Minnes et al., 2014) and substance related problems (Min et al., 2014a) at age 15 in adolescents with PCE. We also found that adolescents with PCE were more likely to engage in early (<age 15) sexual intercourse (Min et al., 2015). Thus, in the present study, we examined externalizing behavior, substance use, and early sexual intercourse as pathways mediating the effects of PCE on sexual risk behaviors. We also tested whether PCE had unique effects on sexual risk behavior over and above its indirect influences. We hypothesized that PCE would be related to externalizing behavior and that externalizing behavior would predict substance use and early sexual intercourse, leading to sexual risk behaviors. Since both substance use and early sexual intercourse were assessed at age 15, correlation between these two variables was included in the path model. A direct path from PCE to sexual risk behavior was also specified in the model to indicate other unmeasured mediators of PCE.

2. Method

2.1. Sample

This study included 364 (185 PCE, 179 NCE) adolescents recruited at birth (September 1994–June 1996) from an urban county hospital and their birth mothers or caregivers for a longitudinal investigation of the effects of PCE. Pregnant women who had a urine toxicology screening at delivery due to a lack of prenatal care, behavior suggesting intoxication, self-admitted substance use, or a history of involvement with the Department of Human Services, were eligible for the study. Women with a psychiatric history, low intellectual functioning indicated in medical chart review, HIV-positive status, or chronic medical illness were excluded, as were infants with Down syndrome, fetal alcohol syndrome, or congenital heart defects. A nurse recruiter approached 647 screened women immediately before or after infant birth; of these 647 women, 54 were excluded, 155 refused to participate, and 23 did not come to the enrollment visit.

Maternal and infant urine samples and infant meconium were obtained shortly before or after infant birth and analyzed for cocaine and other drug metabolites, including benzoylecgonine, meta-hydroxybenzoylecgonine, cocaethylene, cannabinoids, opiates, phencyclidine, amphetamines, and benzodiazepines. A total of 415 newborns and their birth mothers were enrolled at birth, of which 218 infants were identified as cocaine-exposed based on positive screens of maternal and infant urine, infant meconium, or maternal self-report to hospital or research staff. Infants who were negative on all indicators of prenatal cocaine exposure were identified as NCE. Subjects and their caregivers were assessed by separate examiners who were blinded to exposure status at follow-up assessments at 6, 12, and 18 months and 2, 4, 6, 9–12, 15, and 17 years postpartum.

Since birth, 12 (9 PCE, 3 NCE) enrolled children died from sudden infant death syndrome (4 PCE, 2 NCE), cardiopulmonary arrest (1 PCE), pneumonia (1 PCE), accidental asphyxia (1 PCE), respiratory distress syndrome (1 PCE, 1 NCE), and unknown illness (1 PCE). The present study utilizes data from 364 adolescents who completed sexual behavior assessment at age 15 and/or 17 years, representing 90% retention of the 403 living participants in the original study. Among the 364 participating adolescents, 92.6% (n = 337) were assessed at both 15 and 17 years of age. Of the 39 adolescents not included in these analyses (19 drop-out, 18 lost contact, 2 low intellectual functioning (IQ < 50)), the 24 PCE adolescents did not differ from the 185 participating PCE adolescents. The 15 NCE adolescents not included in the study were more likely to be white, have birth mothers who were older, more likely to be married, and had more years of education compared to the 179 participating NCE adolescents. The Institutional Review Board of the participating hospital approved this study. All participants were given a monetary stipend, lunch, and transportation costs if needed. Parental written informed consent was obtained, with child assent beginning at age 9. A Certificate of Confidentiality (DA-09-146) was obtained from the U.S. Department of Health and Human Services to protect identifiable research information from forced disclosure.

2.2. Measures

2.2.1. Prenatal cocaine and other substance exposure. At the newborn visit, birth mothers were asked to recall frequency and amount of drug use for the month prior to and for each trimester of pregnancy. The number of tobacco cigarettes and marijuana joints smoked, and the number of drinks of beer, wine, or hard liquor per week was computed, with each drink equivalent to 0.5 oz. of absolute alcohol. For cocaine, as the majority of women (97%) in our study used the crack cocaine form, the number of "rocks" consumed and the amount of money spent per day were noted, which was converted to a standard "unit" of cocaine, referring to $20 worth of cocaine. No sociodemographic difference was found between women (n = 11) who used a non-crack cocaine form and 353 women who used crack cocaine. However, the 11 women used more marijuana, and less alcohol and cocaine compared to the 353 women who reported using crack cocaine. Frequency of use was recorded for each drug on a Likert-type scale ranging from 0 (not at all) to 7 (daily use) and converted to reflect the average number of days per week a drug was used, except for cigarettes, which was

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