Received: 13 June 2016 | Revised: 21 December 2016 | Accepted: 22 May 2017
DOI: 10.1002/dev.21537

RESEARCH ARTICLE

WILEY Developmental Psychobiology

HPA axis linkage in parent–child dyads: Effects of parent sex, autism spectrum diagnosis, and dyadic relationship behavior

Darby E. Saxbe[1] | Ofer Golan[2] | Sharon Ostfeld-Etzion[2] | Yael Hirschler-Guttenberg[2] | Orna Zagoory-Sharon[3] | Ruth Feldman[2,3]

[1] Department of Psychology, University of Southern California, Los Angeles, CA

[2] Department of Psychology, Bar-Ilan University, Ramat Gan, Israel

[3] Gonda Brain Sciences Center, Bar-Ilan University, Ramat Gan, Israel

Correspondence
Darby Saxbe, Department of Psychology, University of Southern California, 3620 McClintock Ave, Los Angeles, CA 90089.
Email: dsaxbe@usc.edu

Abstract

Families of preschoolers participated in two dyadic home visits, once with mother (56 dyads) and once with father (59 dyads). Each member of the dyad provided three cortisol samples and participated in several interaction tasks that were behaviorally coded. Approximately half of the children had been diagnosed with autism spectrum disorders (ASD), whereas half were typically developing (TD).

In a multilevel model, father's cortisol level at each timepoint predicted child cortisol. Father–child linkage was stronger in dyads that showed less reciprocity, in which fathers showed less sensitivity, and in which children showed less self-regulation and more withdrawal. Cortisol levels were not significantly correlated in mother–child dyads, and there was a trend toward moderation by ASD diagnosis, such that linkage was greater in TD children. Mother–child linkage was stronger in dyads that showed less behavioral coordination and less sensitivity. HPA axis linkage may be stronger in less behaviorally attuned dyads.

KEYWORDS
autism spectrum disorders, cortisol, HPA axis, parent–child, parenting, stress

1 | INTRODUCTION

The "affectional tie" (Ainsworth, 1969) that connects parent and child is shaped and strengthened by biobehavioral synchrony (Feldman, 2007a), or the coordination of behavior, emotion, and physiology between child and caregiver. In early infancy, this synchrony unfolds through simple touch, vocalization, and shared gaze; in later childhood, parents and children participate in more complex joint interactions. In both cases, sensitive parents attune and adapt to their child's arousal and emotional state, while children seek out input from parents and coordinate their attention and behavior accordingly. The reciprocal, patterned exchanges that mark parent–child interactions help to provide scaffolding for the child's own developing self-regulatory systems. Synchrony in early childhood may form a foundation for many of the processes that underlie sophisticated social organization, such as empathy, collaboration, and contingency detection (Feldman, 2015).

Biobehavioral synchrony implies not only mirroring or matching but the balancing or modulating of the child's emotional and physiological arousal. When a child becomes distressed, an effective caregiver does not burst into tears, but instead adopts a soothing demeanor. Similarly, when a dyadic interchange become over-stimulating, a child may briefly avert her gaze and turn her head; a sensitive parent might shift to a quieter, more calming tone. As such, parent–child interactions may involve oscillation and continual dyadic adjustment around an optimal set-point. This recalls social baseline theory (Beckes & Coan, 2011), which argues that social groups can maintain a more energy-efficient homeostasis by jointly regulating emotion, distributing risk, and sharing goals in order to converge around a calm baseline marked by a decreased need for vigilance to threat.

Given this, biobehavioral synchrony may flourish within conditions of "low intensity" (Feldman, 2015; Schneirla, 1946) in which synchronous interactions feature a calm, neutral, or positive tone. Such interactions may lay the groundwork for infants to experience positive emotion, which are typically first expressed in social contexts (e.g., social smiling, laughing). Positive interchanges may also support the release of affiliative neuropeptides such as oxytocin and prolactin which facilitate bonding and nurturant activities (breastfeeding, touch) and may provide a biological foundation for attachment. In contrast, conditions of threat and stress may disrupt synchrony and threaten bonding. At the hormonal level, the stress hormone cortisol and oxytocin appear to have suppressive effects on each other (Ditzen et al., 2009; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). Consistent with this, close relationships appear to have "social buffering" effects on the stress response; parent–child attachment relationships, in particular, act as "hidden regulators" of child behavior and physiology and help to mitigate distress (Hofer, 1984; Hostinar, Sullivan, & Gunnar, 2014).

A growing literature has found evidence for HPA axis (cortisol) linkage or covariation in dyads including parent–child pairs (Granger, 1998; Hibel, Granger, Blair, & Cox, 2009; Hibel, Granger, Blair, & Finegood, 2015; Papp, Pendry, & Adam, 2009; Spratt et al., 2016) and adult couples (Liu, Rovine, Cousino Klein, & Almeida, 2013; Papp, Pendry, Simon, & Adam, 2013; Saxbe & Repetti, 2010; Saxbe, Negriff, Susman, & Trickett, 2015). However, given that cortisol is a stress hormone that is most reliably activated by social evaluative threat (Dickerson & Kemeny, 2004), it is unclear whether cortisol linkage reflects a form of beneficial biobehavioral synchrony or, alternatively, reflects poor, asynchronous dyadic functioning. The research on parent–child HPA linkage has been mixed to date, with some studies suggesting that linkage is bolstered under conditions of parental sensitivity, as well as proximity and closeness. Hibel et al. (2015) found that parent–child linkage during a stress task was greater when mothers showed more sensitivity and positive engagement, whereas linkage was disrupted by child distress. Consistent with this, several studies (Atkinson et al., 2013; Sethre-Hofstad, Stansbury, & Rice, 2002; van Ijzendoorn et al., 2007) found that more sensitive parents showed stronger correlations in cortisol with children participating in stress and challenge paradigms (such as a balance-beam task, a robot-stranger task, a task in which a desirable toy was removed, and a Strange Situation task). However, other studies have reported greater attunement under conditions of stress or adversity; Laurent, Ablow, and Measelle (2011) found linkage in a sample of depressed mothers and their children, and Hibel and co-workers (2009) found stronger cortisol attunement in mother–child dyads with a history of intimate partner violence and punitive parenting. In a study that measured diurnal cortisol patterns in mothers and their 6-year-old children over 2 days, Pratt et al. (under review) found stronger mother–child cortisol linkage in dyads with greater maternal parenting stress, lower mother–child behavioral reciprocity, and greater father–child intrusiveness. Moreover, maternal depression was correlated with lower behavioral reciprocity. Papp et al. (2009) found that time together, shared activities, and parental monitoring bolstered mother–adolescent cortisol synchrony over several days, but so did elevations in both maternal negative affect and adolescent negative affect.

Additional evidence regarding the implications of physiological linkage for dyadic relationship quality comes from research on couples. Within this literature, linkage in cortisol has been most consistently associated with relationship dysfunction (Timmons, Margolin, & Saxbe, 2015). For example, positive associations in cortisol have been linked with relationship distress (Liu et al., 2013; Saxbe & Repetti, 2010), intimate partner aggression (Saxbe et al., 2015), and decreased empathy and increased risk of relationship dissolution (Schneiderman, Kanat-Maymon, Ebstein, & Feldman, 2014). Levenson and Gottman (1983) have suggested that more strongly linked couples engage in negative affect reciprocity, in which partners become locked in escalating exchanges of negative emotion and stress. In other words, rather than modulating each others' negative emotions or stress states, dyads that show stronger linkage may be more reactive to each other and show stress contagion rather than stress buffering. The current study aims to test this theory of stress contagion within a sample of parent–child dyads.

The literature on parent–child HPA axis linkage has almost exclusively focused on mother–child pairs: all of the above-cited studies recruited mothers only (with the exception of van Bakel and Riksen-Walraven (2008), whose sample included 81 mothers and 2 fathers). Given that children share the same physiological environment as their mothers during pregnancy, and that maternal cortisol appears to influence early child development (Davis & Sandman, 2010), HPA axis linkage might be expected to be stronger in mother–child vs. father–child dyads. Indeed, in a sample of families with 6-month-old infants, Stenius and co-workers (2008) found that mother and infant cortisol were strongly correlated in the morning, afternoon, and evening, whereas father and infant cortisol were more weakly correlated only at the afternoon and evening sampling timepoints. However, this study focused on single-timepoint correlations rather than patterns of cortisol over time, did not include a parent–child interaction paradigm, and did not examine how characteristics of the parents or children moderated the degree of cortisol linkage. In a study of family triads including older children that did include a laboratory-based interaction, Saxbe et al. (2014) found cross-sectional linkage between the father, mother, and child. In time-lagged analyses, only fathers uniquely predicted child cortisol, suggesting that fathers had stronger influence on child cortisol than mothers. Given that only two studies have compared father–child and mother–child HPA axis linkage, more investigation is warranted.

To date, studies of parent–child HPA axis linkage have focused on neurotypical children. However, children with developmental disabilities may show different behavioral and physiological responses when interacting with parents. Autism spectrum disorder (ASD) is a neurodevelopmental condition characterized by difficulties with communication and with social relationships. Compared with typically developing (TD) children, children with autism appear to show less coordination of gaze and attention with their caregivers and make fewer nonverbal gestures. Interestingly, although children with ASD are often less involved and less responsive in parent–child interactions,

several studies have reported that parents show similar degrees of sensitivity to TD children and children with ASD (e.g., van Ijzendoorn et al., 2007), suggesting that parents of children with ASD may "work harder" and show more compensatory behaviors to maintain a connection with their children. Consistent with this, another study found that parents of children with ASD, compared to parents of TD children, showed more physical, high-intensity approach behaviors during parent–child interactions (Doussard-Roosevelt, Joe, Bazhenova, & Porges, 2003). In terms of HPA axis functioning, some studies have reported higher cortisol reactivity to stress in children with ASD versus typically developing (TD) children (Spratt et al., 2012), while others have reported greater variability in diurnal cortisol (Carmean, 2008), and still others have found similar patterns of cortisol responses in children with ASD and TD children (Ostfeld-Etzion, Golan, Hirschler-Guttenberg, Zagoory-Sharon, & Feldman, 2015). Given that ASD may affect attachment and social interactions with parents, it may also be associated with parent–child cortisol linkage, but this possibility has not yet been tested.

The current study focused on the families of young children who engaged in both mother–child and father–child home visits that included several behaviorally coded interactions. Half of these children had been diagnosed with ASD. Therefore, we were able to build on the preexisting literature by examining parent–child linkage in both mothers and fathers, and ASD and TD children. The fact that children participated in the home visit protocol twice, once with the father and once with the mother, offers the opportunity to examine how parent–child cortisol linkage differs and stays the same within different dyadic configurations. Understanding parent–child linkage has clinical relevance because stress contagion processes within families may affect physical health, given that HPA axis dysregulation can compromise metabolism, immunity, sleep, and inflammation (McEwen, 1998). Moreoever, understanding how physiological interconnectedness may differ between mother–child and father–child dyads sheds light on the unique contribution of fathers to family systems and may have implications for family-level interventions. Finally, as the first study to compare parent–child cortisol linkage in dyads in which children did and did not have an ASD linkage, this study may inform intervention and theory development in the field of ASD research.

We investigated three research questions:

1) Does parent–child linkage in cortisol emerge during the home visits? Given above-cited evidence that HPA axis linkage has been found in a number of parent–child studies with a wide range of child ages, we expect parent cortisol to positively predict child cortisol at the same timepoint when sampling time is controlled.
2) Are these effects moderated by ASD diagnosis, parent sex, or behavioral interaction during the parent–child visit? Although we expect that each of these moderators may influence HPA axis linkage, we do not present directional hypotheses due to a lack of prior evidence. To date, no studies have compared parent–child HPA axis linkage among children with ASD to typically developing children. Only two studies have directly compared father–child and mother–child linkage, and of these, one study found stronger mother–child linkage (Stenius et al., 2008) and one study found stronger father–child linkage (Saxbe et al., 2014). Studies that have examined moderators of dyadic linkage (both in parent–child and in couple dyads) have also found mixed results, with evidence of stronger linkage in both better-functioning and worse-functioning dyads.
3) Do children show stability across visits? In other words, if a child has high cortisol and/or high linkage with the parent in the father–child visit, does that predict higher cortisol or higher linkage in the mother–child visit? This question may help clarify the extent to which cortisol patterns and parent–child linkage are trait—rather than state—dependent. Given the lack of prior research testing this question, we do not have a directional hypothesis.

2 | METHODS

2.1 | Participants

Eighty families of mothers, fathers, and their preschool-aged child participated in two groups. The *ASD group* included 40 preschoolers (five females) diagnosed with ASD by trained clinicians according to DSM-IV-TR criteria (APA, 2003) and their parents. Families were recruited from psychiatric clinics and special-needs kindergartens in central Israel. Diagnosis was confirmed using the 2nd edition of the Autism Diagnostic Observation Schedule (ADOS 2; Gotham, Risi, Pickles, & Lord, 2007). One child failed to meet ASD criteria and was excluded. The *Typically Developing group* included 40 preschoolers (six females) and their parents with no neuro-psychiatric disorders who matched the ASD group on mental age, child sex, and family demographics. TD participants were screened for ASD using the Childhood Autism Spectrum Test (CAST; Scott, Baron-Cohen, Bolton, & Brayne, 2002). Because emotion regulation abilities are sensitive to developmental stage, children were matched on mental age consistent with prior research (e.g., Jahromi, Meek, & Ober-Reynolds, 2012). Groups were matched on raw scores of the *Stanford-Binet Intelligence Test* (Thorndike, Hagen, & Sattler, 1986), a standardized test assessing IQ in children aged 2 years and above. The study was approved by the Institutional Review Board and all parents signed informed consent.

2.2 | Procedure

2.2.1 | Diagnostic and cognitive assessment

During a visit to kindergarten, children were tested with the Stanford-Binet Intelligence test and those with ASD were administered the ADOS by trained psychologists.

2.2.2 | Home visits

Two identical home visits were conducted within the same month with mother or father (counterbalanced), each lasting approximately 2 h,

conducted at the same time and by the same research assistants, and including cortisol collection, parent–child interactions, several ER procedures, and self-reports. Several parent–child interaction protocols were adapted from the widely used Lab-TAB (Laboratory Temperament Assessment Battery), Goldsmith and Rothbart (1999). These protocols have been used in numerous studies of parent–child interaction with both neurotypical children and children with behavior problems (e.g., Bridges, Palmer, Morales, Hurtado, & Tsai, 1993; Hane, Fox, Polak-Toste, Ghera, & Guner, 2006; Hane, Fox, Henderson, & Marshall, 2008).

2.3 | Behavioral measures

a) Parent–child free play: Parent and child engaged in a 7-min free play with preselected toys known to elicit symbolic play at this age (Feldman, 2007b). Instructions were "Play with your child as you typically do."
b) Emotion regulation: Mask condition (Goldsmith, 1996). Child and parent sat in front of experimenter who put on four masks of increasing fearfulness: rabbit, lion, alligator, and monster. Each mask was put on for 15 s.
c) Emotion regulation: Puppet condition (Goldsmith & Rothbart, 1999). Child and parent sat face-to-face, were given five colorful hand puppets, and were asked to play with the puppets for 5 min.

2.4 | Cortisol (CT) data

Three saliva samples were collected from parent and child in each home visit. Ten minutes after arrival at home and following child acquaintance with the RA, a baseline saliva sample was collected from each parent and each child by parent placing a Salivette (Sarstedt, Rommelsdorft, Germany) in their own and the child's mouth for one minute. Baseline cortisol was measured for all participants at 5 pm on school days. All children attended school on the day of the home visit, waking time at approximately 7 am. Children did not nap at noon in order to avoid circadian changes in cortisol levels. The second CT sample was collected 10 min after the end of the stressful ER paradigms (masks). The final CT sample was collected 10 min after the termination of the entire social battery. Six children with ASD and one TD child refused to place the Salivette in their mouth, so cortisol was not collected for these children for either the mother–child or father–child home visit. Cortisol from another eight home visits could not be analyzed due to technical problems. Finally, another 23 individual cortisol samples (5% of the total number of samples collected) were dropped due to insufficient quantities of saliva to analyze. In total, complete cortisol data was available for 56 dyads who participated in the mother–child visit and for 59 dyads who participated in the father–child visit.

Salivettes were kept cooled until thawed before being centrifuged at 4°C at 1000*g* for 15 min. The samples were then stored at −20°C until assayed. Cortisol levels were assayed using a commercial ELISA kit (Assay Desigs, Ann Arbor, MI). Measurements were performed in duplicates according to the kit's instructions and consistent with our prior research (e.g., Apter-Levy et al., 2016; Feldman, Vengrober, Eidelman-Rothman, & Zagoory-Sharon, 2013). CT levels were calculated by Matlab 7 according to relevant standard curves. The intra- and inter-assay coefficients are <10.5 and 13.4%.

2.5 | Behavioral coding

2.5.1 | Parent–child interactions

The Coding Interactive Behavior (CIB, Feldman, 1998) was used. The CIB includes 42 scales each rated from 1 (low) to 5 (high) that aggregate into several parent, child, and dyadic composites. The system has been used in many studies of typically developing and high-risk children and showed good psychometric properties for children at this age (Feldman, 2012). The following factors were used.

a) Parental sensitivity: Acknowledgment of child's signals, elaboration of communication, positive affect, appropriate vocal quality, resourcefulness in handling child's distress or expanding interaction, appropriate range of affect, supportive presence. *Mother alpha* = .83, *Father alpha* = .90.
b) Child involvement: Maintaining eye contact, joint attention, positive affect, affection to parent, alertness, social initiation, vocalizations, symbolic play. *Mother alpha* = .72, *Father alpha* = .72.
c) Child withdrawal: Child withdraws or disconnects, child avoids parent.
d) Dyadic reciprocity: Mutual adaptation to partner's state, give-and-receive reciprocity, fluent and rhythmic interactions. *Mother alpha* = .82, *Father alpha* = .82.
Two coders were trained to 90% reliability. Inter-rater reliability conducted for 20 interactions (25.3%), averaged *intraclass r* = .93 (range = .84–1.00).
e) Emotion regulation paradigms: Micro-coding of child and parent behavior was conducted on computerized system. Codes were based on ER research in preschoolers with ADS following extensive piloting. Each variable was computed as the sum proportions of time out of the entire episode this behavior was observed.
f) Child self-regulation: Included the following self-regulatory behaviors: withdrawal, physical self-soothing, verbal self-comfort, idiosyncratic behavior, substitutive play. These codes were recorded separately for the Masks condition and the Puppets condition.
g) Parent–child behavioral coordination: Included behaviors indicating that child seeks the parent in order to jointly regulate the negative or positive emotion. *Coordination during Masks* included child's social gaze to parent, physical proximity seeking, and engaging parent for distraction. *Coordination during Puppets* included social gaze to parent, interactive communications, and joint symbolic play.
Two coders blind to group membership coded each episode and trained to 90% reliability. Inter-rater reliability computed for 15

observations in each episodes averaged *kappa* = .91, .83 for masks and puppets respectively (range = .75–.94).

2.6 | Data analysis approach

Cortisol levels were checked for outliers and values >3SD were dropped. We elected to drop rather than winsorize outliers due to concerns that extreme values might reflect error. This resulted in 9 (5%) child with mother, 7 (4%) of child with father, 9 (5%) of mother, and 9 (5%) of father cortisol samples being dropped. Cortisol values were significantly positively skewed so natural log transformation was applied to the cleaned data. For the analyses using Empirical Bayes coefficients, we also dropped one multivariate outlier (Mahalanobis distance >3SDs from mean) from the mother–child visits. There were no multivariate outliers detected in the father–child visits.

Multilevel modeling (HLM 7.0; Raudenbush et al., 2011) was used to test change in cortisol levels and parent–child cortisol synchrony within each visit. This approach is well suited for data that have a nested structure, such as multiple cortisol sampling occasions nested within participants.

At Level 1, we included all of the cortisol data from the children and parents, along with a sample timepoint variable (coded as 1, 2, or 3 for the first, second, and third sample).

At Level 2, we included group (ASD vs. not ASD) and child sex.

We then ran a two-level HLM model predicting child cortisol from timepoint (1, 2, or 3) and from parent cortisol (at the same timepoint). Since they occurred in separate sessions, the mother–child visits and father–child visits were analyzed separately, in separate models.

Since the cortisol pattern might be expected to be non-linear (e.g., cortisol might rise and then fall given the laboratory protocol), we also tried adding a dummy variability coded 1 (peak cortisol, aka second sample) and 0 (first and third sample). However, this dummy variable was non-significant in both the mother–child and father–child models and did not significantly affect any of the key results reported here, so we dropped it from final analyses for parsimony.

3 | RESULTS

Figure 1 depicts average trajectories of cortisol across the visit for mothers, fathers, and children in each of the dyadic visits.

Before conducting HLM analyses, we ran independent samples *t*-tests to see whether the ASD and TD groups differed in cortisol or in behavior during the home visit. There were no differences by ASD versus TD group for either child or parent cortisol, with one exception (mothers of typically developing children had higher cortisol at the third sampling timepoint, $t(53) = 2.17$, $p = .035$). Consistent with the results reported by Ostfeld-Etzion et al. (2015), children with TD and children with ASD showed very similar behavioral responses to the home visit paradigm. Of the eight total behavioral codes for the mother–child visit, two significantly differed by TD/ASD group for the mother–child visits: children in the ASD group showed lower involvement ($t(51) = 2.76$, $p = .01$) and greater withdrawal

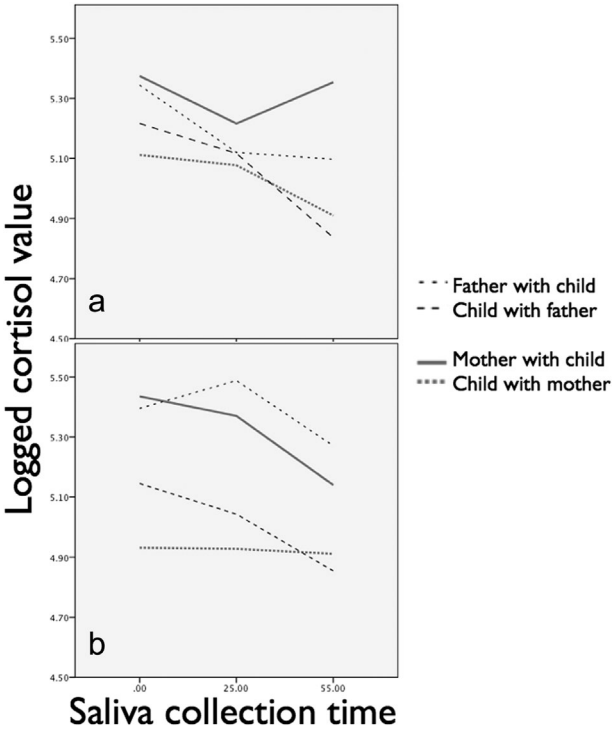

FIGURE 1 Average cortisol trajectories across the visit for father–child and mother–child dyads. Panel A shows typically developing children (TD group); Panel B shows children with autism spectrum diagnosis (ASD group)

($t(51) = -2.58$, $p = .01$). Of the eight behavioral codes for the father–child visit, four significantly differed by TD/ASD group: children in the ASD group showed greater self-regulation in the masks condition ($t(56) = -2.23$, $p = .03$); father–child dyads in the ASD group showed more positivity in the puppets condition ($t(56) = -2.44$, $p = .02$; children in the ASD group showed lower involvement ($t(55) = 3.07$, $p = .003$; and children in the ASD group showed greater withdrawal ($t(55) = -3.11$, $p = .003$.

Next, we ran zero-order correlations to examine correlations between parent and child cortisol during the home visit. In the father–child visit, father and child cortisol were positively correlated with each other at all three timepoints: at the first timepoint, $r(50) = .41$, $p = .003$; at the second timepoint, $r(51) = .39$, $p = .01$; and at the third timepoint ($r(55) = .26$, $p = .05$. Mother and child cortisol were positively correlated with each other at the second timepoint, $r(49) = .29$, $p = .05$, and, at a marginal level of significance, at the third timepoint, $r(48) = .28$, $p = .06$.

We also examined zero-order correlations between parent and child cortisol and behavior during the home visit. Child cortisol was not correlated with any of the behavioral codes, with one exception: in the mother–child visit, child withdrawal was positively associated with cortisol at the first timepoint, $r(49) = .29$, $p = .05$. In the father–child visit, father cortisol at the first timepoint was negatively correlated with child self-regulation in the masks condition, $r(60) = -.25$, $p = .05$, and positively correlated with positivity in the puppets condition, $r(60) = .28$, $p = .03$. In the mother–child visit, mother intrusiveness was

negatively associated with mother cortisol at the third timepoint, $r(54) = -.27$, $p = .05$. None of the other behavioral codes were associated with parent or child cortisol. Given that there were 16 behavioral codes and three cortisol timepoint measures for each parent and child, this rate of four significant associations is at chance-level, suggesting that parent–child behavior during the home visits was not reliably linked with cortisol.

3.1 | Multilevel modeling results

Next, we analyzed the data using multilevel modeling (HLM 6.0) in order to examine parent–child synchrony in cortisol within a model that included all three timepoints and that controlled for cortisol intercept and slope (change over the course of the visit). We also tested the behavioral codes as potential moderators of parent–child synchrony. Results are presented separately for each parent. All HLM analyses used child cortisol at the outcome variable as predicted by visit timepoint (reflecting sample 1, 2, or 3) and parent cortisol.

3.1.1 | Father–child visits

Fathers' cortisol significantly predicted child cortisol in the father–child visits, $b(169) = 0.24$, $t = 3.18$, $p = .002$ (note that the degrees of freedom reflect the total number of Level 1 units, e.g., the number of cortisol samples analyzed). Timepoint also predicted child cortisol, with a general decrease (as would be expected given the diurnal slope of cortisol): $b(169) = -.13$, $t = -3.92$, $p = .001$. These results remained significant, and coefficients were unchanged, when ASD group and child sex were added as Level 2 coefficients. Neither ASD group or child sex significantly moderated children's intercept of cortisol, slope by timepoint, or the influence of father cortisol on child cortisol (all p values >.50).

Next, we examined whether the behavioral codes moderated these results, with results shown in Table 1. To summarize the results, children who showed more *self-regulation* in the puppets condition had cortisol levels that were less correlated with fathers, whereas children who showed more *withdrawal* across the father–child visit had stronger correlations with fathers' cortisol, at a marginal level of significance. If the father showed more *sensitivity* or if the dyad was characterized by greater *reciprocity*, the child's cortisol was less correlated with the father. More withdrawn children also started with lower cortisol and had less of a drop in cortisol over the task, whereas children in dyads with more child self-regulation, dyadic reciprocity, and father sensitivity started with higher cortisol and (in the case of father sensitivity), had more of a drop in cortisol across the task. Child sex and ASD group did not moderate any of these results.

3.1.2 | Mother–child visits

Mothers' cortisol did not significantly predict child cortisol in the mother–child visits, $b(170)=.09$, $t = 0.86$, $p = .39$. Timepoint predicted child cortisol in the mother–child visits, with a general decrease similar in magnitude to the father–child visits ($b(170) = -.12$, $t = -3.68$, $p = .001$. These results were unchanged (timepoint was a significant predictor of child cortisol and mother cortisol was not) when ASD group and child sex were included as Level 2 coefficients. At a marginal level of significance, both ASD group and child sex moderated mother–child linkage, $b(164) = -.37$, $t = -1.78$, $p = .08$ for ASD group, such that typically developing children had stronger linkage with mothers; $b(164) = -.76$, $t = -1.83$, $p = .07$ for sex, such that boys had stronger linkage with mothers.

As shown in Table 2, children whose mothers showed more *behavioral coordination* in the puppets condition had weaker linkage with their mothers; at a marginal level of significance, maternal *sensitivity* during the home visit was also associated with weaker linkage. Children in dyads characterized by more behavioral coordination and more maternal sensitivity also had a higher starting value of child cortisol.

Given that ASD group was associated with mother–child linkage, we also tried splitting the sample and running separate analyses of children with ASD and TD children. For typically developing children, mother cortisol was positively associated with child cortisol at a marginal level of significance, $b(78) = .22$, $t = 1.83$, $p = .07$. For children with ASD, mother and child cortisol were not associated, $b(85) = -.05$, $t = -0.30$, $p = .77$.

3.2 | Analyses of aggregate parent–child synchrony coefficients

As a follow-up analysis, we extracted Empirical Bayes (EB) coefficients from both the mother–child and the father–child visits (reflecting the aggregate degree of parent–child linkage in cortisol over the entire visit). We then ran t-tests comparing these linkage coefficients by both ASD group and by child sex. For mother–child dyads, typically developing children showed significantly greater mother–child linkage during the home visit, $t(49) = 2.22$, $p = .03$. For father–child linkage, there was no significant difference by ASD group, $t(55) = 1.08$, $p = .28$.

Next, we examined whether EB coefficients extracted from the mother–child and father–child visits were correlated with each other. Children's total cortisol output, based on the EB intercept terms from the "unconditional model," was correlated across the two visits, $r(54) = .64$, $p = .001$. The parent–child linkage terms were correlated at a marginal level of significance, $r(49) = .24$, $p = .09$. In other words, children who had higher overall cortisol in the mother–child visit tended to have higher cortisol in the father–child visit as well, and children who showed stronger linkage with their mothers also tended to show stronger linkage with fathers.

Finally, we ran paired-samples t-tests in order to determine whether child cortisol or parent–child linkage differed across the mother–child and father–child visits. There were no significant differences in children's overall cortisol between the mother–child and the parent–child visit (t-test for EB intercept term: $r(53) = 0.25$, $p = .80$). However, children showed significantly stronger cortisol linkage with fathers than with mothers (t-test for EB linkage term: $r(49) = 5.55$, $p = .001$).

TABLE 1 Two-level model showing father cortisol as a predictor of child cortisol, with moderators: fixed effects with robust standard errors (n = 55 dyads)

	Model 1 child self-regulation			Model 2 dyadic reciprocity			Model 3 father sensitivity			Model 4 child withdrawal		
Fixed effects	**Estimate**	**(SE)**	***t* ratio**	**Estimate**	**SE**	***t* ratio**	**Estimate**	**SE**	***t* ratio**	**Estimate**	**SE**	***t* ratio**
Cortisol intercept	4.09	(0.35)	11.68***	4.09	(0.35)	11.68***	4.27	(0.38)	11.17***	4.03	(0.43)	9.43***
Level 2 covariates[a]												
ASD group	−0.15	(0.73)	−0.21	−0.15	(0.73)	−0.21	0.75	(0.81)	0.93	0.90	(0.97)	0.92
Child sex	1.03	(1.03)	1.00	1.03	(1.03)	1.00	−0.24	(0.93)	−0.26	−0.51	(1.10)	−0.47
Moderator	1.82	(0.49)	3.73***	1.82	(0.49)	3.73***	1.83	(0.59)	3.11**	−1.10	(0.52)	−2.12*
Cortisol slope by timepoint	−0.14	(.03)	−4.34***	−0.14	(0.03)	−4.34***	−0.14	(0.03)	4.69***	−0.12	(0.03)	−4.25***
Level 2 covariates												
ASD group	0.06	(.06)	0.93	0.06	(0.06)	0.93	0.04	(.06)	0.59	−0.002	(0.06)	−0.03
Child sex	−0.11	(.09)	−1.16	-0.11	(0.09)	0.25	−0.08	(.09)	−0.93	−0.09	(0.09)	−1.04
Moderator	−0.06	(.04)	−1.48	-0.06	(0.04)	−1.48	−0.08	(.04)	−2.15*	0.10	(0.04)	2.95**
Fathers' cortisol	0.23	(.06)	3.68***	0.23	(.06)	3.68**	0.20	(.07)	2.88**	0.24	(.08)	3.05**
Level 2 covariates												
ASD group	−0.01	(.13)	−0.06	-0.01	(.13)	−0.06	−0.18	(.14)	−1.25	−0.19	(.18)	−1.08
Child sex	−0.13	(.19)	−0.65	0.13	(.19)	−0.65	0.11	(.18)	−0.60	0.14	(.22)	0.66
Moderator	−0.31	(.09)	−3.47***	-0.31	(.09)	−3.47***	−0.32	(.11)	2.99**	0.17	(.09)	1.80+

+p < .10; *p < .05; **p < .01; ***p < .001.

[a]Level 2 covariates refer to effects tested on each of the bolded Level 1 indices.

TABLE 2 Two-level model showing mother cortisol as a predictor of child cortisol, with moderators: fixed effects with robust standard errors (n = 52 dyads)

	Model 1 behavioral coordination			Model 2 maternal censitivity		
Fixed effects	**Estimate**	**(SE)**	***t* ratio**	**Estimate**	**SE**	***t* ratio**
Cortisol intercept	5.16	(0.58)	8.96***	4.95	(0.56)	8.79***
Level 2 covariates[a]						
ASD group	0.80	(1.09)	−0.21	0.50	(1.09)	0.46
Child sex	4.16	(2.44)	1.70	4.28	(2.24)	1.91+
Moderator	0.07	(0.03)	2.50*	1.86	(0.87)	2.13*
Cortisol slope by timepoint	−0.12	(.03)	−3.41***	−0.11	(0.03)	−3.51***
Level 2 covariates						
ASD group	0.04	(.07)	0.53	0.07	(0.06)	1.29
Child sex	−0.09	(.14)	−0.66	−0.09	(0.13)	−0.72
Moderator	−0.01	(.01)	−0.94	−0.08	(0.05)	−1.76
Fathers' cortisol	0.01	(.10)	0.14	0.06	(.10)	0.60
Level 2 covariates						
ASD group	−0.19	(.20)	−0.94	−0.16	(.19)	−0.82
Child sex	−0.78	(.42)	−1.84+	−0.80	(.39)	−2.08*
Moderator	−0.01	(.01)	−2.61**	−0.29	(0.16)	−1.84+

+p < .10; *p < .05; **p < .01; ***p < .001.

[a]Level 2 covariates refer to effects tested on each of the bolded Level 1 indices.

4 | DISCUSSION

This study examined linkage in cortisol among parent–child dyads participating in a home visit. The fact that the same children did the same home visit protocol twice, once with their father and once with their mother, offers an unusual opportunity to explore patterns of HPA axis linkage in both father–child and mother–child dyads. Indeed, children appear to show stability across visits in their overall cortisol output and in the degree of parent–child linkage. However, only father–child dyads showed significantly correlated cortisol levels across the entire visit, and linkage in cortisol appeared stronger in father–child than in mother–child pairs. These results are consistent with Saxbe et al. (2014), which found that only father cortisol uniquely predicted child cortisol when mothers were included in the same statistical model.

In mother–child dyads, autism spectrum diagnosis appeared to moderate linkage in cortisol, such that mothers had stronger linkage with typically developing children. Indeed, when we split the sample and analyzed only dyads including typically developing children, mothers showed cortisol levels that were positively correlated with their children, at a marginal level of significance. In contrast, in dyads including children with ASD, mothers' cortisol was not linked with child cortisol. One possible reason we did not find cortisol linkage in mother–child dyads with ASD is that mothers may need to "work harder" in order to buffer the stress responses of children with ASD. Our previous work (Ostfeld-Etzion et al., 2015) found that fathers tended to interact more similarly with children with ASD and TD children, and children's cortisol patterns were similar to each other in both groups; in contrast, mothers showed different regulation-facilitation strategies with ASD children, and ASD children showed more blunted cortisol than TD children in the mother–child visit. If mother–child interactions are more informed by ASD diagnosis, it makes sense that we would find more different patterns of linkage between TD and ASD dyads for mothers, but not for fathers.

Both parent and child behaviors during the home visit were associated with parent–child HPA axis linkage. Specifically, father–child linkage was weaker if the father–child dyad showed more reciprocity and more sensitive father behavior, as well as more child self-regulation and less child withdrawal. Similarly, mother–child linkage was weaker if the dyad showed more behavioral coordination and if the mother showed more sensitivity. Children who showed more self-regulation and were in in more sensitive, reciprocal, coordinated dyads also tended to show markers of a more robust pattern of cortisol (higher starting values and more of a drop over the course of the visit), while more withdrawn children showed the opposite pattern (lower starting cortisol and less of a drop over the visit). The consistency of our findings across mother–child and father–child visits is striking. Parental sensitivity was a moderator in both cases, and behavioral synchrony was also a moderator although it was coded in different ways (for mothers, linkage was moderated by behavioral coordination, which was coded on the micro-level within the emotion regulation paradigms; for fathers, linkage was moderated by reciprocity, which was coded on the macro-level within the free play task). Taken together, these results suggest that when parents and children showed better, more coordinated dyadic functioning, they had less correlated levels of cortisol across their home visit. This finding is consistent with

studies that have reported stronger within-couple correlations in cortisol in distressed and aggressive couples (Saxbe & Repetti, 2010; Saxbe et al., 2015). This may also be consistent with Hibel et al (2009)'s finding that mother–child dyads under more stress (e.g., those in households characterized by intimate partner aggression) showed stronger linkage in cortisol, and with Pratt et al. (under review)'s finding that parent–child dyads lower in behavioral reciprocity and higher parental intrusiveness showed stronger diurnal cortisol linkage. In better-functioning dyads, parents and children may be able to balance out each other's stress and arousal and maintain a homeostatic baseline. In contrast, dyads characterized by stronger HPA axis linkage may escalate, rather than modulate, negative emotional states. Although it may seem intuitive that cortisol levels would be more similar in better-attuned dyads, cortisol is a stress hormone, particularly sensitive to social threat, and exaggerated HPA axis linkage may point to "stress contagion" processes.

Strengths of the study include inclusion of both father–child and mother–child dyads in separate visits that had similarly timed protocols. Another strength is that both typically developing children and children with autism spectrum diagnoses were represented in the sample. The detailed behavioral observation and coding is another strength. The data were analyzed using multilevel modeling, which adjusts for the nesting of cortisol levels within individuals within dyads, and that allows for modeling of parent–child linkage while simultaneously controlling for the child's starting value of cortisol and change-by-timepoint across the visit. Many early studies of parent–child cortisol linkage have focused only on zero-order correlations at separate sampling timepoints, but this approach does not adjust for each member of the dyad's initial level of cortisol, or control for each individual's change in cortisol over time. Therefore, our approach offers a stronger test of HPA linkage.

This study is limited by the fact that only three cortisol samples were taken over the course of a relatively brief home visit. A diurnal data collection protocol over several days would provide a richer picture of parent–child cortisol linkage. The small number of samples also means that time-lagged analyses could not be performed. However, this limitation is offset by the fact that the visits were conducted in an ecologically valid environment (the home) and used a structured protocol with standardized timing. Moreover, three cortisol samples represents an improvement over published studies that measured cortisol from parents and children only once or twice (e.g., Granger, 1998; Spratt et al., 2016) and allows us to use multilevel modeling instead of single-timepoint correlation analyses. Another limitation of the study is the small number of dyads including girls. The overrepresentation of boys in our sample reflects the greater prevalence of ASD in males and the need to recruit a gender-matched sample of TD children. However, it may account for the null results we found for mother–child cortisol linkage, since cortisol linkage may be higher in same-sex parent–child dyads (Saxbe et al., 2014). Additionally, we standardized the protocol by ensuring that children did not nap at noon on the saliva collection day. Although most children in the sample were not ordinarily napping at this time, it is possible some children who would have otherwise napped were more fatigued during the parent–child interaction. Finally, our interpretation of the results is limited by the fact that parents and children did not show large increases in cortisol in response to the home visit tasks. Instead, consistent with the normal diurnal decline in cortisol, most participants showed flat or decreasing cortisol over the visit. Many cortisol studies employing mild relational stressors, for example, parent–child interactions, have failed to elicit significant cortisol increases (Gunnar, Talge, & Herrera, 2009), so this issue is not uncommon, but may affect interpretation of our results.

Despite its limitations, this paper offers an intriguing preliminary account of parent–child cortisol linkage. This is the first study to contrast HPA axis linkage in children with ASD and typically developing children. It is also one of the first studies to compare HPA axis linkage in mother–child and father–child dyads that included the same children. Our results are compelling and consistent. Across parents (mother and father), across arousal state (play and emotion regulation), and across level of observation (micro- and macro-level) parent–child cortisol linkage was associated with markers of less coordinated dyadic functioning. Our results support a "stress contagion" account of parent–child cortisol linkage and illustrate the importance of stress in shaping parent–child physiological interplay.

REFERENCES

Ainsworth, M. D. S. (1969). Individual differences in strange-Situational behaviour of one-year-olds. In H. R. Schaffer (Ed.), *The origins of human social relations*. London: Academic Press.

Apter-Levy, Y., Pratt, M., Vakart, A., Feldman, M., Zagoory-Sharon, O., & Feldman, R. (2016). Maternal depression across the first years of life compromises child psychosocial adjustment; relations to child HPA-axis functioning. *Psychoneuroendocrinology*, *64*, 47–56.

Atkinson, L., Gonzalez, A., Kashy, D. A., Santo Basile, V., Masellis, M., Pereira, J., & Levitan, R. (2013). Maternal sensitivity and infant and mother adrenocortical function across challenges. *Psychoneuroendocrinology*, *38*(12), 2943–2951.

Beckes, L., & Coan, J. A. (2011). Social baseline theory: The role of social proximity in emotion and economy of action. *Social and Personality Psychology Compass*, *5*(12), 976–988.

Bridges, L. J., Palmer, S. A., Morales, M., Hurtado, M., & Tsai, D. (1993). Agreement between affectively based observational and parent-report measures of temperament at infant age 6 months. *Infant Behavior and Development*, *16*(4), 501–506.

Carmean, V. (2008). Variable cortisol circadian rhythms in children with autism and anticipatory stress. *Journal of Psychiatry & Neuroscience: JPN*, *33*(3), 227–234.

Davis, E. P., & Sandman, C. A. (2010). The timing of prenatal exposure to maternal cortisol and psychosocial stress is associated with human infant cognitive development. *Child Development*, *81*(1), 131–148.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355–391.

Ditzen, B., Schaer, M., Gabriel, B., Bodenmann, G., Ehlert, U., & Heinrichs, M. (2009). Intranasal oxytocin increases positive communication and reduces cortisol levels during couple conflict. *Biological Psychiatry*, *65*(9), 728–731.

Doussard-Roosevelt, J. A., Joe, C. M., Bazhenova, O. V., & Porges, S. W. (2003). Mother-child interaction in autistic and nonautistic children:

Characteristics of maternal approach behaviors and child social responses. *Development and Psychopathology*, *15*(02), 277–295.

Feldman, R. (1998). Coding interactive behavior manual. *Unpublished Manual*.

Feldman, R. (2007a). Parent-infant synchrony biological foundations and developmental outcomes. *Current Directions in Psychological Science*, *16*(6), 340–345.

Feldman, R. (2007b). Parent–infant synchrony and the construction of shared timing; physiological precursors, developmental outcomes, and risk conditions. *Journal of Child Psychology and Psychiatry*, *4*, 329–354.

Feldman, R. (2012). Oxytocin and social affiliation in humans. *Hormones and Behavior*, *61*(3), 380–391.

Feldman, R. (2015). Sensitive periods in human social development: New insights from research on oxytocin, synchrony, and high-risk parenting. *Development and Psychopathology*, *27*(02), 369–395.

Feldman, R., Vengrober, A., Eidelman-Rothman, M., & Zagoory-Sharon, O. (2013). Stress reactivity in war-exposed young children with and without posttraumatic stress disorder: Relations to maternal stress hormones, parenting, and child emotionality and regulation. *Development and Psychopathology*, *25*, 943–955.

Goldsmith, H. H. (1996). Studying temperament via construction of the toddler behavior assessment questionnaire. *Child Development*, *67*(1), 218–235.

Goldsmith, H. H., & Rothbart, M. K. (1999). The laboratory temperament assessment battery. Locomotor Version, 3.

Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The Autism Diagnostic Observation Schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, *37*(4), 613–627.

Granger, D. A. (1998). Children's salivary cortisol, internalising behaviour problems, and family environment: Results from the Concordia Longitudinal Risk Project. *International Journal of Behavioral Development*, *22*(4), 707–728.

Gunnar, M. R., Talge, N. M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology*, *34*(7), 953–967.

Hane, A. A., Fox, N. A., Polak-Toste, C., Ghera, M. M., & Guner, B. M. (2006). Contextual basis of maternal perceptions of infant temperament. *Developmental Psychology*, *42*(6), 1077.

Hane, A. A., Fox, N. A., Henderson, H. A., & Marshall, P. J. (2008). Behavioral reactivity and approach-withdrawal bias in infancy. *Developmental Psychology*, *44*(5), 1491.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry*, *54*(12), 1389–1398.

Hibel, L. C., Granger, D. A., Blair, C., & Cox, M. J. (2009). Intimate partner violence moderates the association between mother-infant adrenocortical activity across an emotional challenge. *Journal of Family Psychology*, *23*(5), 61–25.

Hibel, L. C., Granger, D. A., Blair, C., & Finegood, E. D. (2015). Maternal-child adrenocortical attunement in early childhood: Continuity and change. *Developmental Psychobiology*, *57*(1), 83–95.

Hofer, M. A. (1984). Relationships as regulators: A psychobiologic perspective on bereavement. *Psychosomatic Medicine*, *46*(3), 183–197.

Hostinar, C. E., Sullivan, R. M., & Gunnar, M. R. (2014). Psychobiological mechanisms underlying the social buffering of the hypothalamic-pituitary-adrenocortical axis: A review of animal models and human studies across development. *Psychological Bulletin*, *140*(1), 256–282.

Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry*, *53*(12), 1250–1258.

Laurent, H. K., Ablow, J. C., & Measelle, J. (2011). Risky shifts: How the timing and course of mothers' depressive symptoms across the perinatal period shape their own and infant's stress response profiles. *Development and Psychopathology*, *23*(02), 521–538.

Levenson, R. W., & Gottman, J. M. (1983). Marital interaction: Physiological linkage and affective exchange. *Journal of Personality and Social Psychology*, *45*(3), 587–597.

Liu, S., Rovine, M. J., Cousino Klein, L., & Almeida, D. M. (2013). Synchrony of diurnal cortisol pattern in couples. *Journal of Family Psychology*, *27*(4), 579–588.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *840*(1), 33–44.

Ostfeld-Etzion, S., Golan, O., Hirschler-Guttenberg, Y., Zagoory-Sharon, O., & Feldman, R. (2015). Neuroendocrine and behavioral response to social rupture and repair in preschoolers with autism spectrum disorders interacting with mother and father. *Molecular Autism*, *6*(11), 11–24.

Papp, L. M., Pendry, P., & Adam, E. K. (2009). Mother-adolescent physiological synchrony in naturalistic settings: Within-family cortisol associations and moderators. *Journal of Family Psychology*, *23*(6), 882–894.

Papp, L. M., Pendry, P., Simon, C. D., & Adam, E. K. (2013). Spouses' cortisol associations and moderators: Testing physiological synchrony and connectedness in everyday life. *Family Process*, *52*(2), 284–298.

Pratt, M., Apter-Levi, Y., Vakart, A., Kanat-Maymon, Y., Zagoory-Sharon, O., & Feldman, R. (2017). Mother-child adrenocortical synchrony; Moderation by dyadic relational behavior. *Hormones and behavior*. *89*, 167–175.

Raudenbush, S. W., Spybrook, J., Congdon, R., Liu, X. F., Martinez, A., & Bloom, H. (2011). Optimal design software for multi-level and longitudinal research (Version 3.01) [Software].

Saxbe, D. E., Margolin, G., Spies Shapiro, L., Ramos, M., Rodriguez, A., & Iturralde, E. (2014). Relative influences: Patterns of HPA axis concordance during triadic family interaction. *Health Psychology*, *33*(3), 273–281.

Saxbe, D. E., Negriff, S., Susman, E. J., & Trickett, P. K. (2015). Attenuated hypothalamic-pituitary-adrenal axis functioning predicts accelerated pubertal development in girls 1 year later. *Development and Psychopathology*, *27*(03), 819–828.

Saxbe, D., & Repetti, R. L. (2010). For better or worse? Coregulation of couples' cortisol levels and mood states. *Journal of Personality and Social Psychology*, *98*(1), 92–103.

Schneiderman, I., Kanat-Maymon, Y., Ebstein, R. P., & Feldman, R. (2014). Cumulative risk on the oxytocin receptor gene (OXTR) underpins empathic communication difficulties at the first stages of romantic love. *Social Cognitive and Affective Neuroscience*, *9*(10), 1524–1529.

Schneirla, T. C. (1946). Problems in the bio-psychology of social organization. *The Journal of Abnormal and Social Psychology*, *41*(4), 385–402.

Scott, F. J., Baron-Cohen, S., Bolton, P., & Brayne, C. (2002). The CAST (Childhood Asperger Syndrome Test) preliminary development of a UK screen for mainstream primary-school-age children. *Autism*, *6*(1), 9–31.

Sethre-Hofstad, L., Stansbury, K, & Rice, M. A. (2002). Attunement of maternal and child adrenocortical response to child challenge. *Psychoneuroendocrinology*, *27*(6), 731–747.

Spratt, E. G., Nicholas, J. S., Brady, K. T., Carpenter, L. A., Hatcher, C. R., Meekins, K. A., & Charles, J. M. (2012). Enhanced cortisol response to stress in children in autism. *Journal of Autism and Developmental Disorders*, *42*(1), 75–81.

Spratt, E. G., Marsh, C., Wahlquist, A. E., Papa, C. E., Nietert, P. J., Brady, K. T., … Wagner, C. (2016). Biologic effects of stress and bonding in mother-infant pairs. *International Journal of Psychiatry in Medicine*, *51*(3), 246.

Stenius, F., Theorell, T., Lilja, G., Scheynius, A., Alm, J., & Lindblad, F. (2008). Comparisons between salivary cortisol levels in six-months-olds and their parents. *Psychoneuroendocrinology*, *33*(3), 352–359.

Thorndike, R. L., Hagen, E. P., & Sattler, J. M. (1986). *Stanford-binet intelligence scale*. Rolling Meadows, IL: Riverside Publishing Company.

Timmons, A. C., Margolin, G., & Saxbe, D. E. (2015). Physiological linkage in couples and its implications for individual and interpersonal functioning: A literature review. *Journal of Family Psychology*, *29*(5), 720–732.

van Bakel, H. J., & Riksen-Walraven, J. M. (2008). Adrenocortical and behavioral attunement in parents with 1-year-old infants. *Developmental Psychobiology*, *50*(2), 196–201.

van IJzendoorn, M. H., Rutgers, A. H., Bakermans-Kranenburg, M. J., Swinkels, S. H., Van Daalen, E., Dietz, C., . . . Van Engeland, H. (2007). Parental sensitivity and attachment in children with autism spectrum disorder: Comparison with children with mental retardation, with language delays, and with typical development. *Child Development*, *78*(2), 597–608.

How to cite this article: Saxbe DE, Golan O, Ostfeld-Etzion S, Hirschler-Guttenberg Y, Zagoory-Sharon O, Feldman R. HPA axis linkage in parent–child dyads: Effects of parent sex, autism spectrum diagnosis, and dyadic relationship behavior. *Dev Psychobiol*. 2017;9999:1–11. https://doi.org/10.1002/dev.21537