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Young Children's Physiological Reactivity During Memory Recall: Associations with Posttraumatic Stress and Parent Physiological Synchrony

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Abstract

Autonomic reactivity is implicated in stress response and social engagement – both key components of posttraumatic stress disorder (PTSD) – but few studies have examined autonomic reactivity in pediatric samples, and no known studies have examined physiological synchrony among children with PTSD and caregivers. In a sample of 247 young children (94 girls, 153 boys), most (85%) of whom had exposure to trauma and 40% who met criteria for PTSD, we examined children's patterns of respiratory sinus arrhythmia (RSA) at baseline and in response to a memory recall task, as well as correspondence between parents' and children's RSA. Children with PTSD demonstrated significantly higher reactivity than other groups during their recollection of a traumatic memory, but not during other memory tasks. Regarding synchrony, caregivers' and children's RSA were more significantly and positively correlated during the trauma recall task among children who had had exposure to a potentially traumatic event but did not meet PTSD criteria, suggesting physiological synchrony may be protective in contexts of trauma. Overall, findings demonstrate physiological reactivity differences among young children with PTSD. While more work is needed to understand the meaning of parent-child physiological synchrony, these data suggest that children's psychopathology is associated with physiological synchrony processes among young children with exposure to trauma.

Keywords

Trauma; Posttraumatic Stress Disorder; Parent-Child Relationship; Respiratory Sinus Arrhythmia; Emotion Regulation; Vagal Tone

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Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

Potentially traumatic events, even in early childhood, are not rare occurrences (Briggs-Gowan et al., 2010), and for many children, are followed by post-traumatic stress symptoms that do not spontaneously remit. Posttraumatic stress disorder (PTSD) can be reliably diagnosed even among young children with developmentally appropriate criteria (Scheeringa, 2008). Reactions to trauma reminders, as well as indicators of physiological arousal such as enhanced startle response and hypervigilance, are core symptoms of PTSD, suggesting that physiology may differentiate diagnostically between individuals with and without the disorder. Indeed, the adult literature suggests that physiological response differentiates PTSD from non-PTSD subjects (Cohen et al., 1998; Hauschildt, Peters, Moritz, & Jelinek, 2011) – but there is a relative lack of research on physiological reactivity in pediatric PTSD (Grasso & Simons, 2012). The parasympathetic nervous system (PNS) marker of respiratory sinus arrhythmia (RSA) in particular indexes self-regulation and is associated with stress and anxiety (Beauchaine & Thayer, 2015) – hallmark processes related to PTSD – however, very little work describes RSA patterns among children with PTSD (Kirsch, Wilhelm, & Goldbeck, 2015; Scheeringa, Zeanah, Myers, & Putnam, 2004). Understanding physiological reactivity in the presentation of pediatric PTSD may help to further describe phenotype, as well as potential biological markers to guide clinical prevention and intervention.

Moreover, although relations between parents' and children's physiological reactivity have shown interesting patterns in relation to children's risk and resilience (Giuliano, Skowron, & Berkman, 2015; Woody et al., 2016), no known reports have investigated parent-child RSA synchrony in children with PTSD. Given the theoretical and empirical links between parent-child relationship processes and both RSA (Kennedy, Rubin, Hastings, & Maisel, 2004) and PTSD (Scheeringa & Zeanah, 2001), understanding how parents' and children's autonomic physiology relates in contexts of PTSD may illuminate dyadic processes that may additionally inform prevention and intervention. This study integrates across physiological and relational frameworks of understanding PTSD, investigating young children's autonomic reactivity to a trauma reminder task, as well as how caregivers' and children's physiology relate to one another in a sample with high exposure to potentially traumatic events.

PTSD and PNS Activity

Studies with adults have demonstrated links between posttraumatic stress disorder (PTSD) and altered autonomic system reactivity in response to trauma reminders (Cohen et al., 1998), including RSA, a reliable marker of high-frequency heart rate variability (HF-HRV) thought to index parasympathetic control of the heart via the vagus nerve (Porges, 2007). RSA can be quantified both at rest (tonic response), with higher RSA indexing greater vagal control, as well as during evocative stimuli (phasic response) such as trauma-specific stimuli. A substantial body of work associates higher resting (tonic) RSA with enhanced emotion regulation, whereas lower resting RSA has been associated with anxiety disorders (Beauchaine, 2015; Beauchaine & Thayer, 2015; Cohen et al., 2000). Regarding phasic shifts in RSA, Porges's polyvagal theory posits that withdrawing RSA under stress may adaptively prepare the body for activation and response to the stressful context (Porges, 2007); however, others have further noted that excessive RSA withdrawal can be associated

with higher psychopathology, including among children (Beauchaine, 2001; Calkins, Graziano, & Keane, 2007). Others have suggested that HF-HRV, indexed by RSA, may serve as a transdiagnostic marker for self-regulation and cognitive control (Beauchaine & Thayer, 2015); however, we know little about patterns of HF-HRV in pediatric samples of children with PTSD, limiting our understanding of the developmental course of HF-HRV in posttraumatic stress. Importantly, PNS activity acts in concert with sympathetic nervous system (SNS) activity, which some evidence suggests may relate to psychological outcomes in contexts of trauma; for example, a prospective study on the Boston Marathon bombing indicated that youth with heightened SNS activity prior to the bombing interacted with media exposure to predict PTSD symptom development (Busso, McLaughlin, & Sheridan, 2014).

Among adults, PTSD diagnosis has been associated with lower RSA at rest (Cohen et al., 1998; Hauschildt et al., 2011). A twin study on combat-related PTSD has suggested that alteration in PNS activity may be a component of PTSD rather than reflective of a genetic predisposition that may put individuals at risk (Shah et al., 2013). In addition, alterations in RSA reactivity to stressors, including traumatic reminders, have been noted among adult PTSD samples. However, patterns of RSA reactivity are inconsistent: both increased and blunted reactivity have been observed among adults with PTSD (Cohen et al., 1998; Cohen et al., 2000; Hauschildt et al., 2011; Sack, Hopper, & Lamprecht, 2004).

In the only two known studies of RSA in pediatric PTSD populations, PTSD-specific effects have not been found. One study among children aged 6–17 with exposure to trauma ($n = 34$; Kirsch et al., 2015) failed to note differences by PTSD vs. control children in RSA during a trauma script task. Additionally, Scheeringa et al., 2004 found no differences between PTSD vs. non-PTSD trauma exposed groups, though they did find differences between trauma-exposed children and controls ($n = 117$). This dearth of literature in PNS activity in pediatric PTSD is despite the conceptual and empirical links between PNS physiology and children's psychopathology broadly, as well as between PNS physiology and self-regulation and cognitive control specifically (Beauchaine & Thayer, 2015; Beauchaine, 2015) – both hallmark features of posttraumatic stress presentation (Frewen & Lanius, 2006). The current study seeks to extend these studies to a larger sample of younger children, examining reactivity across a range of memory recall tasks.

Physiological Synchrony

In addition to its demonstrated association to psychopathology and psychophysiological arousal, RSA is associated with social interactions. As theorized by Porges in his polyvagal theory, dynamic parasympathetic control of arousal supports appropriate engagement in social interactions (Porges, 2007) – including parenting and parent-child interactions (Skowron, Cipriano-Essel, Benjamin, Pincus, & Van Ryzin, 2013). Dyadic and interactive stressors have been shown to be associated with decreases in RSA, which, according to polyvagal theory, are consistent with a social disengagement and resultant emotion regulation through parasympathetic de-activation (Porges, 2007). To some extent, patterns related to RSA suppression during social disengagement appear to vary in contexts of psychopathology, with a recent meta-analysis showing that children with psychopathology

are less likely to suppress during social stressors than children without psychopathology (Shahrestani, Stewart, Quintana, Hickie, & Guastella, 2014). Thus, as a physiological marker, RSA holds interest both for psychopathology and for dyadic social processes.

This coupling of psychopathology and social relations vis a vis RSA makes it a particularly interesting marker in the context of childhood PTSD. The family environment has been implicated in the emergence and maintenance of posttraumatic stress in early childhood, suggesting that looking to relationship-level factors may illuminate PTSD's course (Scheeringa & Zeanah, 2001; Thakar, Coffino, & Lieberman, 2013), although data on this hypothesis have been mixed (Feldman & Vengrober, 2011; Scheeringa, Myers, Putnam, & Zeanah, 2015). Physiologically, synchrony or – correspondence between parent and child physiological responses, as well as their affect and behavior – is a process that integrates across parent-child relationship processes and physiology. Synchrony begins in infancy as mothers and children learn to coordinate affective and physiological states. In general, evidence suggests that physiological synchrony is a health-promoting factor (Feldman, 2007), with meaningful correspondence to relationship quality.

In the broader developmental literature, studies have shown that physiological synchrony tends to be less evident in relationships qualified by risk factors. For example, in dyadic challenge tasks with toddlers and mothers, cortisol synchrony was observed only among dyads with highly sensitive mothers (Sethre-Hofstad, Stansbury, & Rice, 2002). Similarly, among young children, parent-child RSA was less synchronous over the course of an interaction in dyads marked by children's externalizing behavior (Lunkenheimer et al., 2015).

No studies that we are aware of have examined the relation between caregiver and child RSA in the context of child PTSD. Still, evidence from studies of children with exposure to adversity suggest that examining the family context in concert with RSA may yield interesting results. In the one previous study that has examined PTSD diagnosis in relation to young children's RSA, an interaction between PTSD symptoms and parents' positive discipline was observed: children with caregivers who demonstrated less positive discipline during a clean-up task had higher PTSD symptoms when they demonstrated strong RSA reactivity (Scheeringa et al., 2004). In other, non-PTSD samples, maltreatment and quality of observed relationship were both linked to dampened tonic RSA among young children (Skowron et al., 2011), and mothers with histories of maltreatment, an extreme form of negative discipline, have been shown to exhibit RSA decreases following hostile behavior towards children (Skowron et al., 2013).

Thus, correspondence between parent and child RSA has demonstrated meaningful correspondence to psychopathology and family dynamics (Lunkenheimer et al., 2015; Woody et al., 2016). Given evidence of the role of the parent-child relationship in children's posttraumatic stress reactions (Feldman & Vengrober, 2011), parent-child physiological synchrony seems appropriate for investigation in contexts of PTSD.

The Current Study

The current study was designed to address the above-reviewed gaps in the literature. Specifically, it leverages a study on young children's PTSD to examine children's RSA reactivity patterns in the context of a memory recall task, and preliminarily to examine relations between children's RSA and parents' RSA. We asked two questions:

Question 1: Does child RSA change in response to memory recall, and if so, does this vary for children with PTSD? We examined reactivity to children's recollection of a pleasant recall, children's recollection of an unpleasant or traumatic memory, and parent's recollection of the same unpleasant or traumatic memory. Given that both blunted and increased reactivity have been observed in the adult literature, directional hypotheses were not made.

Question 2: Do caregivers' and children's RSA correspond across tasks, and do these patterns vary according to children's trauma exposure or diagnostic status? Based on work from other samples with psychopathology demonstrating lower synchrony among dyads marked by psychopathology (e.g., Woody et al., 2016), we hypothesized that parents' and children's RSA would be lower in dyads where the children had PTSD.

Method

Procedures

Data come from a larger study focused on PTSD in very young children. Study visits were conducted at a university lab site. Study procedures were approved by the university IRB. Signed informed consent was obtained from primary caregivers, who were compensated for participation.

Participants

The analytic sample for this report included 247 female caregivers and young children (94 girls and 153 boys) who were between ages 3–6 years (mean age = 5.08 years, $SD = 1.06$) and who had valid electrocardiogram (EKG) data; 67% of children were identified by parents as Black, 19% as White, and 14% as Mixed Race or Other. Participants were recruited from a range of community outreach techniques, including newspaper advertisements and flyers in pediatrician's offices, as well as direct recruitment through a Level I Trauma Center registry and three battered women's programs in the New Orleans area. Exclusion criteria included head trauma with Glasgow Coma Scale score ≤ 7 ; intellectual disability or autistic disorder by caregiver report or clinician observation; blindness; deafness; and non-English speaking family; and trauma occurred prior to 36 months of age.

At study intake, participants were intentionally sampled across a range of potentially traumatic exposures. Some children ($n = 62$) had single incident exposures such as accidental injuries or witnessing community assaults. Others experienced Hurricane Katrina ($n = 137$). Still others ($n = 85$) had exposure to repeated potentially traumatic events, primarily domestic violence. Additionally, trauma-exposed participants were asked for

neighbors and acquaintances with children in the 3–6 year old age range and families were then contacted by phone; through this method, a comparison group of children ($n = 46$) with no parent-reported exposure to potentially traumatic events was recruited. For more details on the sample, see Scheeringa, Myers, Putnam, & Zeanah (2012). The study consisted of two visits to the lab spaced 1 week apart. Of these 330 participants, 264 returned for the second visit when the RSA data were collected (223 trauma group and 41 comparison group). Data for some stimuli were missing due to technical difficulties or lack of cooperation from the children. Valid RSA baseline data for all stimuli were available on 247 children (38 comparison group, 209 trauma; 52 single incident, 54 repeated exposure, 103 Hurricane Katrina).

Measures

The *Preschool-Aged Psychiatric Assessment* (PAPA; Egger et al., 2006), a semi-structured diagnostic interview, was used to assess child PTSD diagnosis, including exposure to potentially traumatic events. Caregivers provided information about exposure to 12 life events, DSM-IV criteria for PTSD, as well as developmentally modified symptom-level criteria consistent with the PTSD for children 6 years and younger criteria in DSM-5. Test-retest reliability for PTSD using the PAPA in a community sample of 307 preschool-aged children was .73. For this study, diagnoses of PTSD were determined using the diagnostic algorithm for PTSD in children six years and younger in the DSM-5, which required one intrusive symptom, one avoidance or numbing symptom, and two increased arousal symptoms, in addition to functional impairment.

EKG data were collected from caregivers and children simultaneously across a range of tasks using equipment and software the James Long Company. Electrodes were placed axially on left and right rib cage and centrally on the stomach. The bioamplifier was set for bandpass filtering at frequencies of .1 and 1000 Hz. The rising edges of R waves were automatically identified with a multiple-pass, self-scaling algorithm and were then visually inspected and corrected manually when possible. Distances between successive R waves were the interbeat intervals (IBI).

Caregivers and children were asked to sit still for approximately 45–50 seconds to watch an affectively neutral scene of an airplane flying accompanied by soft music, used as baseline in this report. Immediately following, caregiver and child participated in a memory recall task with study personnel (Fivush & Hudson, 1990). First, children completed a 3-minute semi-structured interview regarding a remote pleasant memory of a real-life, pleasant event that occurred within the past 2–6 months, such as a trip to an amusement park or zoo. Next, children were asked to recall an unpleasant event. For the children with exposure to potentially traumatic events, the memory of the event identified by parents as the “worst event” in PTSD diagnostic interviewing was used; for children without a potentially traumatic experience, children were asked to recall unpleasant events from their past, such as trips to the hospital or falling off a bike. Component features assessed in the interview included location, children’s activity at the time, other persons present, method of injury if injury present, children’s feelings at the time, and children’s outward reactions at the time; again, recall task was 3 minutes. Finally, parents were interviewed for 3 minutes about their

recall of their children's unpleasant events. Across all memory tasks, the interviewer began with open-ended questions, progressed to more specific questions, then asked yes-no questions (Merritt, Ornstein, & Spicker, 1994); interviewers used probing to ensure that parents and children remained on-task for memory recall. Parent and child EKG were collected simultaneously across tasks.

Respiratory Sinus Arrhythmia data were derived from IBI data using spectral analyses. Prorated IBIs were detrended with a high-pass filter, then Fourier analyses were applied to residual IBI data for each epoch. RSA was estimated from power spectral analysis, using the 0.3–0.75 Hz band for 3 year-old children, the 0.2–0.65 Hz frequency band for older children, and the 0.12–0.40 Hz band for adults (Bar-Haim, Marshall, & Fox, 2000). Data were non-normal and were log-transformed for analyses. Because shifts in tonic RSA across development in early childhood have been noted (Alkon et al., 2003) and, less consistently, sex differences (Beauchaine, Hong, & Marsh, 2008; Gray, Theall, Lipschutz & Drury, 2016), age and sex were included as potential covariates in analyses using RSA.

Results

Preliminary Analyses

Of all 247 participating young children with valid RSA data, 98 met criteria for PTSD using DSM-5 algorithms (39.8% of the overall sample, 47% of sample exposed to potentially traumatic events; boys $n = 59$, girls $n = 39$); correspondingly, 111 children exposed to potentially traumatic events did not meet criteria for PTSD with impairment (53% of the sample exposed to potentially traumatic events; boys $n = 68$, girls $n = 43$). Trauma groupings for this report therefore included children who met criteria for PTSD (PTSD group; $n = 98$), children with exposure to a potentially traumatic event who did not meet criteria for PTSD (exposure no PTSD group; $n = 111$); and a comparison, no trauma exposure or PTSD group ($n = 38$).

Among children who had exposure to a single-incident trauma, 48% met criteria for PTSD ($n = 75$); among the repeated exposure group, the rate was 43% ($n = 23$); this group difference was not significant, $\chi^2(1, N = 247) = 0.66, p = .44$. There were no PTSD vs. non-PTSD group differences on child age, sex, or race; caregivers' employment or education; or father living in the home, $ps = .23-.63$. Caregivers of children with PTSD were older on average, $t(246) = 3.48, p < .00$. Looking across trauma exposure groups (single vs. repeated vs. no exposure), there were no differences on child age or sex; caregivers in the no exposure group were on average younger than single-incident exposure caregivers, $p = .02$, and less well-educated than either of the exposed groups, $ps = .02, < .00$; given recruitment strategies from battered women's shelters, it is not surprising that the repeated traumas group was less likely than either other group to have fathers residing with children, $ps < .00$. Finally, children in the comparison, no exposure group were marginally more likely to be White than children in the repeated trauma group, $p = .05$. See Table 1 for descriptive data and correlations between study variables. Valid parent RSA data was available for 242 of the 247 children. Correlations between parent and child RSA across tasks are reported in Table 2.

Question 1: Does Child RSA Change in Response to Recall Tasks, and if so, Does this Vary for Children with PTSD?

A repeated measures mixed model analysis using the PROC MIXED statement in SAS 9.4 was used to assess change from baseline to recall tasks, as well as between-group differences across comparison, exposure without PTSD, and PTSD groups. Separate models were run from baseline to each recall task. Child trauma grouping was examined as a between subjects factor (comparison; exposure without PTSD; and PTSD), and child age and sex were included as covariates. Because the AIC and BIC values indicated improved fit, compound symmetry covariance structure was used.

From baseline to all three recall tasks, a significant main effect of time was observed, suggesting that on average, children's RSA was significantly lower from baseline during their recall of the pleasant memory, their own recall of the unpleasant/traumatic memory, and their parent's recall of the unpleasant/traumatic memory (see Table 3). No main effects of group were observed in any recall task, suggesting no average differences in RSA across the trauma groups (comparison no trauma, exposure no PTSD, and PTSD; see Table 3). A main effect of age was observed, with older age associated with higher RSA. Sex differences were in the direction of girls demonstrating higher RSA, but did not reach statistical significance, $ps = .07-.18$.

Additionally, a significant time-by-group interaction was observed only in the context of children's own recall of an unpleasant or potentially traumatic memory, $F(2, 235) = 3.05$, $p = .05$. During recall of a potentially traumatic event, children in the PTSD group demonstrated a significantly steeper rate of decline (stronger RSA reactivity or suppression) compared to other children with trauma exposure but no PTSD (see Table 3, Figure 1).

Question 2: Do Caregivers' and Children's RSA Correspond Across Recall Tasks, and Do These Patterns Vary for Children with PTSD?

To examine whether children and caregivers' RSA values corresponded during the recall tasks, we ran a series of generalized linear models for each task (baseline, pleasant recall, child unpleasant/trauma recall, parent unpleasant/trauma recall). For each model, child RSA during the specified epoch was the outcome; predictors included caregivers' RSA during the specified epoch, trauma grouping (comparison no trauma, exposure / no PTSD, and PTSD), a caregiver RSA-by-group interaction, and child sex and age as covariates. When all variables were included in the model, no main effects of caregiver RSA on child RSA were observed in any task; however, significant Caregiver RSA * Group interactions were observed in both the child unpleasant/trauma recall, $F(2, 235) = 3.05$, $p = .05$, and the caregiver unpleasant/trauma recall tasks, $F(2, 235) = 4.90$, $p = .01$, suggesting that caregiver RSA relates differentially to child RSA depending on trauma status only during the trauma recall tasks. Covariates of age were significant in all models, and sex in the trauma recall task models only; however, patterns did not vary by age or sex (3-way interactions all *ns*).

To decompose these interactions, we examined partial correlations between caregiver and child RSA during recall tasks stratified by trauma group, controlling for child age and sex. To compare correlation values within task but across trauma grouping, the Fisher *r*-to-*z*

transformation was used (see Table 4). During children's recollection of their own unpleasant or traumatic memory, children with trauma exposure but *without* PTSD demonstrated significantly higher physiological synchrony with their caregivers than children who met criteria for PTSD, and also than comparison children with no trauma. During the caregiver's recollection of the children's unpleasant or traumatic events, children with no trauma exposure demonstrated significantly lower physiological synchrony with their caregivers than either of the trauma-exposed groups. Children with exposure to potentially traumatic events, including both those who met criteria for PTSD and those who did not, on average had RSA values that were positively correlated with their caregivers' RSA during recall tasks, whereas correlation coefficients for children's and caregivers' RSA among dyads in the no trauma exposure, comparison group were negative in direction, though not significant.

Discussion

This report examined very young children's autonomic reactivity during a recall task, examining both group differences by trauma grouping (no exposure to potentially traumatic event, exposure but no PTSD, and exposure with PTSD) as well as the relation between children's and caregivers' RSA. On average, all children in this sample demonstrated autonomic reactivity during recall tasks, suppressing RSA compared to baseline during the task of pleasant memories, their own recollection of unpleasant or traumatic memories, and their parents' recollection of the same unpleasant or traumatic event. However, children with PTSD demonstrated greater RSA reactivity than children without PTSD, but only during their own recollection of traumatic memories.

This finding contributes to the limited other work in autonomic reactivity among children with PTSD, here observing differences that were not observed in smaller and less diverse samples (Kirsch et al., 2015; Scheeringa et al., 2004). While previous work on RSA broadly has suggested that stronger RSA reactivity is adaptive in regards to some outcomes, the patterns observed here are consistent with other emergent data that suggests that *excessive* RSA reactivity may be an indicator of poor emotion regulation capabilities in contexts of psychopathology (Beauchaine, 2015). While a previous meta-analysis found that children from clinical samples had lower levels of RSA reactivity, *within* clinical samples, excessive RSA withdrawal was associated with higher levels of social problems (Graziano & Derefinko, 2013). These data, then, are consistent with the emerging view of excessive RSA reactivity as an indicator of clinical emotion dysregulation (Beauchaine, 2015). The complexity of these patterns in the wider literature reminds us to attend closely to populations studied (clinical vs. community), stimuli used to assess reactivity (trauma-specific, cognitive, affective), and the behavioral features assessed (internalizing, externalizing, social problems).

This is also the first known study to examine physiological synchrony between young children with PTSD and their caregivers. Interesting patterns emerged in the relation between children's and parents' autonomic reactivity. First, specifically during children's recollection of an unpleasant or potentially traumatic event, children with trauma exposure but *not* PTSD demonstrated significantly higher parasympathetic physiological synchrony

with their parents than dyads with PTSD or than dyads with no trauma exposure. Additionally, during the parent's recollection of a potentially traumatic event, children with trauma exposure (both those with and without PTSD) demonstrated significantly higher synchrony than those with no exposure.

Broadly speaking, the finding that dyads characterized by clinically significant psychopathology demonstrated lower levels of synchrony than dyads with exposure but no psychopathology is consistent with the other limited work on psychopathology and physiological synchrony (Lunkenheimer et al., 2015; Woody et al., 2016), where in general, psychopathology is associated with lower synchrony between parents' and children's physiology. Interestingly, in this sample, the children and caregivers with no trauma exposure showed the strongest decoupling of parasympathetic activity; this may in part be due to the salience of the recall task for those dyads, who were asked to recall unpleasant tasks as opposed to potentially traumatic ones. Perhaps most interesting, though, is the significantly higher synchrony patterns in trauma-exposed dyads without PTSD. The positive divergence of this vulnerable but non-clinical group may reflect an effective recruitment of the parent-child relationship to regulate physiologically. Alternatively, it may reflect a protective similarity in parent and child parasympathetic functioning that existed prior to traumatic events that could be due either to shared genetic effects or parent-child bidirectional influences. Examining these processes in greater detail would require multi-method (e.g., behavioral and physiological) and time-sequenced analyses of parent-child physiology to disentangle, but may help to illuminate the developmental course of post-traumatic stress and dysregulation among young children exposed to trauma. A critical next step would be validating these physiological findings with observed parenting measures, such as indicators of warmth, sensitivity, and intrusiveness, which have been linked to children's behavioral self-regulation as well as their RSA functioning (Hastings et al., 2008). Additionally, it may be that parental involvement in and reactions to children's traumatic experiences, as well as parents' own psychopathology, also may be important variables to consider in the link between parents' and children's physiology, and future work may be well served to characterize these processes in more detail to explore potential relations.

Limitations of the current study include a cross-sectional design, which limits understanding of how these relations may unfold temporally across development. Moreover, a limitation of the study methodology is that the recall task may have different effects across subjects who focused on a potentially traumatic stressor, which was the case for dyads for whom children had exposure to a potentially traumatic event, as compared to subjects who focused on a more mild, unpleasant memory, which was the case for the comparison children with no such exposure. However, the observance of differences between the trauma-PTSD and trauma-no-PTSD groups suggests that differences in physiology during the recollection may in fact reflect physiological arousal that corresponds with psychopathology. Our measurement of baseline was shorter than other reports indexing RSA in youth (Musser, Galloway-Long, Frick, & Nigg, 2013).

Additionally, we focused this report on only one index of physiology – RSA – which may be an oversimplification of physiological systems that act in concert with one another to impact psychopathology presentation (El-Sheikh et al., 2011). In the future, research in childhood

PTSD would benefit from the inclusion of both parasympathetic and sympathetic markers of physiological reactivity, as there is evidence that SNS overactivity also may be associated with symptom development following trauma exposure (Busso et al., 2014). Examining these systems in concert—including potential interacting effects – holds promise for yielding interesting mechanistic findings related to physiological reactivity beyond examining any one of these systems in isolation.

Developmentally, these patterns, together with the newly developing work on synchrony and psychopathology from other groups (e.g., Woody et al., 2016) begs the question of how we interpret synchrony – and negative synchrony – in contexts of risk. Our understanding of physiological and behavioral synchrony in infancy has progressed greatly over the past decade (Feldman, 2012); given this and other promising data from early childhood, this work should be extended to understand the developmental unfolding of these patterns over time, in contexts of adversity, to determine the utility of these patterns for developmental psychopathology.

Overall, the present findings address two major conceptual and empirical questions, namely physiological dysregulation phenotypes in pediatric PTSD and the relational context of autonomic regulation in conditions of stress. Our findings were particularly robust for children's recollection of potentially traumatic events, which was the only recall task that differentiated children without PTSD from clinical children, who appeared to be hyper-reactive compared to other children in the context of that task. Additionally, it was during children's recollection of a potentially traumatic event, as well as during parents' recollections of the event, that group differences in physiological synchrony were observed. Together, these findings indicate preliminarily that in contexts of psychopathology, parent and child autonomic activity may be more likely to diverge from one another. Continuing to explore parent-child synchrony patterns in stressed samples across multiple physiological domains may represent a promising area of continued research regarding children's developing physiology and post-traumatic stress. Additionally, given that recalling trauma exposure memories in the presence of a caring adult is a key component of some evidence-based therapies with children with PTSD (Scheeringa, Weems, Cohen, Amaya-Jackson, & Guthrie, 2011), exploring these dyadic and physiological patterns in treatment contexts for children with PTSD may hold promise for illuminating mechanistic markers.

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Figure 1.

Children with PTSD demonstrate higher RSA reactivity during a child traumatic memory recall task.

Note: * indicates slope of line is significantly different between Trauma / No PTSD and Trauma / PTSD groups during second recall task.

Table 1

Correlations and descriptive statistics for study variables.

	Child sex	Child age	Parent age	Child RSA	Parent RSA	Child PTSD symptoms	Child PTSD diagnosis
Child age	.03						
Parent age	.00	.14*					
Child RSA	-.06	.21**	.02				
Parent RSA	-.03	-.10	-.21**	.15*			
Child PTSD symptoms	-.01	.03	.21**	.00	.00		
Child PTSD diagnosis	-.03	.03	.25***	.07	-.01	.69***	
Mean (SD) or %	38% girls	5.08 (1.06)	32.41 (9.71)	4.64 (0.71)	4.11 (0.70)	4.96 (3.53)	40% PTSD diagnosis

Note: Correlations are Pearson's r with exception of dichotomous variables (child sex, PTSD diagnosis) which are point biserial. Age presented in years. Child sex coded as 0 = female, 1 = male; PTSD diagnosis coded as 0 = absent, 1 = present. RSA = baseline RSA value. $N = 247$ except for caregiver RSA, $n = 242$.

Table 2

Correlations between RSA values across task for entire sample.

	1.	2.	3.	4.	5.	6.	7.	8.
1. Child: baseline								
2. Child: pleasant recall	.87***							
3. Child: child unpleasant recall	.83***	.94***						
4. Child: parent unpleasant recall	.85***	.92***	.92***					
5. Parent: baseline	.15*	.15*	.16*	.14*				
6. Parent: pleasant recall	.15*	.16*	.16*	.16*	.76***			
7. Parent: child unpleasant recall	.20**	.18**	.20**	.19**	.77***	.89***		
8. Parent: parent unpleasant recall	.20**	.17**	.18**	.19**	.66***	.84***	.83***	
Mean (SD)	4.64 (0.71)	4.44 (0.70)	4.39 (0.71)	4.46 (0.71)	4.11 (0.70)	4.12 (0.71)	4.09 (0.69)	4.30 (0.62)

Note: Correlations are Pearson's *r*.

Table 3

Repeated measures mixed model analysis of RSA suggests children with PTSD show differential reactivity to traumatic memory recall task but not other recall tasks.

Child RSA	Estimate (SE)	p value
Pleasant Recall		
Intercept	4.21 (0.22)	<.001
Time	−0.23 (0.02)	<.001
Group: Trauma PTSD (referent)	0	--
Trauma No PTSD	−0.16 (0.10)	.12
Control	0.02 (0.14)	.86
Time*Group: Trauma PTSD (referent)	0	--
Trauma No PTSD	0.05 (0.03)	.11
Control	0.01 (0.04)	.89
Sex: Girls (referent)	0	--
Boys	−0.14 (0.09)	.09
Age	0.16 (0.04)	<.001
Unpleasant Child Recall		
Intercept	4.26 (0.22)	<.001
Time	−0.31 (0.02)	<.001
Group: Trauma PTSD (referent)	0	--
Trauma No PTSD	−0.19 (0.10)	.06
Control	0.00 (0.14)	.99
Time*Group: Trauma PTSD (referent)	0	--
Trauma No PTSD	0.08 (0.03)	.01
Control	0.04 (0.05)	.38
Sex: Girls (referent)	0	--
Boys	−0.15 (0.08)	.07
Age	0.17 (0.04)	<.001
Sex	1.79	.18
Unpleasant Parent Recall		
Intercept	4.17 (0.22)	<.001
Time	−0.22 (0.02)	<.001
Group: Trauma PTSD (referent)	0	--
Trauma No PTSD	−0.17 (0.10)	.10
Control	0.04 (0.14)	.77
Time*Group: Trauma PTSD (referent)	0	--
Trauma No PTSD	0.06 (0.03)	.06
Control	0.06 (0.04)	.18
Sex: Girls (referent)	0	--
Boys	−0.12 (0.09)	.18
Age	0.16 (0.04)	<.001

Table 4
Caregiver-child physiological synchrony varies across trauma groups only during negative memory recall tasks

	Child RSA during task by group				
	No trauma _a	Trauma No PTSD _b	Trauma with PTSD _c	<i>z-value</i> _{bc}	<i>z-value</i> _{ac}
Baseline	-.14	.24 [*]	.15	<i>ns</i>	<i>ns</i>
Parent RSA during tasks					
Pleasant Memory Recall	-.01	.25 ^{**}	.17	<i>ns</i>	<i>ns</i>
Child Unpleasant Memory Recall	-.15	.42 ^{***}	.16	3.07 ^{**}	2.01 [*]
Parent Unpleasant Memory Recall	-.20	.35 ^{***}	.27	2.91 ^{**}	0.62
					2.42 [*]

Note:

^{*} $p < .05$,

^{**}

$p < .01$,

^{***}

$p < .001$. Values are partial Correlation coefficients for caregiver and child RSA values during tasks, controlling for child age and sex. Z values indicate significant differences in correlation coefficients compared between groups. Subscript letters indicate comparison groups.