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The Interpersonal Neurobiology of Child Maltreatment: Parasympathetic Substrates of Interactive Repair in Maltreating and Nonmaltreating Mother–Child Dyads

Erika Lunkenheimer[1], **Alex Busuito**[1], **Kayla M. Brown**[1], **Carlomagno Panlilio**[1], **Elizabeth A. Skowron**[2]

[1]Pennsylvania State University, University Park, PA, USA

[2]University of Oregon, Eugene, OR, USA

Abstract

Children's repair of conflict with parents may be particularly challenging in maltreating families, and early, stressful parent–child interactions may contribute to children's altered neurobiological regulatory systems. To explore neurobiological signatures of repair processes, we examined whether mother and child individual and dyadic respiratory sinus arrhythmia (RSA) covaried with interactive repair differently in maltreating versus nonmaltreating mother–preschooler dyads (N = 101), accounting for whether repair was mother or child initiated. Mother-initiated repair was equally frequent and protective across groups, associated with no change in mother or child RSA at higher levels of repair. But lower levels of mother repair were associated with child RSA withdrawal in nonmaltreating dyads versus child RSA augmentation in maltreating dyads. In maltreating dyads only, higher child-initiated repair was associated with higher mean mother RSA, whereas lower child repair was associated with mother RSA withdrawal. Findings suggest that interactive repair may have a buffering effect on neurobiological regulation but also that maltreating mothers and children show atypical neurobiological response to interpersonal challenges including differences related to children conducting the work of interactive repair that maltreating parents are less able to provide. We conclude by considering the role of maladaptive parent–child relationship processes in the biological embedding of early adversity.

Keywords

child maltreatment; parent–child relationships; physiological processes; preschoolers

Conflict with caregivers is common in early childhood. As children develop autonomy and explore the limits of their behavior, conflict may manifest as negative emotion expression, child misbehavior in response to a parental command, or the mismatch of goals during face-

Corresponding Author: Erika Lunkenheimer, Pennsylvania State University, 140 Moore Building, University Park, PA 16801, USA, ezl5238@psu.edu.

Authors' Note

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to-face interactions (E. Z. Tronick, 1989). These moments pose an opportunity for the development of self-regulation (SR) in a relational context as children learn how to repair conflict. Interactive repair, defined as moving from a negative or mismatched emotional or behavioral state to a mutually positive or matched state, may contribute to homeostasis in the dyad, which in turn may serve to regulate the individual (E. Tronick & Reck, 2009). Caregivers are the primary context for child experiences of interpersonal repair, beginning in infancy as parent and child coordinate affect and behavior around the child's needs (Feldman, 2007). Conflict with caregivers may be stressful as it may threaten the child's primary source of support, underscoring the child's drive to repair (Macfie, Fitzpatrick, Rivas, & Cox, 2008). Repair processes may be more challenging or different in maltreating families (Skowron, Kozlowski, & Pincus, 2010) in which repair is less certain or the parent is perceived as a source of threat rather than support. Thus, understanding how regulatory neurobiology covaries with parent–child repair processes could be one window by which to understand regulatory differences in maltreating and nonmaltreating families. Such relations may shed light on the interpersonal mechanisms by which adversity is biologically embedded (McLaughlin et al., 2015). Accordingly, we examined how individual and dyadic neurobiological regulation covaried with child- and mother-initiated repair during challenging interactions and whether these relations differed in maltreating versus nonmaltreating dyads.

Interactive Repair in Early Parent–Child Interactions

Most interpersonal interactions involve mismatch and the occasional bout of conflict (E. Z. Tronick, 1989). This mismatch and conflict are normative as caregiver and child work out how to coordinate emotion and behavior in the earliest years of the child's life (E. Z. Tronick, 1989). The process of resolving this conflict serves to preserve the relationship and regulate one's own emotions and is a skill that is learned over repeated interpersonal interactions (Seifer & Schiller, 1995). In early interactions, caregivers do the majority of the work of resolving negativity by responding in a sensitive and timely way to child cues, using positive facial affect and vocal tone to redirect the child in a positive manner (Jameson, Gelfand, Kulcsar, & Teti, 1997). By doing so, caregivers externally regulate the child and model interactive repair.

The repair of interpersonal conflict plays an important role in child development. It lays the foundation for attachment security, such that when a caregiver consistently resolves mismatches by attuning to the infant's needs, the infant internalizes a working model of relationships as safe, trustworthy spaces where negative emotions can be managed (E. Tronick & Reck, 2009). Further, the affective exchange processes characteristic of conflict and repair cycles are thought to support children's understanding and regulation of emotions (Rosenblum, Dayton, & Muzik, 2009). As the child ages, his or her ability to repair social interactions may evoke higher quality interactions and reinforce regulatory development (Seifer & Schiller, 1995). For example, greater parent–child interactive repair at age 3 predicts children's higher SR skills in preschool at age 4 (Kemp, Lunkenheimer, Albrecht, & Chen, 2016), thus also conferring individual regulatory benefits to the child across social settings.

In contrast, when interactive repair is inconsistent or absent, it can lead to insecure attachment between parent and child and prevent the opportunities needed to develop SR in social contexts (Siegel, 2001). As a result of these disruptions, unrepaired conflict also puts children at risk of externalizing and internalizing behavior problems (Biringen, Emde, & Pipp-Siegel, 1997). In some cases, children take on the primary responsibility for repairing the conflict, resulting in role reversal, wherein young children have developmentally inappropriate relationship responsibilities (Macfie et al., 2008). Role reversal is primarily a function of the caregiver's attempts to get their own needs met at the expense of the child's needs and represents a clear distortion of the parent–child relationship (Sroufe, Jacobvitz, Mangelsdorf, DeAngelo, & Ward, 1985); it is also associated with maladaptive relationship processes such as disorganized attachment (Macfie et al., 2008).

Interactive repair may be altered in families who maltreat their children, given that the normative expectations of caregiving are violated. Maltreating parents display less positive affect, respond less sensitively to child cues, and have higher levels of insecure attachment with their children (Cicchetti, 2016; Wilson, Shi, Rack, & Norris, 2008). Maltreating dyads also show greater interactive mismatch (Skowron et al., 2011) and repair less than nonmaltreating dyads (Skowron et al., 2010). Additionally, role reversal occurs in maltreating families, even as early as the toddler period (Macfie et al., 2008). For example, maltreating mothers are more likely to rupture interactions, and their preschoolers are more likely to repair them (Skowron et al., 2010), which is the opposite of patterns found in typical mother–child dyads. The present study expanded upon this work to examine whether there were differing neurobiological signatures in maltreating versus nonmaltreating mother–child interactions that were associated with these varying patterns of interactive repair.

Regulatory Neurobiology in Interpersonal Contexts

The polyvagal perspective (Porges, 2007) suggests that social engagement and stress regulation in social contexts are supported by the myelinated vagus nerve, which connects the central nervous system to the heart via parasympathetic regulation of cardiac activity. The vagus nerve is regulated by a wider network of cortical and subcortical brain regions involved in threat detection and SR including the amygdala and medial prefrontal cortex (Thayer & Lane, 2000). Porges (2007) argued this system evolved to support human engagement in social behaviors and regulatory behaviors such as self-soothing and impulse inhibition with which to manage social interactions. The neurovisceral integration perspective states that vagally mediated heart rate variability may be a marker of top-down SR skills, given that the prefrontal cortex regulates subcortical structures that influence vagal input to the heart (Thayer, Hansen, Saus-Rose, & Johnsen, 2009). Thus, indices of parasympathetic regulation of cardiac activity are considered markers of stress reactivity in response to social threat and markers of the top-down SR behaviors that promote social bonding (Holzman & Bridgett, 2017).

The parasympathetic nervous system supports heart rate, breathing, and digestion in the body at rest and is often measured via respiratory sinus arrhythmia (RSA), an index of the degree of variability in heart rate associated with the respiration rate (Berntson, Cacioppo, &

Quigley, 1993). Baseline RSA during resting states is thought to reflect the maintenance of homeostasis in the body and the capacity to respond when challenge occurs (Porges, 2007). When challenge occurs, a typical response is a decrease in RSA, which reflects the relaxation of the inhibitory influence of parasympathetic processes in order to mobilize resources with which to respond (Shahrestani, Stewart, Quintana, Hickie, & Guastella, 2014), including engagement of the sympathetic nervous system to activate fight-or-flight responses to threat (Kahle & Hastings, 2015). In contrast, an excessive decrease in RSA in the absence of threat is considered an atypical response (Beauchaine, 2001) as is an RSA increase in response to social challenge, considered an insufficient response to social demands and associated with disengagement from or avoidance of the challenging stimulus (Thayer & Lane, 2000). Low baseline RSA levels are also thought to reflect atypically blunted parasympathetic processes, tied to chronic stress exposure (Beauchaine, 2001). However, it should be noted that these response patterns are not empirically robust across all studies and may differ by factors such as age, assessment task, and/or risk level and type (e.g., Leerkes et al., 2015; Ostlund, Measelle, Laurent, Conradt, & Ablow, 2017).

A strong implication of polyvagal and neurovisceral integration models is that we should see neurobiological signatures of challenging social interactions in real time. There is ample evidence on parasympathetic responses to social challenge, most notably in work on parent–child interactions involving experimentally induced stressors (e.g., Moore et al., 2009; Ostlund et al., 2017). A meta-analysis showed that dyadic parent–child tasks with stressor conditions such as the still face paradigm and strange situation were associated with significant declines in individual mother and child RSA, indicating that both mother and child were biologically stressed by these tasks (Shahrestani et al., 2014). Notably, these tasks exemplify situations in which mothers are prevented from repairing child distress, raising the question as to whether the absence of expected maternal repair may be associated with decreases in RSA.

Additionally, recent work argues for the importance of assessing dyadic patterns of RSA between parent and child. Early biological synchrony is thought to support behaviors critical for the child's developing biobehavioral regulatory systems and is associated with behaviors characteristic of repair processes in infancy such as maternal sensitivity and shared positive affect (Feldman, Magori-Cohen, Galili, Singer, & Louzoun, 2011). Lunkenheimer, Tiberio, Skoranski, Buss, and Cole (2018) have shown that RSA concordance declines over time in the context of more challenging social goals in mother–preschooler interactions, which may imply that the repair of conflict will be associated with weaker concordance in RSA. From a polyvagal or neurovisceral integration perspective, it is possible that individual regulatory deficits in the parent or child manifest as atypical individual RSA patterns, which in turn disrupt the dyadic concordance of RSA. Accordingly, both individual and dyadic patterns of RSA were examined in the present study to understand their relations with interactive repair in the preschool period.

Polyvagal and neurovisceral integration models also have implications for acute social threat, such as in the case of child maltreatment (McLaughlin et al., 2015). When there is a disconnect in the safety cues between parent and child, it triggers the child's biobehavioral defense systems (Porges, 2007), which in turn alter social awareness and vigilance to threat

(Holochwost & Jaffee, 2017). Perhaps as a result, individual and dyadic parasympathetic processes are disrupted in maltreating families. For example, maltreating mothers and their children show more blunted resting RSA (Skowron et al., 2011) thought to reflect more chronic stress exposure. Maltreating mothers and children also show atypical RSA increases or decreases in response to challenging tasks relative to nonmaltreating controls, suggesting atypical parasympathetic responding during parent–child interactions (Creaven, Skowron, Hughes, Howard, & Loken, 2014; Skowron et al., 2011). Additionally, maltreating dyads do not show the dyadic RSA concordance shown in typical families during challenging tasks (Creaven et al., 2014) and show dyadic discordance in RSA at higher levels of maltreatment severity (Lunkenheimer, Busuito, Brown, & Skowron, 2018). Together, these findings imply that disruptions to parasympathetic regulatory processes may be related to the interpersonal challenges characteristic of maltreating families. The case of interactive repair may offer a unique test of how regulatory neurobiology underlies behavioral relationship processes differently for maltreating and nonmaltreating parent–child dyads and whether it does so in ways that map onto theoretical expectations about biomarkers of early adversity in children (Cicchetti, 2016).

Present Study

This study had descriptive goals of understanding the neurobiological signatures of interactive repair during maltreating and nonmaltreating mother–child challenging interactions, specifically with respect to the SR and coregulation (CO) of RSA as indices of socially related regulatory neurobiology. Multilevel models captured the effects of mean RSA, the SR of RSA (i.e., the effect of one's own prior RSA, reflecting intraindividual variability in RSA over time), and the CO of RSA (i.e., the effect of the partner's concurrent RSA, reflecting concordance) at the within level, and the effects of the frequency of mother-initiated and child-initiated dyadic interactive repair sequences at the between level. Models were performed separately by mother versus child RSA to examine differences in each partner's respective RSA.

This work was exploratory, given that prior research could suggest multiple potential associations. On one hand, socially challenging tasks have been shown to be associated with declines in individual and dyadic RSA (Lunkenheimer, Tiberio, et al., 2018), with some linear variation in these processes by maltreatment status (Lunkenheimer, Busuito, et al., 2018), as well as lower resting RSA in maltreating families (Creaven et al., 2014). So there could be a quantitative difference in mean levels or the degree of change in RSA associated with repair by maltreatment status. Alternatively, children in maltreating dyads conduct more repair than their mothers, suggesting role reversal (Skowron et al., 2010), and maltreating mothers are stressed by effortful parenting, showing RSA decreases following their own positive parenting moments (Skowron, Cipriano-Essel, Benjamin, Pincus, & Van Ryzin, 2013). Thus, it was also possible we would see qualitative differences in terms of RSA increases versus decreases or the initiator of the repair. Accordingly, we made no specific hypotheses about differences by maltreatment status, and models were performed separately by maltreatment status, given qualitatively differential processes in RSA and behavior by maltreatment group suggested by prior research (e.g., Creaven et al., 2014; Skowron et al., 2013).

Method

Participants

Participants were a subsample of 101 mothers and children (M_{age} = 3.61 years, SD_{age} = 0.70 years, range = 3–5 years) from a study on parent–child interaction and child maltreatment (N = 222). Subsample inclusion criteria were that (a) mother–child dyads exhibited negative behaviors during dyadic interaction tasks, thus allowing the chance to repair and (b) at least two or more consecutive epochs of concurrent RSA data for both mother and child were available for analysis. Of the original sample, 144 dyads displayed negative behavior, and of those, 101 had two or more consecutive epochs of concurrent RSA data for both mother and child, making up the analytic subsample. The analytic subsample did not differ from the original sample on any demographic characteristics including child sex, child age, child or mother ethnicity, family income, maternal education, or marital status. Maltreating families were recruited through public welfare offices and had a documented Child Protective Services (CPS) record by the mother that could be coded with the Maltreatment Classification System (MCS; see Measures; Barnett, Manly, & Cicchetti, 1993). Nonmaltreating families were recruited from a birth database, oversampled for lower levels of family income and maternal education, and excluded if they had a CPS record. To participate, mothers had to be 18 years or older, speak English, and live with their preschooler. Parents and children with severe physical health problems or disabilities were excluded.

For maltreating families, mothers' age ranged from 20 to 34 years (M = 27.37, SD = 5.35). Ethnicity was 91.5% non-Hispanic White, 3.4% Hispanic, and 3.4% multiethnic. Mothers were married (32.2%) or in a committed relationship (23.7%), separated or divorced (25.5%), or single (18.6%). Annual income ranged from US$10,000 or less (40.7%), US $10,000–US$30,000 (47.5%), US$30,000–US$50,000 (6.8%), to more than US$50,000 (3.4%). Mothers' education included 20.3% completing some high school, 71.2% with high school degrees, 3.4% completing some college, 3.4% with college degrees, and 1.7% with graduate degrees. For nonmaltreating families, mothers' age ranged from 23 to 49 years (M = 30.31, SD = 5.78). Ethnicity was 100% non-Hispanic White. Mothers were married (54.8%) or in a committed relationship (23.8%), separated or divorced (16.7%), or single (4.8%). Annual income ranged from US$10,000 or less (14.3%), US$10,000–US$30,000 (40.5%), US$30,000–US$50,000 (16.7%), to more than US$50,000 (26.2%). Mothers' education levels included 4.8% completing some high school, 50% high school degrees, 16.7% some college, 21.4% with college degrees, and 7.1% with a graduate degree.

Procedure

Mothers and their children came for a 2.5-hr laboratory visit as part of a larger study, which involved two prior home visits. They engaged in a baseline resting task to assess RSA and two dyadic puzzle tasks during which RSA data were collected. The resting task involved 5 min with lights dimmed, while mother and child were seated closely and watched an animated video. Puzzle tasks were designed to be challenging for 3- to 5-year-olds, which in turn prompted effort on the parent's part to aid in the task and/or respond to the child's difficulty; thus, the task prompted the potential for negative affect, behavior, or conflict

during the tasks. Prior research has utilized similar tasks to prompt variation in emotion and behavior, as well as to elicit changes in parent and child RSA (e.g., Lunkenheimer, Tiberio, et al., 2018).

In the first puzzle task, mother and child were asked to complete a puzzle of a train with nine pieces. In the second puzzle task (Duplo Puzzle Task; Hoffman, Crnic, & Baker, 2006), dyads were shown a three-dimensional figure made out of Duplo Lego blocks, and then parent and child were asked to construct a replica using the blocks provided. For both tasks, mothers were asked to help the child using only their words and not touch the pieces. Each task was terminated after 3 min if the puzzle had been successfully completed and lasted a maximum of 5 min. Mothers were compensated US$150, provided transportation to sessions as needed, and children were given a small toy. The study and all procedures were approved by the sponsoring university's institutional review board.

Measures

Structural analysis of social behavior (SASB) coding system—Similar to prior work (Skowron et al., 2010), the SASB (Benjamin, 1996) was used to code mother and child behavior. It is a circumflex model based on the orthogonal dimensions of affiliation (from loving to hostile) and interdependence (from differentiated to enmeshed). Coding a behavior involves determining the focus of the behavior (other or self), the degree of affiliation, and the degree of interdependence. When integrated, these three dimensions form eight "clusters," one of which is assigned to each behavior. Each cluster can manifest as other-oriented or self-oriented (listed first and second, respectively, in cluster descriptions below). Behaviors in Clusters 1 (freeing/emancipating, asserting/separating), 2 (affirming/understanding, disclosing/expressing), 3 (loving/approaching, joyfully connecting), and 4 (nurturing/protecting, trusting/relying) were aggregated to form a "positive" behavior category. Behaviors in Clusters 6 (blame/criticize, sulk/ appease), 7 (attack/reject, protest/recoil), and 8 (ignore/neglect, wall-off/avoid) were aggregated to form a "negative" behavior category. Cluster 5 (controlling/managing, deferring/submitting) was excluded. These positive and negative aggregate codes were then used to define interactive repair. Coders were advanced graduate students in counseling psychology, were blind to child maltreatment status, and received more than 75 hr of training from experienced SASB coders. Initial reliability prior to coding and subsequent drift reliability on a total of 18% of the sample was acceptable (weighted κ = .75; see Benjamin, 1996, for more detail on the SASB).

Interactive repair—We calculated repair from the positive and negative SASB aggregate codes. Similar to prior approaches (Kemp et al., 2016; Skowron et al., 2010), we conceptualized repair as the return to a dyadic positive state following a negative state by either mother or child, accounting for who initiated the repair. Mother-initiated repair was defined as a two-step sequence of SASB-coded mother positive behavior followed by a child positive behavior, occurring immediately after either mother or child exhibited a negative behavior (i.e., Mother negative → Mother positive → Child positive OR Child negative → Mother positive → Child positive). Child-initiated repair was defined as a two-step sequence of SASB-coded child positive behavior followed by a mother positive behavior that occurred after a mother or child negative behavior (i.e., Mother negative → Child positive → Mother

positive OR Child negative → Child positive → Mother positive). Thus, the initiator was the first to express positive behavior following a negative behavior, and then to be considered a return to a dyadic positive state, the partner must then have reciprocated with a positive behavior. The frequency of mother- and child-initiated repair sequences was used in primary analyses to represent interactive repair.

Child maltreatment status—CPS records were coded using the MCS (Barnett et al., 1993), where maltreatment reflected the presence of a substantiated record of physical abuse, neglect, emotional maltreatment, or sexual abuse by the mother prior to study entry. Of the 59 dyads available for analysis, their classifications were 64% neglect, 36% physical abuse, and there was no emotional maltreatment or sexual abuse. Based on MCS coding, severity levels were normally distributed, $M = 3.12$, $SD = 1.07$, range = 1–5.

RSA—The Mindware wireless electrocardiograph (ECG) MW1000A was used to collect high-frequency heart rate variability (HF-HRV), which served as an index of RSA. Three pregelled Ag/AgCl electrodes were placed on the right clavicle, lower left rib, and lower abdomen. ECG signals were passed through an A/D converter sampled at 500 ms to obtain heart rate, processed with Mindware Heart Rate Variability 3.0 software. RSA was operationalized as HF-HRV, the natural log of the variance of heart period within the frequency band related to respiration (0.12–0.40 for parents and 0.24–1.04 for children; Fracasso, Lamb, Porges, & Rosenberg, 1994). A fast Fourier transformation of the interbeat interval series and power in the respiratory frequency band was derived from the spectral density function. Consistent with prior studies on RSA in preschoolers (Calkins & Keane, 2004), data were parsed into 30-s epochs across the 10 min of tasks, and trained researchers manually corrected missing or erroneous heartbeats using R peak detection and placement functions in Mindware software. If more than 10% of the epoch required editing, it was excluded from analysis. Breathing rate can affect mean-level differences in RSA and can vary by the degree of talking involved in study tasks (Shader et al., 2018); mother and child mean RSA was unrelated to the total utterances made (as a proxy of talking) when evaluated overall and separately by maltreatment status.

Analytic Plan

Multilevel coupled autoregressive models were performed in Mplus 7.3 (Muthén & Muthén, 1998–2012) to analyze the effects of the frequency of mother- and child-initiated repair sequences on individual and dyadic patterns of mother and child RSA, assessed in 30-s epochs over the course of 10 consecutive minutes of dyadic tasks in the laboratory. These models allowed for the examination of the effects of covariates on concurrent time-series patterns in two individuals. Specifically, analyses modeled how interactive repair was related to mother and child mean RSA, SR of RSA (i.e., prediction of one's own current RSA from one's previous RSA at a 30-s lag), and CO of RSA (i.e., prediction of one's own current RSA from the partner's concurrent RSA or concordance), which varied as the function of time (RSA; Level 1) and person (interactive repair; Level 2).

Prior work on differences in RSA in maltreating and nonmaltreating mother–child dyads guided the design of the analytic models (Lunkenheimer, Busuito, et al., 2018). Lagged SR

parameters were selected based on the effects of intraindividual variability of RSA in prior work (Lunkenheimer et al., 2015). Lagged CO parameters were also considered and examined in a preliminary Level 1 model but were not significant and thus omitted for parsimony. Prior work has found that more nuanced time-series analysis may reveal qualitative differences by maltreatment status, which are not evident when examining average or aggregate variables of interest (Skowron et al., 2013). Thus, we first performed a preliminary multilevel model examining maltreatment status as a covariate in relations between repair and RSA (Creaven et al., 2014), but the effects of maltreatment status were not significant. We then conducted our main time-series analyses separately by maltreatment status in an effort to better examine potential qualitative differences in RSA related to child maltreatment that may have been obscured in the preliminary model. Models were also run separately by mother or child RSA, allowing for the examination of differences in the direction of SR and CO effects (i.e., from mother to child or from child to mother). Thus, a total of four primary analytic models were performed (maltreating dyads—mother RSA, nonmaltreating dyads—mother RSA, maltreating dyads—child RSA, nonmaltreating dyads—child RSA).

Within-dyad RSA: Level 1 model—Mean levels, SR, and CO of RSA were modeled with the equations below, in which $m\mathrm{RSA}_{i,t}$ and $c\mathrm{RSA}_{i,t}$ denote the ith mother and child's RSA values, respectively, at time t. Mother and child RSA means at epoch t were modeled as μ_{Mi} and μ_{Ci} (where i indicates random effects were included). SR at a 30-s lag was denoted by $\beta_{M,\mathrm{SR}}$ for mothers and $\beta_{C,\mathrm{SR}}$ for children, and CO was denoted by $\beta_{M,\mathrm{CO}}$ for mothers and $\beta_{C,\mathrm{CO}}$ for children.

Level 1 (within dyad, across time):

$$\begin{aligned} m\mathrm{RSA}_{i,t} &= \mu_{Mi} + \beta_{M,\mathrm{SR1}} m\mathrm{RSA}_{i,t-1} + \beta_{M,\mathrm{CO}} c\mathrm{RSA}_{i,t} + \varepsilon_{Mi,t}, \\ c\mathrm{RSA}_{i,t} &= \mu_{Ci} + \beta_{C,\mathrm{SR1}} c\mathrm{RSA}_{i,t-1} + \beta_{C,\mathrm{CO}} m\mathrm{RSA}_{i,t} + \varepsilon_{Ci,t}. \end{aligned} \quad (1)$$

Between-dyad differences by interactive repair: Level 2 model—In the second level, we examined whether frequency of mother- and child-initiated interactive repair was related to mother and child mean level, SR, and CO of RSA using the equations below. The fixed effects denoted as $\beta_{M/C}$ in the Level 1 equations are represented as $\gamma_{M/C}$ in the Level 2 equations.

Level 2 (Between dyad):

The following equations model the effects of both mother-initiated (α_M) and child-initiated (α_C) interactive repair on mothers' mean RSA (μ_{Mi}), SR of RSA ($\beta_{M,\mathrm{SR}}$), and CO of RSA ($\beta_{M,\mathrm{CO}}$), including random intercepts (u_{Mi}):

$$\begin{aligned} \mu_{Mi} &= u_{Mi} + \gamma_M + \alpha_{M,\mathrm{Mean}}\mathrm{Repair} + \alpha_{C,\mathrm{Mean}}\mathrm{Repair}, \\ \beta_{M,\mathrm{SR}} &= \gamma_{M,\mathrm{SR}} + \alpha_{M,\mathrm{SR}}\mathrm{Repair} + \alpha_{C,\mathrm{SR}}\mathrm{Repair}, \\ \beta_{M,\mathrm{CO}} &= \gamma_{M,\mathrm{CO}} + \alpha_{M,\mathrm{CO}}\mathrm{Repair} + \alpha_{C,\mathrm{CO}}\mathrm{Repair}. \end{aligned} \quad (2)$$

The following equations model the effects of both mother-initiated (α_M) and child-initiated (α_C) interactive repair on children's mean RSA (μ_{Ci}), SR of RSA ($\beta_{C,\mathrm{SR}}$), and CO of RSA ($\beta_{C,\mathrm{CO}}$), including random intercepts (u_{Ci}):

$$\mu_{Ci} = u_{Ci} + \gamma_C + \alpha_{C,\mathrm{Mean}}\mathrm{Repair} + \alpha_{M,\mathrm{Mean}}\mathrm{Repair},$$
$$\beta_{C,\mathrm{SR}} = \gamma_{C,\mathrm{SR}} + \alpha_{C,\mathrm{SR}}\mathrm{Repair} + \alpha_{M,\mathrm{SR}}\mathrm{Repair},$$
$$\beta_{C,\mathrm{CO}} = \gamma_{C,\mathrm{CO}} + \alpha_{C,\mathrm{CO}}\mathrm{Repair} + \alpha_{M,\mathrm{CO}}\mathrm{Repair}.$$

Overall, there were 42 nonmaltreating and 59 maltreating dyads available for analysis; these dyads displayed one or more negative behaviors during the 10 min of dyadic tasks, allowing for the chance for the dyad to repair. Dyads were also excluded if RSA data were completely missing for mother or child or if there were less than two consecutive epochs of concurrent RSA data for both mother and child with which to analyze coupled autoregressive effects. Missing data were handled using full information maximum likelihood estimation in Mplus 7.3 (Muthén & Muthén, 1998–2012). Although separate analyses by maltreatment status reduced sample size, statistical power in time-series models is derived from both sample size and the number of observations per person (Guo, Logan, Glueck, & Muller, 2013). With twenty 30-s epochs per individual, the present models involved 840 person-by-time observations for nonmaltreating dyads and 1,180 person-by-time observations for maltreating dyads, offering adequate statistical power for the proposed analyses.

Results

Descriptive Analyses

All study variables were normally distributed. In the analytic subsample used, including only those dyads with at least one instance of negative behavior, dyads exhibited 10.24 instances of negative behavior on average over 10 min of interaction ($SD = 11.22$, range = 1–59). Mother–child dyads with younger children showed more negative behavior overall, $r = -.24$, $p < .05$. Of all dyads, 62.4% demonstrated any mother-initiated repair and 22.8% demonstrated any child-initiated repair. Thus, instances of mother-initiated ($M = 1.30$, $SD = 1.30$, range = 0–5) and child-initiated repair ($M = 0.31$, $SD = 0.64$, range 0–3) were low on average, and only 26.4% of negative behaviors were repaired by either mother or child across the full sample. In dyads who exhibited more negative behavior, both children, $r = .37$, $p < .001$, and mothers, $r = .34$, $p < .001$, repaired more frequently than in dyads who were less negative. Mothers repaired more often than children, $t(100) = -6.80$, $p < .001$, and mother and child repair were not correlated. There were no differences in mean mother and child RSA between dyads who were included versus excluded from the study on the basis of negative behavior displays.

With respect to sociodemographic factors, there were significant differences between the maltreating and nonmaltreating groups, such that maltreating mothers were younger, $r = -.36$, $p < .001$, and their income was lower on average, $\chi^2(4) = 18.19$, $p < .001$. However, income was not related to any primary study variables of interest (mother or child RSA, negative behaviors, or repair) for either group and so was not considered as a covariate in main analyses. Primary study variables were not related to sociodemographic factors with the exception of a negative relation between mother RSA and maternal age, $r = -.35$, p

< .01. However, when included as a covariate in mother RSA models, maternal age had no significant effects. Thus, to simplify models and examine comparable models across mother and child, it was not included as a covariate in main analyses.

Maltreating and nonmaltreating dyads did not differ as a function of any primary study variables, nor in the total frequency or in proportion of repair or in who repaired (i.e., mother repaired more than child in both groups). Average mother RSA (M = 6.00, SD = 1.27) and child RSA (M = 5.60, SD = 1.09) were not correlated with each other, nor with negative behavior or repair.

Relations Between Mother- and Child-Initiated Repair and RSA Over Time

Multilevel coupled autoregressive analyses tested how the overall frequency of mother- and child-initiated repair during 10 min of dyadic tasks was associated with change over time in mother and child mean RSA, SR of RSA, and CO of RSA during the same dyadic tasks. Models were conducted separately for mother RSA in maltreating dyads, mother RSA in nonmaltreating dyads, child RSA in maltreating dyads, and child RSA in nonmaltreating dyads, resulting in four models (Table 1). For both maltreating and nonmaltreating dyads, all intercepts for the effects of mean RSA and SR of RSA were significant in relation to current RSA for both mothers and children. With respect to RSA CO, intercepts were positive or trending for nonmaltreating dyads and nonsignificant for maltreating dyads. Collectively, these findings replicated prior work illustrating stability in one's own RSA over time, positive concordance in RSA for typical mother–child dyads, and no concordance in RSA for maltreating dyads, provided the maltreating group is not broken down by maltreatment subtype (Creaven et al., 2014; Lunkenheimer, Busuito, et al., 2018).

For nonmaltreating mother–child dyads, mother-initiated repair had multiple associations with mother and child RSA. Mother-initiated repair was significantly negatively related to children's SR of RSA, reflected as children's decreasing RSA over the course of the tasks. Thus, higher levels of mother repair were related to children's greater RSA withdrawal, reflecting the child's greater regulatory challenge over the course of the tasks. Mother-initiated repair was also observed more frequently in the presence of weakening RSA concordance for mothers (the effect of child on mother) and to marginally significantly weaker RSA CO for children (the effect of mother on child), reflecting weaker concordance in mother and child RSA associated with greater social challenge. Child-initiated repair in nonmaltreating dyads was marginally negatively associated with decreasing child RSA over time, which could reflect the greater regulatory load associated with the effort to repair conflict with mothers. Mother-initiated repair was not associated with mean mother and child RSA, and child-initiated repair was not associated with mean RSA nor concordance in RSA for mother or child.

For maltreating mother–child dyads, mother-initiated repair was positively associated with children's SR of RSA, reflected as children's increasing RSA over the course of the tasks. Thus, higher levels of mother repair were related to children's greater RSA augmentation, reflecting children experiencing less regulatory challenge over the course of the dyadic tasks. Child-initiated repair was negatively associated with mothers' SR of RSA, reflected as mothers' decreasing RSA over the course of the tasks. Thus, higher levels of child repair

were related to mothers' greater RSA withdrawal, reflecting greater regulatory challenge over the course of the dyadic tasks with their children. Child-initiated repair was also marginally negatively associated with dyadic CO of RSA, such that greater child repair was related to negative concordance in RSA. Thus, although maltreating mother–child dyads showed no concordance in RSA overall, there was a trend toward negative concordance (i.e., divergence) at higher levels of child-initiated repair. Child-initiated repair was not associated with mean mother and child RSA, and mother-initiated repair was not associated with mean RSA nor concordance in RSA for mother or child in maltreating dyads.

The aforementioned coupled autoregressive analyses examined linear relations between Levels 1 and 2 of the multilevel model; to further unpack the effects found, mother and child RSA patterns predicted by the models were graphed according to high (+1 *SD*) and low (−1 SD) levels of interactive repair, separately by maltreatment status and repair initiator (Figure 1). The effects of all primary variables in the model (repair, mean RSA, SR of RSA, and CO of RSA) were included in estimating graphical representations of mother and child RSA time-series data. These graphs illustrated that (a) lower mother-initiated repair was related to group differences, associated with decreases in child RSA over time in nonmaltreating dyads versus increases in child RSA over time in maltreating dyads; (b) in comparison, higher mother-initiated repair in both maltreating and nonmaltreating dyads was associated with little change in RSA for mother or child, suggesting a potential buffering effect of higher mother repair; and (c) higher child-initiated repair in maltreating dyads was associated with higher than average mother mean RSA, whereas lower child-initiated repair was associated with decreases in mother RSA over time, suggesting a potential protective effect of child repair on maltreating mothers' neurobiology.

Discussion

In maltreating families, relationship processes are altered and caregiving expectations are violated. Given heterogeneity in child maltreatment risk and incidence, it is worth researchers' efforts to examine whether commonalities in these relationship disruptions can better inform our understanding of child maltreatment. Getting under the skin to understand the neurobiological regulatory processes that underlie relationship disturbances may be fruitful in delineating atypical developmental mechanisms. We argued that dyadic interactive repair could be challenging in maltreating families and accordingly had the potential to reveal neurobiological regulatory differences by maltreatment status. We found that there were both commonalities and differences across groups in the relations between the frequency of interactive repair and patterns of individual and dyadic mother and child RSA over time.

In nonmaltreating dyads, lower mother-initiated repair was associated with children's declining RSA and weaker dyadic concordance in RSA. Further probing revealed that weaker dyadic concordance was a function of the declines in individual child RSA, and child RSA declines were demonstrated at a 1 SD unit decrease in mother repair. In contrast, higher mother repair was associated with little change in mother or child RSA. This suggests that maternal repair of conflict is a developmentally normative and expected caregiving process in the preschool years (Kemp et al., 2016) and offers new evidence that when it is low or

absent, particularly during challenging problem-solving interactions in high-risk families, children experience greater challenge indexed by their regulatory neurobiology. This finding is in line with prior theory on the importance of mothers serving an external regulatory function via sensitive and timely responding to the child's needs, the absence of which is linked with biological and behavioral challenges (Feldman, 2007). Given that RSA declines are expected in response to social stressors (Shahrestani et al., 2014) and mother–preschooler dyadic RSA concordance weakens during more challenging tasks (Lunkenheimer, Tiberio, et al., 2018), this finding implies a buffering effect of mother-initiated repair on child regulatory neurobiology in the context of challenging mother–preschooler interactions.

Interestingly, maltreating dyads also showed little change in mother or child RSA at higher levels of mother-initiated repair, implying that this buffering effect may also operate in maltreating dyads. We also found no differences in overall rates or success of repair or in repair initiators by maltreatment status. This latter finding ran contrary to prior research, showing less overall repair and greater child initiation of repair in maltreating families (Skowron et al., 2010). However, the present study also showed lower total rates of repair than prior studies, with an average of only 26.4% negative behaviors repaired as compared to 50% or more in prior work (Kemp et al., 2016; Skowron et al., 2010). Although we operationalized repair similarly, commonalities in the rates and protective effects of mother repair across groups could be considered in light of the homogeneity of the sample characteristics including majority White, low-income families in both maltreating and nonmaltreating groups. Also, constraining analysis to dyads who had the opportunity to repair could lead to differences in rates across studies. Regardless, the implication that mother repair may operate similarly in maltreating families and may buffer children from regulatory challenges, when it occurs, is promising for family-based intervention. For example, interventions shown to help maltreating parents recognize a rupture in the parent–child relationship (Gurney-Smith, Granger, Randle, & Fletcher, 2010) could potentially indirectly support neurobiological regulation in addition to targeted behavioral changes.

In contrast, when mother-initiated repair was low in maltreating families, children showed increases, or augmentation, in RSA over time, in contrast to the decreases seen in nonmaltreated children. RSA augmentation in response to social challenge is considered maladaptive and associated with social disengagement (Thayer & Lane, 2000) and linked to higher levels of developmental psychopathology in children (Kahle & Hastings, 2015). Prior research suggests that children's RSA augmentation in challenges with mothers is associated with higher child neglect, which may reflect adaptive disengagement in cases where support is not available (Lunkenheimer, Busuito, et al., 2018). Thus, this finding suggested a qualitative difference in regulatory neurobiology associated with interactive repair across maltreating and nonmaltreating families. It expands upon prior work to suggest that perhaps interpersonal mechanisms by which early regulatory neurobiology is altered in maltreating families are not only major violations such as child maltreatment episodes but also the ongoing or cumulative absence of expected positive relationship processes day-to-day between parent and child.

Differences in regulatory neurobiology by maltreatment status were also exemplified by the effects of child-initiated repair on maltreating mothers. At high levels, child repair was associated with higher mother mean RSA, whereas at low levels, it was associated with mother RSA withdrawal. The implication is that child repair is protective for maltreating mothers' regulatory burden in challenging tasks with their children. Maltreating mothers have shown low and declining RSA in tasks with children, related to the greater effort needed for them to support parenting behaviors (Skowron et al., 2013). Maltreated children have also shown role reversal, conducting the work of interactive repair that their parents are less able to provide (Macfie et al., 2008). The adaptive calibration model (Del Giudice, Ellis, & Shirtcliff, 2011) suggests children exposed to adversity adapt to threatening situations in the short term, but that these adaptations tax their resources for the long term, particularly their stress neurobiology. To date, a largely unanswered question is what role moment-to-moment relationship processes play in this neurobiological embedding of adversity. The present findings suggest that preschoolers conducting the work of repair for their maltreating parents may be a candidate for a calibration process associated with alterations to children's regulatory neurobiology, but it is not yet clear how this operates. We found a marginally significant negative association between child repair and RSA CO, indicating that at higher child repair, mother RSA had negative effects on child RSA. Thus, one possibility is that the presence of child repair buffers mother RSA, which in turn drives down child RSA, implying a resulting physiological cost to the child. Future work will be needed to determine whether and when children's neurobiological alterations emerge and whether the developmental sequelae of such alterations are maladaptive in anticipated ways. As the parasympathetic nervous system is only one aspect of regulatory neurobiology, this work must also consider other systems integral to the biological embedding of adversity, such as neural, immune, endocrine, and metabolic systems, as well as interrelations among these systems (Berens, Jensen, & Nelson, 2017).

Limitations and Future Directions

By restricting analysis only to dyads that had a chance to repair and testing differences by maltreatment status and mother versus child RSA, some subgroups were small and may have limited power to detect effects. The frequency of child-initiated repair was low. Our repair definition was highly specific in order to make stronger claims about dyadic sequences and distinguish repair from negative behavior, but this choice also limited episodes for analysis. Most work to date has centered on linear changes in RSA in relation to environmental risk and stress conditions (e.g., Calkins & Keane, 2004; Moore et al., 2009), but an interesting direction for future research is whether RSA changes in nonlinear ways in response to stress or whether relations between RSA and social behavior operate on a shorter time scale, for example, in the seconds before, during, or after the moment of repair. Due to the relatively low base rate of repair and the calculation of RSA in 30-s bins, we were not able to examine how RSA corresponded with precise moments of repair. The present analysis was correlational, and thus we could not make conclusions about causal relations between interactive repair and regulatory neurobiology. For example, it is equally possible that repair is a response to a partner's greater stress response as compared to a driver of changes in regulatory neurobiology in real time. Future longitudinal work would help to delineate causal relations among these processes.

We were also statistically underpowered to examine differences by maltreatment subtype (physical abuse vs. neglect), but this could be an important next step, given that research suggests subtype differences in RSA and behavior (Lunkenheimer, Busuito, et al., 2018; Skowron et al., 2013). These qualitative differences may be why our preliminary analyses examining differences by overall maltreatment status were not significant, if differences between physically abusing and neglectful families washed out the effects by overall status. To conclude that the present differences found are in fact due to maltreatment status and not subtype, future work could replicate the present study on larger samples distinguished by subtype; replication would also help to ensure that group differences found in the present work were not overly inflated by the use of separate models by maltreatment status. Also, other variables not addressed could have affected the processes of interest such as attachment status, SR skills, and mother and child perceptions of conflict or task difficulty. Additionally, our sample was limited to primarily low-income White families, which limits generalizability to other socioeconomic and ethnic groups. Despite these limitations, the present findings offer exciting initial evidence regarding the role that interactive relationship processes in maltreating families play in the neurobiological embedding of early adversity. This work could inform novel directions for family-based intervention that centers on improving parent–child interactions and children's regulatory processes in maltreating families.

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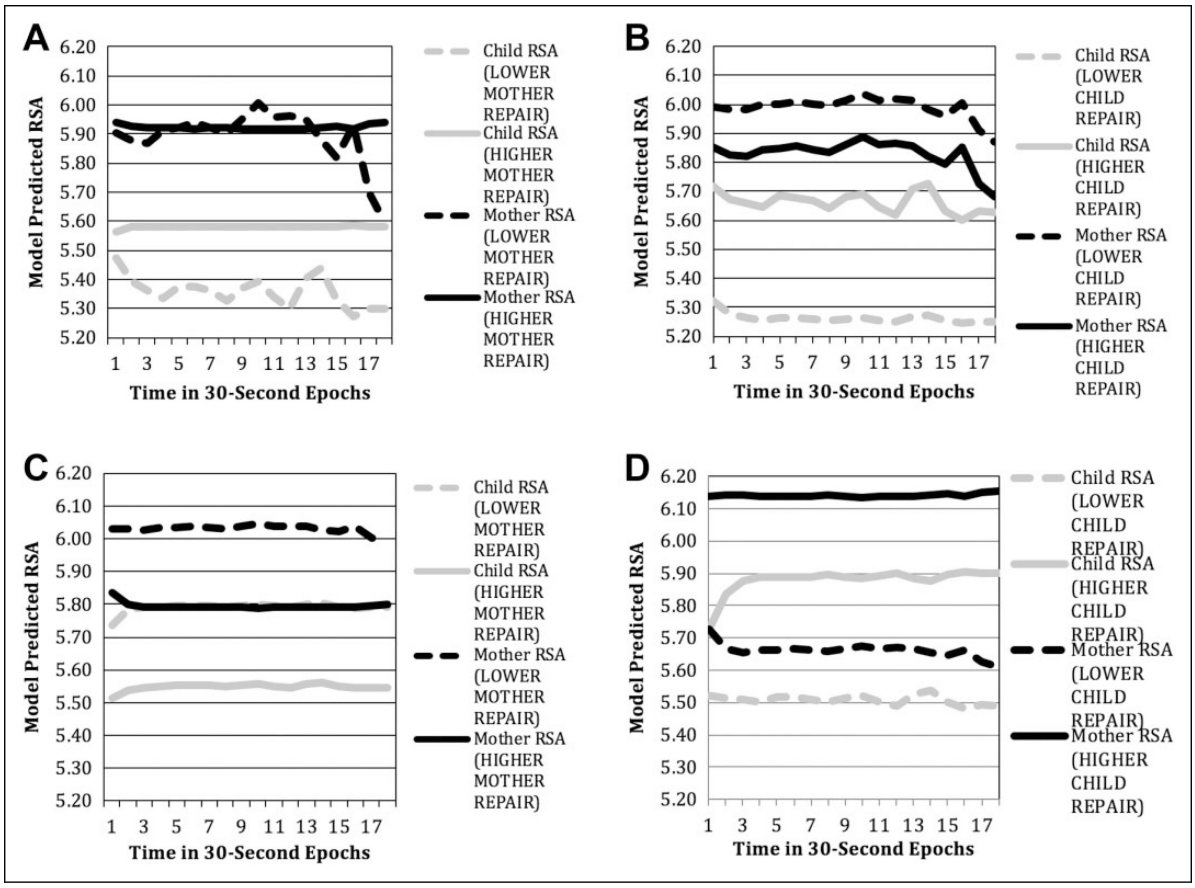

Figure 1.

Model-predicted RSA by maltreatment status and repair initiator. (A) Nonmaltreating dyads—mother repair. (B) Nonmaltreating dyads—child repair. (C) Maltreating dyads—mother repair. (D) Maltreating dyads—child repair. Higher = +1 *SD* and lower = −1 *SD*. SD= standard deviation; RSA = respiratory sinus arrhythmia.

Table 1.

Effects of Mother- and Child-Initiated Dyadic Repair on Mother and Child Respiratory Sinus Arrhythmia, Separately by Maltreatment Status.

Effects	Nonmaltreating Mother RSA (N = 42)	Nonmaltreating Child RSA (N = 41)	Maltreating Mother RSA (N = 59)	Maltreating Child RSA (N = 59)
Estimated intercepts				
Mean RSA	5.966 (.152)***	5.514 (.138)***	5.927 (.146)***	5.658 (.096)***
RSA self-regulation	0.159 (.059)**	0.185 (.057)**	0.118 (.061)*	0.284 (.065)***
RSA coregulation	0.111 (.057)*	0.108 (.062)†	0.014 (.036)	0.019 (.042)
Estimated regression coefficients and variance components				
Level 1 (time)				
Level-1 residual variance	0.550 (.056)***	0.710 (.097)***	0.555 (.051)***	0.687 (.103)***
Level 2 (interactive repair)				
Mother-initiated repair				
Mean RSA	−0.037 (.132)	0.050 (.100)	−0.087 (.118)	−0.075 (.084)
RSA self-regulation	−0.043 (.035)	−0.069 (.033)*	0.034 (.048)	0.074 (.028)**
RSA coregulation	−0.097 (.048)*	−0.088 (.049)†	−0.015 (.042)	0.001 (.039)
Child-initiated repair				
Mean RSA	−0.096 (.246)	0.263 (.250)	0.310 (.291)	0.192 (.125)
RSA Self-regulation	−0.023 (.054)	−0.122 (.070)†	−.101 (.048)*	0.060 (.048)
RSA coregulation	0.020 (.065)	0.111 (.08l)	−0.040 (.042)	−0.086 (.052)†
Level-2 residual variance	0.927 (.258)***	0.674 (.207)**	1.177 (.312)***	0.484 (.140)**
Intraclass correlation	0.597	0.437	0.689	0.400

Note. Values in table denote *b(SE)* unless noted otherwise. RSA = respiratory sinus arrhythmia; *SE* = standard error.

† $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.