

ORIGINAL ARTICLE

Physiological contagion in parent–child dyads during an emotional challenge

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Abstract

Parents and children mutually influence each other's behavior, but little work has examined how parent–child dyads influence one another's physiological responding under conditions of emotional challenge. This is important to examine because physiological substrates underlie the development of self-regulation. We examined this in 97 parent–child dyads who participated in a frustrating laboratory challenge that first perturbed the affective state of the dyad and then allowed recalibration. Children were between 3 and 7 years of age ($M = 5.80$ years, $SD = 1.25$ years; 41 boys). Respiratory sinus arrhythmia (RSA) and pre-ejection period (PEP) were assessed continuously as measures of parasympathetic and sympathetic nervous system activity, respectively. Actor–partner interdependence models were used to quantify the extent of physiological contagion between partners. The expected actor effects were found for RSA and PEP, and a partner effect was found for RSA only. Specifically, parents' RSA reactivity to perturbation influenced children's RSA reactivity during recalibration. This provides additional evidence for parent–child coregulation, during which parents support their children's regulatory efforts. We found no partner effects for PEP. Taken together, our results suggest that the two ANS branches may serve different regulatory functions, with parasympathetic functioning relating more strongly to social interactions and

dyadic challenges like those in the current study. This study provides the first evidence of physiological contagion in an emotionally challenging context between parents and children.

KEYWORDS

actor–partner interdependence model, autonomic nervous system, parent–child relationship, PEP, physiological contagion, RSA

1 | INTRODUCTION

Parents and children influence one another's behavior and emotional states (e.g., Grusec & Hastings, 2014; Hofer, 2006), but much less is known about whether and how they influence each other's physiological functioning in an emotional context. The nature of the dyadic relationship between parent and child is implicated in interpersonal outcomes across the lifespan, from mother–infant affective synchrony (Feldman, 2007) to physical health concordance in long-married couples (Meyler, Stimpson, & Peek, 2007). But little work has examined how parent–child dyads influence one another's physiological responding under conditions of emotional challenge. This is important to understand more clearly because physiological substrates undergird children's development of self-regulatory processes (e.g., Shih, Quiñones-Camacho, & Davis, 2018).

Many terms have been used previously to describe mutual influences between two members of a relationship dyad, like attunement (Ruttle, Serbin, Stack, Schwartzman, & Shirtcliff, 2011), synchrony (Feldman, 2012), concordance (Bornstein & Suess, 2000), and coregulation (Saxbe & Repetti, 2010). These terms have guided the conceptualization of the mutual association within a dyad usefully by describing patterns of convergence and divergence over time in different contexts, but the goal of this paper was not to examine convergent and divergent physiological patterns. Guided by a systems perspective, in which we consider the reciprocal, transactional, and dynamic influence of members in a dyad across time (e.g., Butler, 2015; Lunkenheimer, Olson, Hollenstein, Sameroff, & Winter, 2011), we instead examined whether parents' physiological activity would influence children's subsequent physiological activity more strongly than vice versa. This view of dynamic physiological contagion across distinct contexts is closely aligned with the concept of physiological *direction*, the predictability of one person's physiology from another's (see Palumbo et al., 2017, for a review). The goal of this study was to examine the extent to which physiological contagion between the members of a parent–child dyad occurs across the changing contexts of a structured emotional challenge task (i.e., perturbation followed by recalibration) to address this gap in our knowledge.

1.1 | The autonomic nervous system supports social interaction

The mammalian autonomic nervous system (ANS) is critical in aiding survival by enabling response to potential threats, but also is charged with promoting social interactions and bonds, like the ones between a parent and child (e.g., Porges, 2009). Humans maintain a need for social interaction throughout their lives. Social separation and isolation lead to disruption in the ability to regulate one's physiological state and compromise both physical and mental health across the lifespan (Porges & Furman, 2011). The ANS supports children's attempts to engage their caregivers and learn about social exchanges as a mechanism that regulates physiology and behavior (e.g., Porges, 2003; Porges & Furman, 2011).

The ANS has two coordinated branches that jointly support adaptive functioning. The parasympathetic nervous system (PNS) is responsible for modulating the visceral and neuroendocrine systems to maintain homeostasis

and self-regulation, as well as to regulate recovery from a stressor or challenge. The sympathetic nervous system (SNS) is responsible for mobilizing resources to meet environmental demands (Berntson, Quigley, & Lozano, 2007). Under conditions of immediate challenge, the suppression of the parasympathetic input to the heart may not be sufficient to enable adaptive responding, and thus the activation of the sympathetic system (equipping the body by increasing heart rate and oxygen flow) is necessary for response mobilization. In other words, both systems are activated by environmental stress or challenges, but serve distinct functions. Porges' polyvagal theory (e.g., Porges, 2003, 2009) suggests that the PNS is responsible for social engagement processes whereas the SNS is responsible for challenge-related responses (Dickerson & Kemeny, 2004). Despite the important insights from studies that have been guided by the polyvagal theory, little is known about the dynamic operation of the PNS and the SNS within parent-child dyads undergoing emotional challenge. To date, the study of the ANS physiology as a measure of human experience has been focused largely on intrapersonal processes, wherein temporal changes are explored "within-subject" (Beauchaine, 2001).

More recently, studies have begun to offer evidence that the ANS is externally responsive to, and in some instances, dependent on or shaped by the ANS of others "across-subject" (e.g., Butler, 2011; Ferrer & Helm, 2013). The latter framework suggests that social interactions may be understood better when autonomic processes of all participants are evaluated; this idea has been expanded on in recent years. For example, Butler (2011) introduced the temporal interpersonal emotion system (TIES) model to suggest that the temporal ordering of emotion subcomponents (e.g., subjective experience, physiology) in one person is directly related to a parallel stream of temporally ordered subcomponents in another person. Empirical developmental work has supported this reasoning (e.g., Lee, Miernicki, & Telzer, 2017; Lunkenheimer, Kemp, Lucas-Thompson, Cole, & Albrecht, 2017). For instance, mothers and infants demonstrate time-sensitive positive concordance in heart rate that increases around episodes of positive, synchronous behavior (Feldman, Magori-Cohen, Galili, Singer, & Louzoun, 2011). And, physiological synchrony between parents and children is sensitive to emotional context during dyadic interactions, such that dyads who demonstrated physiological synchrony showed higher levels of repair (recovery) following a discussion on a contentious topic (Woltering, Lishak, Elliott, Ferraro, & Granic, 2015). By including both parents' and children's physiological responses, we aim to provide a clearer understanding of the bidirectional processes occurring within a dyadic social interaction.

1.2 | Physiological contagion within parent-child dyads

The influence that parents' practices and characteristics have on children's regulatory physiology may be a mechanism by which parenting shapes children's adjustment. For example, animal models have shown that variations in parental socialization practices can affect the development of autonomic regulation in offspring (Parent et al., 2005). Environmental adversity results in patterns of parent-offspring interactions that increase stress reactivity through sustained effects on gene expression in brain regions known to regulate behavioral, endocrine, and autonomic responses to stress. Parallel mechanisms with humans have been proposed, supported by emerging evidence that children and parents dynamically influence one another's physiology. Many studies have examined this using respiratory sinus arrhythmia (RSA), an index of parasympathetic influence on the heart (e.g., Berntson, Cacioppo, & Grossman, 2007).

Basal RSA represents an individual's regulatory capacity whereas RSA reactivity to a task or perturbation is thought to represent the increase in arousal that is facilitated by PNS withdrawal (Porges, 2003). Inconsistent findings, however, have emerged across studies examining the relation between RSA and behavioral measures of children's adjustment. For example, we see inconsistencies across findings reporting RSA patterns and children's psychopathology, such that both baseline RSA and RSA reactivity in response to challenge tasks have been reported as negatively, positively, or nonsignificantly related to children's externalizing (Beauchaine, Gatzke-Kopp, & Mead, 2007; Calkins, Graziano, & Keane, 2007; Dietrich et al., 2007) and internalizing problems (El-Sheikh, 2001; Hastings & De, 2008). We also see mixed results looking at RSA patterns and children's social functioning, with

some studies finding that higher levels of RSA withdrawal were associated with higher social preference scores (Graziano, Keane, & Calkins, 2007), and other studies finding an inverse relation (Blair, 2003). These inconsistencies could be attributed to the varying contextual demands of the studies (e.g., types of stressor), or individual differences between children (e.g., children's baseline physiology; Porges, 2007). The current study provides insight into these variations by highlighting how the assessment of both individual differences and careful consideration of the context can be leveraged to characterize different patterns of physiology.

A relationship dyad composed of a parent and child theoretically should function differently than a dyad composed of two adults. Adult dyads typically would be characterized by more equal, bidirectional responses. Within a parent-child dyad however, inherently it may be adaptive for parents to influence children's physiology in challenge contexts more strongly than for children to influence parents. Yet, child-directed effects on parents' behavior and physiology also have been shown. For example, children's behavioral problems predict more negative parenting behaviors (e.g., Caspi & Moffitt, 1995; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005; Pettit, Laird, Dodge, Bates, & Criss, 2001), and child temperament predicts a wide range of parenting behaviors (for a review, see Lengua & Kovacs, 2005). Children's physiology also predicts later parenting behaviors (Kennedy, Rubin, Hastings, & Maisel, 2004). Given that parents are particularly important sources of instruction and information for children during challenging contexts, parents may have stronger influences on their children than children would have on their parents. Prior research has demonstrated that parents' physiological states underlie and support positive engagement with children, which in turn provides support for children's physiological regulation. For example, Moore et al. (2009) examined parents' and children's physiological regulation during a dyadic interaction to highlight the role that parents' physiological regulation played in driving a mutually positive, arousing interaction. They examined mothers' and 6-month-old infants' RSA reactivity throughout the face-to-face still face paradigm (FFSFP; Tronick, Als, Adamson, Wise, & Brazelton, 1978), and whether maternal sensitivity moderated infants' physiological recovery from the stressor during the reunion episode. They found that a greater decrease in sensitive mothers' RSA from baseline (indicative of more pronounced social engagement) was associated with higher levels of behavioral dyadic synchrony in both normal play and reunion episodes. Thus, during early infancy, responsive social engagement behavior from parents, indexed physiologically, can drive and support a mutually positive interaction between sensitive mothers and their infants. Through repeated interactions like these, parents may transmit a more adaptive style of autonomic responsiveness to their children.

Later in childhood, greater parent-child equivalence in the dyad's behavioral transactions tends to emerge (e.g., parents may let children decide for themselves how to respond to provocations like not being invited to a friend's party). These increases in behavioral autonomy may be one source of variation in children's developing physiology. A similar developmental pattern may exist for physiology. Bornstein and Suess (2000) assessed the relationship between mother and child RSA reactivity to an environmental challenge at 2 months and 5 years of age. RSA reactivity was concordant between child and mother at both time points, though the magnitude of the correlation increased (2 months $r = 0.23$; 5 years $r = 0.42$) over time. Thus, a shared style of physiological responding to environmental challenges may develop within the parent-child dyad over time. But children's physiology is still, even later in childhood, a developmental domain in which parents may have substantial influence. For example, mothers with elevated heart rates showed less physiological synchrony with their children (Creaven, Skowron, Hughes, Howard, & Loken, 2014) whereas mothers demonstrating greater RSA withdrawal during a stressful interaction displayed greater behavioral synchrony with their children (Giuliano, Skowron, & Berkman, 2015). Similarly, a recent study with preschool children showed that heightened maternal baseline RSA was related to divergent coregulation patterns in mother and child RSA over time (Skoranski, Lunkenheimer, & Lucas-Thompson, 2017). Despite the bidirectional mutual processes that tend to occur as children gain more autonomy, parents' physiological patterns still may be important in supporting adaptive parent-child interactions and children's adjustment, though studies have not yet examined this in later phases of childhood.

Fewer studies consider dyadic sympathetic activity, and no studies of which we are aware have examined it in the context of a parent-child dyad. Pre-ejection period (PEP) indexes the SNS activity, representing the time

between the onset of the heart beat to the ejection of blood into the aorta (Beauchaine et al., 2007; Fox, Schmidt, Henderson, & Marshall, 2007). Shorter PEP intervals indicate higher SNS activation and are correlated with faster heart rate and increased cardiac output (Berntson et al., 1994; Mendes, Reis, Seery, & Blascovich, 2003). SNS activity is investigated rarely within a social interaction context. One study that examined a group of adult women considered how social support might influence cardiovascular reactivity during an acute stressor (Uno, Uchino, & Smith, 2002). The effectiveness of social support depended on the quality of the women's friendships. Women who interacted with an ambivalent female friend had shortening PEP (increases in sympathetic activation) compared to those interacting with either a supportive female friend or an ambivalent male friend. This pattern of findings provides a foundation for reasoning about how the sympathetic system might respond within another established relationship context like a parent-child dyad, by highlighting the importance of variability in the quality of the social relationship for characterizing functioning. Although PEP specifically is not well studied in dyadic contexts, children's temperament (the biological basis of which is often assessed with physiological measures including PEP; Stifter, Dollar, & Cipriano, 2011) appears to influence functioning within a parent-child dyad. For example, Kim and Kochanska (2012) found that temperamental negative emotionality moderated the effects of mother-child mutually responsive orientation, such that highly negative infants were less self-regulated when they were in unresponsive relationships, but more self-regulated when in responsive relationships. Thus, interpersonal relationships research with adults and children suggests that PEP may be implicated in the functioning of parent-child dyads, though no studies have tested this directly. The current study addressed this gap in knowledge by examining the activity of the sympathetic system within the established parent-child relationship.

1.3 | The current study

Individuals involved in social relationships, even brief dyadic interactions, influence each other's cognitions, emotions, and behaviors over time, creating non-independence between the members of the dyad (e.g., one person's behavior—or physiology—is contingent on the other's; Levenson & Gottman, 1983). The Actor-Partner Interdependence Model (APIM; Kenny, Kashy, & Cook, 2006) was developed as a statistical framework for collecting and analyzing dyadic data that stresses the importance of accounting for the interdependence between dyad members. The APIM uses the *dyad* as the unit of analysis rather than the individual, and estimates both "actor" effects (e.g., the effect of each person's physiology during a challenge on their own physiology after the challenge) and "partner" effects (e.g., the effect of one person's physiology during a challenge on their partner's physiology after the challenge). This approach allows us to evaluate the nature of dyadic parent-child influences on physiology by parsing the effects of each actor in the dyad and testing the contagion effects between partners.

This study was designed to investigate physiological contagion between parents and their 3- to 7-year-old children, using APIMs. Behavioral research on the dynamics of the child-parent dyad in this age range suggests that exchanges during early and middle childhood become more equal or mutual. This mutuality in early and middle childhood has included constructs like dyad cooperativeness (discussion and carrying out cooperative acts) and dyad behavioral and emotional reciprocity (eye contact, matched positive and neutral affect, and organized turn-taking quality to verbal and nonverbal behaviors) (e.g., Deater-Deckard & O'Connor, 2000). Although a shift toward more dyadic mutuality in behavior has been identified within this age range (e.g., Deater-Deckard & O'Connor, 2000), it is not clear whether the same pattern would emerge for physiology. We assessed parasympathetic and sympathetic contagion in separate models. Given the intraindividual associations between baseline RSA and RSA reactivity to challenge (Porges, 1995; Porges, Doussard-Roosevelt, & Maiti, 1994; Salomon, 2005), we expected individuals' own physiology to be relatively stable across time (i.e., an actor effect). Because activity of the PNS is more dominant during social contexts, and the activity of the SNS is activated during perceived stressors, we hypothesized that we would obtain a different pattern of parent and child physiological contagion for the PNS and the SNS. Because dyadic patterns of physiological activity in the PNS are documented consistently in the literature, we expected to see physiological influence from parent to child for the PNS. Lastly,

because no empirical work has tested dyadic patterns of physiological activity in the SNS, we had no specific predictions but sought to explore this dynamic social interaction.

2 | METHOD

2.1 | Participants

Participants were 97 children 3–7 years of age ($M = 5.80$ years, $SD = 1.25$ years; 41 boys), along with one parent (84% were mothers) who took part in a larger study of emotional development. Families were recruited from a sociodemographically diverse area of the southwestern United States. Parents reported children's races and ethnicities as: multiracial (37%), Hispanic (27%), Caucasian (19%), African American (8%), Asian American (3%), or other (3%). Mothers' race/ethnicity was reported as Hispanic (36%), Caucasian (30%), multiracial (11%), African American (6%), Asian American (5%), or other (4%). Fathers' race and ethnicity was reported as Hispanic (33%), Caucasian (27%), African American (16%), multiracial (10%), Asian American (5%), or other (2%). Thirty-five percent of the families who chose to report annual income had a household income below \$30,000; 30% reported income above \$50,000. In our sample, 38.4% of mothers and 42.4% of fathers had a high school diploma or less education, 56.5% of mothers and 46.5% of fathers obtained a college (or higher) degree. Education level was not reported for 5.1% of mothers and 11.1% of fathers.

Data were partially missing for 19 participant dyads; these dyads did not have usable physiological data (due to electrodes coming loose or technical issues with acquisition). Data were completely missing for 14 participant dyads (due to refusals to wear the electrodes). Independent t tests revealed no differences across the primary measures ($-1.29 < t < 0.71$; $p > 0.28$) and demographics ($-1.42 < t < -0.91$; $p > 0.16$) between families with missing data and families with no missing data. Results from Little's MCAR test were nonsignificant ($p = 0.18$), indicating that the missingness in the current study did not significantly deviate from a missing completely at random pattern. Missing data were multiply imputed using the expectation method (EM) algorithm in SPSS. This approach is superior to listwise deletion, mean substitution, or multiple regression techniques for handling missing data (Musil, Warner, Yobas, & Jones, 2002). Twenty imputations were generated and the pooled estimates were used for analyses.

2.2 | Procedure

Families visited the lab once for a 3.5-hr session that included parent surveys (about themselves and their children), interactive tasks between the experimenter and the child, and interactive tasks between the parent and the child. The university's institutional review board approved the study before research procedures began. Parent consent and child assent were secured at the beginning of the study. All assessments were videotaped for later behavioral coding. Physiology was collected from children continuously throughout the visit, starting with an initial baseline phase acquired at the beginning of the visit, after a warm-up period that allowed children to acclimate to the sensors and the equipment. Physiology was additionally collected from parents in the second half of the lab visit.

2.2.1 | Emotional challenge task

Children and parents were instructed to attempt to complete a complex Lego puzzle within a specified amount of time so that the child could win a prize. This Lego set is designed for ages 12+ and would be nearly impossible to finish within the very short time allotted for them, though children and parents were not told that the puzzle would be difficult to complete. In pilot testing, the Lego puzzle was given to three adult research assistants, who required an average of 40 min to complete the puzzle. Thus, it was highly unlikely that unaided young children

would complete it within 10 min. The task consisted of two phases. During the first phase (dyad perturbation; 5 min), parents were instructed to allow their child to attempt the puzzle independently and not to help build it. This phase can be viewed as a perturbation to the dyad's typical interaction, by restricting parents' behavior. During the second phase (dyad recalibration), the experimenter returned, said the dyad could have an additional 5 min to work on the puzzle, and invited parents to help their children or interact with the puzzle however they liked. This phase provided parents and children the opportunity to return to a more typical interactive state. At the end of the task, children were told that because they tried their best and did such a great job, they would receive a prize.

2.2.2 | Psychophysiology acquisition

Physiological data, electrocardiogram (ECG), and impedance cardiography (ICG) were collected during a resting baseline immediately before the frustration task, in which children and parents sat together quietly for 3 min, and continuously throughout the frustrating challenge task phases. ECG and ICG were wirelessly transmitted to a nearby computer using an ambulatory impedance cardiograph (MindWare Technologies, Westerville, OH, USA) and MindWare Wi-Fi ACQ Version 3.0.21 acquisition software. Data were collected via self-adhesive electrodes placed on the participants' rib cages. The experimenter introduced the sticky electrodes along with colorful, attractive stickers and asked children to help "decorate" the back of the electrodes with stickers they selected. A second experimenter entered the room and explained that children would wear the sticky sensors on their bodies so that the experimenters could listen to their hearts during the study. Seven disposable pre-gelled electrodes were placed on the children's torsos in ECG and ICG configuration. Three electrodes were placed over the children's distal right collarbone, lower left rib, and lower right rib to acquire an electrocardiograph (ECG) signal. Four additional electrodes derived impedance data. Two voltage electrodes were placed below the suprasternal notch and xiphoid process, and two current electrodes were placed on the back with one 3–4 cm above and one 3–4 cm below the voltage electrodes. The ambulatory monitor was secured in a small backpack to allow children to move freely during the tasks. Once electrodes were attached and participants acclimated to wearing the sensors, physiological recording began for the resting baseline measure. Similar procedures were followed to acquire ECG/ICG from parents. For the current study, physiological data acquired from children and parents during the resting baseline immediately before the emotional challenge task (parents and children sat quietly in a room together for this baseline), and during phases 1 and 2 of the task were used.

2.3 | Data reduction and coding

2.3.1 | Processing and coding of cardiac physiology

The ECG data were processed offline using a multi-pass algorithm designed to detect R-waves. Heart rate was quantified from the ECG as the number of R–R intervals per minute. RSA was used as a measure of parasympathetic activity. RSA spectral power was integrated over the appropriate frequency band for respiration (the 0.15–0.80 Hz range was used for the children in our sample, and the 0.12–0.24 Hz range was used for the parents), and calculated in 30-s epochs. Each 30-s epoch was inspected visually for errors (most often these were missed R-waves or peaks misidentified as R-waves), which were manually corrected as needed. Research assistants achieved RSA values for each epoch of data within 0.1 of the master coder's (first author) values before they were considered reliable. Inter-rater reliability was calculated on 25% of the files, and was excellent (percent agreement within 0.1 = 98%).

PEP was derived from ECG and ICG and impedance data were ensemble averaged within 30-s epochs, and each waveform was verified or edited prior to the analyses. Data were coded offline using MindWare ICG V. 3.0.21 (ANS Suites; Mindware, Westerville, OH), allowing for simultaneous editing of the data obtained from

ECG and ICG. PEP was qualified as the time interval in milliseconds from the onset of the Q-wave to the B point of the dZ/dt wave, using the method outlined by Berntson, Lozano, Chen, and Cacioppo (2004). The Q-onset in the ECG was placed using a validated automated scoring algorithm. Artifacts were visually inspected to ensure accurate placement and adjusted if needed. Inter-rater reliability was calculated on 25% of the files (absolute agreement = 90%). Mean PEP was calculated for each 30-s epoch for each subject.

2.4 | Behavioral coding for dyadic distress

Trained research assistants, supervised by the first author, coded video recordings of the parent-child interactions during the perturbation and recalibration phases of the task to determine distress at the level of the dyad. Distress was globally coded on a 1 to 5 scale (1 = low; 5 = high) across the entire phase, and was based on the duration and intensity of both parents' and children's distress-related behaviors. Two separate codes were given, one for distress during the perturbation (stressor) phase and another for distress during the recalibration phase. During the perturbation phase, we coded distress behaviors and verbalized complaints about the task from both the child and the parent. For example, children were distressed in the attempt to elicit help from their parents (e.g., "You really can't help me? I can't do this, I can't do it dad."), and parents complained about how difficult it was for them to refrain from helping (e.g., "Hey, don't get mad at dad."). During the recalibration phase, distress included children's verbalized concerns that they would not finish the task in time (e.g., "We aren't getting it.") and parents' complaints of distress (e.g., "We don't come here to argue."). We also coded for nonverbal distress behaviors in children (e.g., pouting, throwing LEGO pieces) and in parents (e.g., frowning, furrowed brows, arms crossed) for both the phases. Distinctions between levels of distress for both the phases were based on the duration and intensity of children's and parents' expressions. For example, a child who was visibly distressed for nearly the entire duration of each phase and exhibiting behaviors such as complaining and throwing the LEGO pieces was given a distress score of 5 whereas a child who was calm and obediently listening to the parent give instructions was given a distress score of 1. Children who showed both more verbal and nonverbal signs of worry and distress throughout the task were coded as more distressed than a child who showed only one or two signs of concern. Inter-rater reliability was calculated for 80% of the files with >93% agreement for dyadic distress codes in both the phases. Disagreements between coders were discussed and resolved by the first author.

3 | RESULTS

We analyzed data from both children and parents to examine the relations between their individual patterns of autonomic nervous system functioning (i.e., parasympathetic nervous system, sympathetic nervous system) during a two-part parent-child dyadic frustration task. To model the interdependence of the dyad members and the mutual influence between them, our analyses utilized the two-intercept multilevel actor-partner interdependence model (APIM; Cook & Kenny, 2005; Kenny et al., 2006). Pearson's correlations were used to first assess the associations between parents' and children's physiology during phase 1 and the outcome (parents' and children's physiology during phase 2).

Tables 1 and 2 present the descriptives and zero-order correlations among age, gender, observed dyadic distress for Phase 1 and Phase 2, and the main physiological variables, (i.e., parents' and children's RSA and PEP values during Phase 1, perturbation, and Phase 2, recalibration, of the frustration task). Children's RSA levels in Phase 1 were correlated positively with their parents' RSA levels in Phase 2 ($r = 0.24, p = 0.02$) and parents' RSA levels in Phase 1 were correlated positively with their children's RSA levels in Phase 2 ($r = 0.32, p = 0.002$), justifying the use of APIMs. In contrast, parents' and children's PEP during Phase 1 was not correlated with their partners' PEP during Phase 2 ($r_s > -0.14, p_s > 0.17$). We ran the PEP model using a standard linear regression, yielding similar estimates as the APIM and it did not alter the obtained pattern of results. Thus, only coefficients for the APIM

are included in the text. Age and gender did not associate significantly with the physiological variables, and thus were excluded from the APIMs. Dyadic distress during Phase 1 was correlated negatively with parents' RSA during Phase 1, and correlated positively with children's PEP during Phase 2 of the task. Dyadic distress during Phase 2 was correlated negatively with parents' RSA during Phase 1 and Phase 2 of the task. To enable us to better statistically isolate the hypothesized physiological associations of interest, the dyadic behavioral distress codes for both phase 1 and phase 2 were included as covariates for both the RSA (PNS) and PEP (SNS) models.

Two separate APIMs were tested, one for RSA and one for PEP. We used a two-intercept APIM that allowed parents and children to have separate slopes and intercepts (Kenny et al., 2006). In the first model, we examined the contagion effects in the PNS using RSA. Parents' and children's RSA values during Phase 2 were regressed on parents' and children's RSA values during Phase 1. Both actor effects were significant which showed that parents', $\beta = 0.90$, $p = 0.0001$, and children's, $\beta = 0.91$, $p = 0.0001$, RSA during Phase 1 significantly and positively related to their own RSA during Phase 2. In other words, children and parents with high RSA during Phase 1 also showed high RSA during Phase 2. One partner effect was significant, such that only the parents' RSA during Phase 1 had a partner effect on children's RSA during Phase 2. Parents' lower RSA during Phase 1 predicted their children's lower RSA during Phase 2; $\beta = 0.09$, $p = 0.012$. Children's RSA values during Phase 1 were not significantly related to parents' RSA during Phase 2; $\beta = 0.0005$, $p = 0.99$. Coefficients are presented and modeled in Figure 1.

To examine the contagion effects of the sympathetic nervous system using PEP, we used a second APIM in which parents' and children's PEP values during Phase 2 were regressed on parents' and children's PEP values during Phase 1. A main actor effect for children's, $\beta = 0.93$; $p = 0.0001$, and parents', $\beta = 0.85$; $p = 0.0001$, PEP during Phase 1 revealed a positive association with their own PEP values during Phase 2 whereas no significant partner effects emerged ($ps > 0.33$). Coefficients are presented and modeled in Figure 2.

4 | DISCUSSION

The goal of this investigation was to examine the ANS contagion within a parent-child dyad undergoing an emotional challenge. We predicted that parents' physiology would play a more active role in influencing their children's physiology, specifically in the PNS branch. This was supported by our results, such that parents' RSA during the

TABLE 1 Descriptive statistics of main variables

	M	SD
1. Age	5.80	1.25
2. Gender (41 boys, 56 girls)	0.58	0.50
3. Dyadic distress Phase 1	1.72	1.08
4. Dyadic distress Phase 2	1.81	1.10
5. Child RSA Phase 1	6.69	1.15
6. Child RSA Phase 2	6.64	1.11
7. Parent RSA Phase 1	5.60	0.99
8. Parent RSA Phase 2	5.33	1.02
9. Child PEP Phase 1	100.84	15.32
10. Child PEP Phase 2	100.19	14.94
11. Parent PEP Phase 1	131.94	13.89
12. Parent PEP Phase 2	130.43	14.77

Note. RSA = Respiratory sinus arrhythmia.

PEP = Pre-ejection period.

Dyadic Distress coded on a scale of 1 (low) to 5 (high).

TABLE 2 Zero-order correlations between main variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. Age		-0.05	0.33**	0.22*	0.01	-0.02	0.01	-0.10	0.05	0.02	0.05	0.06
2. Gender			0.06	0.08	0.00	0.07	-0.04	-0.06	0.16	-0.07	0.14	-0.12
3. Dyadic Distress Phase 1				0.76**	0.06	-0.26*	0.07	-0.20	0.20	-0.03	0.25*	0.04
4. Dyadic Distress Phase 2					-0.05	-0.30**	-0.06	-0.30**	0.06	-0.01	0.10	-0.01
5. Child RSA Phase 1						0.25*	0.94**	0.24*	0.14	0.10	0.12	0.09
6. Parent RSA Phase 1							0.32**	0.89**	-0.08	-0.02	-0.09	-0.02
7. Child RSA Phase 2								0.32**	0.13	0.10	0.12	0.10
8. Parent RSA Phase 2									0.00	-0.06	0.01	0.02
9. Child PEP Phase 1										-0.15	0.94**	-0.06
10. Parent PEP Phase 1											-0.14	0.84**
11. Child PEP Phase 2												-0.07
12. Parent PEP Phase 2												

Note. Respiratory Sinus Arrhythmia (RSA); Pre-ejection Period (PEP).

* $p < 0.05$. ** $p < 0.01$.

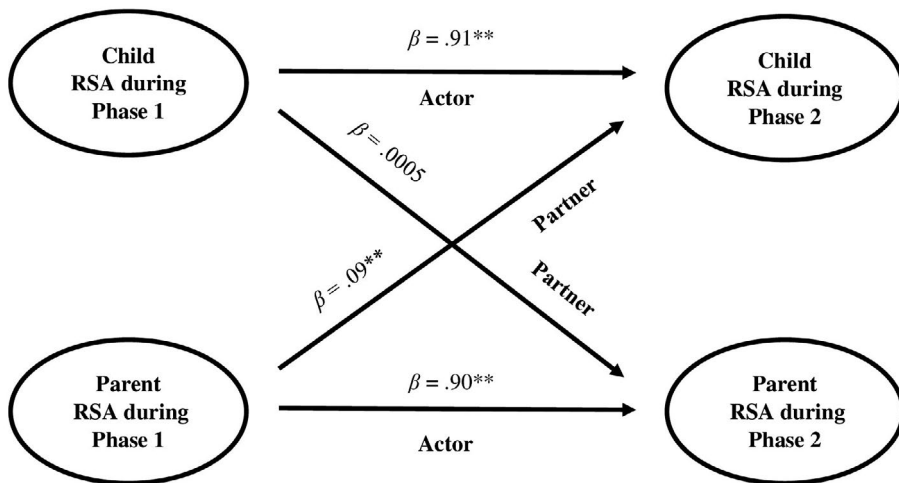


FIGURE 1 Actor-partner interdependence model for respiratory sinus arrhythmia. Note. Dyad Distress for Phase 1 ($\beta = 0.08$, $p = 0.11$) and Phase 2 ($\beta = -0.06$, $p = 0.24$) were included as covariates in this model. * $p < 0.05$; ** $p < 0.01$

perturbation phase predicted children's subsequent RSA during the recalibration phase. In contrast, our exploration of the SNS physiology contagion showed no partner effects—parents' PEP during the perturbation phase did not predict children's subsequent PEP. Children's physiology during the perturbation did not predict parents' subsequent physiological activity for either branch of the ANS. We discuss each of these results in the following sections.

4.1 | Parasympathetic contagion

We found the hypothesized effect of physiological contagion from parent to child in the parasympathetic system. This finding is novel, but aligns with behavioral work showing that parents are highly influential in shaping their

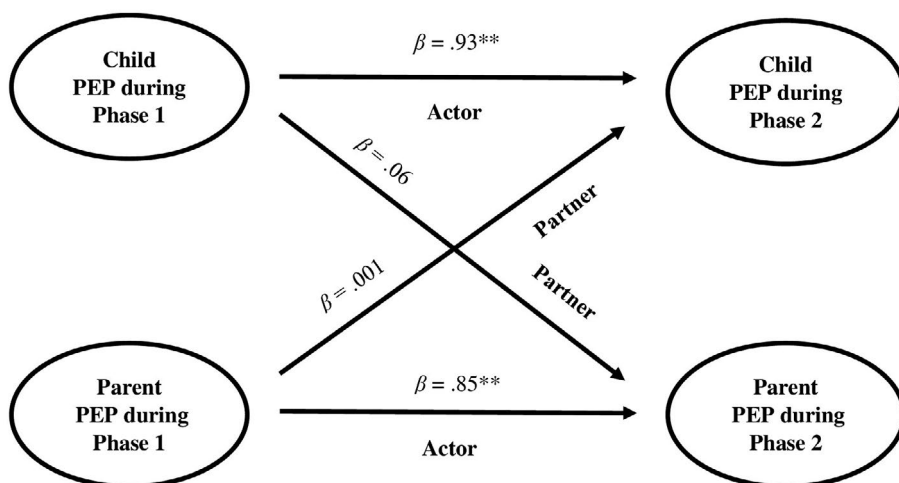


FIGURE 2 Actor-partner interdependence model for pre-ejection period. Note. Dyad Distress for Phase 1 ($\beta = 0.10$, $p = 0.03$) and Phase 2 ($\beta = -0.04$, $p = 0.39$) were included as covariates in this model; * $p < 0.05$; ** $p < 0.01$

children's emotional development and adjustment (e.g., Grusec & Hastings, 2014; Hofer, 2006). Because the focus of this investigation was to assess these dyadic processes at a physiological level, we included dyadic behavioral distress as a covariate. Of course, examining distress more thoroughly, perhaps as a focal predictor, would also provide meaningful information regarding socialization processes and could be done in future research. And although prior research had shown behavioral dyadic mutuality between parents and school-age children (e.g., Deater-Deckard & O'Connor, 2000), our findings suggest that at the physiological level, parents play more of a driving role within the dyad. These findings are consistent with the dyadic coregulation literature that has shown that parents' physiological activity can shape their children's emotional adjustment via developing self-regulation (e.g., Moore et al., 2009).

Our study provides unique insight by examining both directions of possible contagion (i.e., parent to child, child to parent) in one model, allowing us to identify the more robust direction. Few studies have assessed children's influences on parents' physiology. Most work examining parents' adjustment in response to children's temperament and behaviors has focused on atypically developing children or those with special needs (e.g., Barnett, Clements, Kaplan-Estrin, & Fialka, 2003), such as autism (e.g., Gray, 2006) and chronic illnesses (Popp, Robinson, & Britner, 2014). Less research has examined this in typically developing children (e.g., Premo & Kiel, 2014), and even less has assessed the physiological level. We found that children's physiology did not influence parents' subsequent physiological activity. It is possible that parents are less physiologically susceptible to their children's distress, which functionally would equip them to serve as resilient emotion socialization agents for their children.

Taken together, these findings are in line with our understanding of parental emotion socialization processes that shape children's developing emotion regulation abilities. We used physiological indexes to describe further how and through which mechanisms children might learn ways to manage undesirable emotions. Specifically, children were managing negative emotions evoked by the frustrating task in part through physiological coregulation with their parents (e.g., Moore et al., 2009), and we showed that the direction of this effect was specific to parents' parasympathetic physiology influencing children's subsequent physiological regulation. This pattern of findings provides additional evidence for physiological mechanisms that may underlie parent socialization of children's growing self-regulation abilities.

What is particularly interesting and novel about our findings is the specificity of the parent-child partner effect to the PNS. These findings support theorizing about the role of the PNS (and RSA) as an index of regulatory ability that is related particularly strongly to social functioning (Porges, 2003). The parasympathetic system enables flexible emotional responding that is required for mammalian social behavior, such that changes in the parasympathetic input to the heart give rise to increases in metabolic output sufficient for participating in social interactions (Porges, 2007). Because parasympathetic processes are thought to be the physiological substrates of emotional and behavioral self-regulatory processes throughout development (Bornstein & Suess, 2000), the specific patterning of parasympathetic contagion in the parent-child dyad in early childhood that we detected may support effective self-regulatory development.

4.2 | Sympathetic contagion

We sought only to explore contagion effects in the sympathetic system, given the paucity of empirical evidence that exists on this topic. Little work to our knowledge has examined sympathetic coregulation in parent-child dyads. One study utilizing salivary alpha-amylase (sAA) as a marker of sympathetic activation showed patterns of attunement in sAA activity between parents and children (Laurent, Ablow, & Measelle, 2012). But these findings were specific to infants and using sAA as an index of SNS rather than PEP. We found no evidence of sympathetic contagion from one member of the dyad to the other. One explanation for this could be the differences in contextual demands across studies. For instance, sympathetic synchrony occurs during specific forms of social interaction, like cooperation (Strang, Funke, Russell, Dukes, & Middendorf, 2014) and conflict (Levenson & Gottman,

1983). Coregulation of sympathetic activity has been studied with adults, examining the covariation of PEP levels in adult non-romantic dyads across time (Danyluck & Page-Gould, 2018). This study demonstrated that intergroup dissimilarity, not similarity, predicted physiological synchrony, and facilitated friendship initiation. The mildly frustrating challenge task we used in our study may represent a useful novel context for assessing a particular kind of social interaction, specifically one that results when the typical interaction between a parent and child is strained. But more research would be needed to clarify the implications of the SNS patterns we observed.

4.3 | Limitations and future directions

This study provides important insight about physiological contagion between children and parents, but some limitations must be considered. First, findings from this study should be interpreted with caution, given the modest sample size and modest effect size of the partner effect for RSA. The parent participants for the current study were predominantly mothers, so we could not examine sex differences in the dyadic composition that could be related to contagion effects between parents and children. Sex differences in emotion have been widely documented (Brody & Hall, 2000), and there appear to be different developmental pathways for boys' and girls' emotions (Brody & Hall, 2000; Chaplin, Cole, & Zahn-Waxler, 2005) that arise, in part, from the input of socialization agents like parents. It will be important for future studies to parse potential sex differences in parent-child dyad composition to further characterize physiological contagion effects. Additionally, our study assessed dyadic physiological contagion within a context of frustration. A next step for research in this area will be to examine physiological contagion in different contexts (e.g., disappointment, fear, sadness). Importantly, we opted to examine physiology at the level of the task rather than using more fine-grained (e.g., dynamic time series) information because of our specific interest in a frustration context. Future research into parent-child physiological contagion during challenge tasks would no doubt benefit from dynamic analyses of data streams, and this represents a promising direction for investigation. We also considered behavioral distress only at the level of the dyad, and not at the individual level (i.e., we did not code or analyze parent and child distress as separate variables). Given our specific interest in understanding the ANS physiological processes at a dyadic level, this focus on dyadic behavior was justified in this investigation. But future work should examine behavioral distress for each member of the parent-child dyad to clarify fully the role of the parent, the child, and the dyadic distress in shaping patterns of the ANS physiology during a frustration challenge. Interestingly, we found no consistent convergence between behavioral and physiological measures in this study—distress was correlated with some but not all of the physiological measures. This is consistent with prior work that reported inconsistent relations between observational and physiological components (e.g., Smith, Hubbard, & Laurenceau, 2011), suggesting the importance of considering individual differences and context. Finally, parenting behaviors (e.g., parental intrusiveness; parental coaching of strategies) could influence children's physiology causally, though this was not tested in the current investigation. Future research thus also could investigate mediating variables to clarify the patterns reported here.

4.4 | Conclusion

Findings from this study contribute knowledge about how parents and children mutually influence one another's physiology during an emotional challenge. Our results provide some of the first empirical evidence for theoretical perspectives that view the parasympathetic (but not sympathetic) system as foundational for effective social functioning. This investigation allowed us to identify a more robust direction of physiological influence from parent to child, which highlights a novel aspect of the parent-child relationship that has implications for children's developing self-regulation abilities. Parents' interactions with children during conditions of challenge shape children's developing physiological self-regulatory abilities, so results from this initial study can also inform prevention and intervention efforts that center on the parent-child dyad.

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