



The Role of Autonomic System Coordination in Relations Between Peer Factors and Aggressive Behavior in Early Childhood

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Abstract

This study tested biological sensitivity to context theory in the peer context. Respiratory sinus arrhythmia (RSA-R) and skin conductance level (SCL-R) reactivity to a peer stressor were collected for participants ($N = 86$; M age = 45.99 months old; 70.2% White) in the summer (Time 1). Children's peer risk (i.e., physical and relational victimization) and protective (i.e., received prosocial behavior) factors were examined in the fall (T2) and relational and physical aggression were measured at T2 and in the spring (T3). Interactions were tested in regression analyses. Interactions emerged between relational victimization, RSA-R, and SCL-R in the prediction of T3 relational aggression and between received prosocial behavior, RSA-R, and SCL-R in the prediction of T3 relational and physical aggression, respectively. There was a positive relation between T2 relational victimization and T3 relational aggression for children with a coactivation pattern (i.e., increased RSA and SCL activity to a bullying stressor) but no relation for any other physiological pattern. Conversely, there was a negative relation between T2 received prosocial behavior and both forms of aggression at T3 for children with a reciprocal pattern (i.e., increased RSA and decreased SCL or decreased RSA and increased SCL activity) but no protective benefit of received prosocial behavior on subsequent aggression for children with a coactivation pattern. For children with a coinhibition pattern (i.e., decreased RSA and SCL activity), received prosocial behavior was negatively related to subsequent physical but not relational aggression. In sum, a coactivation pattern in response to stress may represent a vulnerability factor.

Keywords Biological sensitivity to context theory · Early childhood · Relational aggression · Physical aggression · Peer victimization

Children who display continuity in aggressive behavior from early childhood into adulthood are more likely to experience negative outcomes, such as school and occupational problems, diagnosis of a psychological disorder, and arrest or incarceration (Huesmann et al., 2009). In fact, research suggests that from 2 years of age to 8–9 years of age (i.e., 3rd grade), 15% of children are in a moderately stable physical aggression group and 3% of children are in a high physical aggression group (Early Child Care Research Network

(ECCRN), 2004). Children in either of these groups are more likely to experience peer, academic, and psychological problems in 3rd grade compared to their peers (ECCRN, 2004). Notably, an additional 12% of children demonstrate a moderately declining trajectory with moderate levels of physical aggression throughout early childhood and a further decrease in physical aggression at 54 months of age (ECCRN, 2004). These children do not show the same negative outcomes as those in the high or moderately stable trajectories, suggesting that intervening on aggressive behavior in early childhood may reduce the negative impact of this behavior (ECCRN, 2004). These patterns are consistent with data collected in other large longitudinal studies from around the world (e.g., Moffitt, 1993). Therefore, it is critical to understand processes that place young children at risk for the development of aggressive behavior.

The purpose of the present study was to investigate associations between negative (i.e., physical and relational victimization) and positive (i.e., receipt of prosocial behavior)

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peer experiences and the development of aggressive behavior in young children. To gain a more comprehensive understanding of children's behavior, we included both relational and physical forms of aggression at two time points in early childhood. Physical aggression refers to behaviors with the intent to hurt, harm, or injure through physical harm or the threat of physical harm whereas relational aggression refers to behaviors with the intent to hurt, harm, or injure through "damage to a child's peer relationships" and includes behaviors such as social exclusion, gossip, and friendship withdrawal (Crick et al., 1997; p. 597). In addition, we investigated whether, consistent with biological sensitivity to context theory, autonomic nervous system (ANS) reactivity to peer stress moderated these associations. ANS reactivity was examined given that it is a key physiological mechanism assessed in the lab that is associated with children's regulation and responses to a number of different challenges (Obradović et al., 2010) and it has been widely used in studies of biological sensitivity to context theory (e.g., Abaied et al., 2018; Obradović et al., 2010). Given mounting evidence highlighting the importance of interactions between the sympathetic and parasympathetic branches in children's adjustment, we included interactions across these branches.

Peer Experiences and Aggressive Behavior in Early Childhood

Children's social experiences with peers during early childhood may play an important role in the development of their aggressive behaviors. For example, peer victimization (i.e., being the recipient of aggressive behavior) may lead to aggression through modeling of the aggressive behavior and the development of social-cognitive biases (Reijntjes et al., 2011; Yeung & Leadbeater, 2007). In fact, there is a robust literature linking peer victimization to externalizing problems (for a meta-analysis, see Reijntjes et al., 2011). Peer victimization may take several forms; relational victimization is defined as being the recipient of relational aggression and physical victimization is defined as being the recipient of physical aggression (Crick et al., 1999). Children are influenced by their own past experiences and therefore, when a child is a victim of one type of aggressive behavior, they may begin to use that type of aggression. If they are reinforced for using this specific type of aggressive behavior then over time their use of that behavior will increase. The specificity hypothesis of aggression posits that the within form of victimization is related to the development of that form of aggressive behavior (e.g., relational victimization

is related to relational aggression; Ostrov, 2010). Beyond early childhood, prior research has found support for the specificity hypothesis for preadolescents (Yeung & Leadbeater, 2007) and adolescents (Leadbeater et al., 2006). Despite moderate to strong bivariate correlations between the non-specific form of victimization and aggression (e.g., Farrell et al., 2016; Leadbeater et al., 2006), when controlling for the within form of aggression, the non-specific form of aggression is non-significantly or negatively related to aggression (Leadbeater et al., 2006; Ostrov, 2010; Yeung & Leadbeater, 2007).

Peer experiences may also serve as a protective factor against the development of aggression. For instance, received prosocial behavior, defined as being the recipient of a voluntary behavior from a peer that benefits the child (e.g., sharing, helping, or comforting; Eisenberg et al., 2015), may play a protective role against aggression. In fact, children who affiliate with prosocial peers are more likely to display more positive affect and less negative affect in future peer interactions, suggesting that affiliating with prosocial peers may positively shape children's social cognitions in subsequent peer experiences (Fabes et al., 2012). Additionally, children who are recipients of peers' prosocial behavior may develop a larger prosocial behavioral repertoire, reducing their reliance on aggression in future peer interactions.

It is particularly important to understand the role of peer experiences in the development of aggression during early childhood because children are learning how to navigate peer relationships for the first time (Rose-Krasnor & Denham, 2009). Achieving harmonious peer relationships serves as a critical developmental milestone and a precursor to future functioning (Darling-Churchill & Lippman, 2016). Moreover, at the start of early childhood, physical aggression peaks and relational aggression is just beginning to develop (Crick et al., 2006). Although relational aggression presents differently in early childhood (i.e., more direct and overt) relative to later developmental periods, observations of aggression indicate that it is prevalent and is reliably and validly measured across multiple methods and informants (Crick et al., 2006). In the present study, we investigated the association between peer experiences and the development of aggression during the early childhood period. Consistent with specificity hypotheses (Ostrov, 2010), we hypothesized that relational victimization would be uniquely associated with subsequent relational aggression, physical victimization would be uniquely related to subsequent physical aggression, and received prosocial behavior would be negatively associated with both forms of aggressive behavior.

Biological Sensitivity to Context

Although experiences with peers may play an important role in the development of aggression in early childhood, some children may be more affected by peer experiences than others. In fact, biological sensitivity to context theory hypothesizes that the impact of the environment on individuals' behaviors varies based on their neurobiological susceptibility. The central tenet of biological sensitivity to context theory hypothesizes that neurobiological susceptibility functions as a moderator between environmental inputs and developmental outcomes (Boyce & Ellis, 2005; Ellis et al., 2011). Importantly, this theory posits that children who are more reactive to the environment are more susceptible to both supportive and risky environmental contexts (Boyce & Ellis, 2005; Ellis et al., 2011). For example, one study in early childhood found that in the context of high levels of victimization, cortisol activity predicted increased aggressive behavior but in the context of low environmental risk, this same physiological profile predicted reduced aggressive behavior (Valliancourt et al., 2018).

Potential indicators of biological sensitivity to context include indices of autonomic nervous system (ANS) functioning, including indicators of sympathetic nervous system (SNS) activation such as electrodermal activity (i.e., skin conductance level (SCL), a measure of sweat gland activity, Dawson et al., 2016) and indicators of parasympathetic nervous system (PNS) activity, such as respiratory sinus arrhythmia (RSA; variability in heart rate tied to the respiratory cycle, Porges, 2007; for examples, see Erath et al., 2011; Obradović et al., 2010). Historically, researchers have examined SNS and PNS indices in isolation; however, there have been recent calls in the literature to examine interactions across the SNS and PNS (Buss et al., 2018). The interaction of the SNS and PNS generally comprise four different psychophysiological patterns: reciprocal sympathetic, reciprocal parasympathetic, coactivation, and coinhibition (El Sheikh et al., 2009; Murray-Close et al., 2017).

Because the SNS and PNS exert opposing effects on arousal, reciprocal patterns reflect coordinated responses in which both branches are functioning to yield the same directional effects on arousal (El-Sheikh et al., 2009). Reciprocal sympathetic activity, reflecting withdrawal of the PNS and activation of the SNS, functions to increase arousal (e.g., heart rate), whereas reciprocal parasympathetic activity, reflecting activation of the PNS and withdrawal of the SNS, functions to decrease arousal (El Sheikh et al., 2009; Murray-Close et al., 2017). Coactivation reflects an increase in activity in both the PNS and SNS and may reflect trait anxiety or physiological overarousal, whereas coinhibition occurs when there is reduced activity in both the PNS and

SNS in response to a stressor and may reflect a combination of poor emotion regulation and sympathetic underarousal. These nonreciprocal patterns have ambiguous effects on arousal and are hypothesized to reflect uncoordinated patterns of reactivity that increase risk for maladjustment in the context of adversity (El Sheikh et al., 2009; El-Sheikh & Erath, 2011).

Prior research with adolescent samples has used biological sensitivity to context theory to evaluate the role of these patterns in relations between contextual factors and adolescent outcomes. In one study, maternal involvement predicted better adolescent emotion regulation for individuals who exhibited reciprocal sympathetic activity but not those who exhibited coinhibition, coactivation, or reciprocal parasympathetic activity (Abaied et al., 2018). Additionally, maternal psychological control predicted adolescent emotion regulation deficits for those that exhibited reciprocal sympathetic activity but not those who exhibited any other pattern (Abaied et al., 2018). Overall, results suggest that a reciprocal sympathetic activity pattern may be representative of greater sensitivity to context.

An important limitation of prior research in this area is the focus on exposure to risk factors, with a paucity of research explicitly examining supportive or protective contexts. This is a significant limitation, given that biological sensitivity to context theory specifically postulates that highly reactive youth will benefit more from supportive contexts than their less reactive peers (Ellis et al., 2011). To extend this work, in the present study we examined whether ANS reactivity moderated associations between both positive and negative peer experiences and change in aggression.

Nonreciprocal Patterns as a Vulnerability Factor

An alternative perspective to biological sensitivity to context theory, is that nonreciprocal patterns may represent a vulnerability factor, such that they exacerbate risk in the presence of a negative environment. In fact, several studies in middle childhood and adolescence have found that when individuals display nonreciprocal patterns, negative family environment factors are associated with externalizing problems (El-Sheikh et al., 2009; Gordis et al., 2010; McKernan & Lucas-Thompson, 2018; Philbrook et al., 2018). Further, although the majority of research examining the dual impact of the SNS and PNS has examined risk factors within the home environment, some emerging research indicates that similar processes may be evident in the peer context. For instance, in a cross-sectional study with emerging adults, relational victimization was associated with reactive relational aggression for individuals demonstrating a coinhibition, coactivation, or reciprocal PNS activation, whereas reciprocal SNS activation was protective (Wagner & Abaied, 2015).

The Current Study

In sum, the goal of the present study was to investigate associations between negative (i.e., relational and physical victimization) and positive (i.e., receipt of prosocial behavior) peer experiences and changes in relational and physical aggression in an early childhood sample. Given our focus on the peer context, we assessed skin conductance level reactivity (SCL-R) and respiratory sinus arrhythmia reactivity (RSA-R) to a bullying stressor. Based on prior research examining both supportive and risky contexts (i.e., Abaied et al., 2018), it is possible that reciprocal SNS activation would reflect greater sensitivity to these peer experiences. Alternatively, nonreciprocal patterns may serve as a vulnerability factor that increases children's risk in the context of negative environmental influences (El-Sheikh et al., 2009; Gordis et al., 2010; McKernan & Lucas-Thompson, 2018; Philbrook et al., 2018). Given this mixed literature, we tested two hypotheses: (1) a reciprocal sympathetic pattern would be indicative of a sensitivity pattern, consistent with prior research using biological sensitivity to context theory, or (2) nonreciprocal patterns would be indicative of a vulnerability pattern, congruent with prior research evaluating negative aspects of the home environment.

Method

Participants

Children were recruited from ten National Association for the Education of Young Children (NAEYC) accredited or recently accredited early childhood education centers in the Northeastern United States. Four of the schools were university affiliated and six were community based. Ninety-four children were recruited for a summer lab session. Eight children did not assent to the physiological portion of data collection and therefore were not included in the study. The final sample included 86 children (46.0% girls; M age = 45.99 months

old, $SD = 6.11$ months; 3.2% African American or Black, 7.4% Asian or Asian American or Pacific Islander, 3.2% Hispanic or Latinx, 14.9% multi-racial, 1.1% Other race/ethnicity, and 70.2% White) and was middle to upper middle class (64.9% reported household income > \$100,000, 20.2% household income of \$55,000–\$100,000, 9.6% household income of \$36,000–\$54,999, and 8.5% reported household income < \$36,000). This study was drawn from a larger sample of children followed throughout preschool (see Ostrov et al., 2022), and there were not racial or ethnic differences [$\chi^2(4) = 1.38, p = 0.85$] or gender differences [$\chi^2(1) = 0.73, p = 0.80$] between the larger sample and the current subsample. Children with physiological data were slightly older than children without physiological data [$F(1, 294) = 4.13, p = 0.04$, Adjusted $R^2 = 0.01$, M age difference = 1.14 months], because of recruitment methods for the lab session (see below for more information).

Procedures

See Table 1 for information about data collection procedures. In the spring of children's preschool year, parents and children were invited to participate in a summer lab session (T1). Children were eligible to participate in the summer lab session after they turned four years old. Data collection began in the summer (Time 1, T1) and continued into participants' pre-k year in November/December (Time 2, T2) and May/June (Time 3, T3). Teachers provided reports of victimization, received prosocial behavior, and aggression in the fall (T2) and of aggression in the spring (T3). Data were collected between summer of 2015 and the spring of 2019.

During the lab session, physiological data were collected while the child watched 3 short video clips and parents completed a packet of measures. Parents were compensated \$30–\$40 for their time in the lab and children received a small educational toy. Parental consent and child verbal assent were obtained for the lab session. Teacher consent was obtained prior to teacher report completion. Teachers

Table 1 Data Collection Guide

Data Collected at Each Study Time Point		
Preschool Summer (T1)	Pre-Kindergarten Fall (T2)	Pre-Kindergarten Spring (T3)
Baseline SCL	Relational aggression	Relational aggression
Baseline RSA	Physical aggression	Physical aggression
SCL-Reactivity	Relational victimization	
RSA-Reactivity	Physical victimization	
	Received prosocial behavior	

All measures in the fall (T2) and spring (T3) were teacher report

T1 Time 1, *T2* Time 2, *T3* Time 3, *SCL* Skin Conductance Level, *RSA* Respiratory Sinus Arrhythmia

received \$5–\$30 based on the number of enrolled children in their classrooms. All procedures in the study were approved by the University at Buffalo IRB.

Due to the longitudinal nature of the study across school years (e.g., children changed schools for free or reduced cost universal pre-kindergarten programs or attended kindergarten), missing data was expected. At T2 (fall), there was missing data for 22% of the sample and from T2 to T3 retention was strong (98.5% of the sample). It was expected that data would be missing at random (MAR) given that missingness was not randomly assigned (i.e., MCAR; Baraldi & Enders, 2010). Thus, sources of systematic missingness within our dataset were identified and included in the model to facilitate the maximum likelihood process (Baraldi & Enders, 2010). The MAR assumption was tested using t-tests for continuous variables and chi-squared tests for categorical variables to examine if missing data was related to pertinent study variables. Baseline SCL was related to missing data at T2 [$F(1, 81) = 5.76, p = 0.02, \text{Adjusted } R^2 = 0.06$] and T3 [$F(1, 81) = 4.45, p = 0.04, \text{Adjusted } R^2 = 0.04$]. Children with higher baseline SCL scores were more likely to have missing data at T2 and T3. Therefore, baseline SCL was controlled. Missing data was accommodated using Full Information Maximum Likelihood (FIML) estimation.

Measures

ANS Reactivity

In the lab session at T1, the child's ANS reactivity was assessed using developmentally appropriate techniques (see Perhamus et al., 2022; Ostrov et al., 2022 for more details), while watching three video clips. The first video was a three-minute cartoon clip of "Spot the dog" playing with his favorite toys, which was designed to generate a baseline level of physiological arousal (Calkins & Keane, 2004). The second clip was a three-minute Sesame Street clip of Big Bird being excluded from the 'Good Birds Club', which was designed to get an index of reactivity to social exclusion. The third clip resolved the previous exclusion by having experts talk to the birds and other *Sesame Street* characters about the bullying. This clip was included in part for ethical reasons, so that children were not distressed by the conflict in the previous clip. Physiological data from the first two clips were used in the current study.

The procedure was explained and shown to the child in detail, using a stuffed bear to make the information developmentally appropriate. Children were then asked to give assent for the psychophysiology portion of the study. The child's caregiver was present when the physiological equipment was placed on the child and when it was removed from the child. A research assistant was present with the child while the video clips were being played to remind them to

sit still and to press event markers on the recording device (i.e., Biolog, see below) to indicate when clips began and ended. The parent was able to observe the child while they watched the video clips with project staff on a TV monitor in an adjacent room.

SCL-R and RSA-R were assessed separately. SCL-R was measured with skin conductance electrodes and adhesive collars that were put on the distal phalanges of the child's nondominant hand and recorded using Biolog equipment developed by UFI (Model 3991 with 3 channels). To measure RSA, disposable ECG electrodes were attached to participants' right and left rib in an axial configuration, with a ground lead attached to the participant's sternum. The ECG sampling rate was 1000 Hz, close to the 1024 Hz recommended value (Beauchaine et al., 2019) and the frequency band-pass parameters were set to 0.24 to 1.04 Hz. Cardiac inter-beat intervals (IBI) were measured as time in milliseconds between successive R waves of the electrocardiogram. The data were examined for artifacts and cleaned by trained and reliable graduate students using CardioEdit (Brain-Body Center, 2007). Session notes were also taken during the physiology data collection and were available during editing to see if outliers were due to movement or other problems, such as excessive talking. However, these data were almost entirely free of artifacts and needed minimal cleaning. After the ECG data were cleaned, RSA values were calculated using a time series method in the CardioBatch software (Porges, 1985). Respiration was also examined using a Biolog Pneumotrace respiration transducer. Consistent with prior work in this sample (Perhamus et al., 2022), reactivity was calculated by subtracting the mean score while viewing exclusion (clip 2) from the mean score of each physiological index at baseline (clip 1), such that positive values represent activation of the branch of the ANS to exclusion whereas negative values represent inhibition. This approach was chosen given that the sample size precludes a latent variable approach and prior work in this sample has demonstrated that SCL values are correlated but significantly different at baseline and reactivity video conditions and the variance of SCL was statistically equal across these two conditions (Perhamus et al., 2022). These statistics favor a difference-score approach (Perhamus et al., 2022).

Victimization and Received Prosocial Behavior-Teacher Report

At T2, relational victimization, physical victimization, and received prosocial behavior were measured using teacher reports of the Preschool Peer Victimization Measure-Teacher Report Revised (PPVM-TR-R, Crick et al., 1999; Godleski et al., 2015). Physical victimization (4 items; e.g., "This child gets hit, kicked, or pinched by peers"), relational victimization (4 items; e.g., "This child gets told 'you can't play' by peers when they are angry at him/her"), and

received prosocial behavior (4 items; e.g., “This child gets help from peers when they need it”) were rated on a 5-point Likert scale (1- *never or almost never true* to 5- *always or almost always true*). Higher mean scores reflect greater levels of victimization or received prosocial behavior. The subscales have demonstrated good psychometric properties in the past, including acceptable reliability (e.g., Cronbach’s α s = 0.85 and 0.90 for relational and physical victimization, respectively; Ostrov, 2010) and associations with observer informants of victimization (e.g., r s range from 0.13–0.35; Ostrov, 2008). All scales were reliable in the current sample (physical victimization Cronbach’s α = 0.90, relational victimization Cronbach’s α = 0.89, and received prosocial behavior Cronbach’s α = 0.89).

Aggression-Teacher Report

At T2 and T3, aggression was measured using teacher reports of the physical (6 items; e.g., “This child hits or kicks others”) and relational aggression (6 items; e.g., “When mad at a peer, this child keeps that peer from being in the play group”) subscales from the Preschool Social Behavior Scale-Teacher Form (PSBS-TF, Crick et al., 1997). Items were rated on a 5-point Likert scale (1- *never or almost never true* to 5- *always or almost always true*). Items from each subscale were summed and higher scores represent higher levels of aggression. The scales were reliable in the current sample (physical aggression Cronbach’s α > 0.86 and relational aggression Cronbach’s α > 0.90) congruent with prior research which has demonstrated acceptable reliability (e.g., Cronbach’s α s > 0.90) and validity through significant correlations with observer reports (e.g., r s range from 0.30–0.53; Perry & Ostrov, 2018).

Data Analysis

Descriptive statistics and zero-order correlations were examined for all study variables. Skewed values were adjusted to ± 3 standard deviations from the mean and skew and kurtosis statistics were assessed. Bivariate correlations were examined among all continuous variables used in the study. Gender and age were assessed as potential covariates and were included in subsequent models if they were related to any of the pertinent study variables at a value of 0.30 or higher. Respiration and temperature were considered as covariates for the physiological variables consistent with recommendations by Berntson et al. (2017). Baseline levels of RSA and SCL were controlled in the ANS reactivity models based on the law of initial values which indicates that an individual’s initial physiological activity at baseline reflects how reactive they are to a stressor, as well as meta-analytic work demonstrating that controlling for baseline values

increases associations between RSA reactivity and outcomes (Graziano & Derefinko, 2013). As a robustness test, we also ran the final three-way interaction models without baseline RSA and SCL to ensure that this was not conflating results.

Regression models were estimated in Mplus version 8.6 (Muthén & Muthén, 1998–2021) using the Maximum Likelihood with Robust Standard Errors (MLR) estimator to account for any skewness. For all models, model fit was evaluated using several indicators, including the likelihood ratio χ^2 test of overall model fit where $p > 0.05$ indicates good model fit, the comparative fit index (CFI), where values greater than 0.90 suggest adequate fit and values greater than 0.95 suggest good fit, the standardized root mean-square residual (SRMR) fit index where values less than 0.08 represent adequate model fit and values less than 0.05 represent good model fit (Hu & Bentler, 1999), and the root mean square error of approximation (RMSEA; Steiger, 1990), where values greater than 0.10 represent poor fit, values less than 0.08 represent mediocre fit, and values less than 0.05 represent close fit (Browne & Cudeck, 1992). Four sets of models were tested (i.e., relational victimization at T2 as a predictor of relational aggression at T3, physical victimization at T2 as a predictor of physical aggression at T3, received prosocial behavior at T2 as a predictor of relational aggression at T3, received prosocial behavior at T2 as a predictor of physical aggression at T3). For each set of models, a hierarchical approach was used, such that first a base model was examined which tested the main effects of the peer factors, SCL-R, and RSA-R on aggressive behavior. Second, two-way interactions between the peer variables and SCL-R or RSA-R were tested before moving on to test a third model with the three-way interaction. Gender, room temperature, baseline RSA, baseline SCL, and physical and relational aggression at T2 were controlled in analyses. Both forms of victimization were controlled in the base models for the peer risk analyses, but the non-specific form of aggression or victimization was removed if it was not significant in the interaction analyses to increase model parsimony. The T2 variables and SCL-R and RSA-R were regressed on the covariates.

If there was a significant two- or three-way interaction then follow-up simple slopes were conducted. Consistent with prior research (i.e., Abaied et al., 2018; Lafko et al., 2015), simple slopes were probed at one standard deviation (SD) above and below the mean to identify the following physiological patterns: (1) coactivation (both SCL-R and RSA-R are 1 SD above the mean); (2) reciprocal parasympathetic activity (SCL-R is 1 SD below the mean and RSA-R is 1 SD above the mean); (3) reciprocal sympathetic activity (SCL-R is 1 SD above the mean and RSA-R is 1 SD below the mean); and (4) coinhibition (both SCL-R and RSA-R are 1 SD below the mean).

Results

Preliminary Analyses

Bivariate correlations and descriptive statistics for the key variables are provided in Table 2. The correlations for the potential control variables for the physiological data are included in the supplementary material document and are noted below. For the key variables, skew values (-0.22 to 1.92) and kurtosis values (-0.33 to 3.23) were slightly skewed but within accepted ranges for normally distributed variables (Kline, 2015). Gender was considered as a categorical covariate, coded as -1 = girls, 1 = boys. Child gender was related to relational aggression at T2 [$F(1, 65) = 4.55$, $p = 0.04$, Adjusted $R^2 = 0.04$] and physical aggression at T2 [$F(1, 65) = 6.30$, $p = 0.02$, Adjusted $R^2 = 0.07$], such that girls had higher relational aggression scores than boys and boys had higher physical aggression scores than girls. No other significant gender differences were found. Room temperature was associated with SCL-R ($r = 0.30$, $p < 0.01$) and RSA-R ($r = -.29$, $p < 0.01$). Baseline SCL was also controlled given that it was related to missing data.

Primary Analyses¹

Relational Victimization and Relational Aggression Models

First, a model was tested which examined relational victimization at T2, SCL-R, and RSA-R as predictors of relational aggression at T3. Results provided an acceptable fit to the data [$\chi^2(9) = 14.71$, $p = 0.12$, CFI = 0.95, SRMR = 0.06, RMSEA = 0.09]. Relational victimization at T2 was non-significantly but marginally related to higher levels of relational aggression at T3 ($\beta = 0.36$, $p = 0.06$) and there was stability in relational aggression from T2 to T3 ($\beta = 0.49$, $p = 0.001$). See Table 3 for unstandardized estimates. Physical victimization at T2 was not related to relational aggression at T3, so was removed from subsequent models to increase parsimony.

A model with all two-way interactions included provided an acceptable fit to the data [$\chi^2(17) = 22.88$, $p = 0.15$, CFI = 0.92, SRMR = 0.06, RMSEA = 0.06]. None of the two-way interaction terms emerged as significant in models testing these interactions (see Table 3). Next, the three-way interaction term between relational victimization, SCL-R, and RSA-R was tested controlling for all lower order interaction terms. The model provided an acceptable fit to the

¹ The base and final interaction models were also run controlling for nesting within school. These analyses are included in the supplemental materials and mirrored the results in the main text, with the exception of the main effect of SCL-R on physical aggression at T3, which was no longer significant.

Table 2 Descriptive Statistics and Correlations

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. SCL-R T1	—									
2. RSA-R T1	-0.05	—								
3. Ragg TR T2	0.28*	-0.03	—							
4. Pagg TR T2	0.13	-0.22	0.47**	—						
5. Rvict TR T2	0.33*	-0.13	0.55**	0.46**	—					
6. Pvict TR T2	0.29*	-0.31*	0.34**	0.56**	0.66**	—				
7. Rec Pro TR T2	0.20	0.05	0.11	-0.20	-0.11	-0.18	—			
8. Ragg TR T3	0.13	0.12	0.57**	0.26*	0.42**	0.10	-0.03	—		
9. Pagg TR T3	-0.05	-0.06	0.22	0.57**	0.37**	0.35**	-0.42**	0.41**	—	
10. Age (months)	-0.004	0.09	-0.04	0.10	-0.10	-0.06	-0.23	-0.02	-0.02	—
<i>M</i>	2.14	-0.11	9.26	7.72	1.69	1.39	3.45	10.54	8.53	45.58
<i>SD</i>	3.64	0.40	4.34	2.66	0.80	0.62	0.87	4.64	3.49	3.86
<i>Range</i>	-7.95–13.10	-1.16–1.25	6.00–20.87	6.00–16.48	1.00–3.50	1.00–2.95	1.40–5.00	6.00–22.89	6.00–18.74	36.13–61.97

Ragg Relational aggression, Pagg Physical aggression, SCL-R Skin Conductance Level reactivity, RSA-R Respiratory Sinus Arrhythmia reactivity, TR Teacher report, Rec Pro Received prosocial behavior, Rvict Relational victimization, Pvict Physical victimization, T1 Time 1, T2 Time 2, T3 Time 3

* $p < 0.05$, ** $p < 0.01$

Table 3 Unstandardized Estimates From the Peer Risk Models

Model 1 – Relational Aggression Base Model				Model 2 – Relational Aggression Two-way Interaction Model				Model 3 – Relational Aggression Three-way Interaction Model			
<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²
Relational Aggression T3				Relational Aggression T3				Relational Aggression T3			
Ragg T2	0.53**	0.17	0.001	Ragg T2	0.53**	0.17	0.002	Ragg T2	0.50**	0.17	0.003
Rvict T2	1.71	0.90	0.06	Rvict T2	1.06	0.60	0.08	Rvict T2	1.07	0.59	0.07
Pvict T2	-1.02	0.91	0.26	Pvict T2	—	—	—	Pvict T2	—	—	—
C Gender	0.06	0.49	0.90	C Gender	-0.14	0.47	0.76	C Gender	-0.22	0.46	0.64
RSA-R	0.86	0.57	0.13	RSA-R	1.18	0.71	0.09	RSA-R	0.81	0.69	0.24
SCL-R	-0.37	0.48	0.43	SCL-R	-0.34	0.50	0.50	SCL-R	-0.18	0.45	0.69
				RVict T2 x RSA-R	-0.04	0.45	0.93	RVict T2 x RSA-R	0.35	0.50	0.48
				RVict T2 x SCL-R	-0.01	0.38	0.98	RVict T2 x SCL-R	0.55	0.42	0.19
				RSA-R x SCL-R	0.44	0.88	0.61	RSA-R x SCL-R	0.41	0.80	0.61
								RVict T2 x	1.22*	0.58	0.04
								SCL-R x RSA-R			
Model 4 – Physical Aggression Base Model				Model 5 – Physical Aggression Two-way Interaction Model				Model 6 – Physical Aggression Three-way Interaction Model			
<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²
Physical Aggression T3				Physical Aggression T3				Physical Aggression T3			
Pagg T2	0.60**	0.17	< 0.001	Pagg T2	0.70	.15	< 0.001	Pagg T2	0.70	0.16	< 0.001
Rvict T2	0.93	0.69	0.18	Rvict T2	—	—	—	Rvict T2	—	—	—
Pvict T2	0.03	0.68	0.96	Pvict T2	0.41	0.68	0.54	Pvict T2	0.50	0.69	0.47
C Gender	0.53	0.35	0.13	C Gender	0.35	0.41	0.39	C Gender	0.37	0.41	0.36
RSA-R	0.44	0.26	0.09	RSA-R	0.42	0.27	0.12	RSA-R	0.64*	0.31	0.04
SCL-R	-0.69*	0.30	0.02	SCL-R	-0.59*	0.27	0.03	SCL-R	-0.63*	0.28	0.03
				PVict T2 x RSA-R	0.05	0.50	0.92	PVict T2 x RSA-R	-0.13	0.41	0.75
				PVict T2 x SCL-R	0.20	0.44	0.65	PVict T2 x SCL-R	-0.08	0.45	0.86
				RSA-R x SCL-R	-0.05	0.32	0.88	RSA-R x SCL-R	0.10	0.30	0.74
								PVict T2 x	-0.54	0.34	0.11
								SCL-R x RSA-R			

Bolded coefficients denote significant effects

Baseline RSA, baseline SCL, and room temperature were controlled. Gender was coded -1 = girls, 1 = boys

SCL-R Skin Conductance Level reactivity, RSA-R Respiratory Sinus Arrhythmia reactivity, Ragg Relational aggression, Pagg Physical aggression, Rvict Relational victimization, Pvict Physical victimization, C Child, T2 Time 2, T3 Time 3

p* < 0.05; *p* < 0.01

data [$\chi^2(19)=25.07$, $p=0.16$, CFI=0.92, SRMR=0.06, RMSEA=0.06] and the three-way interaction was significant ($b=1.22$, $SE=0.58$, $p=0.04$). The interaction was probed further to see whether the relation between relational victimization at T2 and relational aggression at T3 was significant for each of the different patterns (see Fig. 1). Results demonstrated that there was a significant positive relation between relational victimization at T2 and relational aggression at T3 among children exhibiting a coactivation pattern (i.e., high levels of SCL-R and high levels of RSA-R; $b=3.19$, $SE=1.13$, $p=0.005$). However, there were no significant effect for children displaying reciprocal PNS activation (i.e., low levels of SCL-R and high levels of RSA-R; $b=-.35$, $SE=1.29$, $p=0.79$), coinhibition (i.e., low levels of SCL-R and low levels of RSA-R; $b=1.39$, $SE=0.82$, $p=0.09$), or reciprocal SNS activation (i.e., high levels of SCL-R and low levels of RSA-R; $b=0.05$, $SE=0.91$, $p=0.96$). When removing baseline RSA and SCL from the model as a robustness test, the three-way interaction was still significant.

Physical Victimization and Physical Aggression Models

First, a model was tested which examined physical victimization at T2, SCL-R, and RSA-R as predictors of

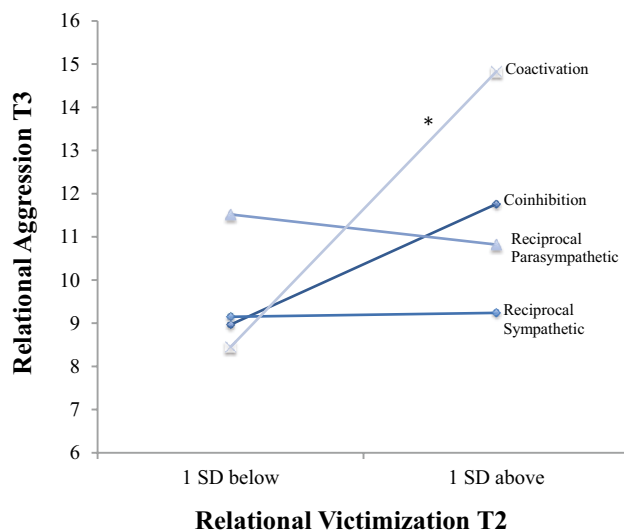


Fig. 1 Interaction Between Relational Victimization at T2, SCL-R at T1, and RSA-R at T1 in Predicting Relational Aggression at T3. Note. $**p < 0.01$. SCL-R = Skin Conductance Level reactivity, RSA-R = Respiratory Sinus Arrhythmia reactivity, T1 = Time 1, T2 = Time 2, T3 = Time 3. In response to a bullying (i.e., social exclusion) stimulus a coactivation pattern represents high SCL-R and high RSA-R, a coinhibition pattern represents low SCL-R and low RSA-R, a reciprocal sympathetic pattern represents high SCL-R and low RSA-R, and a reciprocal parasympathetic pattern represents low SCL-R and high RSA-R. Relational aggression at T2 and other relevant covariates were controlled (see text)

physical aggression at T3. Results provided a good fit to the data [$\chi^2(9)=7.43$, $p=0.59$, CFI=1.00, SRMR=0.06, RMSEA=0.00]. Higher levels of SCL-R were associated with lower levels of physical aggression at T3 ($\beta=-.19$, $p=0.02$) and there was stability in aggression from T2 to T3 ($\beta=0.45$, $p < 0.001$). Relational victimization at T2 was not related to physical aggression at T3, so was removed in subsequent models to increase parsimony. See Table 3 for unstandardized estimates.

A model with all two-way interactions included [$\chi^2(17)=36.34$, $p=0.004$, CFI=0.83, SRMR=0.08, RMSEA=0.12] provided a poor fit to the data. Modification indices and residual covariances were examined to determine sources of model misfit. Adding a covariance between physical aggression at T2 and the physical victimization and RSA-R interaction term resulted in a model that provided acceptable fit to the data [$\chi^2(16)=26.17$, $p=0.05$, CFI=0.91, SRMR=0.07, RMSEA=0.09]. This covariance was retained in the three-way interaction model. None of the two-way interactions were significant (see Table 3). Next, a three-way interaction between received physical victimization, SCL-R, and RSA-R was tested. The model provided marginal fit to the data [$\chi^2(18)=30.87$, $p=0.03$, CFI=0.89, SRMR=0.07, RMSEA=0.09] but the three-way interaction was not significant ($b=-.54$, $SE=0.34$, $p=0.11$). When removing baseline RSA and SCL as a robustness test, the three-way interaction remained non-significant.

Received Prosocial Behavior and Relational Aggression

First, a model was tested which examined received prosocial behavior, SCL-R and RSA-R as predictors of relational aggression at T3. Results provided an acceptable fit to the data [$\chi^2(9)=13.27$, $p=0.15$, CFI=0.95, SRMR=0.06, RMSEA=0.07]. There was stability in relational aggression from T2 to T3 ($\beta=0.56$, $p=0.001$) but no other predictor emerged as significant. See Table 4 for unstandardized estimates. Physical aggression at T2 was not related to relational aggression at T3, so was removed from subsequent models.

A model with all two-way interactions included provided an acceptable fit to the data [$\chi^2(17)=23.40$, $p=0.14$, CFI=0.91, SRMR=0.06, RMSEA=0.07]. None of the two-way interaction terms emerged as significant in the model testing these interactions (see Table 4). Next, a three-way interaction between received prosocial behavior, SCL-R, and RSA-R was tested. The model provided an acceptable fit to the data [$\chi^2(19)=25.55$, $p=0.14$, CFI=0.91, SRMR=0.06, RMSEA=0.06] and the three-way interaction was significant ($b=1.68$, $SE=0.62$, $p=0.007$). Follow-up simple slope analyses (see Fig. 2a) indicated that received prosocial behavior at T2 was related to lower relational aggression at T3 among children exhibiting reciprocal sympathetic activation (i.e., high levels of SCL-R and low levels of RSA-R; $b=-2.51$,

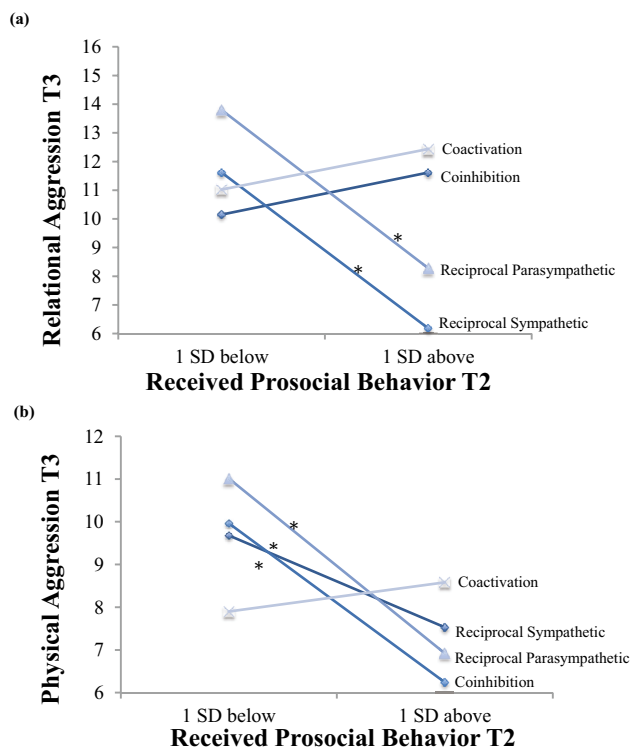


Fig. 2 Interaction Between Received Prosocial Behavior at T2, SCL-R at T1, and RSA-R at T1 in Predicting (a) Relational Aggression at T3 and (b) Physical Aggression at T3. Note. * $p < 0.05$. SCL-R = Skin Conductance Level reactivity, RSA-R = Respiratory Sinus Arrhythmia reactivity, T1 = Time 1, T2 = Time 2, T3 = Time 3

$SE = 1.02$, $p = 0.01$) and children exhibiting reciprocal PNS activation (i.e., low levels of SCL-R and high levels of RSA-R; $b = -2.76$, $SE = 1.13$, $p = 0.02$). Received prosocial behavior was not associated with T3 relational aggression among children exhibiting coinhibition (i.e., low levels of SCL-R and low levels of RSA-R; $b = 0.73$, $SE = 0.69$, $p = 0.29$) or coactivation (i.e., high levels of SCL-R and high levels of RSA-R; $b = 0.71$, $SE = 1.47$, $p = 0.63$). When removing baseline RSA and SCL as a robustness test, the three-way interaction remained significant.

Received Prosocial Behavior and Physical Aggression

First, a model was tested which examined received prosocial behavior, SCL-R, and RSA-R as predictors of physical aggression at T3. Results provided a good fit to the data [$\chi^2(9) = 7.83$, $p = 0.55$, CFI = 1.00, SRMR = 0.05, RMSEA = 0.00]. Received prosocial behavior at T2 predicted lower physical aggression scores at T3 ($\beta = -0.30$, $p = 0.01$). There was also stability in physical aggression from T2 to T3 ($\beta = 0.51$, $p < 0.001$). See Table 4 for unstandardized estimates. Relational aggression at T2 was not related to physical aggression at T3 so was dropped from subsequent models.

A model with all two-way interactions included provided an acceptable fit to the data [$\chi^2(17) = 21.44$, $p = 0.21$, CFI = 0.94, SRMR = 0.06, RMSEA = 0.06]. None of the two-way interaction terms emerged as significant. Next, a three-way interaction between received prosocial behavior, SCL-R, and RSA-R was added to the model. The model provided an acceptable fit to the data [$\chi^2(19) = 23.33$, $p = 0.22$, CFI = 0.95, SRMR = 0.06, RMSEA = 0.05] and the three-way interaction was significant ($b = 0.79$, $SE = 0.36$, $p = 0.03$). Follow-up simple slope analyses (see Fig. 2b) indicated that among children exhibiting coactivation, there was no relation between received prosocial behavior at T2 and physical aggression at T3 ($b = 0.34$, $SE = 0.77$, $p = 0.66$). Received prosocial behavior at T2 was negatively related to physical aggression at T3 among children exhibiting coinhibition ($b = -1.07$, $SE = 0.49$, $p = 0.03$), reciprocal PNS activation ($b = -2.04$, $SE = 0.97$, $p = 0.04$), and reciprocal SNS activation ($b = -1.86$, $SE = 0.72$, $p = 0.01$). When removing baseline RSA and SCL as a robustness test, the three-way interaction remained significant.

Discussion

The goal of the current study was to test whether there were interactions between the SNS and PNS systems in moderating longitudinal relations between peer risk and protective factors and aggression. Two competing hypotheses were examined. First, based on biological sensitivity to context theory, we examined whether a reciprocal SNS pattern would serve as an indicator of greater sensitivity to the environment, and thus would make youth more susceptible to both negative and positive peer treatment. Second, based on prior research, which has found nonreciprocal patterns to be a vulnerability factor, we examined whether non-reciprocal patterns would reflect greater vulnerability to negative peer contexts. Results indicated that interactions between SNS and PNS systems moderated associations between peer treatment and aggression, such that a coactivation pattern (i.e., high levels of SCL-R and RSA-R) in response to a bullying stimulus conferred risk in the context of peer victimization, whereas reciprocal patterns conferred benefits in the context of received prosocial behavior from peers. These findings are not consistent with biological sensitivity to context theory, as different physiological patterns were associated with vulnerability to peer victimization versus sensitivity to positive peer experiences. Instead, findings suggest that adaptive ANS patterns may make youth especially likely to benefit from positive peer experiences, whereas maladaptive patterns may place them at risk in the context of peer adversity.

With respect to vulnerability to peer victimization, relational victimization at T2 was related to higher relational aggression at T3 among children exhibiting coactivation. In

Table 4 Unstandardized Effects From the Received Prosocial Behavior Models

Model 1 – Relational Aggression Base Model				Model 2 – Relational Aggression Two-way Interaction Model				Model 3 – Relational Aggression Three-way Interaction Model			
<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²
Relational Aggression T3				Relational Aggression T3				Relational Aggression T3			
Ragg T2	0.61**	0.18	< 0.001	Ragg T2	0.68**	0.15	< 0.001	Ragg T2	0.72**	0.15	< 0.001
Pagg T2	0.10	0.21	0.64	Pagg T2	—	—	—	Pagg T2	—	—	—
Rec Pro T2	-0.46	0.48	0.33	Rec Pro T2	-0.62	0.57	0.28	Rec Pro T2	-0.96*	0.47	0.04
C Gender	-0.32	0.45	0.47	C Gender	-0.27	0.47	0.57	C Gender	-0.23	0.46	0.61
RSA-R	0.95	0.57	0.09	RSA-R	1.12	0.66	0.09	RSA-R	0.80	0.55	0.14
SCL-R	-0.13	0.44	0.77	SCL-R	-0.14	0.52	0.79	SCL-R	-0.38	0.54	0.49
				RecPro T2 x RSA-R	-0.18	0.74	0.81	RecPro T2 x RSA-R	-0.07	0.60	0.91
				RecPro T2 x SCL-R	-0.56	0.48	0.25	RecPro T2 x SCL-R	0.06	0.52	0.91
				RSA-R x SCL-R	0.63	0.97	0.52	RSA-R x SCL-R	0.72	0.74	0.33
								RecPro T2 x SCL-R x RSA-R	1.68**	0.62	0.007
Model 4 – Physical Aggression Base Model				Model 5 – Physical Aggression Two-way Interaction Model				Model 6 – Physical Aggression Three-way Interaction Model			
<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²	<i>b</i>	<i>SE</i>	<i>p</i>	Overall <i>R</i> ²
Physical Aggression T3				Physical Aggression T3				Physical Aggression T3			
Ragg T2	0.04	0.11	0.69	Ragg T2	—	—	—	Ragg T2	—	—	—
Pagg T2	0.66**	0.15	< 0.001	Pagg T2	0.69**	0.13	< 0.001	Pagg T2	0.68**	0.13	< 0.001
Rec Pro T2	-1.05*	0.41	0.01	Rec Pro T2	-1.00*	0.39	0.01	Rec Pro T2	-1.16**	0.39	0.003
C Gender	0.18	0.32	0.59	C Gender	0.10	0.36	0.79	C Gender	0.11	0.36	0.77
RSA-R	0.33	0.29	0.26	RSA-R	0.28	0.32	0.38	RSA-R	0.12	0.34	0.72
SCL-R	-0.29	0.29	0.33	SCL-R	-0.21	0.25	0.39	SCL-R	-0.31	0.26	0.24
				RecPro T2 x RSA-R	0.24	0.48	0.62	RecPro T2 x RSA-R	0.31	0.45	0.49
				RecPro T2 x SCL-R	0.11	0.27	0.69	RecPro T2 x SCL-R	0.40	0.30	0.18
				RSA-R x SCL-R	-0.10	0.39	0.81	RSA-R x SCL-R	-0.05	0.37	0.88
								RecPro T2 x SCL-R x RSA-R	0.79*	0.36	0.03

Bolded coefficients denote significant effects

Baseline RSA and SCL and room temperature were controlled. Gender was coded -1 = girls, 1 = boys

SCL-R Skin Conductance Level reactivity, RSA-R Respiratory Sinus Arrhythmia reactivity, Ragg Relational aggression, Pagg Physical aggression, RecPro Received Prosocial behavior, C Child, T2 Time 2, T3 = Time 3

* $p < 0.05$; ** $p < 0.01$

contrast, children exhibiting coordinated, reciprocal physiological responses to exclusion (i.e., reciprocal sympathetic or parasympathetic activation), as well as children exhibiting coinhibition, appeared resilient in the face of peer victimization; in fact, among these youth, relational victimization at T2 was unrelated to relational aggression at T3. Coactivation may be indicative of SNS activation when the PNS response is not sufficient for regulation, reflecting dysregulated emotion (Beauchaine et al., 2007; El-Sheikh & Erath, 2011; El-Sheikh et al., 2009). Researchers have argued that this response is maladaptive and may facilitate dysregulated fight-flight-freeze responses to stress (El-Sheikh & Erath, 2011). In the context of peer relational victimization, children who exhibit coactivation to peer stress may be at particularly high risk for responding with aggression.

These findings are also congruent with several other studies examining psychophysiology as a moderator of relations between environmental factors and outcomes. In fact, although some findings are mixed (e.g., Abaied et al., 2018), researchers have found that in middle childhood and adolescence, coinhibition and coactivation may serve as vulnerability factors (El-Sheikh et al., 2009; Gordis et al., 2010; McKernan & Lucas-Thompson, 2018; Philbrook et al., 2018). For example, prior work has found that there is a link between child maltreatment and aggression among girls with non-reciprocal ANS activation (i.e., coactivation or coinhibition pattern; Gordis et al., 2010). Similarly, children demonstrating a coactivation pattern who have parents with high levels of marital conflict experienced increased internalizing and externalizing behavior problems across adolescence (Philbrook et al., 2018). Our novel work extends these findings to the peer domain in early childhood, such that relational victimization predicted increased relational aggression, but only among children displaying a coactivation pattern to a bullying stimulus. However, our findings for coinhibition were in contrast with these prior findings, such that for these children relational victimization did not enhance risk for subsequent relational aggression. Additionally, children exhibiting coinhibition benefited from positive peer treatment, as received prosocial behavior at T2 was related to lower physical (though not relational) aggression at T3 among these youth. In prior research, a coinhibition pattern at baseline in combination with parent marital conflict, has been found to be related to elevated risk for internalizing problems in middle childhood, whereas a coactivation pattern was not related to internalizing problems (El-Sheikh et al., 2013). Therefore, even though prior research has supported that either nonreciprocal pattern may represent a vulnerability factor, children may express this vulnerability in different ways resulting in various maladaptive outcomes.

A different pattern of effects emerged in analyses investigating which children benefited most from received prosocial behavior. Children exhibiting coordinated, reciprocal

physiological responses to exclusion (i.e., reciprocal SNS or PNS activation) appeared to be sensitive to positive peer experiences, such that received prosocial behavior at T2 was related to lower physical and relational aggression at T3 among these children. In contrast, received prosocial behavior was unrelated to physical or relational aggression at T3 among children with coactivation. These results suggest that reciprocal patterns facilitate sensitivity to protective environmental effects. Prior researchers have posited that reciprocal patterns in the context of stress promote adaptive responses, such as mobilization of resources and emotion regulation (El-Sheikh et al., 2009). In addition, these findings are consistent with prior work indicating that adolescents who experienced a reciprocal SNS pattern and had higher levels of maternal involvement exhibited lower levels of depression (Abaied et al., 2018). Findings from the present study extend this prior work and underscore the potential benefits of these adaptive physiological responses in the context of supportive peer experiences.

Importantly, no physiological pattern conferred sensitivity to both protective and risky peer contexts. These findings are inconsistent with biological sensitivity to context theory, which posits that there are plasticity factors that make youth more susceptible to both supportive and risky environmental contexts (Boyce & Ellis, 2005; Ellis et al., 2011). Instead, our findings suggest that a coactivation pattern to peer exclusion may make youth vulnerable to peer victimization, whereas adaptive patterns (i.e., reciprocal; El-Sheikh & Erath, 2011) may make youth especially likely to benefit from positive peer treatment. It is possible that children with a coactivation pattern in response to an exclusion event may attend to negative and not positive stimuli in their peer environment, making them less likely to benefit from received prosocial behavior, but susceptible to the negative effects of relational victimization. Conversely, children with a reciprocal pattern may have adaptive responses to positive and stressful peer events, thus increasing the likelihood that they benefit from positive peer events and reducing the impact of stressful peer events, such as victimization. This possibility is consistent with the emotion integrated Social Information Processing (SIP) model, which posits that arousal influences how a child interprets a social situation with children often incorporating a mood congruent interpretation (Lemerise & Arsenio, 2000). Future research integrating patterns of SIP are necessary to investigate this possibility.

There was no evidence that ANS patterns moderated associations between physical victimization and physical aggression. This may be because of the context of the physiology stressor, which was relational in nature. However, there was a direct association between SCL-R and physical aggression at T3, such that lower levels of SCL-R were associated with higher levels of physical aggression. This main effect is consistent with fearlessness and stimulation-seeking

theories of aggression, which posit that physiological underarousal is associated with aggressive behavior (Raine et al., 1998). However, as this association was not significant in the analyses controlling for nesting within school, this effect should be interpreted with caution.

Implications

Overall, results demonstrate that children with a coactivation pattern may be more sensitive to negative peer contexts and less sensitive to positive peer contexts. In contrast, children with reciprocal patterns may be less sensitive to a negative peer context and more sensitive to positive peer contexts. These findings have important initial implications for intervention efforts. Specifically, children with a coactivation pattern may experience limited benefits from interventions targeting peer treatment, such as classroom-level interventions aimed at reducing peer victimization experiences. This is congruent with prior research, which has found that children with certain patterns of physiological reactivity (i.e., higher SCL reactivity) receive less benefit from psychological treatments (Dieleman et al., 2016). Examining these different physiological patterns is a novel area of study and therefore, more research is needed to understand how children who are at an increased physiological risk may best respond to interventions. Determining how interventions could target this physiological risk and adapting interventions to address this risk are important avenues for future research.

Limitations and Future Directions

Despite strengths from the study, including the longitudinal design, the use of multiple methods and informants, and the inclusion of psychophysiology data, there are a number of important limitations. Due to the school-based nature of the data collection, missing data was expected given that children frequently change preschools and transition to formal schooling (e.g., 22% of the sample from T1 to T2). Although missing data was accommodated using FIML and any variables related to missing data were included in the model, consistent with best practice recommendations (i.e., Little et al., 2014), it is not possible to test the impact of missing data on study findings. Additionally, this sample was small ($n=86$), although commensurate with other samples examining three-way interactions between environmental effects and psychophysiological variables in childhood and adolescence (e.g., $N=68$, Benito-Gomez et al., 2018; $N=61$; Lafko et al., 2015) and previous published work from this sample (Perhamus et al., 2022). We did not run a post-hoc power analysis given significant concerns regarding this approach (Zhang et al., 2019, p. 4). Overall, the three-way interaction effects were small to moderate in size (i.e., effect

size estimates ranged from approximately 0.03 to 0.08 based on change in R^2) and future research should be implemented accordingly. A sensitivity analysis determined that we were reliably powered to detect a medium effect (i.e., partial R^2 of 0.135) for the regression estimates with our sample size. Therefore, we cannot conclude whether the null effects observed in this study are the result of reduced power to detect small effects or whether they are null effects. This novel study offers preliminary evidence of the dual influence of the SNS and PNS systems in the peer domain and we hope that future research replicates findings from this study and extends this research to examine gender effects and interactions with non-specific relations between victimization and aggression (e.g., relational victimization on physical aggression).

The context of the peer stressor likely played a role. Meta-analytic work has demonstrated that discrepancies in the RSA-R literature may partially reflect differences in reactivity paradigms (Beauchaine et al., 2019). Children's physiological reactivity was assessed using a developmentally appropriate stressor, where they were exposed to a relatively benign peer stressor (i.e., video of a TV character being excluded). This may have dampened effects because children were not actually experiencing the exclusion themselves and physiological reactivity in this context may not be generalizable to other contexts. Moreover, in the current study we were unable to examine reciprocal relations between aggression, peer victimization, and received prosocial behavior despite a large body of research that suggests these associations are bidirectional (e.g., Ostrov, 2008, 2010). Future research should test whether the patterns moderate links between aggression and subsequent victimization and received prosocial behavior. Additionally, consistent with the specificity hypothesis, we only tested the moderation analyses for the within form of aggression (e.g., relational victimization on relational aggression). This is congruent with other research which has only examined the within form of victimization on aggression (Kawabata et al., 2014). Nonetheless, there are often bivariate associations across forms of aggression and victimization (Farrell et al., 2016; Leadbeater et al., 2006) and future research with a larger sample should test the moderation findings for these non-specific relations while controlling for specific relations.

In terms of demographic limitations of the sample, children were recruited from NAEYC accredited or recently accredited childcare centers, were predominately White (70.2% White), and were middle to upper middle class, and therefore, it will be important to replicate findings with more ethnically/racially and economically diverse samples. Additionally, the sample was typically developing and therefore victimization and aggression values were lower than they would be in a clinical sample and the physiology range may

also have been limited, which may impact our ability to test biological sensitivity to context theory. Therefore, results are not necessarily generalizable to other geographic regions or higher risk groups.

Future research should continue to examine the dual role of both branches of the ANS in relations between peer risk and protective factors and aggressive behaviors. In the current study, we used a statistical cutoff (e.g., one standard deviation above and below the mean) to assess the various patterns. Future research with larger sample sizes may benefit from using a person-centered approach to evaluate whether there are different profiles of autonomic nervous system response. Additionally, there is much less work on the role of protective factors and the dual role of the SNS and PNS, and future research would benefit from greater attention to whether physiological factors confer sensitivity to positive contexts. Early childhood has generally been understudied in this area of research. It is possible that different physiological patterns confer risk or protection at different developmental periods. Therefore, it is essential that research continue to include interactions among the SNS and PNS across developmental periods.

Conclusions

The goal of the current study was to examine the role of ANS reactivity in relations between peer factors and aggressive behavior. In contrast to hypotheses, biological sensitivity to context theory was not supported; instead, children displaying a coactivation pattern were more sensitive to a negative peer context (i.e., relational victimization) but less sensitive to a positive peer context (i.e., received prosocial behavior). Conversely, children demonstrating reciprocal SNS or PNS activation had lower levels of aggression when received prosocial behavior was high, but did not experience negative effects from peer victimization factors. In sum, this novel study demonstrates the importance of examining interactions among the SNS and PNS, the significance of studying these constructs in early childhood, and the power of the peer context in predicting changes in aggressive behavior.

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Data Availability Data is available by contacting the authors.

Compliance with Ethical Standards

Ethical Approval The content is solely the responsibility of the authors and does not represent the official views of the National Science Foundation.

Informed Consent All procedures in the study were approved by the University at Buffalo IRB and all participants provided informed consent.

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