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Oxytocin and parenting behavior among impoverished mothers with low vs. high early life stress

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Abstract

Purpose—Recent work suggests that key aspects of sensitive parenting (e.g., warmth, emotional attunement) may be shaped in part by biology, specifically the neuropeptide oxytocin. However, some studies have found that oxytocin may not act in expected ways in higher-risk populations (e.g., those with postnatal depression or borderline personality disorder). This study examined the relation between oxytocin and parenting among mothers with varying levels of early life stress.

Methods—Forty low-income mothers and their 34 to 48-month-old child participated in this study. Mother-child dyads were observed in an interaction task in their home, and videos of these interactions were later coded for parenting behaviors. Mothers' oxytocin production before and after the interaction task was assessed through saliva. Mothers' early stress was assessed via the Adverse Childhood Experiences Scale (ACES; Felitti et al., 1998).

Results—For mothers with low ACEs, higher oxytocin secretion was associated with more positive parenting. For mothers with high ACEs, higher oxytocin secretion was associated with lower levels of positive parenting.

Conclusions—Oxytocin may be operating differently for mothers who experienced harsh early social environments, supporting more defensive behaviors and harsh parenting than anxiolytic and prosocial behaviors.

Keywords

Parenting; Stress; Oxytocin; Parent-child interaction; Adverse Childhood Experiences

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Young children depend on their caregivers to scaffold their development and help them cope with negative arousal by reading child cues, providing comfort, anticipating challenges, and promptly responding to the child's needs. Parenting can play a protective role for young children who are at higher familial (Raby, Roisman, Fraley, & Simpson, 2015) or genetic risk (Kochanska, Boldt, Kim, Yoon, & Philibert, 2015), and parenting behaviors are associated with children's self-regulation (Brophy-Herb, Stansbury, Bocknek, & Horodynski, 2012; Eiden, Edwards, & Leonard, 2007), behavior problems (Lunkenheimer, Olson, Hollenstein, Sameroff, & Winter, 2011), internalizing problems (Rose, Roman, Mwaba, & Ismail, 2017), and executive function (Lucassen et al., 2015). Recent work suggests that key aspects of sensitive parenting (e.g., warmth, emotional attunement) may be shaped in part by biology, specifically the neuropeptide oxytocin. In the current study we consider associations of oxytocin and observed positive and negative parenting in a low-income sample of mother-child dyads.

Biological basis of parenting

Studies examining oxytocin and parenting have increased rapidly in recent years. Animal studies suggest that oxytocin production is associated with maternal behavior and bonding (Dwyer, 2008; Febo, Numan, & Ferris, 2005), and rats who have been administered oxytocin provide more maternal care to their offspring (Pedersen & Boccia, 2002). In humans, oxytocin levels increase during pregnancy, and oxytocin facilitates mother-child bonding and reduces maternal stress reactivity (Galbally, Lewis, van IJzendoorn, & Permezel, 2011). The increase in oxytocin just prior to birth is thought to prime maternal care (Flinn, Ward, & Noone, 2015).

Oxytocin and maternal behavior are related during infancy and early childhood, yet associations are complex. During mother-child interaction, maternal salivary oxytocin increases, but only when mothers provide affectionate physical contact (Feldman, Gordon, Schneiderman, Weisman, & Zagoory-Sharon, 2010). Likewise, plasma and salivary oxytocin levels are both higher among parents who are more socially engaged, and have more synchronous and positive interactions with their infants (Feldman, Gordon, & Zagoory-Sharon, 2011). Relative to insecure-dismissing mothers, mothers with secure attachment styles show elevations in serum oxytocin after playing with their infants, and show more activation of dopaminergic reward circuits in the brain in response to a picture of their infant smiling or sad (Strathearn, Fonagy, Amico, & Montague, 2009). When shown a picture of their infant with sad affect, insecure-dismissing mothers show less activation of reward circuits, and more activation of neural circuits associated with negative feelings. Thus, for mothers with secure attachments, but not those with insecure attachments, oxytocin may contribute to the experience of reward when mothers interact positively with their infants, which may facilitate mothers' ability to continue to provide nurturing care (Galbally et al., 2011).

Some human studies utilize intranasal oxytocin administration to test the effect of oxytocin on behavior. Intranasal oxytocin administration is associated with better recognition of facial expressions of emotions, and increased trust to one's in-group (Graustella & MacLeod,

2012; Van IJzendoorn & Bakermans-Kranenburg, 2012). Oxytocin may prime parents for positive parenting beyond infancy, yet results are somewhat mixed. When oxytocin is produced, low-risk mothers are typically in a more affiliative state and more attuned to facial expressions of emotions, which is hypothesized to make warm, sensitive caregiving more likely. Yet, associations between oxytocin and positive behavior may be attenuated or even reverse direction among high risk groups, as early aberrant social experiences have been associated with altered oxytocin activity throughout the lifespan (Bartz et al., 2010, 2011; Wismer Fries et al., 2005). Further, a study of oxytocin administration in postnatally depressed mothers found that oxytocin administration was associated with increased reports of positive relationships with their infant, but also increased negative affect and views of the infant as difficult (Mah, Van IJzendoorn, Smith, & Bakermans-Kranenburg, 2013). Thus, researchers should consider under what circumstances oxytocin promotes prosocial behavior and positive parenting; mothers' childhood experiences have been identified as a potentially important moderating factor (Bakermans-Kranenburg, van IJzendoorn, Riem, Tops, & Alink, 2012; Huffmeijer et al., 2013).

Trauma and Parenting

Mothers who have experienced abuse, neglect, or other forms of childhood stress are at higher risk for poor parenting (Knutson, 1995; Maestripieri, 2005; Patterson, 1998), tend to be more intrusive, engage in more negative parenting strategies, and appear disengaged when interacting with their infant (Banyard, 1997; Lyons-Ruth & Block, 1996; Moehler, Biringen, & Poustka, 2007). Trauma may enact effects on parenting through modeling of harsh parenting practices (Serbin & Karp, 2003; Van Ijzendoorn, 1992) and increased psychopathology risks (Enns, Cox, & Clara, 2002), which are associated with negative parenting (Zahn-Waxler, Duggal, & Gruber, 2002). Animal studies have demonstrated that quality of early mothering affects the development of endocrine systems (Champagne & Meaney, 2001) and brain structures that are associated with later expressed maternal behavior (Barrett & Fleming, 2011). In human studies as well, early adversity has been shown to alter both biology (e.g., cortisol, adrenocorticotrophic hormone) and parenting behavior (Barrett & Fleming, 2011). Yet, it is not known how biology, particularly oxytocin biology, may relate to parenting under conditions of high and low levels of early-life stress.

The Current Study

We examined the relation between oxytocin and parenting in a naturalistic setting among low-income mothers with varying levels of early stress. Prior research has shown that while oxytocin is typically associated with positive affect and behavior among low-risk samples, oxytocin may be associated with more negative affect and behavior among higher risk samples such as anxiously attached individuals (Bartz et al., 2010), individuals with borderline personality disorder (Bartz, Simeon, et al., 2011), and mothers with postnatal depression (Mah et al., 2013). Thus, it was hypothesized that higher oxytocin production would be associated with more positive parenting and less negative parenting in mothers with lower levels of early life stress, but that the reverse would be true among mothers with higher levels of early life stress.

Methods

Participants

Participants were a subset of families who had participated in a longitudinal study of child self-regulation and eating behavior (Miller, Rosenblum, Retzliff, & Lumeng, 2016). Families were low-income at original study enrollment, and were recruited from Women, Infants and Children (WIC) clinics and Early Head Start programs. Inclusion criteria for the original study were that the child was born at 35 weeks of gestation without significant complications at birth; had no history of food allergies, serious medical problems, or significant developmental delays; both mother and child were fluent in English; child was in custody of the biological mother; mother was at least 18 years old at recruitment and had less than a 4-year college degree; and family income was <185% of the federal poverty line. Families who had completed the final wave of the original study were recruited to participate in the current study. Additional eligibility criteria for this study were that the mother was not pregnant or nursing, as this can affect oxytocin production (Levine, Zagoory-Sharon, Feldman, & Weller, 2007; Matthiesen, Ransjo-Arvidson, Nissen, & Uvnas-Moberg, 2001).

Forty participants completed the study protocol (December 2012-May 2013). Five participants were excluded from analyses due to missing data. Two sets of twins were in the dataset; the first twin to complete the protocol was included in analyses. The resulting sample consisted of 33 mother-child dyads; this sample did not differ from the sample excluded with regard to maternal age, race/ethnicity, educational attainment, income-to-needs ratio, or child age, race or sex ($ps > .05$).

Participants were children aged 34 to 48 months ($M = 39.21$, $SD = 3.37$; 48.5% male; 48.5% non-Hispanic white) and their mothers (aged 21 to 38 years; $M = 27.18$, $SD = 4.60$). At enrollment, the average income-to-needs ratio was below the poverty line ($M = 0.87$, $SD = 0.65$), and the mean annual income was \$20,148 ($SD = \$13,012$). About half (48.5%) of mothers had more than a high school education.

Procedure & Measures

This study was approved by the [redacted for blind review] Institutional Review Board (protocol number [redacted for blind review]). Mothers provided written informed consent. Families were compensated \$50 for participating. During a home visit, mother-child dyads participated in an interactive task during which saliva was sampled, then engaged in a 5-minute videotaped free play session, and then interviewers administered questionnaires to mothers. The interactive task consisted of storybook reading and games that were designed to promote positive mother-child interactions (20 minutes). Mother-child dyads were provided age-appropriate toys (e.g., plastic animals) for the free-play, and these videotapes were later coded for observed parenting.

Predictor: Oxytocin—Oxytocin was measured in saliva; salivary oxytocin correlates with serum values (Carter et al., 2007; Feldman et al., 2010). Saliva was collected by having mothers chew on a sorbette until it was saturated. No stimulants were used. Samples were gathered prior to the interactive task (after mothers and children had not interacted for at

least 30 minutes) and 20 minutes after the end of the task, following others' similar behavioral paradigms to assess salivary oxytocin (e.g., Feldman et al., 2010).

Visits occurred in the afternoon to avoid interference with diurnal pattern of oxytocin that typically peaks in the morning and then declines across the day (Amico, Levin, & Cameron, 2008; Forsling, Montgomery, Halpin, Windle, & Treacher, 1998). Participants were asked to refrain from eating for at least 2 hours prior to the visit. We queried mothers at each saliva collection regarding factors that could influence oxytocin (e.g. medications, sleep, eating; Gunnar & Vazquez, 2001).

Once collected, samples were immediately placed on ice and refrigerated in a portable cooler. Upon arrival at the lab, they were centrifuged and stored at -20°C until assayed. Salivary oxytocin was determined utilizing a high sensitivity ELISA kit (Enzo Life Science™ RIA Kits, formally Assay Design, Ann Arbor, MI). The detection limit of this assay is 11.7 pg/mL, with little to no cross-reactivity with vasopressin. The intra^(a) and inter assay^(b) coefficients of variation vary with the concentration of oxytocin as follows: Low 9.1%^a, 14.5%^b; Medium 8.7%^a, 8.7%^b; High 12.4%^a, 5.2%^b. This study utilized a variable representing the total amount of oxytocin secreted during the assessment (in pg*minutes/mL), specifically area under the curve (AUC). The pre-assessment oxytocin level was used entered into the first step of regression analyses to control for baseline levels of oxytocin.

Predictor: Maternal Adverse Childhood Experiences—The *Adverse Childhood Experiences Scale* (ACES; Felitti et al., 1998) was used to assess mother's early life stress. Mothers responded yes or no to a series of questions about experiences prior to age 18 that covered physical and emotional abuse and neglect, domestic violence, household substance abuse, household mental illness, parental separation or divorce, and incarceration (9 items; the sexual abuse item was not administered). Responses were summed to create a total score ($\alpha=.82$) with higher values indicating more ACEs. For some analyses, this variable was dichotomized into high (3+ ACEs endorsed; $N=15$) vs. low (0 to 2 ACEs endorsed; $N=18$) approximately equally sized groups.

Outcomes: Maternal Affect and Behavior—Maternal affect and behavior were coded from videotapes of the free play using a modified version of the Maternal Warmth and Control Scale (Booth, Rose-Krasnor, McKinnon, & Rubin, 1994). Videos were coded in 10-second intervals for: *Positive Affect* (e.g., pleasant, joyful; 0=none, 1=moderate positivity, 2=outright positivity), *Negative Affect* (e.g., sad, anxious; 0-2), *Negative Control* (e.g., intrusive, ill-timed behaviors; 0-2), *Sensitivity & Guidance* (e.g., supportive, well-timed behavior; -1=miss or inappropriate response, 0=none, 1=minimal, 2=extended) and *Hostile Affect* (e.g., anger, irritability; 0-2). These variables were averaged to create *Positive Parenting* (Positive Affect, Sensitivity & Guidance) and *Negative Parenting* (Negative Affect, Negative Control, Hostile Affect) composite variables.

Possible Covariates—Mothers provided demographic information and indicated time since waking, eating, or taking any medications/caffeine or hormonal birth control, date of

last menses, and whether they were healthy or had a cold or fever, as these could influence oxytocin production (Gunnar & Vazquez, 2001).

Results

Preliminary Analyses

Correlations revealed that maternal oxytocin AUC was not associated with mothers' medications, sleep, or eating (all $ps > .05$). There were no associations between oxytocin AUC and demographic variables (race/ethnicity, maternal education, maternal age, income-to-needs ratio; all $ps > .05$). Thus, these variables were not included as covariates in the regression models.

Main Analyses

Results are presented for effects that attain at least a “small” effect size. Effect sizes were considered small, medium, or large if they surpassed the cutoffs of .02, .13, and .26 for R^2 , or .10, .30, and .50 for r (Cohen, 1992).

Correlations—In the full sample, maternal oxytocin AUC was positively associated with pre-assessment maternal oxytocin, $r = .92$, and negatively associated with observed negative parenting, $r = -.30$. Pre-assessment maternal oxytocin was positively associated with Total ACEs, $r = .10$ and observed positive parenting, $r = .10$, and negatively associated with observed negative parenting, $r = -.29$. There was a negative association between observed positive parenting and observed negative parenting, $r = -.19$.

Correlations with Maternal Adversity Groups—When correlations were examined separately in the low (0-2) vs. high (3+) ACEs subsamples, observed positive parenting was positively associated with maternal oxytocin AUC, $r = .41$, and maternal pre-assessment oxytocin, $r = .53$, among mothers with low ACE scores. The association was of a similar magnitude but inversely associated among mothers with high ACE scores, $rs = -.33, -.41$. The association between observed negative parenting and maternal oxytocin AUC or maternal pre-assessment oxytocin was negative in both the low ACE, $rs = -.34, -.34$, and high ACE $rs = -.29, -.26$, subsamples. Among mothers with high ACE scores, but not those with low ACE scores, there was also a negative association between observed positive parenting and observed negative parenting, $r = -.31$.

Regressions—Linear regression analyses were conducted to test how oxytocin AUC and ACEs related to observed parenting. Pre-assessment maternal oxytocin was entered in Step 1, and maternal oxytocin AUC and Total ACEs were entered in Step 2. The oxytocin AUC and ACEs interaction was added in Step 3. The final model (Table 2), though nonsignificant, $F(4,32) = 1.28, p = .30$, explained a medium effect size of variance, $R^2 = .15$, with most of the variance attributable to the maternal oxytocin AUC \times ACEs interaction, $R^2 = .14$. The interaction effect was statistically significant, $\beta = -.66, p = .04, sr = -.38$, and suggests a positive association between positive parenting and oxytocin production among mothers with low ACEs, but a negative association for mothers with high ACEs (Figure 1). A parallel

regression run with negative parenting as the outcome variable was nonsignificant for all variables (Table 2).

Discussion

This study examined the relation between oxytocin and observed parenting in a naturalistic setting in low-income mothers with varying levels of early adversity. While the sample size is small, and only some effects attained statistical significance, effect sizes were in the small to medium range. The primary finding of this study was that the effect of maternal oxytocin on positive parenting, differs based on maternal early adversity. For mothers with low ACEs, oxytocin related to positive parenting in the expected manner—higher oxytocin was associated with more positive parenting. For mothers with high ACEs, however, higher oxytocin was associated with lower levels of positive parenting. While there was a direct negative association between maternal oxytocin AUC and observed negative parenting, this association was no longer detected in the full regression model.

Our results suggest that the expected connection between oxytocin and positive parenting behavior is disrupted among mothers who have had high levels of early adversity. These findings are in line with previous work that has found that oxytocin may not function as expected in individuals who had harsh early experiences (Bartz et al., 2010; Bartz, Simeon, et al., 2011). Oxytocin administration can increase social awareness and perception (Graustella & MacLeod, 2012), has also been shown to increase perceived social stress in men (Eckstein et al., 2014), and has been associated with negative affect in depressed mothers (Mah et al., 2013). Thus, it is possible that the inconsistencies in prior research on oxytocin (Bartz, Zaki, Bolger, & Ochsner, 2011; Graustella & MacLeod, 2012) are in part due to studies not taking into account an individual's social context, particularly for individuals who have experienced early-life adversity.

Mothers with low levels of early adversity may get a “boost” from their biology that helps them to exhibit positive parenting behaviors. Consistent with intranasal oxytocin studies of (low risk) individuals' behaviors (Graustella & MacLeod, 2012; Van IJzendoorn & BakermansKranenburg, 2012), close, affectionate interaction with their children is associated with higher oxytocin levels, which is then associated with more positive parenting behaviors. For mothers with high levels of early adversity, oxytocin levels are associated with lower levels of positive parenting. These mothers may have to work against their biology to provide positive parenting, thus, it may be more difficult for high-risk mothers to show positive parenting behaviors.

A possible explanation for oxytocin by ACEs interaction lies in the association between oxytocin and vasopressin, a related hormone that is linked to anxiety and defense behaviors. A large increase in oxytocin may lead to occupation of vasopressin receptors, producing defensive behaviors typically associated with vasopressin release (Carter, 2014), which could contribute to the current findings for mothers with high ACEs. Similar paradoxical effects of oxytocin have been reported in other studies of at-risk mothers; in mothers with postnatal depression, oxytocin administration is associated with increased negative affect, presumably because oxytocin can make an individual's emotional state (in this case

depressed) more salient (Mah et al., 2013). The early social context has been associated with individual differences in oxytocin release and its role in mediating or buffering distress (Bartz, Zaki, et al., 2011; Neumann, 2007; Uvnäs-Moberg, 1998; Wismer Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005; Zheng et al., 2010). As a result, oxytocin may be operating differently for mothers who experienced harsh early social environments, perhaps promoting more defensive behaviors and harsh parenting than anxiolytic and prosocial behaviors.

Recently, oxytocin has received attention as a potential target for intervention. Studies have used intranasal oxytocin to improve emotion recognition in individuals with autism spectrum disorders (Guastella et al., 2010), improve higher-level social cognition in individuals with schizophrenia (Davis et al., 2013), improve trust toward one's ingroup (Van IJzendoorn & Bakermans-Kranenburg, 2012), and improve positive communication during couple conflict (Ditzen et al., 2009). Yet, our findings suggest that oxytocin may not operate as expected in individuals with high levels of early adversity. Oxytocin interventions should therefore carefully consider individuals' social context, as effects of oxytocin may differ among different subgroups (Graustella & MacLeod, 2012). Future work might also consider exploring whether the negative relationship between oxytocin and positive parenting among high-risk parents might be ameliorated through trauma-informed interventions that target early parent-child relationships (e.g., Child-Parent Psychotherapy).

Strengths and Limitations

This study is limited by its small sample size, so findings will need to be replicated with larger samples. Although a strength of the study is the low-income sample, which is uncommon in existing studies of oxytocin and parenting, findings may not generalize to higher-income samples. Other strengths include use of observational and biological data in a naturalistic home setting, which builds upon extant literature that primarily uses lab-based protocols. Thus, despite the small sample size, this study expands our knowledge about how oxytocin relates to parenting in high-risk populations, and suggests that maternal early adversity may affect the association between oxytocin and parenting.

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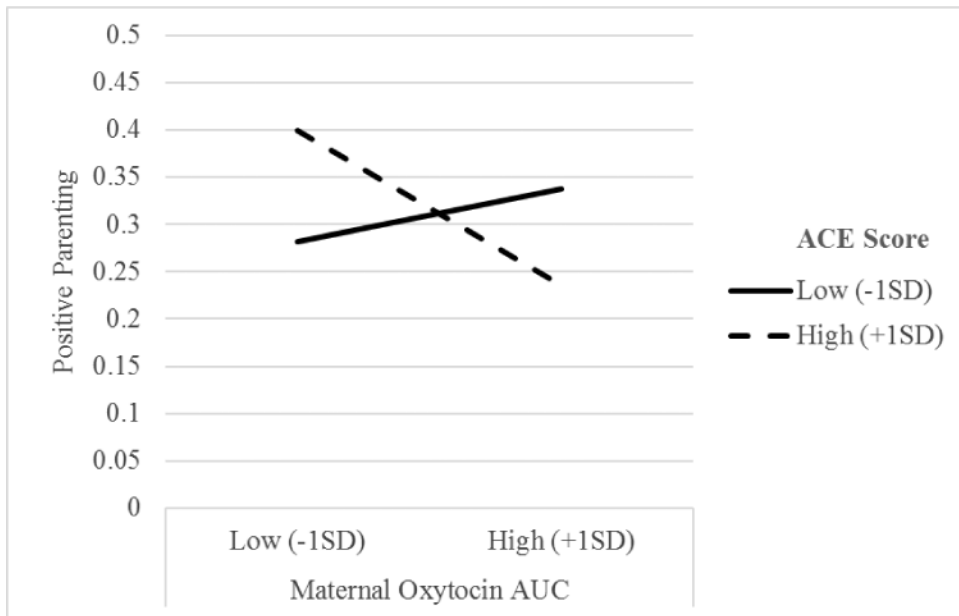


Figure 1. Positive Parenting as a function of maternal oxytocin production and mothers' adverse childhood experiences, controlling for pre-test maternal oxytocin level

Table 1

Descriptive statistics and correlations of maternal oxytocin AUC (pg/mL), pre-assessment maternal oxytocin (pg/mL), ACES total score, CES-D total score, and observed parenting (N = 33)

	1	2	3	4	M (SD)
1. Maternal Oxytocin AUC					325,306.68 (145,570.51)
2. Pre-Assessment Maternal Oxytocin	<u>.92*</u>				100.89 (49.55)
3. Total ACEs	.05	.10			2.88 (2.41)
4. Observed Positive Parenting	.09	.10	.04		.31 (.12)
5. Observed Negative Parenting	-.30⁺	-.29	.07	-.19	.02 (.03)

* $p < .05$

⁺ $p < .10$

Small effect sizes ($r = .10$) are bolded. Medium effect sizes ($r = .30$) are bolded and underlined. Large effect sizes are bolded, underlined, and italicized.

Hierarchical regressions of observed parenting behavior on pre-assessment maternal oxytocin, maternal oxytocin AUC, ACEs, and maternal oxytocin AUC × ACEs interaction.

Table 2

	Positive Parenting				Negative Parenting			
	<i>F</i>	<i>R</i> ²	<i>β</i>	<i>sr</i>	<i>F</i>	<i>R</i> ²	<i>β</i>	<i>sr</i>
Model 1								
Pre-Assessment Maternal Oxytocin	0.29	.01	.01	.10	2.82	.08	.08	-.29
Model 2								
Pre-Assessment Maternal Oxytocin	0.10	.01	.01	.03	1.07	.10	.02	-.10
Maternal Oxytocin AUC			.01	.00			-.22	-.08
Total ACEs			.03	-.03			.09	.09
Model 3								
Pre-Assessment Maternal Oxytocin	1.28	.15	.14	.11	0.78	.10	.00	-.12
Maternal Oxytocin AUC			.34	.12			-.25	-.09
Total ACEs			.07	.07			.08	.08
Maternal Oxytocin AUC × Total ACEs			-.66*	-.38			.06	.03

* $p < .05$, *sr* = semipartial *r*

Small effect sizes (R^2 .02, *sr* .10) are bolded. Medium effect sizes (R^2 .13, *sr* .30) are bolded and underlined.