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Maternal cocaine use and mother-infant interactions: Direct and moderated associations

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Abstract

This study examined the associations between prenatal cocaine exposure and quality of mother-infant play interactions at 13 months of infant ages. We investigated whether maternal psychological distress and infant reactivity mediated or moderated this association. Participants consisted of 220 (119 cocaine exposed, 101 non-cocaine exposed) mother-infant dyads participating in an ongoing longitudinal study of prenatal cocaine exposure. Results indicated that mothers who used cocaine during pregnancy displayed higher negative affect and lower sensitivity toward their infant during play interactions at 13 months, and that their infants were less responsive toward them. Contrary to hypothesis, this association was not mediated by maternal psychological distress or by infant reactivity. However, results for both the cocaine and non-cocaine exposed infants were supportive of a transactional model where lower maternal sensitivity at 1 month was predictive of higher infant reactivity at 7 months, which in turn was predictive of lower maternal warmth/sensitivity at 13 months, controlling for potential stability in maternal behavior. Results also indicated that as hypothesized, infant reactivity moderated the association between maternal cocaine use during pregnancy and maternal warmth/sensitivity at 13 months of age. Cocaine using mothers who experienced their infants as being more reactive in early infancy were less warm/sensitive toward them in later infancy. Results have implications for parenting interventions that may be targeted toward improving maternal sensitivity among cocaine using mothers with more reactive infants.

Keywords

Infant Reactivity; Prenatal Substance exposure; Maternal Behavior

There is a growing consensus that the teratological effects of prenatal cocaine exposure are subtle, and may occur through several pathways [41]. One such pathway may be through problematic mother-infant interactions. Cocaine using mothers are more disengaged and passive during mother-infant interactions in the neonatal period [31], are less flexible and engaged during feeding interactions [40], have lower responsiveness and enthusiasm in later

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infancy [11,12], are less emotionally engaged in the toddler period [50], use fewer positive reinforcements and more threats of physical discipline in the toddler/preschool period [2], and are more hostile and intrusive in a structured teaching situation at 3 years of age [37]. The animal literature is supportive of these results indicating dose-dependent associations between cocaine treatment during gestation and disruptions in maternal caretaking behavior in the immediate post-partum period [52,61,66], perhaps due to decreased oxytocin levels [34,35,36,44,49].

However, the literature is by no means unequivocal. Several studies have noted no associations between maternal cocaine use and quality of mother-child interactions [7,38,53,63]. These mixed results may be a function of differences in timing of measurement, varying sample sizes, large within group heterogeneity on variables such as placement in foster care, and other maternal and infant risk characteristics. Among these risk characteristics, some of the most critical factors are maternal psychological distress, use of other substances in addition to cocaine, continued postnatal substance use, and individual differences in infant reactivity. Some of these variables such as other substance use and continued postnatal substance use may be better viewed as additional predictors or covariates, while others such as infant reactivity and maternal psychological distress may be better viewed as process variables and better conceptualized as potential mediators of this association.

Cocaine using mothers report higher postpartum general psychiatric symptoms [5,57,58,68], higher antisocial behavior [25], and more symptoms of post-traumatic stress disorder (PTSD) [25]. There is a large body of literature documenting the effects of maternal psychological distress on the quality of mother-infant interactions [22,23]. Thus, in addition to prenatal cocaine and other substance exposure, maternal psychological distress may be a significant predictor of the quality of mother-infant interactions.

Prenatal cocaine exposure is also consistently associated with greater infant negative affect and poorer regulation [41]. For instance, prenatal cocaine exposure is associated with higher infant state lability, orienting, and attention problems in the neonatal period [15,28,39,42]. Even beyond the neonatal period cocaine exposed infants exhibit higher negative affect in response to novel stimulation or stress [6,26,48]. Results at older ages indicate that cocaine exposed toddlers and preschoolers demonstrate higher behavioral reactivity during testing [32]; higher impulsivity at 2 years of age [6]; higher frustration and disruptive behavior at 4 years [20]; and in the school setting [18,19].

Transactional models of development have emphasized mothers' and infants' influence each other over time, and that the quality of mother-infant interactions are a product of both [4]. Thus, individual differences in infant reactivity are both a product of prior maternal behavior and a source of influence for subsequent maternal and infant behaviors [66]. Among maternal behaviors, maternal sensitivity or contingent responsiveness to infant cues has received considerable attention as a longitudinal predictor of infant reactivity [60,62,64]. Fish et al. [29] reported that lower maternal sensitivity was associated with an increase in negative reactivity from birth to 5 months of infant age. Similarly, infant reactivity may be longitudinally predictive of maternal behavior over time, although fewer studies have examined this direction of influence. For instance, Crockenberg and Smith [16,17] reported longitudinal, bidirectional associations between infant negative reactivity and maternal contingent responsiveness over the first 3 months of life, with infant reactivity and maternal responsiveness influencing each other over time. Spinrad and Stifter [60] reported that infants who were more emotionally reactive to frustration at 5 months had mothers who were more intrusive during free play at 10 months. These bidirectional associations may be particularly salient in high risk samples.

Maternal psychological distress and infant reactivity may also moderate the association between prenatal cocaine and other substance exposure and the quality of mother-infant interactions. For instance, cocaine exposed infants who are more reactive are more likely to elicit negative maternal behaviors and be less responsive during mother-infant interactions, compared with less reactive cocaine exposed infants. Similarly, cocaine using mothers with higher levels of psychological distress may be less emotionally available during mother-infant interactions than cocaine using mothers who are less distressed. As noted by previous research on cocaine using women and their infants, moderators of risk may be particularly useful to examine in order to better target prevention and intervention efforts on those at highest risk for disorder [56].

The purpose of this study was to examine the associations between prenatal cocaine exposure and quality of mother-infant play interactions at 13 months of infant ages. We hypothesized that cocaine using mothers would display higher negative affect, lower positive involvement, and sensitivity compared to mothers in the NE group. We also hypothesized that cocaine exposed infants would display lower responsiveness and higher negative affect during play interactions with their mothers. We hypothesized that two pathways to lower maternal sensitivity and infant responsiveness at 13 months would be via higher maternal psychological distress and higher infant reactivity among cocaine exposed infants. Thus, we tested a model that included both these variables, and included predictive paths from prior maternal behavior at 1 month to infant reactivity at 7 months, and a path from infant reactivity to maternal behavior at 13 months. Finally, we examined if maternal psychological distress and infant reactivity were moderators of the association between maternal cocaine and other substance use and mother-infant interactions. We hypothesized that cocaine using mothers who reported their infants to be more reactive or those who had higher psychological distress would exhibit less optimal parenting behavior and would have infants who were less responsive. Thus, the association between maternal cocaine use and quality of mother-infant interactions would be stronger under conditions of higher infant reactivity or higher maternal psychological distress.

Method

Participants

Participants consisted of mother-infant dyads recruited postpartum from two local area hospitals into a longitudinal study of maternal substance use and child development. The initial sample consisted of 220 mother-infant dyads (108 boys, 112 girls). Infants were classified as either cocaine-exposed (CE, $n = 119$) or non-cocaine exposed (NE, $n = 101$). Of these 220 infants, 18% were in foster care by 13 months of infant age, and with one exception, all of these families were in the cocaine group. Because of previously noted group differences between foster care mother-infant dyads and cocaine using mothers on a number of variables included in this study (e.g., maternal psychological distress) [25], foster care mother-infant dyads were excluded from data analyses. Thus, the final sample consisted of 180 mother-infant dyads (87 boys, 93 girls), with 81 in the CE group and 99 in the NE group.

The two groups were matched on maternal education, age, race/ethnicity and on infant gender. 11% of the CE infants (ranged from 33 to 41 weeks) and 3% of the NE infants (ranged from 36 to 42 weeks) were preterm (<37 weeks gestational age). CE infants were significantly more likely to have been preterm than NE infants, Pearson chi-square = 7.76, $p < .01$. All testing was conducted after age correction for prematurity. Infants ranged from 1531 to 5072 grams at birth ($M = 3142.01$, $SD = 567.33$). Additional exclusionary criteria consisted of maternal age less than 18 years, use of illicit substances other than cocaine or marijuana during pregnancy, and significant medical problems in the infant (e.g., congenital

anomalies, HIV+ status, prolonged respiratory distress). Mothers ranged in age from 18 to 42 ($M = 29.34$, $SD = 2.18$). 70% of mothers were African-American, 17% were Caucasian, 10% were Hispanic-American and the remaining were other. The two groups did not differ significantly with regard to ethnicity. 81% of mothers were receiving Temporary Assistance for Needy Families and 90.3% were single.

The study received approval from the institutional review boards of the hospitals as well as the primary institutions with which the authors are affiliated. Informed written consent was obtained from all recruited participants. Mothers were paid for participation in the form of toys, checks, and gift cards.

Procedure

All mothers were screened after delivery for initial eligibility and matching criteria. Interested and eligible mothers were given detailed information about the study and asked to sign consent forms. About 2 weeks after delivery, mothers were contacted and scheduled for their first laboratory visit, which took place at the time that their infant was approximately 4–8 weeks old. Subsequent visits were scheduled when the infant was 7 (range: 6–8) and 13 (range: 12–15) months old. All visits consisted of a combination of maternal interviews, observations of mother-infant interactions, assessments of infant arousal and arousal regulation. In the circumstance of a change in custody arrangements, the person who had legal guardianship of the child was contacted and asked to participate. Biological mothers were interviewed at the 4–8 week assessment in order to obtain accurate information about prenatal substance use.

Once a family was recruited into the cocaine group, the closest matching non-cocaine group family was recruited. However, a significantly higher proportion of mothers in the NE group declined participation or withdrew before formal enrollment, resulting in a smaller number of families in the control group. Of the 4,800 women screened at delivery, 340 were eligible for participation in either group. Of these 340 women, 35% either declined participation or were not enrolled in the study because they expressed initial interest but later withdrew. Of the 220 women who were formally enrolled in the study, 180 biological mother-infant dyads were used in data analyses as described above.

Assessment of growth and risk status

Three measures of growth were used in this study: birth weight (gm), birth length (cm), and head circumference (cm). All measurements were taken by obstetrical nurses in the delivery room and recorded in the infant's medical chart. Research staff recorded this information from the charts after recruiting the mother-infant dyad. There is some unreliability in this measurement as most obstetric nurses need to be trained to research criteria to ensure that length and head circumference are measured accurately and reliably. Medical chart review at the time of recruitment also was used to complete the Obstetrical Complications Scale (OCS) [46], a scale designed to assess the number of perinatal risk factors experienced by the infant. Higher numbers on this scale indicate a more optimal obstetric score. Gestational age was calculated by maternal report of last menstrual period and extracted from medical records.

Identification of Substance Use

Cocaine status was determined by a combination of maternal report, chart review, and maternal hair analysis. Urine toxicologies were routinely conducted at the first prenatal visit on maternal urine and/or at delivery (for those mothers who tested positive prenatally, obtained prenatal care elsewhere, or did not receive any prenatal care) on infant and maternal urine by participating hospitals. Mothers were included in the cocaine group if self-

reports were positive, regardless of urine toxicology or hair-sample results. Similarly, mothers who reported that they did not use cocaine but had positive urine toxicology or hair samples were included in the cocaine group.

Urine toxicologies consisted of standard urine screening for drug level or metabolites of cocaine, opiates, benzodiazepines, and tetrahydrocannabinol. Urine was rated positive if the quantity of drug or metabolite was >300 g/ml. Hair samples were collected from the mothers at the first laboratory visit and sent to the Psychomedics Corporation for radioimmunoanalyses (RIAH). Hair samples were screened for cocaine followed by a gas chromatography/mass spectrometry (GC/MS) confirmation for positive cocaine screens. Drugs and their metabolites are absorbed into the hair and can be extracted and measured. As hair grows at an average rate of 1/2 inch per month, it can record a pattern of drug consumption related to the amount and frequency of use [3]. Thus, a 2-inch length of hair could contain a record of approximately 4 months of use, and given adequate hair length (i.e., about 4–5 inches), use per trimester may be recorded. Drugs become detectable in hair about 3 to 4 days after use, a time when cocaine is rendered undetectable by urinalysis. RIAH is the most well-established hair-analysis technique and has been replicated by independent laboratories across the world [47]. GC/MS confirmations of RIAH have not revealed any false positives because of testing errors [47].

Approximately 32% of mothers in the study (55% of the mothers in the cocaine group) had positive urine toxicologies at delivery, and 25% of mothers (79% of the mothers in the cocaine group) had hair samples that tested positive for cocaine during pregnancy. The remainder of mothers in the cocaine group admitted having used cocaine in the brief self-report screening instrument administered after delivery. Mothers in the comparison group reported not having used any illicit substances other than marijuana. Additional exclusionary criteria for all mothers were (a) maternal age younger than 18 years, (b) use of illicit substances other than cocaine or marijuana, and (c) significant medical problems for the infant (e.g., genetic disorders, major perinatal complications, baby in critical care for over 48 hours).

The Timeline Follow-Back Interview (TLFB) [59] was used to assess maternal substance use before, during, and after pregnancy at recruitment, and in the postnatal period at 7 and 13 months of infant age. Participants were provided a calendar and asked to identify events of personal interest (i.e., holidays, birthdays, vacations, etc.) as anchor points to aid recall. This method has been established as a reliable and valid method of obtaining longitudinal data on substance-use patterns, has good test-retest reliability, and is highly correlated with other intensive self-report measures [10]. The TLFB yielded data about the average number of days of cocaine use per week, average number of joints smoked per week, average number of cigarettes smoked per week, and average number of standard drinks per week, during pregnancy and for the postnatal period.

Maternal Psychological Distress—The Brief Symptom Inventory (BSI) [21], a brief form of the Symptom Checklist 90-Revised was administered at the 7 month visit. The BSI consists of 53 items rated on a five-point scale. The items are grouped into nine scales of Anxiety, Hostility, Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Phobic Anxiety, Paranoid Ideation, and Psychoticism. A positive symptoms distress index was computed by summing the items for all the subscales and dividing by the number of items endorsed with a positive response. The BSI has been reported to have high internal consistency and has been used in a large number of studies, including studies of maternal cocaine use [58]. Symptoms of Post-Traumatic Stress Disorder (PTSD) were measured using the Impact of Events Scale-Revised (IES-R) [67] at the 7 month visit. The items in this scale parallel the DSM-IV criteria for PTSD and assess current subjective

distress for any specific life event. The scale consists of 22 items and has been shown to have good psychometric properties [9,33,67]. Finally, antisocial behavior was assessed with the Antisocial Behavior Checklist (ASB) [69] at 7 months. Caregivers were asked to rate their frequency of participation in a variety of aggressive and antisocial activities along a 4-point scale rating from 1 (never) to 4 (very often). The measure has been found to discriminate among groups with major histories of antisocial behavior (e.g., prison inmates, individuals with minor offenses in district court, and university students) [69] and between alcoholic and non-alcoholic adult males [30].

We conducted confirmatory factor analyses using these three measures as indicators of a latent construct reflecting maternal psychological distress. Confirmatory factor analyses of this measurement model indicated that the three measures loaded on one factor reflecting maternal psychological distress (high positive symptoms on the BSI, high PTSD symptoms, and more antisocial behavior, with standardized factor loadings ranging from .52 to .62).

Infant Reactivity—Maternal reports on the Distress to Limitations subscale of the Infant Behavior Questionnaire (IBQ) [54] was used as a measure for infant reactivity at the 7 month visit. The IBQ yields six subscales: Activity Level, Distress to Limitations, Latency to Approach, Duration of Orientation, Smiling and Laughter, and Soothability. The IBQ was administered to mothers by trained interviewers due to the complexity of the scale and concerns about literacy. The internal consistency of this scale for this sample was *Cronbach's alpha* = .69.

Mother-Infant Interactions—Mother-infant interactions were assessed at 1 and 13 months of infant age, corrected for prematurity. At 1 month, mothers were asked to feed their infants as they normally would at home and were videotaped. The first 10 minutes of these interactions were coded using the Mother-Infant Feeding Scale [13]. For the purposes of this study, 18 of the original 26 maternal items were coded. The remaining items were not included due to low variability. Principal components analysis with varimax rotation yielded eigenvalues greater than 3 for the first two factors, and lower eigenvalues on five other factors. Thus, this analysis was repeated with a two-factor solution, resulting in the factors of maternal intrusiveness (with items such as *restricts movements, handles excessively, handles roughly*) and maternal warmth or sensitivity [27]. The factor labeled maternal sensitivity was used in this study and included items such as *talking to infant, making positive remarks to infant, pleasure toward infant, high positive affect, low detachment or sadness*. The internal consistency of this scale was *Cronbach's alpha* = .79.

At 13 months, mothers were asked to interact with their infants as they normally would at home for 10 minutes in a room filled with toys. These interactions were videotaped and coded using a collection of global 5-point rating scales called the *Parent-Child Early Relational Assessment* [14]. Three composite maternal scales labeled positive involvement, sensitivity, and low negative affect, and three infant scales labeled positive affect, negative affect, and responsiveness were derived from these items [24]. The internal consistencies of these scales were *Cronbach's alpha* = .94 for all three maternal behavior scales, and ranged from .86 to .94 for the infant behavior scales.

Two coders blind to group status rated the feeding interactions at 1 month, and two different coders rated the play interactions at 13 months. Both groups of coders were trained by the first author until inter-rater reliability criterion was reached (agreement of 90% or above). Subsequently inter-rater reliability was established on 22% of the tapes. Inter-rater reliability on the individual items of the feeding scales ranged from intraclass correlation coefficients of .85 to .96. Inter-rater reliability on the composite Clark scales ranged from intraclass correlations of .82 to .90.

Results

Missing Data and Data Analytic Approach

As would be expected of any longitudinal study involving high risk families, there were incomplete data for some participants at one or more of the three assessment points used in this study. Of the 180 infants who were not in foster care, 160 completed the 7 month assessments, and 145 completed the mother-infant play interactions at 13 months. At 7 months of infant age, 7 families declined to participate or did not show up after repeated reschedules, 10 were unable to be located, 2 were dropped from the study (one because of a diagnosis of fetal alcohol syndrome, and another because of a diagnosis of Shaken Baby Syndrome), and 1 was in the middle of new custody arrangements. At 13 months, an additional 4 infants were fussy and unable to complete the free play assessment, 1 was an outlier because the mother did not interact with the infant at all during free play and thus was not coded, 5 were unable to be located, and 5 passed the target dates for the lab assessment. There were no significant differences between families with complete vs. missing data at 7 or 13 months on any demographic or substance use variable ($p > .20$).

Although it is not clear if the data were missing completely at random, data did meet criteria for missing at random (MAR). Little and Rubin [45] defined data as MAR when cases with incomplete data differed from cases with complete data, but the pattern of missingness could be predicted from other variables in the database. Discussions of missing data have also noted that the assumption for MAR can never be definitively assessed. However, given that no differences were found between families with missing data and those with complete data on parenting or child outcome variables, the assumption of MAR seems tenable for these data. To take advantage of all data provided by all participants, we used full-information maximum likelihood (FIML) to estimate parameters during model testing [1]. MANCOVAs were used to test group differences, gender differences, and group by gender interactions on study variables. Structural equations modeling (SEM) was used to test the mediational model. All SEM analyses were conducted using Mplus (Version 4.0) [51]. FIML estimation procedures were used and standardized parameter estimates are presented. The goodness of fit of the models was estimated by using the comparative fit index (CFI) and the root-mean-square error of approximation (RMSEA). Given the limits of sample size, we chose to examine moderation using hierarchical regressions instead of multiple group models in SEM.

Demographic and Descriptive Information

Results from MANOVA with maternal age, education, and parity yielded a significant multivariate effect of group status, $F(3, 176) = 8.08, p < .001$. Results from univariate analyses indicated that control group mothers were younger and had lower parity compared to those in the cocaine group (see Table 1)¹. Correlational analyses were used to examine the association between maternal age, education, and parity and mother-infant interaction variables. Results indicated no significant associations between these measures. Maternal age, education, and parity were included in the initial SEM model, but given lack of association with mediators or outcomes, were dropped from final model testing in order to have a parsimonious final model. A second MANOVA was conducted with infant birth outcomes as the dependent measures. Results indicated a significant effect of group status on infant birth outcomes, $F(5, 174) = 6.70, p < .05$. Univariate analyses indicated that cocaine exposed infants had lower gestational age, birth weight, birth length, and cocaine

¹Once a woman was recruited into the cocaine group, the closest matching non-cocaine using woman with regard to maternal education, age, race/ethnicity and infant gender was recruited into the control group. This method resulted in small but significant group differences in maternal age.

using mothers had less optimal scores on the Obstetrical Complications Scale compared to those in the control group (see Table 1). 87% of the cocaine exposed, and 97% of the comparison infants were full term (≥ 37 weeks gestational age). Correlational analyses were used to examine the association between infant birth outcomes and mother-infant interaction variables. There were no significant associations. MANOVAs were used to examine if there were gender differences in maternal or infant behavior. No gender differences were obtained.

Maternal Substance Use

Results from MANOVA with prenatal substance use variables as the dependent measures and group status as the independent variable yielded a significant multivariate effect of group status, $F(4, 175) = 6.00, p < .01$. As expected, mothers in the cocaine group were heavier users of cigarettes, alcohol, and cocaine during pregnancy (see Table 2). There was no group difference in marijuana use. Results of MANOVA with postnatal substance use measures at 7 months yielded a significant effect of group status, $F(4, 175) = 6.29, p < .001$. Mothers in the cocaine group continued to be heavier users of cigarettes and cocaine, but not marijuana, during the postnatal period (see Table 2).

Group Differences in Mother-Infant Interactions

Results from MANOVA with the three maternal behavior variables as the dependent measures and group status as the independent variable yielded a significant multivariate effect of group status, $F(3, 141) = 2.68, p < .05$. Mothers in the cocaine group had higher negative affect and lower sensitivity compared to mothers in the control group. Maternal sensitivity and maternal low negative affect were positively correlated with each other ($r = .64, p < .001$) and were averaged into a single composite measure for purposes of model testing and labeled maternal warmth/sensitivity.

These analyses were repeated with other prenatal substance use variables as covariates (number of cigarettes per day, number of standard drinks per day, and number of joints per day). Results remained unchanged. Results from MANOVA with the three infant behavior variables as the dependent measures and group status as the independent variable indicated a marginal multivariate effect of group status $F(3, 141) = 2.34, p < .10$. Mothers in the cocaine group had infants who were less responsive toward them (see Table 2). These analyses were repeated with other prenatal substance use variables as covariates (number of cigarettes per day, number of standard drinks per day, and number of joints per day). The group difference on infant responsiveness remained significant, with cocaine exposed infants displaying lower responsiveness compared to the control group infants.

Associations among Study Variables

Associations among the variables in the hypothesized model are presented in Table 3. We used the dummy coded group status variable in these analyses (0 = control group). We chose to use the average number of drinks per week as an indicator of risky drinking during pregnancy. The initial model included prenatal and postnatal marijuana use (number of joints per week), but these two variables were not associated with either other substance use or any of the other variables in the model. Thus, marijuana use was dropped from model testing. Maternal substance use during pregnancy and average drinks per week in the postnatal period were consistently associated with higher antisocial behavior. Postnatal, but not prenatal substance use (alcohol and cigarettes) were associated with higher PTSD symptoms and higher BSI scores. Higher infant reactivity was associated with higher prenatal alcohol use. Higher maternal antisocial behavior was associated with higher infant reactivity and higher BSI score at 7 months was associated with lower child responsiveness at 13 months.

Model Testing

SEM was used to examine the fit of a hypothesized model that included maternal psychological distress and infant reactivity at 7 months as potential mediators of the association between maternal cocaine use and mother-infant interactions. The model included predictive paths from prenatal and postnatal substance use variables to the latent variable for maternal psychological distress and to infant reactivity, and predictive paths from these two variables to the composite measures for maternal warmth/sensitivity and child responsiveness. The model included predictive paths from maternal sensitivity at 1 month to infant reactivity at 7 months and maternal warmth/sensitivity at 13 months. The model also included covariances among the pre and postnatal substance use variables, covariances among postnatal substance use at 7 months and the residuals for maternal psychological distress and infant reactivity, between the residuals of maternal psychological distress and infant reactivity, and the covariance between the residuals of the two interaction variables. Goodness of fit indices revealed that this hypothesized model was an adequate explanation of the data (i.e., $\chi^2_{(39)} = 50.31$, $p = .11$, CFI = .97, RMSEA = .04). Contrary to hypotheses, maternal cocaine use during pregnancy was not associated with infant reactivity at 7 months, or with maternal psychological distress. However, lower maternal sensitivity during feeding interactions at 1 month was predictive of higher infant reactivity at 7 months. Higher infant reactivity at 7 months was predictive of lower maternal warmth/sensitivity at 13 months. The association between maternal sensitivity during feeding at 1 month and during play at 13 months was non-significant. Given the lack of association between maternal cocaine use and the two hypothesized mediators and group differences in maternal and child behavior during interactions at 13 months, we added direct paths from cocaine group status to the two interaction variables at 13 months, and a path from maternal sensitivity at 1 month to infant responsiveness at 13 months. This final model is depicted in Figure 1. The addition of these three paths resulted in a significant improvement in fit, $\Delta\chi^2_{(3)} = 9.83$, $p < .05$. Goodness of fit indices revealed that this model fit the data well, $\chi^2_{(36)} = 40.48$, $p = .28$, CFI = .99, RMSEA = .026). In addition to the significant paths from the previous model, cocaine group status was directly associated with lower maternal sensitivity and infant responsiveness at 13 months.

Moderation

Separate hierarchical regression analyses were used to examine the roles of maternal psychological distress and infant reactivity as potential moderators of the association between maternal cocaine use and maternal warmth/sensitivity and infant responsiveness at 13 months. Maternal substance use variables including the dummy coded group status variable were entered in the first step, the moderators were entered in the second step, and the two-way interaction term in the third step. For maternal warmth/sensitivity, there was a significant interaction effect of group status and infant reactivity ($\beta = -.47$, $p < .001$). As depicted in Figure 2, cocaine using mothers who reported their infants as being more reactive were less warm/sensitive toward them compared to cocaine using mothers with less reactive infants. In other words, the association between maternal cocaine use and maternal warmth/sensitivity was significant only when mothers reported their infants to be more reactive. These analyses were repeated with infant responsiveness as the dependent variable. No significant interaction effects were obtained.

Discussion

Results from this study indicate that mothers who used cocaine during pregnancy display higher negative affect and lower sensitivity toward their infant during play interactions at 13 months, and their infants were less responsive toward them. The results lend further support to a body of literature indicating that maternal cocaine use may compromise the quality of

mother-infant interactions. Maternal cocaine use was directly associated with maternal and infant behaviors, even after controlling the within time or concurrent association between maternal warmth/sensitivity and infant responsiveness. Results are supportive of animal literature suggesting direct teratological effects of cocaine on maternal behavior [61]. It is also supportive of human studies indicating that cocaine using mothers were less emotionally engaged with their infants in the infant/toddler period [50].

Contrary to hypothesis, the association between maternal cocaine use and maternal warmth/sensitivity was not mediated by maternal psychological distress or by infant reactivity. However, results were supportive of a transactional model for both cocaine exposed and non-cocaine exposed infants where lower maternal sensitivity at 1 month was predictive of higher infant reactivity at 7 months, which in turn was predictive of lower maternal warmth/sensitivity at 13 months, controlling for the direct association between maternal behavior at 1 and 13 months and independent of maternal cocaine use. The direction of influence in parent-infant interactions has historically been assumed to be from parent to infant, although some studies have emphasized the role of infant behavior as influencing parent behavior as well [8]. Transactional models of development [43,55] have emphasized that both infant and environment (parenting and other risk factors) play a role in determining the course of development. These models favor the idea that there is an active environment and an active child adapting to and influencing the environment, and that parents and children influence each other over time. Results from this study indicate that *poorer* early maternal sensitivity is associated with *greater* infant reactivity in later infancy, which in turn is predictive of changes in maternal sensitivity from 1 month to 13 months of child age. It is important to note that the measure of infant reactivity was a maternal report measure, and thus was reflective of both infant behavior as well as maternal perceptions of the infant, and that this transactional model was supported for the sample as a whole and was not associated with prenatal cocaine exposure. Maternal reports of the infant are likely to be based on a wider sampling of behavior compared to the few minutes of laboratory based assessment. In addition, maternal perception of the infant is likely to reflect mothers' actual experience with the infant and be more predictive of maternal behavior toward the infant than laboratory based measures. Thus, one interpretation of the findings is that cocaine using mothers who display lower warmth toward their infants perceive their infants as more reactive.

On the other hand, these findings are also consistent with an interpretation that cocaine-using mothers who display lower warmth toward their infants may merely perceive their infants as more reactive. For instance, previous studies with cocaine using samples have reported significant and positive associations between maternal reports of infant reactivity and parenting stress, but not between objective measures of infant reactivity and parenting [56]. Similarly, initial examination of objective measures of infant reactivity (behavioral or physiological indices) in this data set indicated no associations with maternal behavior, indicating that maternal perception of infant reactivity is an important predictor of how mothers behave toward their infants during play interactions.

Results also indicated that as hypothesized, infant reactivity moderated the association between maternal cocaine use during pregnancy and maternal warmth/sensitivity at 13 months of age. The association between maternal cocaine use and maternal warmth/sensitivity was stronger at higher levels of infant reactivity. One interpretation of these findings is that unlike control group mothers, the behavior of cocaine using mothers is more influenced by their experiences with and perceptions of their infants. If their perceptions are more negative, their behavior toward their infant tends to be less warm/sensitive. Thus, cocaine using mothers who reported their infants as being more reactive in early infancy were less warm/sensitive toward them in later infancy. Few studies have examined if the

association between maternal cocaine use and maternal behavior are mediated or moderated by other variables. This is important because it speaks to the process or mechanisms by which maternal cocaine use may be associated with poor maternal behavior. These results indicate that maternal perceptions of the infant may play an important role in this area.

Several studies have reported associations between cocaine exposure and infant reactivity using more objective measures [6,26,48]. Although no direct associations were obtained in this study using maternal reports, results indicate that maternal sensitivity in early infancy longitudinally predicts maternal reports of infant reactivity, and these perceptions may be particularly salient for eliciting warm/sensitive maternal behavior.

This study has several limitations. First, accurate assessment of maternal substance use is difficult. Pregnant and postpartum women are often hesitant to divulge substance use information, particularly illicit substances such as cocaine. One strength of this study is the use of multiple methods to ascertain prenatal substance use. However, measures of prenatal use were retrospective and measures of postnatal use were based on self-report alone. A second limitation is that the measurement of mother-infant interaction was brief, and limited to a single play session. However, this was an objective measure of parenting, as opposed to parent reports of their own parenting behavior. A third limitation is that results may be generalized only to infants exposed to crack/cocaine, as the majority of the women used this form of cocaine during pregnancy. A fourth limitation is that our results regarding infant reactivity was based in maternal report. On the one hand, maternal perceptions may reflect maternal experiences with the infant which are likely to be based on a larger sampling of behavior. On the other hand, we did not include laboratory measures of infant reactivity to test if maternal reports or lab based measures of infant reactivity would be more significant predictors of maternal behavior. Finally, we did not have adequate power to test the hypothesis that the association between prenatal cocaine exposure and mother-infant interactions would be stronger under conditions of high maternal psychological distress and higher infant reactivity. Future studies with larger sample sizes may examine the possibility of three-way interactions.

Despite these limitations, these findings lend further empirical support to animal and human studies indicating significant effects of prenatal cocaine exposure on maternal and child behavior. They suggest that infant reactivity, particularly parental experiences and perceptions of infant reactivity may moderate the association between maternal cocaine use and maternal sensitivity. Highly reactive cocaine exposed infants may be at higher risk for child abuse. Parenting interventions that teach parents strategies to regulate high reactivity may be most beneficial for this group. The findings also indicate that one pathway to later child outcomes may be through the detrimental effects of maternal cocaine use on maternal sensitivity. Thus, interventions designed to increase maternal sensitivity may lead to improved child outcomes.

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References

1. Arbuckle, JL. Full information estimation in the presence of incomplete data. In: Marcoulides, GA.; Schumacker, RE., editors. *Advanced structural equation modeling: issues and techniques*. Lawrence Erlbaum Associates; Mahwah, NJ: 1996. p. 243-277.
2. Bauman PS, Dougherty FE. Drug-addicted mothers' parenting and their children's development. *Int J Addict* 1983;18:291–302. [PubMed: 6874152]
3. Baumgartner WA, Hill VA, Blahd WH. Hair analysis for drugs of abuse. *J Forensic Sci* 1989;34:1433–1453.
4. Bell RQ. A reinterpretation of the direction of effects in studies of socialization. *Psychol Rev* 1968;75:81–95. [PubMed: 4870552]
5. Bendersky M, Alessandri S, Gilbert P, Lewis M. Characteristics of pregnant substance abusers in two cities in the Northeast. *Am J Drug Alcohol Abuse* 1996;22:349–362. [PubMed: 8841684]
6. Bendersky M, Lewis M. Arousal modulation in cocaine-exposed infants. *Dev Psychol* 1998;34:555–564. [PubMed: 9597364]
7. Black M, Schuler M, Nair P. Prenatal drug exposure: neurodevelopmental outcome and parenting environment. *J Pediatr Psychol* 1993;18:605–620. [PubMed: 7507525]
8. Bornstein, MH. Parenting infants. In: Bornstein, MH., editor. *Handbook of parenting: Vol. 1: children and parenting*. 2. Erlbaum; Mahwah, NJ: 2002.
9. Briere J. Methodological issues in the study of sexual abuse effects. *J Consult Clin Psychol* 1997;60:196–203. [PubMed: 1592948]
10. Brown RA, Burgess ES, Sales SD, Whiteley JA, Evans DM, Miller IW. Reliability and validity of a smoking timeline follow-back interview. *Psychol Addict Behav* 1998;12:101–112.
11. Burns K, Chethik L, Burns WJ, Clark R. Dyadic disturbances on cocaine-abusing mothers and their infants. *J Clin Psychol* 1991;47:316–319. [PubMed: 2030140]
12. Burns KA, Chethik L, Burns WJ, Clark R. The early relationship of drug abusing mothers and their infants: an assessment at eight to twelve months of age. *J Clin Psychol* 1997;53:279–287. [PubMed: 9075056]
13. Chatoor, I. Mother-infant/toddler feeding scale. unpublished manuscript
14. Clark R. The parent-child early relational assessment: a factorial validity study. *Educ Psychol Meas* 1999;59:821–846.
15. Coles CD, Platzman KA, Smith I, James M, Falek A. Effects of cocaine and alcohol use in pregnancy on neonatal growth and neurobehavioral status. *Neurotoxicol Teratol* 1992;14:23–33. [PubMed: 1593976]
16. Crockenberg S, Smith P. Antecedents of mother–infant interaction and infant irritability in the first three months of life. *Infant Behav Dev* 1982;5:105–119.
17. Crockenberg S, Smith P. Antecedents of mother-infant interaction and infant irritability in the first 3 months of life. *Infant Behav Dev* 2002;25:2–15. Reprinted from 1982.
18. Delaney-Black V, Covington C, Nordstrom B, Ager J, Janisse J, Hannigan JH, Chiodo L, Sokol RJ. Prenatal cocaine: Quantity of exposure and gender moderation. *Dev Behav Pediatr* 2004;25:254–263.
19. Delaney-Black V, Covington C, Templin T, Kershaw T, Nordstrom-Klee B, Ager J, et al. Expressive language development of children exposed to cocaine prenatally: literature review and report of a prospective study. *J Commun Dis* 2000;33:463–481.
20. Dennis T, Bendersky M, Ramsay DS, Lewis M. Reactivity and regulation in children prenatally exposed to cocaine. *Dev Psychol* 2006;42:688–697. [PubMed: 16802901]
21. Derogatis, LR. *The Brief Symptom Inventory (BSI) administration, scoring, and procedures manual*. National Computer Systems; Minneapolis, MN: 1993.
22. Donovan WL, Leavitt LA, Walsh RO. Conflict and depression predict maternal sensitivity to infant cries. *Infant Behav Dev* 1998;21:505–517.
23. Downey G, Coyne JC. Children of depressed parents: an integrative review. *Psychol Bull* 1990;104:50–76. [PubMed: 2200073]

24. Eiden RD, Chavez F, Leonard KE. Parent-infant interactions in alcoholic and control families. *Dev Psychopathology* 1999;11:745–762.
25. Eiden RD, Foote A, Schuetze P. Maternal cocaine use and caregiving status: Group differences in caregiver and infant risk variables. *Addict Behav* 2007;32:465–476. [PubMed: 16837139]
26. Eiden RE, McAuliffe S, Kachadourian L, Coles C, Colder C, Schuetze P. Effects of prenatal cocaine exposure on infant reactivity and regulation. *Neurotoxicol Teratol* 2009;31:60–68. [PubMed: 18822371]
27. Eiden RD, Stevens A, Schuetze P, Dombkowski LE. A conceptual model for maternal behavior among polydrug cocaine-using mothers: the role of postnatal cocaine use and maternal depression. *Psychol Addict Behav* 2006;20:1–10. [PubMed: 16536660]
28. Eyler FD, Behnke M, Garvan CW, Woods NS, Wobie K, Conlon M. Newborn evaluations of toxicity and withdrawal related to prenatal cocaine exposure. *Neurotoxicol Teratol* 2001;23:399–411. [PubMed: 11711242]
29. Fish M, Stifter CA, Belsky J. Conditions of continuity and discontinuity in infant negative emotionality: newborn to five months. *Child Dev* 1991;62:1525–1537. [PubMed: 1786733]
30. Fitzgerald, HE.; Jones, MA.; Maguin, E.; Zucker, RA.; Noll, RB. Assessing parental antisocial behavior in alcoholic and nonalcoholic families. Michigan State University, Department of Psychology; East Lansing, MI: unpublished manuscript
31. Gottwald SR, Thurman SK. The effects of prenatal cocaine exposure on mother-infant interaction and infant arousal in the newborn period. *Topics Early Child Spec Educ* 1994;14:217–231.
32. Heffelfinger AK, Craft S, White DA, Shyken J. Visual attention in preschool children prenatally exposed to cocaine: implications for behavioral regulation. *J Int Neuropsychol Soc* 2002;8:12–21. [PubMed: 11843069]
33. Horowitz M, Wilner N, Alvarez W. Impact of event scale: a measure of subjective stress. *Psychosom Med* 1979;41:209–218. [PubMed: 472086]
34. Johns JM, Elliott DL, Hofler VE, Joyner PW, McMurray MS, Jarrett TM, Haslup AM, Middleton CL, Elliott JC, Walker CH. Cocaine treatment and prenatal environment interact to disrupt intergenerational maternal behavior in rats. *Behav Neurosci* 2005a;19:1605–1618.
35. Johns JM, Joyner PW, McMurray MS, Elliott DL, Hofler VE, Middleton CL, Knupp K, Greenhill KW, Lomas LM, Walker CH. The effects of dopaminergic/serotonergic reuptake inhibition on maternal behavior, maternal aggression, and oxytocin in the rat. *Pharmacol Biochem Behav* 2005b;81:769–785. [PubMed: 15996723]
36. Johns JM, Lubin DA, Walker CH, Meter KE, Mason GA. Chronic gestational cocaine treatment decreases oxytocin levels in the medial preoptic area, ventral tegmental area and hippocampus in Sprague-Dawley rats. *Neuropeptides* 1997;31:439–443. [PubMed: 9413020]
37. Johnson AL, Morrow C, Accornero VH, Xue L, Anthony JC, Bandstra ES. Maternal cocaine use: estimated effects on mother-child play interactions in the preschool period. *J Dev Behav Pediatr* 2002;23:191–202. [PubMed: 12177564]
38. Johnson HL, Rosen TS. Mother-infant interaction in a multirisk population. *Am J Orthopsychiatry* 1990;60:281–288. [PubMed: 2098001]
39. Karmel BZ, Gardner JM. Prenatal cocaine exposure effects on arousal-modulated attention during the neonatal period. *Dev Psychobiol* 1996;29:463–480. [PubMed: 8809496]
40. LaGasse LL, Messinger D, Lester BM, Seifer R, Tronick EZ, Bauer CR, Shankaran S, Bada HS, Wright LL, Smeriglio VL, Finnegan LP, Maza PL, Liu J. Prenatal drug exposure and maternal and infant feeding behaviour. *Arch Dis Child Fetal Neo Educ* 2003;88:F391–F399.
41. Lester BM, Padbury JF. Third pathophysiology of prenatal cocaine exposure. *Dev Neurosci* 2009;31:23–35. [PubMed: 19372684]
42. Lester BM, Tronick EZ, LaGasse L, Seifer R, Bauer CR, Shankaran S, et al. The maternal lifestyle study: effects of substance exposure during pregnancy on neurodevelopmental outcome in 1-month-old infants. *Pediatr* 2002;110:1181–1192.
43. Lewis, M. Toward a development of psychopathology: models, definitions, and prediction. In: Sameroff, AJ.; Lewis, M.; Miller, S., editors. *Handbook of developmental psychopathology*. Vol. 2. Kluwer Academic; Dordrecht, the Netherlands: 2000. p. 3-22.

44. Light KC, Grewen KM, Amico JA, Boccia M, Brownley KA, Johns JM. Deficits in plasma oxytocin responses and increased negative affect, stress, and blood pressure in mothers with cocaine exposure during pregnancy. *Addict Behav* 2004;29:1541–1564. [PubMed: 15451123]
45. Little R, Rubin D. The analysis of social science data with missing values. *Sociol Methods Res* 1989;18:292–326.
46. Littman A, Parmelee B. Medical correlation of infant development. *Pediatrics* 1978;61:470–474. [PubMed: 643420]
47. Magura S, Freeman RC, Siddiqi Q, Lipton DS. The validity of hair analysis for detecting cocaine and heroin use among addicts. *Int J Addict* 1992;27:51–69. [PubMed: 1537640]
48. Mayes LC, Bornstein MH, Chawarska K, Haynes OM, Granger RH. Impaired regulation of arousal in 3-month-old infants exposed prenatally to cocaine and other drugs. *Dev Psychopathol* 1996;8:29–42.
49. McMurray MS, Joyner PW, Middleton CW, Jarrett TM, Elliott DL, Black MA, Hofler VE, Walker CH, Johns JM. Intergenerational effects of cocaine treatment and rearing environment on aggressive behavior and oxytocin in rats. *Stress* 2008;11:398–410. [PubMed: 18609307]
50. Molitor A, Mayes LC, Ward A. Emotion regulation behavior during a separation procedure in 18-month-old children of mothers using cocaine and other drugs. *Dev Psychopathol* 2003;15:39–54. [PubMed: 12848434]
51. Muthen, LK.; Muthen, BO. *Mplus User's Guide* (1998–2006). Muthen & Muthen; Los Angeles, CA: 2006.
52. Nelson C, Ayers A, Meter KE, Walker CH, Johns JM. The effects of multiple doses of chronic gestational cocaine treatment on the onset and maintenance of maternal behavior in Sprague-Dawley rats. *Neurotoxicol Teratol* 1998;20:657–660. [PubMed: 9831128]
53. Neuspiel DR, Hamel SC, Hochberg E, Greene J, Campbell D. Maternal cocaine use and infant behavior. *Neurotoxicol Teratol* 1991;13:229–233. [PubMed: 2046640]
54. Rothbart MK. Measurement of temperament in infancy. *Child Dev* 1981;52:569–578.
55. Sameroff, AJ.; Fiese, BH. Transactional regulation: The development ecology of early intervention. In: Shonkoff, JP.; Meisels, SJ., editors. *Handbook of early childhood intervention*. Cambridge University Press; Cambridge, UK: 2000. p. 135-159.
56. Sheinkopf SJ, Lester BM, LaGasse LL, Seifer R, Bauer CR, Shankaran S, Bada HS, Poole WK, Wright LL. Interactions between maternal characteristics and neonatal behavior in the prediction of parenting stress and perception of infant temperament. *J Pediatr Psychol* 2006;31:27–40. [PubMed: 15827350]
57. Singer L, Arendt R, Minnes S, Farkas K, Yamashita T, Kliegman R. Increased psychological distress in post-partum, cocaine-using mothers. *J Subst Abuse* 1995;7:165–174. [PubMed: 7580227]
58. Singer LT, Salvator A, Arendt R, Minnes S, Farkas K, Kliegman R. Effects of cocaine/polydrug exposure and maternal psychological distress on infant birth outcomes. *Neurotoxicol Teratol* 2002;24:127–135. [PubMed: 11943500]
59. Sobell MB, Sobell LC, Klajner F, Pavan D, Basian E. The reliability of a timeline method for assessing normal drinker college students' recent drinking history. *Addict Behav* 1986;11:149–162. [PubMed: 3739800]
60. Spinrad, TL.; Stifter, CA. Maternal sensitivity and infant emotional reactivity: concurrent and longitudinal relations. In: Fabes, RA., editor. *Emotions and the Family*. Haworth Press; New York: 2002. p. 243-263.
61. Swain JE, Lorberbaum JP, Kose S, Strathearn L. Brain basis of early parent-infant interactions: psychology, physiology, and in vivo functional neuroimaging studies. *J Child Psychol Psychiatry* 2007;48:262–287. [PubMed: 17355399]
62. Thompson, RA. Early sociopersonality development. In: Damon, W.; Eisenberg, N., editors. *Handbook of child psychology, Vol. 3: social, emotional and personality development*. 5. Wiley; New York: 1998. p. 25-104.
63. Ukeje I, Bendersky M, Lewis M. Mother-infant interactions at 12 months in prenatally cocaine-exposed children. *Am J Drug Alcohol Abuse* 27(2):203–224. [PubMed: 11417936]

64. van de Boom DC. The influence of temperament and mothering on attachment and exploration: an experimental manipulation of sensitive responsiveness among lower-class mothers with irritable infants. *Child Dev* 1984;65:1457–1477.
65. Vernotica EM, Lisciotto CA, Rosenblatt JS, Morrell J. Cocaine transiently impairs maternal behavior in the rat. *Behav Neurosci* 1996;110:315–323. [PubMed: 8731058]
66. Wachs, TD.; Kohnstamm, GA. The bidirectional nature of temperament-context links. In: Wachs, TD.; Kohnstamm, GA., editors. *Temperament in context*. Lawrence Erlbaum Associates; Mahwah, NJ: 2001. p. 201-222.
67. Weiss, DS.; Marmar, CR. The Impact of Event Scale—Revised. In: Wilson, JP.; Keane, TM., editors. *Assessing psychological trauma and PTSD*. Guilford Press; New York: 1997. p. 399-411.
68. Woods NS, Eyler FD, Behnke M, Conlon M. Cocaine use during pregnancy: Maternal depressive symptoms and infant neurobehavior over the first month. *Infant Behav Dev* 1993;16:83–98.
69. Zucker, RA.; Noll, RB. The antisocial behavior checklist. University of Michigan; unpublished manuscript

Figure 1.

Structural model for mother-infant interactions. Maternal age, parity, and education were initially included as predictors in this model, but were not associated with the mediators or the outcomes. Thus, the final model was estimated without these three variables. The model included predictive paths from postnatal substance use variables to infant reactivity and maternal psychological distress, but these non-significant paths are not depicted in the figure for ease of presentation.

Figure 2.

Interaction of group status and infant reactivity on maternal warmth/sensitivity at 13 months. Non-significant within time covariances (e.g., between infant reactivity and maternal psychological distress) were included in the model but not depicted in the figure.

Table 1

Group Differences in Demographic Variables and Birth Outcomes

Variables	Non-Cocaine		Cocaine		F value	η^2
	M	SD	M	SD		
<u>Demographics:</u>						
BM Age	27.89	5.57	31.11	6.03	13.85**	.07
BM Parity	3.19	1.63	4.23	2.60	10.72**	.06
Years Education	12.03	1.88	11.56	1.79	2.96	.02
<u>Birth Outcomes</u>						
Gestational Age	39.37	1.22	38.30	1.69	6.61*	.04
Birth weight	3342.76	504.38	3026.36	542.72	15.57**	.08
Birth length	50.01	2.92	48.35	3.23	12.46**	.07
Head circumference	33.62	1.40	33.26	2.36	1.60	.01
OCS	100.69	17.53	88.73	15.92	21.31**	.11

* $p < .05$ ** $p < .01$

BM: Biological Mother, OCS: Obstetrical Complications Scale.

Table 2

Group Differences in Maternal Substance Use and Parenting Variables.

Variables	Non-Cocaine		Cocaine		F value	η^2
	M	SD	M	SD		
<u>Pregnancy:</u>						
Cigarettes/week	13.02	25.95	31.75	41.11	13.77**	.07
Drinks/week	.19	.82	4.30	11.51	12.51**	.07
Joints/week	1.48	7.39	1.28	4.41	.04	.01
Days cocaine/week	0	0	.57	1.26	20.55**	.10
<u>Postnatal</u>						
Cigarettes/week	18.12	31.42	30.88	33.32	6.96**	.04
Drinks/week	.54	.99	2.18	5.55	8.31**	.05
Joints/week	1.53	5.14	1.62	5.89	.01	.00
Days cocaine/week	0	0	.10	.37	6.99**	.04
<u>Mother-Infant Interaction</u>						
<u>Mother:</u>						
Positive Involvement	3.85	.78	3.61	.97	2.73+	.02
Negative Affect	4.70	.52	4.39	.80	7.79**	.05
Sensitivity	4.65	.94	4.24	1.30	4.86*	.03
<u>Infant:</u>						
Positive Affect	3.80	.85	3.59	.97	1.96	.01
Negative Affect	4.60	.69	4.52	.84	.45	.00
Responsiveness	4.32	.68	4.02	.69	6.34**	.04

*
 $p < .05$ **
 $p < .01$ +
 $p < .10$

High scores on all Clark scales are good. Thus, higher scores on negative affect indicate lower negative affect.

Table 3

	AA/Week: Preg	# Cigs/Week: Preg	Cocaine use: 7m	AA/Week: 7m	# Cigs/Week: 7m	Maternal Warmth: 1m	PTSD Symptoms: 7m	ASB: 7m	BSI - PSI: 7m	Infant Reactivity: 7m	Maternal Warmth/Sens: 13m
.09***											
.07***											
.14***			.66***								
.06***		.08	.19*	.28***							
.04		.14	-.03	-.01	.04						
.0		.03	.07	.21***	.19*	.09					
.00		.13	.08	.20*	.09	.11	.29***				
.0		.22***	.02	.13	.18*	-.06	.38***	.36***			
.06*		.20*	.05	.00	.11	-.14	.07	.18*	.11		
.06		.14	-.06	.04	-.05	.07	-.03	-.15	-.06	-.27***	
.10		-.15	.00	-.03	-.14	.04	.01	-.09	-.23***	-.07	.38***

tes, Mat: Maternal, PTSD: Post Traumatic Stress Disorder, ASB: Antisocial Behavior, PSI: Positive Symptom Index; Sens: Sensitivity; Resp: