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Association of prenatal cocaine exposure, childhood maltreatment, and responses to stress in adolescence

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ABSTRACT

Background: Prenatal cocaine exposure (PCE) may alter responses to stress. Children with PCE tend to grow up in suboptimal caregiving environments, conducive to child maltreatment (CM). Guided by the diathesis-stress model, the present study examined differences in self-reported responses to stress and coping in adolescents with and without PCE and explored whether childhood maltreatment (CM) moderated the effects of PCE.

Methods: Adolescents (N = 363; 184 PCE, 179 non-cocaine exposed (NCE)), primarily African-American and of low socioeconomic status, were prospectively enrolled in a longitudinal study at birth. The Responses to Stress Questionnaire was used to assess volitional coping (primary control, secondary control, disengagement) and involuntary responses (involuntary engagement, involuntary disengagement) to stress at the 15- and 17-year follow-up visits. CM was assessed retrospectively at age 17 using the Juvenile Victimization Questionnaire.

Results: Findings from longitudinal mixed model analyses indicated that PCE was associated with poorer coping strategies only among adolescents with a history of CM. Adolescents with PCE who experienced CM reported less dominant use of primary (e.g., problem solving, emotional regulation) and secondary control (e.g., cognitive restructuring) and more dominant use of disengagement (e.g., denial, avoidance) and involuntary disengagement (e.g., inaction) than adolescents with PCE who did not experience CM or NCE adolescents regardless of CM. CM was associated with more dominant use of involuntary engagement (e.g., intrusive thoughts).

Conclusions: PCE may increase sensitivity to CM, predisposing increased vulnerability to environmental risk. Continued studies into adulthood will elucidate how coping and involuntary stress responses affect social, vocational, and behavioral adjustment.

1. Introduction

Adolescence is a period of heightened stress (Spear, 2000) due to a confluence of social, academic, cognitive, physiological, and physical changes and demands, marked by physical maturation, drive for independence, increased importance of social and peer relationships, and academic challenges. Given the well-known myriad effects of stress on health (Schneiderman et al., 2005; Ystgaard et al., 1999), understanding how adolescents respond to stress and adversity may provide critical knowledge about the linkage between stress and health, with implications for preventive interventions. Responses to stress are considered a self-regulatory process (Compas et al., 2001; Eisenberg et al., 1997), comprising effortful, volitional coping strategies as well as involuntary, automatic physiological, cognitive, behavioral and affective reactions to stress (Connor-Smith et al., 2000). The degree to which adolescents are able to regulate their emotions, behaviors, thoughts, and physiological responses to stress may function as a mediator and/or moderator of the impact of stress on current and future adjustment and psychopathology, explaining individual differences in the effects of stress (Compas et al., 2001; McLaughlin and Hatzenbuehler, 2009; Sontag et al., 2008).

Although responses to stress and coping could be classified in various ways, a primary dimension is between engagement with vs. disengagement from the stressor, reflecting the "fight or flight" response to threat (Compas et al., 2001). This classification yields engagement coping, disengagement coping (e.g., denial, avoidance), involuntary engagement (e.g., intrusive thoughts, physiological arousal), and involuntary disengagement (e.g., emotional numbing, inaction), although multiple stress responses could be utilized simultaneously and/or successively. Engagement coping can be further divided into primary control strategies involving attempts to control the stressor or emotions related to the stressor (e.g., problem solving, seeking social support) and secondary control strategies involving efforts to adapt to the stressor (e.g., cognitive reconstruction, acceptance) (Compas et al.,

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2001; Connor-Smith et al., 2000). Studies indicate that both primary and secondary engagement coping tend to be associated with better psychological adjustment (Compas et al., 2001; Sontag et al., 2008), while disengagement (Compas et al., 2001; Min et al., 2007) and involuntary engagement and disengagement (Troop-Gordon et al., 2015; Sontag et al., 2008) are related to poorer adjustment (e.g., more depressive symptoms, aggression, substance use).

Accumulating evidence suggests that prenatal cocaine exposure (PCE) alters responses to stress in reaction to environmental insults (Lester and Padbury, 2009) by disrupting the monoamine neurotransmitter systems important for directing fetal brain development (Kosofsky et al., 1994; McCarthy et al., 2014), particularly in brain areas known to impact emotional and behavioral arousal and regulation and stress response (McCarthy et al., 2014). Studies from multiple longitudinal prospective birth cohorts collectively indicate that PCE is related to greater externalizing behavior problems. PCE effects have been found on teacher- and caregiver-rated externalizing behavior problems at age 7, 9, 11, and 13 years (Bada et al., 2011) and on adolescent-reported externalizing behavior at 12 (Min et al., 2014a) and 15 years of age (Min et al., 2014a,b), with greater effects related to heavier (> 70th percentile) exposure (Min et al., 2014a). Further, PCE is also related to early onset of substance use (Delaney-Black et al., 2011; Frank et al., 2011; Minnes et al., 2014; Richardson et al., 2013) and risky sexual behavior (De Genna et al., 2014; Lambert et al., 2013; Min et al., 2015, 2016), all of which collectively suggest PCE-related altered responses to stress. However, no study has specifically examined response to stress and coping abilities in PCE adolescents to date.

Children with PCE tend to grow up in suboptimal parenting and caregiving environments, characterized by lower levels of maternal education (Singer et al., 2002, 2004), ongoing caregiver substance use and psychological distress (Singer et al., 1997; Minnes et al., 2008; Molnar et al., 2014), and lack of social support (Min et al., 2013a; Nordstrom et al., 2005), conducive to potential child maltreatment. The deleterious effects of childhood maltreatment (CM) have been widely recognized (Teicher and Samson, 2016; De Bellis, 2001), with both retrospective and prospective studies reporting associations between CM and poorer psychological (Buckingham and Daniolos, 2013; Keyes et al., 2011; Min et al., 2007) and physical functioning in adulthood (Felitti et al., 1998; Min et al., 2013b). CM may adversely affect the volume and functionality of brain structures, including the reduction of the hippocampus and corpus callosum, and alter neuroendocrinological mechanisms involved in mediating the stress response such as the hypothalamic-pituitary-adrenal axis (Nemeroff, 2004). However, few studies have examined how CM may interact with PCE to affect stress response and coping abilities in adolescence.

According to the diathesis-stress model, those with a biological vulnerability are disproportionately likely to be affected adversely by an environmental stressor/adversity (Zahn-Waxler et al., 2008). The effects of environmental stressors such as CM are greater among those with a biological vulnerability, as stressors activate the vulnerability. Thus, adolescents with PCE may experience worse outcomes than non-cocaine exposed (NCE) adolescents when exposed to environmental stress such as CM. The current study assessed differences in self-reported involuntary responses to stress and effortful coping in adolescents with and without PCE and explored whether CM moderates the effects of PCE on stress response. Multiple biological and environmental confounders of PCE were assessed and controlled to isolate the effects of PCE and CM, including prenatal exposure to other substances such as alcohol (Larkby et al., 2011), tobacco (Maughan et al., 2004), and marijuana (Goldschmidt et al., 2000), elevated lead (≥10 μg/dL) levels (Lane et al., 2008; Min et al., 2009; Singer et al., 2008), ongoing caregiver postpartum substance abuse (Elkington et al., 2011) and psychological distress (Minnes et al., 2010), poor quality of the home environment (Singer et al., 2008; Min et al., 2014b), and violence exposure (Kobulsky et al., 2016; Frank et al., 2011). Further, lack of ecological resources and support from family, school, and the neighborhood/community which tend to confound with CM (Sippel et al., 2015), were also controlled. We hypothesized that adolescents with PCE would be more reactive to stress (more involuntary engagement and disengagement) and utilize coping strategies less effectively (less primary and secondary control and more disengagement) than adolescents without PCE. Adolescents with PCE who experienced CM were further hypothesized to have poorer stress responses than non-cocaine exposed (NCE) adolescents or maltreated NCE adolescents.

2. Methods

2.1. Sample and procedure

This study included 363 (184 PCE, 179 NCE) adolescents and their birth mothers or caregivers recruited at birth (September 1994–June 1996) from an urban county hospital for a longitudinal investigation of the effects of PCE. All recruited mothers were identified from a high-risk population screened for drug use. Urine drug toxicology screens were performed by the hospital on women who received no prenatal care, seemed to be intoxicated or taking drugs, had a history of involvement with the Department of Human Services in previous pregnancies due to drug use, self-admitted drug use, or appeared to be at high risk for drug use after an interview with hospital staff. 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 PCE based on positive screens of maternal and infant urine, infant meconium, or maternal self-report of cocaine use during pregnancy to hospital or research staff. Infants who were negative on all indicators of PCE were identified as NCE, but they may have been exposed to other substances (i.e., alcohol, tobacco, marijuana), forming a comparison group. 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 363 adolescents who completed stress response assessment at age 15 and/or 17 years, representing 90% retention of the 403 living participants in the original study. Among the 363 participating adolescents, 92% (n = 335) were assessed at both 15 and 17 years of age. Of the 40 adolescents not included in this analysis (19 drop-out, 18 lost contact, 2 low intellectual functioning (IQ < 50), 1 missing data), the 25 PCE adolescents did not differ from the 184 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. Fig. 1 charts the flow of participants through the study. 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 U.S. Department of Health and Human Services to protect

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