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Early Community Violence Exposure and Adolescent Aggressive Behavior: Moderation by Autonomic Nervous System Reactivity

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Childhood community violence exposure (CVE) is longitudinally associated with later aggression, but questions remain regarding how CVE influences different forms (i.e., physical and relational) and functions (i.e., proactive and reactive) of aggression. Additionally, the moderating influence of children's autonomic nervous system reactivity to fear on associations between CVE and aggression subtypes remains unclear. The present study investigated the joint role of CVE and autonomic nervous system reactivity to fear at age 7 in the development of aggression subtypes across 5 years in a diverse community sample of 197 children (50% assigned female, 46% Latine). Results indicated CVE was longitudinally related to greater reactive physical aggression among boys exhibiting parasympathetic nervous system (PNS) inhibition but lower reactive physical aggression among boys exhibiting PNS activation. Conversely, CVE predicted greater proactive relational aggression for girls exhibiting sympathetic nervous system (SNS) inhibition but not for girls exhibiting SNS activation. Several interactions among CVE, SNS reactivity, and PNS reactivity also emerged. CVE was longitudinally associated with greater proactive relational aggression among boys exhibiting coactivation (i.e., increases in SNS and PNS activation) and with lower proactive relational aggression among boys with reciprocal SNS activation (i.e., SNS activation with PNS inhibition). In contrast, CVE was linked to greater reactive physical aggression among girls with reciprocal SNS activation and with lower reactive physical aggression among girls with coinhibition (i.e., SNS inhibition with PNS inhibition). These findings suggest distinct pathways from CVE to aggression based on gender and autonomic nervous system reactivity, highlighting the need for targeted, context-sensitive interventions that address both environmental and physiological factors.

Public Significance Statement

This study highlights how community violence exposure and children's physiological reactivity to fear jointly predict distinct forms and functions of aggression over time. Findings indicate that physiological reactivity patterns and gender shape the developmental impact of community violence exposure, underscoring the need for tailored interventions that address both environmental and physiological factors to reduce aggression in children.

Keywords: autonomic nervous system reactivity, physical aggression, relational aggression, proactive aggression, reactive aggression

Supplemental materials: https://doi.org/10.1037/dev0002146.supp

Aggressive behaviors are associated with negative psychosocial outcomes, and early intervention is important to mitigate later maladjustment for aggressive youth (Card & Hodges, 2008). Community violence exposure (CVE) in early life is one well-known risk factor for aggressive behavior (Scarpa, 2003). However, less is known about how CVE relates to the development of different forms (i.e., physical and relational) and functions (i.e., proactive and reactive) of aggression, as well as if and how individual characteristics, such as autonomic nervous system (ANS) regulation, might interact with CVE in the prediction of these outcomes. The purpose of this

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longitudinal investigation was to evaluate prospective associations between childhood CVE and subtypes of aggressive behavior expression in early adolescence, as well as the moderating effects of ANS reactivity (ANS-R) on these pathways. We also explored potential differences in these patterns of effects based on children's gender as indicated by their assigned sex at birth.

CVE, defined as witnessing or experiencing violence in settings such as neighborhoods, streets, or schools (Buka et al., 2001), is a significant risk factor for various forms and functions of aggression (McCabe et al., 2005; Perkins & Graham-Bermann, 2012). CVE may contribute to physical forms of aggression (i.e., behaviors that cause physical harm; Henninger, 2004) by normalizing violent behaviors as acceptable means of conflict resolution (Bradshaw et al., 2009; Calvete & Orue, 2011). Similarly, CVE may be associated with relational forms of aggression (i.e., behaviors that damage relationships, such as maliciously spreading rumors) by reinforcing social norms that endorse manipulation and social harm as strategies to gain power (Low & Espelage, 2014). In addition, CVE may be linked with multiple functions of aggression, including proactive functions of aggression (i.e., intentional aggressive behaviors used to obtain a perceived goal or reward; Bandura, 1973) and reactive functions of aggression (i.e., aggression resulting from frustration or anger in response to a perceived threat; Martinelli et al., 2018; Murray-Close & Ostrov, 2009). CVE may be related to proactive functions of aggression due to biased social–cognitive processes including normative beliefs and diminished guilt (Chaux et al., 2012; McMahon et al., 2009) as well as perceptions that aggression will result in positive outcomes (Bradshaw et al., 2009; Calvete & Orue, 2011). CVE may also facilitate reactive functions of aggression because it leads to hostile social cognitions (Martinelli et al., 2018) and primes youth to view aggressive responses as justifiable defenses (McMahon et al., 2009). Importantly, physical and relational forms of aggression can each be proactive and reactive in function. For instance, a relationally aggressive behavior, such as threatening to terminate a friendship, can be used to manipulate others to give the child something they want (i.e., proactive function) or to retaliate against real or perceived slights by causing socioemotional pain (i.e., reactive function).

Importantly, not all children exposed to CVE engage in aggressive behavior (e.g., Guerra et al., 2003), suggesting some youth may be especially vulnerable to the effects of CVE. One potential vulnerability is dysregulated functioning of the ANS, which includes the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS coordinates the "fight or flight" response, which helps mobilize necessary resources for coping (Murray-Close et al., 2017). SNS activity is commonly assessed using pre-ejection period (PEP), an inverse measure of SNS influence on cardiovascular function (Murray-Close, 2013). Shorter PEP reflects SNS activation (e.g., faster heart rate and heightened mobilization of resources), whereas longer PEP reflects SNS inhibition (e.g., slower heart rate and reduced mobilization; El-Sheikh & Erath, 2011). Functioning of the PNS, often referred to as the "rest and digest" system, may also have important implications for the development of aggression subtypes. Respiratory sinus arrhythmia (RSA), defined as heart rate variability linked to breathing, is commonly used to assess PNS activity. Higher RSA reflects PNS activation (e.g., stronger inhibition of heart rate, promoting calming and restoration), whereas lower RSA reflects PNS inhibition (e.g., weaker inhibition of heart rate, supporting cardiac mobilization; Porges, 1995).

Accumulating research indicates that dysregulation of SNS reactivity (SNS-R) is a risk factor for both physical and relational forms of aggression (Murray-Close, 2013), but the direction of this association has been mixed (Sijtsema et al., 2011), perhaps reflecting differences in stimuli used to elicit reactivity and/or a lack of attention to distinct functions of aggression. Indeed, the implications of ANS-R for the development of aggression likely depend on the context in which reactivity is assessed. Prior research indicates that physiological reactivity to stimuli that are negative or aversive (e.g., induce fear or frustration) is more reliably associated with antisocial behavior than physiological reactivity to positive stimuli (Lorber, 2004). Further, CVE is related to increased fear (Patton et al., 2012; Rojas-Flores et al., 2013), and theoretical models underscore the importance of both PNS reactivity (PNS-R) and SNS-R to fear in the development of different functions of aggression (Blankenstein et al., 2022; Gao et al., 2015). However, most studies investigating ANS-R and aggression have used frustration or cognitive tasks rather than reactivity to emotions like fear (e.g., Chong et al., 2022).

According to fearlessness theory (Raine, 2014), underarousal of the SNS reflects an inability to experience appropriate levels of fear, which reduces responsiveness to punishment and ultimately interferes with adaptive social development (Murray-Close et al., 2017; Raine, 1997). Children exhibiting SNS inhibition to fear may not effectively learn from negative consequences, leading to increased risk-taking and aggressive behaviors. These individuals may be at risk for proactive functions of aggression because of their lower sensitivity to potential risks and negative consequences. Indeed, in one recent study, SNS inhibition to fear (measured by skin conductance level reactivity) was related to proactive aggression in men (Thomson et al., 2021). In contrast, the frustration–aggression hypothesis (Berkowitz, 1989) purports that *overarousal* of the SNS following an aversive event reflects a negative emotional state, which leads to aggressive behavior (Hubbard et al., 2002). Although this theory emphasizes SNS activation in response to frustration, Thomson et al. (2021) argued that reactive functions of aggression will be associated with hypersensitivity to threat, including SNS activation to fear. In support of this assertion, these authors reported that SNS activation to fear was associated with reactive aggression in both men and women (Thomson et al., 2021). Importantly, the differential relations between SNS-R and the forms and functions of aggression by gender underscore the need to consider these associations across and within gender groups.

As with the SNS, the meaning and implications of PNS activity also depend on the context in which it is assessed. Porges (2007) suggested that PNS activity that is appropriate to the context will facilitate adaptive behavioral responses. For instance, in safe environments that require social engagement, PNS activation (i.e., higher RSA) may be adaptive because it induces states of relaxation that support social interaction (Porges, 2007). Conversely, in response to threat, PNS inhibition (i.e., RSA withdrawal) may be adaptive because it enables the mobilization of resources for effective coping (Porges, 2007). For instance, Thomson (2022) demonstrated that PNS activation to fear (i.e., increased RSA) was associated with impulsive–antisocial traits. These findings suggest that PNS activation to fear may increase risk for reactive functions of aggression. At the same time, however, PNS activation in response

to fear may also facilitate alert control, increasing risk for proactive functions of aggression (Thomson et al., 2021). In fact, for women, Thomson et al. (2021) found that PNS activation to fear was associated with proactive aggression. Additionally, a recent review by Belfry and Kolla (2021) documents significant associations between PNS activation to stress (i.e., peer exclusion and peer provocation) and proactive aggressive behavior.

Importantly, accumulating research has documented the importance of assessing the joint influence of SNS-R and PNS-R in studies of aggression (e.g., El-Sheikh et al., 2011). Understanding how these branches interact fits well with evidence that the PNS acts to moderate or inhibit other stress response systems, including the SNS (Murray-Close, 2013). The SNS and PNS exert opposing effects on physiological arousal such that their reciprocal activation has the same directional effect on physiological arousal (El-Sheikh et al., 2009). For example, simultaneous SNS activation and PNS inhibition (i.e., reciprocal SNS activation) leads to increased physiological arousal (e.g., higher heart rate), which theorists suggest reflects an adaptive response to stressors and facilitates active coping (El-Sheikh et al., 2009; Murray-Close et al., 2017). Several studies have shown that reciprocal SNS activation to stressors or challenges (e.g., cognitive tasks, peer provocation/rejection) is generally protective against aggression in the context of adversity (e.g., Chong et al., 2022; El-Sheikh et al., 2009). In contrast, simultaneous PNS activation and SNS inhibition (i.e., reciprocal PNS activation) leads to reduced physiological arousal (e.g., lower heart rate), which may be adaptive in situations perceived as nonthreatening (El-Sheikh et al., 2009). Research has also demonstrated that reciprocal PNS activation is generally protective against aggression in adverse contexts, as this physiological profile may signify self-soothing responses (El-Sheikh et al., 2009).

The effects of nonreciprocal activation patterns between the SNS and PNS are less understood but are theorized to be less adaptive than reciprocal patterns, potentially because nonreciprocal patterns may counteract one another (El-Sheikh et al., 2009). For instance, SNS and PNS inhibition (i.e., coinhibition) may represent an unclear response to stress, which has been linked to externalizing behaviors in response to lab challenges (e.g., El-Sheikh et al., 2009). Further, Thomson (2022) found that coinhibition to fear was associated with callous–unemotional traits and lack of empathy, which are closely tied to proactive functions of aggression. More recently, Thomson et al. (2021) found that, among men, coinhibition was associated with heightened proactive aggression, perhaps reflecting a physiological pattern of fearlessness. In addition, simultaneous SNS and PNS activation (i.e., coactivation) may reflect a state of physiological overarousal, wherein an overactive SNS overshadows a weaker PNS response, leading to dysregulated reactions to stress (El-Sheikh & Erath, 2011). Interestingly, in one recent study with two adolescent samples, coactivation to fear was associated with callous–unemotional traits, including lack of empathy (Thomson et al., 2020), which tend to be tied to proactive functions of aggression. SNS and PNS coactivation to fear may facilitate the capacity to remain calm and alert in fearful situations, a state conducive to proactive aggression (Thomson et al., 2020). As signifiers of uncoordinated regulation (El-Sheikh et al., 2009), nonreciprocal patterns of ANS-R may contribute to later aggression (Chong et al., 2022), but more research is needed to understand how nonreciprocal patterns of ANS-R relate to subtypes of aggression forms and functions.

Study Overview

The overarching aim of this study was to investigate longitudinal associations between CVE, ANS-R to fear (via RSA-R for PNS and PEP-R for SNS), and their interactions at age 7 in predicting aggression subtypes at age 12 in a sociodemographically diverse sample with a large percentage of Latine youth. From a developmental psychopathology perspective, early life experiences shape developmental trajectories because they influence physiological systems underlying emotion regulation and behavioral responses (Beauchaine et al., 2007; El-Sheikh & Erath, 2011). Childhood CVE may be especially relevant for understanding aggression during the transition to adolescence as external influences on child behavior become more pronounced during this period (Ostrov & Godleski, 2010). Although aggression is relatively stable from ages 2 to 8 years, rates begin to vary across early adolescence (9–13 years) and are influenced by environmental factors such as CVE (Piquero et al., 2012). Further, Latine youth report significantly higher rates of CVE as compared to their White peers (Crouch et al., 2000) and experience elevations in associated challenges, such as increased posttraumatic stress disorder symptoms, worse academic and physical health, and greater sleep impairments (Mora et al., 2022; Santacrose et al., 2021). Given these trends, it is important to investigate the impact of CVE on youth behavior in groups that may be at heightened risk for experiencing CVE.

The present study tested four primary hypotheses. First, we hypothesized that CVE would be associated with all subtypes of aggression (i.e., physical and relational forms, proactive and reactive functions). Second, we expected that SNS-R to fear would moderate these effects, such that CVE would be most strongly related to proactive functions of both physical and relational aggression among adolescents with SNS inhibition to fear (i.e., PEP lengthening) and to reactive functions of both physical and relational aggression among adolescents with SNS activation to fear (i.e., PEP shortening). Third, we hypothesized that PNS-R to fear would moderate these effects, such that CVE would be most strongly related to all aggression subtypes among adolescents with PNS activation to fear (i.e., RSA activation). Fourth, we expected that the interaction between PNS-R and SNS-R would moderate the association between CVE and aggression subtypes, such that CVE would be most strongly related to all aggression subtypes among adolescents demonstrating nonreciprocal patterns of SNS-R and PNS-R to fear (i.e., coinhibition or coactivation), and this effect would be strongest in the prediction of proactive functions of aggression (e.g., Thomson et al., 2021).

Importantly, we explicitly evaluated the potential influence of children's assigned sex (a proxy for gender) on all hypothesized pathways. Given the literature, it was necessary to assess these pathways separately for male and female adolescents (e.g., Card et al., 2008; Thomson et al., 2021). Although gender differences in relational forms of aggression are too small to be meaningful, boys engage in higher levels of physical forms of aggression than girls (Card et al., 2008), and existing research investigating gender differences in CVE effects on aggression is mixed (e.g., Guerra et al., 2003; Lambert et al., 2005). Further, prior research on ANS-R and aggression points to important gender differences. For example, a study by Thomson et al. (2021) indicated that men's physiological reactivity to fear aligned with fearlessness theory, with hyperarousal to fear being associated with reactive aggression and hypoarousal to

fear being associated with proactive aggression. In contrast, a different physiological basis for proactive aggression emerged among women, suggesting distinct patterns of cognitive or emotional processing (e.g., vigilance; Thomson et al., 2021). This study employed a multigroup approach to explore whether and how associations among CVE, ANS-R to fear, and aggression subtypes differed between boys and girls.

Method

Participants

Participants included 197 children (49.8% assigned female) who were recruited as part of a larger longitudinal study of 250 caregiver–child dyads. Youth were included in the current subsample if they had relevant data on CVE, ANS-R, and/or aggression available at the age 7 assessment ($M = 7.17$, $SD = 0.23$). Regarding race and ethnicity, youth were 46.0% Latine, 17.0% Black, 26.0% multiethnic/racial, and 11.0% White. Of the 197 children who completed assessments at age 7, 184 completed follow-up assessments at age 12 ($M = 12.25$, $SD = 0.35$).

At the age 7 visit, participating caregivers ($M = 34.77$, $SD = 7.29$) were biological mothers (93.8%), adoptive mothers (2.6%), or other kin serving a parental role (3.6%; e.g., grandmothers, aunts). Caregiver education levels were variable (e.g., 16.3% had not completed high school, 15.3% had completed a 4-year college degree or beyond). Most caregivers were married (70.9%) and employed (65.3%). Family socioeconomic status (SES) was assessed via the Hollingshead Four-Factor Index of Social Status, which accounts for parents' highest level of completed education and occupation. The current sample had an average family SES of 31.64 ($SD = 12.26$), which corresponds to a semiskilled worker (e.g., salesperson).

Procedure

At the start of the study, primary caregivers and their preschool-aged child were recruited to participate in "a study of children's learning and development" via flyers posted in community-based childcare centers in Southern California. Exclusionary criteria included children with diagnosed developmental disabilities or delays ($N = 3$), children who were unable to understand English ($N = 4$), and children outside the recruitment age range of 45–54 months at study entry (not tracked). At each data wave, caregiver–child dyads completed a 3-hr laboratory visit that included both observational and survey-based measures of regulation and behavior. Caregivers were compensated with $25/hr for their participation, and children received a small gift. Written informed consent was obtained from the legal guardian at the beginning of each visit, and informed assent was obtained from the participating child. All procedures were approved by the University's Human Research Review Board.

Measures

Child CVE (Age 7)

Children's lifetime CVE was assessed at age 7 using the mean score on the *Things I have Seen and Heard: Child Report* (J. E. Richters & Saltzman, 1990), which has been widely used in CVE research (J. Richters & Martinez, 1992; Trickett et al., 2003). Youth reported their CVE (e.g., "Seen a knife pulled on someone;" "Heard gun shots") across 15 items rated on a 5-point Likert scale (0 = *never*, 1 = *one time*, 2 = *two times*, 3 = *three times*, and 4 = *4 or more times*). Consistent with prior research (see Thompson et al., 2007), the present study omitted four items denoting feelings of safety (e.g., "I feel safe at school"), and one additional item due to mandated reporting concerns (i.e., "Seen someone in the home shot or stabbed"). This measure has shown good internal consistency with diverse samples in prior research (Thompson et al., 2007), as well as in the present sample ($\alpha = .85$).

Child Physical Aggression (Ages 7 and 12)

Caregivers reported on children's physical aggression perpetration using a questionnaire developed by Dodge and Coie (1987). Caregivers rated their child's engagement in proactive physical aggression across three items (e.g., "Your child uses or threatens physical force in order to dominate other kids") and reactive physical aggression across three items (e.g., "When your child has been teased or threatened, s/he gets angry easily and strikes back") on a scale from 0 (*never true*) to 4 (*always true*). Although originally developed for teachers, this measure has been successfully used for caregiver reports and with similarly diverse samples (e.g., Miller-Johnson et al., 2002). The physical aggression subscale was reliable at both assessment points for both proactive physical aggression ($\alpha_{Age\ 7} = .78$, $\alpha_{Age\ 12} = .75$) and reactive physical aggression ($\alpha_{Age\ 7} = .72$, $\alpha_{Age\ 12} = .79$).

Child Relational Aggression (Ages 7 and 12)

Caregivers reported on children's relational aggression perpetration using the Preschool Proactive and Reactive Aggression measure (Ostrov & Crick, 2007). Although this reliable measure is often used for teacher reports (Ostrov & Crick, 2007), it has been successfully adapted for use with caregivers (Baker, 2022). Some items were reworded slightly to improve clarity (e.g., moving "to get what s/he wants" from the beginning to the end of the item). This measure was selected because it was developmentally appropriate for children at the first assessment of the larger longitudinal study (e.g., Hart & Ostrov, 2013). Further, although developed for early childhood samples, the Preschool Proactive and Reactive Aggression contains content and wording that mirror measures used in older samples (e.g., third–sixth graders, Crick, 1996), except that the Preschool Proactive and Reactive Aggression does not include an item assessing gossip and rumor spreading. Caregivers rated their child's engagement in proactive relational aggression across three items (e.g., "Your child will often ignore or stop talking to others to get what s/he wants") and reactive relational aggression across three items ("When your child is angry at others, your child will often tell them that s/he won't be their friend anymore") on a scale from 0 (*never true*) to 4 (*always true*). This scale modified the original scale from 1 (*never or almost never true*) to 5 (*always or almost always true*) used by Ostrov and Crick (2007) to match the physical aggression item response scale. The relational aggression subscale was reliable at both assessment points for both proactive relational aggression ($\alpha_{Age\ 7} = .78$, $\alpha_{Age\ 12} = .72$) and reactive relational aggression ($\alpha_{Age\ 7} = .80$, $\alpha_{Age\ 12} = .63$).

Children's ANS-R to Fear (Age 7)

Caregivers were informed that they would be viewing a series of emotion videos with their child that "show kids in various situations, such as in a fun food fight, running away from a train, and receiving bad news about a sick parent." They were told the clips were "designed to elicit various emotions, but no one gets hurt in the clips." Dyads viewed a series of film clips beginning with a peaceful nature film, followed by emotion inducing video clips, including fear, which was the focus of the present study. Children watched a scary/fear scene from *Stand by Me* depicting a train barreling down on two children. Clips were piloted and confirmed to elicit the intended emotion by Bennett and Lewis (2011) and were administered in a standardized order—sad, happy, fear—with 1-min neutral nature film clips separating each. This standardized order was adopted to ensure that negative emotions were separated by a positive emotion to avoid emotional fatigue (see Rottemberg et al., 2007).

ANS regulation was assessed during each film clip using four spot electrodes, placed on the neck and torso to collect impedance and respiratory measures, and three spot electrodes placed on the right clavicle, left lower rib, and right abdomen for electrocardiogram measures. ANS data were collected using Mindware MW1000A ambulatory cardiography (https://www.mindwaretech.com) via Kendall Medi-Trace #133 spot electrodes. PEP data were extracted and scored using the IMP 3.0.3 analysis program, and the d*Z*/d*t* waveforms were used to obtain impedance-derived PEP measures quantified as the time interval in milliseconds from the onset of the electrocardiogram Q-wave to the *B* point of the d*Z*/d*t* wave (Berntson et al., 2004). RSA data were filtered, extracted, and scored using Mindware's heart rate variability 3.0.10 analysis program. This technique utilizes the Mindware software algorithms to calculate the variance in R-R wave intervals. RSA scores were calculated using the interbeat intervals on the electrocardiogram reading, respiratory rates derived from the impedance (i.e., d*Z*/d*t*) signal, and a specified RSA bandwidth range for 7-year-olds of 0.15–0.80 Hz (Bar-Haim et al., 2000). The appearance of misspecified epochs varied across the broader task, suggesting that sudden changes in respiratory rate likely reflected random alterations in body posture or vocalization across the task (Grossman & Taylor, 2007).

Data cleaning procedures for PEP and RSA included visual inspection of each epoch for errors or abnormalities in the *B* points and R-peaks, respectively. PEP was cleaned using the finalized RSA epochs, which were also visually inspected by trained research assistants. If *B* points were not accurate, the assistant flagged the case for inspection by the doctoral scorer who then manually edited as necessary. For RSA, trained research assistants manually edited R-peaks, which were then scored and finalized by an advanced doctoral student who had completed extensive training in the collection and coding of pediatric cardiography, as well as reliability verification for both PEP and RSA scoring procedures with the current data set under the supervision of the ANS consultant for this study. Further procedures included screening each epoch for outliers (i.e., values greater than 3 standard deviations above or below the mean; Alkon et al., 2011) and deleting a child's data if more than 25% of their epochs were missing due to computer malfunction, electrode conduction problems, or outliers. Of the 197 participants who completed aggression measures at age 7, 137 (70%) had usable PEP data, and 169 (86%) had useable RSA data. Missing ANS data were the result of physiology task noncompletion due to a partial visit ($N = 8$), video errors ($N = 13$), computer/electrode/connection issues ($N = 33$ for PEP, $N = 7$ for RSA), and segment deletion due to artifacts ($N = 9$ for PEP, $N = 4$ for RSA).

Following a 5-min calibration period, baseline ANS activity was indicated by the average of six 30-s epochs across a 3-min film baseline during which children viewed a neutral nature scene. This initial baseline was used to calculate reactivity scores given the greater reliability afforded by its longer duration of 3 min (yielding six, 30-s epochs) as compared to the 1-min intervening neutral nature clips (yielding just two, 30-s epochs). The intervening nature clips also encompassed recovery processes and thus were not true baselines. ANS-R to fear was indicated by standardized residual values. Residual values reflect the extent of reactivity based on one's baseline score relative to the overall sample (Burt & Obradović, 2013; see Supplemental Figure S1 for an illustration of how these scores are calculated). The *expected* level of change is based on the sample regression line of ANS response to fear (i.e., average score across the four 30-s epochs during the 2-min fear-eliciting film) regressed on baseline ANS values. The residual value reflects the difference between the change in ANS relative to expected change and is a common approach in the field (e.g., Rudd & Yates, 2018). We adopted this approach because standardized residuals maximize reliability in cases where the ratio of baseline variance to challenge variance is greater than the correlation between baseline and challenge values (Burt & Obradović, 2013), which was the case in the present study. In addition, this approach for assessing ANS-R has been employed in prior work with the present sample (Murray-Close et al., 2024) and in foundational research focused on the influence of ANS coordination on externalizing behavior (El-Sheikh et al., 2009). For PEP, positive standardized residual scores indicated that, relative to the rest of the sample, the individual exhibited greater PEP lengthening than expected (i.e., SNS inhibition), whereas negative standardized residual scores indicated that the individual exhibited greater PEP shortening (i.e., SNS activation) than expected. For RSA, positive standardized residual scores indicated that, relative to the rest of the sample, the individual exhibited greater RSA activation (i.e., increased PNS activity) than expected, whereas negative standardized residual scores indicated that the individual exhibited greater RSA inhibition (i.e., decreased PNS activity) than expected (see Supplemental Figure S1 for a sample residualized change score).

Data Analytic Plan

Preliminary descriptive statistics were run via SPSS Version 28. Correlational analyses between demographic variables (i.e., gender [0 = *boys*, 1 = *girls*],[1] ethnicity/race [0 = *non-Latine*, 1 = *Latine*], and SES), and the variables of interest (i.e., CVE, the four aggression subtypes, SNS-R, and PNS-R) were conducted. Demographic variables were included as covariates if they were significantly related to key study variables. Primary models were conducted using Mplus Version 8.7. Analyses were run with maximum likelihood estimation

[1] Gender was based on the child's assigned sex as female or male. However, we use "gender" as the focal construct to emphasize the social and psychological processes associated with gender differences in aggression, following the American Psychological Association's guidelines regarding bias-free language (APA; https://apastyle.apa.org/style-grammar-guidelines/bias-free-language/gender).

with robust standard errors to account for missing data. Outliers for SNS-R and PNS-R (i.e., >3 standard deviations from the mean) were winsorized to within 3 standard deviations of the mean to reduce their influence (Kline, 2023). The standardized root-mean-square residual (SRMR), the comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA) were used to evaluate model fit (Hu & Bentler, 1999). Good fit was defined as CFI > 0.95, SRMR < .08, and RMSEA < .06 (Hu & Bentler, 1999), with lower thresholds for acceptable fit (e.g., CFI > 0.90, RMSEA < .08; Little et al., 2014).

We performed a series of multigroup analyses, with gender serving as the grouping variable, to explore potential moderation by child gender. In the model, CVE, SNS-R, PNS-R, and their interactions served as predictors of all four correlated aggression outcomes (i.e., proactive physical, reactive physical, proactive relational, reactive relational) at age 12. Subtypes of aggression at age 7 were included as covariates. Continuous predictors and covariates (i.e., forms and functions of aggression, SNS-R and PNS-R) were mean-centered prior to analyses. In the initial model, pathways linking covariates to age 12 aggression subtypes were constrained across girls and boys to increase model parsimony; all other pathways were freely estimated across groups. Modification indices were utilized to identify whether paths between covariates and outcome variables differed by child gender ($p < .05$); these paths were then sequentially released based on the magnitude of the modification indices. Subsequently, a model with the identified covariate pathways freely estimated across girls and boys was used to investigate gender differences in associations between key predictor variables and outcomes. Specifically, an omnibus Wald chi-square test was conducted to investigate associations between CVE, SNS-R, PNS-R, and their interactions in the prediction of each aggression outcome, respectively. We focused on an omnibus test, rather than testing each parameter separately, as the interactions were contingent upon the main effects. Whenever Wald chi-square tests indicated significant gender moderation ($p < .05$), the entire block (main effects and interactions) was allowed to vary across groups; conversely, when child gender did not moderate associations, the block was constrained across groups. When significant interactions emerged, simple slopes were calculated at +1/−1 standard deviation from the mean for the moderator(s).

Transparency and Openness

This study was not preregistered. Data, study materials, and analysis code are available upon request from Casey Buck.

Results

Descriptive Statistics, Correlations, and Missing Data

Descriptive statistics regarding key study variables, including the number of participants included for each variable and bivariate correlations, are presented in Table 1. Correlations indicated that girls scored higher in reactive relational aggression at age 7 than boys. Latine participants scored lower than non-Latine participants in reactive physical aggression at ages 7 and 12 and were also rated as lower in proactive relational aggression at age 12. SES was associated with greater SNS activation (i.e., greater PEP shortening) to fear. CVE was positively related to proactive physical aggression at age 7, reactive physical aggression at both time points, and both proactive and reactive relational aggression at age 12. PNS-R was negatively associated with SNS-R (i.e., PEP lengthening) and negatively associated with proactive physical aggression at age 7. Correlations between aggression subtypes at ages 7 and 12 ranged from small/moderate to large (rs = .21–.66, all ps < .001), with the exception that proactive physical aggression at age 7 was not significantly associated with proactive and reactive relational aggression at age 12. Given evidence of longitudinal stability across subtypes of aggression, all age 12 aggression subtypes were regressed onto age 7 aggression subtypes in primary analyses.

Regarding missing data, Little's Missing Completely at Random test, which included all four aggression variables at both time

Table 1
Descriptive Statistics and Correlations of Study Variables

Variable	*N*	*M* (*SD*)	Min–max	Skew	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Age (months)	197	7.17 (0.23)	6.57–8.06	1.56	—														
2. Gender	197	0.50 (0.50)		0.01	**.20**	—													
3. Latine ethnicity	197	0.47 (0.50)		0.13	.03	−.08	—												
4. SES	182	31.64 (12.26)	13–66	0.65	.11	.02	−.11	—											
5. CVE	192	0.76 (0.74)	0.00–3.71	1.50	.08	−.05	.00	−.13	—										
6. PEP-R	134	0.02 (0.80)	−3.00 to 3.00	−0.36	.07	−.03	−.01	**−.18**	.00	—									
7. RSA-R	165	0.00 (0.98)	−2.76 to 3.00	0.19	−.15	−.11	−.14	−.11	.03	**.22**	—								
8. Pro Phys age 7	197	0.20 (0.41)	0.00–2.33	2.47	−.01	.08	−.00	.11	**.17**	−.01	**−.17**	—							
9. Re Phys age 7	195	1.31 (0.92)	−0.00 to 4.00	0.56	.02	−.01	**−.15**	.01	**.18**	−.11	−.01	**.40**	—						
10. Pro Rel age 7	197	0.71 (0.77)	0.00–3.33	0.89	.10	.10	−.12	.10	.08	−.10	−.07	**.41**	**.39**	—					
11. Re Rel age 7	196	1.11 (0.82)	0.00–3.33	0.41	.09	**.15**	−.09	.05	.11	.00	−.04	**.41**	**.47**	**.70**	—				
12. Pro Phys age 12	183	0.18 (0.42)	0.00–2.67	3.01	−.11	−.14	−.05	−.04	.12	.08	.03	**.26**	**.34**	**.25**	**.21**	—			
13. Re Phys age 12	184	1.16 (0.96)	0.00–4.00	0.66	−.08	−.07	**−.20**	−.06	**.19**	−.01	−.06	**.23**	**.51**	**.30**	**.30**	**.50**	—		
14. Pro Rel age 12	181	0.38 (0.62)	0.00–4.00	2.45	**.15**	.06	**−.15**	−.04	**.20**	−.02	−.01	.10	**.28**	**.38**	**.25**	**.48**	**.39**	—	
15. Re Rel age 12	183	0.82 (0.74)	0.00–3.00	0.76	−.01	.05	−.12	−.09	**.17**	−.10	.09	.13	**.39**	**.34**	**.33**	**.43**	**.49**	**.66**	—

Note. Values represent winsorized data. Significant correlations (i.e., $p < .05$) are in bold. Gender is coded 0 = *boys*, 1 = *girls*; child ethnicity is coded 0 = *non-Latine*, 1 = *Latine*; SES = socioeconomic status; CVE = community violence exposure; PEP-R = pre-ejection period reactivity to fear at age 7; shortened PEP (i.e., low PEP-R) reflects SNS activation, whereas elongated PEP (i.e., high PEP-R) reflects SNS inhibition. RSA-R = respiratory sinus arrhythmia to fear at age 7; Phys = physical aggression; Rel = relational aggression; Pro = proactive aggression; Re = reactive aggression; SNS = sympathetic nervous system.

points, CVE, SNS-R, PNS-R, ethnicity, SES, and gender, was not significant, suggesting data may be Missing Completely at Random, $\chi^2(199) = 198.54$, $p = .496$. However, as the present study design did not include planned missing data, data were most likely Missing at Random (Little et al., 2014). Consistent with best practices, we used maximum likelihood estimation to handle missing data (Little et al., 2014). In primary analyses, robust standard errors (maximum likelihood estimation) were used to accommodate deviations from normality such as variable skew (see Table 1).

Primary Analyses

This investigation employed a multigroup model using gender based on assigned sex (*girls* = 1; *boys* = 0) as the grouping variable to test associations between CVE, SNS-R, PNS-R, and their interactions in predicting the four correlated aggression outcomes (i.e., proactive physical, reactive physical, proactive relational, reactive relational) at age 12. Ethnicity, SES, and subtypes of aggression at age 7 were included as covariates based on preliminary correlational analyses. Initially, with pathways between covariates and aggression outcomes constrained across girls and boys, the model fit ranged from poor to good (CFI = 0.95, RMSEA = .13, SRMR = .041). Modification indices indicated that releasing several pathways from covariates to outcomes across child gender improved model fit ($p < .05$); pathways that differed between girls and boys are detailed in Table 2. Once indicated pathways were freed across groups, model fit was excellent (CFI = 1.00, RMSEA = .000, SRMR = .018). Next, omnibus Wald tests of gender moderation were performed to determine if associations between CVE, SNS-R, PNS-R, and their interactions were significant in predicting the four aggression subtypes. Child gender moderated predictions to proactive relational aggression, Wald $\chi^2(7) = 15.42$, $p = .031$, and reactive physical aggression, Wald $\chi^2(7) = 14.56$, $p = .042$, but not to reactive relational aggression, Wald $\chi^2(7) = 3.64$, $p = .821$, or proactive physical aggression, Wald $\chi^2(7) = 7.35$, $p = .394$. Thus, for the final model, pathways from CVE, SNS-R, PNS-R, and their interactions were unconstrained across girls and boys in the prediction to proactive relational aggression and reactive physical aggression, but were constrained for predictions to proactive physical aggression and reactive relational aggression. This final model provided excellent fit to the data (CFI = 1.00, RMSEA = .000, SRMR = .027; see Table 2).

Girls

Proactive Physical Aggression. No significant associations emerged between key predictors (i.e., CVE, SNS-R, PNS-R, and their interactions) in the prediction of proactive physical aggression.

Reactive Physical Aggression. The interactions between CVE and PNS-R, as well as between SNS-R and PNS-R, significantly predicted reactive physical aggression at age 12 (see Table 2). However, these two-way interactions were qualified by a significant three-way interaction among CVE, SNS-R, and PNS-R. Specifically, for girls who demonstrated *reciprocal SNS activation* to fear (i.e., SNS activation and PNS inhibition), CVE was positively associated with reactive physical aggression ($b = .68$, $p = .030$). Furthermore, for girls who demonstrated *coinhibition* to fear (i.e., SNS inhibition and PNS inhibition), CVE was negatively associated with reactive physical aggression ($b = -1.54$, $p = .013$; see Figure 1).

Proactive Relational Aggression. CVE and SNS inhibition (i.e., PEP elongation) were longitudinally associated with greater proactive relational aggression. However, these effects were qualified by a significant interaction between CVE and SNS-R such that CVE was positively associated with proactive relational aggression ($b = .73$, $p < .001$) among girls exhibiting SNS inhibition, but not among girls exhibiting SNS activation, to fear (see Figure 2).

Reactive Relational Aggression. No significant associations emerged between key predictors (i.e., CVE, SNS-R, PNS-R, and their interactions) in the prediction of reactive relational aggression.

Boys

Proactive Physical Aggression. No significant associations emerged between key predictors (i.e., CVE, SNS-R, PNS-R, and their interactions) in the prediction of proactive physical aggression.

Reactive Physical Aggression. PNS inhibition was associated with heightened reactive physical aggression at age 12. However, this effect was qualified by a significant interaction between CVE and PNS-R such that CVE was *positively* associated with reactive physical aggression among boys who demonstrated PNS inhibition to fear ($b = .52$, $p = .001$) and *negatively* associated with reactive physical aggression among boys who demonstrated PNS activation to fear ($b = -.46$, $p = .027$; see Figure 3).

Proactive Relational Aggression. SNS inhibition (i.e., PEP elongation) was positively associated with greater proactive relational aggression. Furthermore, there was a significant interaction between CVE and PNS-R, as well as between SNS-R and PNS-R, in the prediction of proactive relational aggression. These two-way interactions were further qualified by a three-way interaction among CVE, SNS-R, and PNS-R. Follow-up simple slope analyses revealed that among boys who displayed *coactivation* to fear (i.e., SNS activation and PNS activation), CVE was associated with heightened proactive relational aggression ($b = .57$, $p = .002$; see Figure 3). In contrast, among boys who demonstrated *reciprocal SNS activation* to fear (i.e., SNS activation and PNS inhibition), CVE was related to decreased proactive relational aggression ($b = -.51$, $p = .004$; see Figure 4).

Reactive Relational Aggression. No significant associations emerged between key predictors (i.e., CVE, SNS-R, PNS-R, and their interactions) in the prediction of reactive relational aggression.

Discussion

This study examined longitudinal associations between children's lifetime CVE assessed at age 7 and four aggression subtypes assessed at age 12 (i.e., proactive physical, reactive physical, proactive relational, and reactive relational). In addition, moderation analyses tested directional hypotheses regarding ANS-R influences on these pathways, which were explored separately among girls and boys. Several findings supported study hypotheses that CVE would be associated with increased risk for subtypes of aggression among youth who demonstrated distinct patterns of ANS-R to fear, supporting previous suggestions that the dynamic regulation of the SNS and PNS in response to fear is an important influence on youths' social functioning, particularly in contexts of

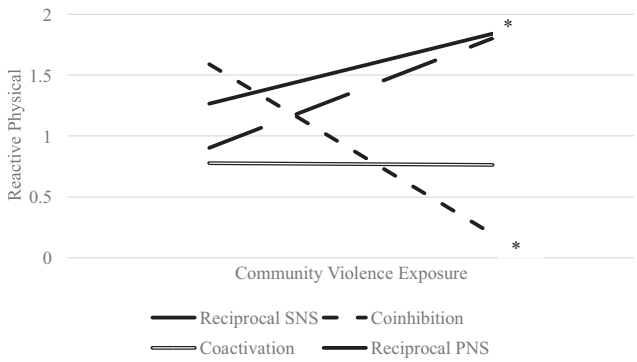
Table 2
CVE, PEP-R, RSA-R, and Their Interaction Predicting Subtypes of Aggression by Gender

Model	*b*	*B*	*p*
Proactive physical			
CVE	.02/.02	.03/.04	.649
PEP-R	.08/.08	.11/.23	.194
RSA-R	−.01/−.01	−.02/−.03	.725
CVE × PEP-R	−.04/−.04	−.04/−.08	.533
CVE × RSA-R	−.01/−.01	−.01/−.02	.822
PEP-R × RSA-R	−.03/−.03	−.06/−.11	.562
CVE × PEP-R × RSA-R	−.13/−.13	−.17/−.21	.197
Covariates			
Age 7 ProPhys	**.31/−.02**	**.22/−.03**	**.027/.818**
Age 7 RePhys	.14/.14	.26/.44	.000
Age 7 ProRel	.11/.11	.17/.29	.020
Age 7 ReRel	**.04/−.10**	**.06/−.30**	**.599/.043**
Latine ethnicity	.03/.03	.03/.04	.638
SES	−.01/.00	−.16/.06	.097
Reactive physical			
CVE	.08/.06	.07/.04	.362/.693
PEP-R	.14/.01	.11/.01	.274/.939
RSA-R	**−.22/−.06**	**−.23/−.05**	**.014/.638**
CVE × PEP-R	−.15/−.21	−.08/−.13	.407/.273
CVE × RSA-R	**−.49/.49**	**−.30/.30**	**.002/.026**
PEP-R × RSA-R	**.08/.45**	**.09/.55**	**.634/.005**
CVE × PEP-R × RSA-R	**.11/.95**	**.07/.47**	**.689/.017**
Covariates			
Age 7 ProPhys	.09/.09	.03/.04	.596
Age 7 RePhys	.50/.50	.47/.48	.000
Age 7 ProRel	**.39/−.15**	**.30/−.12**	**.001/.294**
Age 7 ReRel	.02/.02	.01/.02	.864
Latine ethnicity	−.40/−.40	−.22/−.20	.001
SES	−.01/−.01	−.13/−.12	.059
Proactive relational			
CVE	**.04/.43**	**.07/.41**	**.451/.000**
PEP-R	**.16/.18**	**.24/.22**	**.046/.042**
RSA-R	.03/−.05	.07/−.06	.492/.507
CVE × PEP-R	**.10/.35**	**.10/.29**	**.384/.004**
CVE × RSA-R	**.27/.11**	**.32/.09**	**.002/.431**
PEP-R × RSA-R	**−.29/−.11**	**−.61/−.19**	**.002/.233**
CVE × PEP-R × RSA-R	**−.43/−.07**	**−.54/−.05**	**.008/.757**
Covariates			
Age 7 ProPhys	**.10/−.44**	**.07−.29**	**.491/.003**
Age 7 RePhys	**.06/.29**	**.11/.38**	**.246/.000**
Age 7 ProRel	.32/.32	.48/.35	.000
Age 7 ReRel	−.09/−.09	−.14/−.10	.208
Latine ethnicity	−.14/−.14	−.15/−.10	.063
SES	−.00/−.00	−.06/−.04	.482
Reactive relational			
CVE	.06/.06	.07/.06	.367
PEP-R	−.01/−.01	−.01/−.01	.881
RSA-R	.05/.05	.07/.06	.382
CVE × PEP-R	−.13/−.13	−.09/−.10	.215
CVE × RSA-R	.05/.05	.04/.04	.579
PEP-R × RSA-R	−.04/−.04	−.05/−.06	.630
CVE × PEP-R × RSA-R	.10/.10	.08/.06	.551
Covariates			
Age 7 ProPhys	−.09/−.09	−.05/−.06	. 517
Age 7 RePhys	.21/.21	.27/.27	.001
Age 7 ProRel	.13/.13	.13/.13	.157
Age 7 ReRel	.09/.09	.10/.11	.304
Latine ethnicity	−1.00/−1.00	−.07/−.06	.339
SES	**.00/−.02**	**.02/−.23**	**.836/.005**

Note. Values represent winsorized data. Gender is coded 0 = *boys*, 1 = *girls*; estimates for boys are on the left of the "/" and those for girls are on the right. Effects in bold were significantly different across boys and girls ($p < .05$). When pathways are constrained across groups, unstandardized (*b* values) are constrained to be equal; however, standardized values (i.e., β) may vary across groups due to group differences in variances. Latine ethnicity is coded 0 = *non-Latine*, 1 = *Latine*; SES = socioeconomic status; CVE = community violence exposure; PEP-R = pre-ejection period reactivity to fear at age 7; shortened PEP (i.e., low PEP-R) reflects SNS activation, whereas elongated PEP (i.e., high PEP-R) reflects SNS inhibition. RSA-R = respiratory sinus arrhythmia reactivity to fear at age 7. ProPhys = proactive physical aggression; RePhys = reactive physical aggression; ProRel = proactive relational aggression; ReRel = reactive relational aggression.

Figure 1

Interaction Plot of Community Violence Exposure × SNS-R × PNS-R Predicting Reactive Physical Aggression for Girls



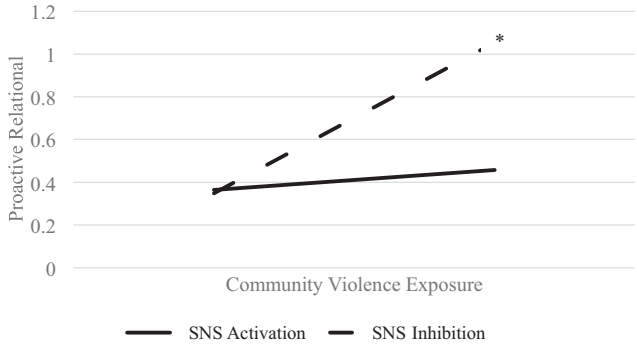
Note. Lines with an * represent significant slopes. Reciprocal SNS = RSA inhibition and PEP shortening indicative of SNS activation. Coactivation = RSA activation and pre-ejection period (PEP) shortening, indicative of SNS activation. Coinhibition = RSA inhibition and PEP lengthening, indicative of SNS inhibition. Reciprocal PNS = RSA activation to fear and PEP lengthening indicative of SNS inhibition. Simple slopes were probed at 1 standard deviation above and below the mean on each moderator (RSA, PEP). SNS = sympathetic nervous system; PNS = parasympathetic nervous system; RSA = respiratory sinus arrhythmia.

adversity (Moore et al., 2018). Further, the obtained patterns of effects differed significantly across girls and boys.

With respect to interactions between CVE and SNS-R, we hypothesized that CVE would be most strongly related to proactive aggression among adolescents exhibiting SNS inhibition to fear and to reactive functions of aggression among adolescents exhibiting SNS activation to fear. Consistent with these hypotheses, SNS inhibition (i.e., PEP lengthening) was related to greater proactive relational aggression among both girls and boys. In addition, CVE was related to higher proactive relational aggression among girls, but not boys, who exhibited SNS inhibition in response to fear. Consistent with fearlessness theory (Frick & Morris, 2004; Murray-Close et al., 2017), these results support prior research indicating blunted SNS-R is a salient risk factor for both proactive and relational subtypes of aggression (e.g., Blankenstein et al., 2022; Moore et al., 2018; Murray-Close et al., 2017; Raine, 1997). Fearlessness may increase risk for proactive functions of aggression in particular because children who do not experience typical levels of fear may be less adept at recognizing fear in others (Thomson, 2022). The obtained findings are also consistent with prior work indicating interactions between adversity (e.g., sexual abuse, negative parenting) and SNS inhibition in the prediction of proactive relational aggression (e.g., Wagner & Abaied, 2016). At the same time, this study extends extant findings by highlighting the moderating role of SNS-R in the association between an additional type of adversity—CVE—and proactive relational aggression among girls. In contrast, neither CVE nor SNS-R were associated with reactive relational aggression. It is possible that other contextual experiences (e.g., being the direct target of violence) or patterns of ANS reactivity (e.g., reactivity to threat) may be more salient predictors of reactive relational aggression.

Figure 2

Interaction Plot of CVE × SNS-R Predicting Proactive Relational Aggression for Girls



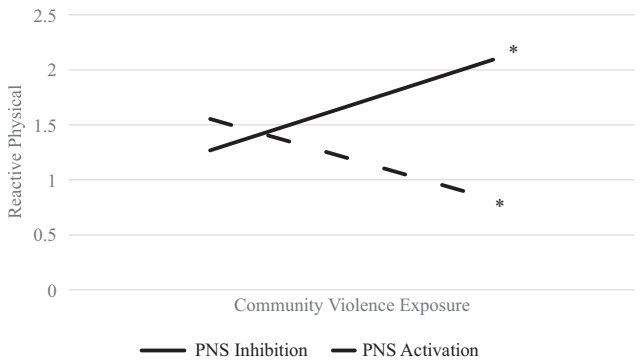
Note. Girls' SNS-R moderates the effect of cumulative CVE at age 7 on proactive relational aggression at age 12. Slopes with an * are significant. SNS inhibition reflects PEP lengthening, whereas SNS activation reflects PEP shortening. Simple slopes were probed at 1 standard deviation above and below the sample mean of PEP reactivity. CVE = community violence exposure; SNS = sympathetic nervous system; PEP = pre-ejection period.

We also expected that PNS activation to fear would serve as a significant risk factor for aggression, such that CVE would be most strongly related to proactive and reactive aggression among adolescents exhibiting PNS activation to fear (indexed by RSA-R). In contrast to hypotheses, PNS *inhibition* to fear was associated with reactive physical aggression among boys. Additionally, CVE was positively associated with reactive physical and proactive relational aggression subtypes among boys who demonstrated PNS inhibition to fear. In contrast, CVE was associated with decreases in reactive physical and proactive relational aggression among boys who demonstrated PNS activation to fear. Although these findings were unexpected, it is notable that Beauchaine and Thayer (2015) asserted that PNS inhibition reflects emotional instability and increases the risk of externalizing problems. In fact, although PNS inhibition in response to threat appears protective against aggression among youth exposed to adversity in some research (Porges, 2007), other work indicates that excessive PNS inhibition to threat reflects a dysregulated response and is associated with greater aggression (e.g., Beauchaine et al., 2007). Gatzke-Kopp et al. (2013) found that children exhibiting PNS inhibition to fear showed lower levels of emotion regulation as compared to children exhibiting PNS activation to fear. Similarly, boys who experience CVE and exhibit PNS inhibition to fear may engage in reactive physical aggression because they have poorly developed emotion regulation skills.

In addition to emotion regulation, PNS inhibition may influence attentional processes in a way that promotes more alert or vigilant focus. By enhancing focused attention and emotional and behavioral responses during social interactions, PNS inhibition to fear (i.e., RSA withdrawal) may support the deliberate use of relational aggression (i.e., proactive functions), providing the necessary resources to effectively gain social power or dominance (Wagner & Abaied, 2016). PNS inhibition facilitates elevated heart rate, which supports physiological mobilization and prepares the body to respond to salient environmental demands (Porges, 2007). This physiological state not only primes the individual for action but also enhances focused attention useful for goal-directed behavior (Wagner & Abaied, 2016). This heightened attentional focus may

Figure 3

Interaction Plot of CVE × PNS-R Predicting Reactive Physical Aggression for Boys

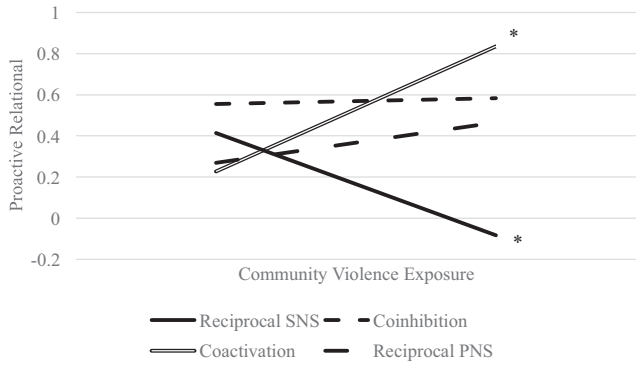


Note. Boys' PNS-R moderates the effect of cumulative community violence exposure at age 7 on reactive physical aggression at age 12. Slopes with an * are significant. The solid line represents PNS inhibition (i.e., RSA inhibition), which represents 1 standard deviation below the sample mean. The solid line indicates PNS activation (i.e., RSA activation), which represents 1 standard deviation above the sample mean. CVE = community violence exposure; PNS = parasympathetic nervous system; RSA = respiratory sinus arrhythmia.

allow for the intentional monitoring of peer dynamics, enabling adolescent boys to utilize relationally aggressive behaviors in a strategic and controlled manner (i.e., proactively). However, this unexpected finding awaits replication in future investigations of the role of PNS inhibition in the development of proactive relational aggression in boys.

Figure 4

Interaction Plot of CVE × SNS-R × PNS-R Predicting Proactive Relational Aggression for Boys



Note. Lines with an * represent significant slopes. Reciprocal SNS = RSA inhibition and PEP shortening indicative of SNS activation. Coactivation = RSA activation and PEP shortening, indicative of SNS activation. Coinhibition = RSA inhibition and PEP lengthening, indicative of SNS inhibition. Reciprocal PNS = RSA activation and PEP lengthening indicative of SNS inhibition. Simple slopes were probed at 1 standard deviation above and below the mean on each moderator (RSA, PEP). CVE = community violence exposure; SNS = sympathetic nervous system; PNS = parasympathetic nervous system; RSA = respiratory sinus arrhythmia; PEP = preejection period.

Finally, we hypothesized that CVE would be most strongly related to aggression among individuals demonstrating nonreciprocal ANS-R patterns to fear (i.e., coinhibition or coactivation) and that these effects would be strongest in the prediction of proactive functions of aggression given findings from prior research (e.g., Thomson et al., 2021). Consistent with this hypothesis, CVE predicted greater proactive relational aggression among boys exhibiting coactivation to fear. This finding is aligned with prior research suggesting that coactivation to fear is associated with callous–unemotional traits and may facilitate the capacity to remain calm and alert during fearful situations (Thomson et al., 2020), such as CVE. This physiological profile may reflect a state of calm alertness, wherein heightened vigilance (SNS activation) is balanced by emotional regulation and attentional control (PNS activation), potentially enabling deliberate and socially strategic proactive functions of aggression (Thomson et al., 2020). For boys in high-adversity environments, this pattern could facilitate goal-directed aggression that is relational in nature and less overtly dysregulated (i.e., relational forms of aggression that subserve proactive vs. reactive functions). These findings align with research emphasizing the instrumental use of aggression when regulatory systems support calm, controlled engagement, even in threatening or socially complex situations (Thomson et al., 2020). In addition, consistent with our hypotheses, reciprocal SNS activation to fear was protective against the effects of CVE on proactive relational aggression among boys. This finding is consistent with the idea that a coordinated pattern of ANS-R that functions to heighten physiological arousal is adaptive in adverse contexts as it enables youth to actively manage threats or stressors (El-Sheikh et al., 2009; Murray-Close et al., 2017).

For girls, a different and unexpected pattern of effects emerged, indicating that CVE was related to greater reactive physical aggression among girls who demonstrated reciprocal SNS activation and to lower reactive physical aggression among girls exhibiting coinhibition. These findings are inconsistent with prior research indicating that reciprocal SNS activation buffers the effects of adversity on aggression (e.g., Chong et al., 2022; El-Sheikh et al., 2009), as well as with prior research suggesting that coinhibition to fear may reflect fearlessness that contributes to aggressive behavior, at least in men (Thomson et al., 2021). Importantly, most extant research on the relation between reciprocal SNS activation and aggression has examined responses to cognitive or frustration tasks (e.g., Chong et al., 2022), rather than responses to fear. Further, there is an emerging literature on distinct relations of ANS-R to fear and aggression among females and males (e.g., Thomson et al., 2021). For instance, it is notable that reciprocal SNS activation functions to increase net arousal, which may be tied to overarousal theories of reactive aggression (Murray-Close et al., 2017), as well as to emotions such as rage and panic (Beauchaine et al., 2007). In effect, when encountering threats, such as CVE, a pattern of overarousal may be indicative of dysregulated emotions, such as panic, that may increase risk for reactive aggression. Further, some prior research suggests that physiological patterns of coinhibition reflect passive vigilance and low stress (El-Sheikh & Erath, 2011), which is an ANS-R pattern that can help youth effectively interact with adverse environments with reduced sensitivity (Thomson et al., 2019). In this way, coinhibition to fear may buffer against extreme feelings of panic or anxiety among girls, thus facilitating emotional control in adverse environments and attenuating dysregulated responses like reactive physical aggression.

Taken together, these findings underscore the importance of considering ANS patterns in context rather than interpreting them as universally adaptive or maladaptive. The functional meaning of these physiological profiles appears to vary as a function of various factors, including child gender, aggression subtype, and the emotional valence of the task. Indeed, although coactivation strengthened the positive association between CVE and proactive relational aggression among boys, some findings among girls suggested protective effects of nonreciprocal ANS-R patterns. Although reciprocal SNS activation is often conceptualized as a coordinated and adaptive physiological pattern, especially in cognitive or frustration tasks (e.g., Chong et al., 2022), in the context of fear, reciprocal SNS activation may reflect emotional overarousal that increases risk for reactive physical aggression among girls with CVE. As another example, although coinhibition has been associated with blunted affect and low engagement in some studies (El-Sheikh & Erath, 2011), in the context of adversity such as CVE, coinhibition might reflect a downregulated or dampened physiological state that protects against emotional flooding or panic-like responses and promotes emotional control and behavioral inhibition (Thomson et al., 2019), at least among girls. This interpretation is consistent with the finding that coinhibition was linked to lower reactive aggression among girls. In sum, what is adaptive in one context (e.g., cognitive challenge) may be maladaptive in another (e.g., fear), and distinct patterns may emerge as a function of gender and history of adversity exposure.

Strengths, Limitations, and Future Directions

This multimethod, multi-informant, longitudinal investigation demonstrated novel and directional associations among childhood CVE, ANS-R to fear, and the development of different forms and functions of aggression in early adolescence in an ethnically and racially diverse sample. Importantly, the diversity of the sample allowed us to examine subgroup differences, adding nuance to existing work. For example, our findings regarding Latine youth revealed lower levels of reactive physical aggression and proactive relational aggression compared to non-Latine peers. This pattern diverges from prior reports suggesting elevated aggression among Latine youth often attributed to acculturation stress and CVE (Fenimore et al., 2019). At the same time, our results align with research indicating that protective cultural factors and social norms are associated with lower aggression rates in Latine youth (Marsiglia et al., 2009). Although we did not directly assess cultural mechanisms, these findings highlight within-group variability and provide a critical counterpoint to deficit-based narratives portraying Latine youth as disproportionately aggressive.

Additionally, our examination of specific forms and functions of aggression extends prior research investigating the effects of CVE and physiological reactivity on externalizing behaviors (e.g., Cooley et al., 2019) and prior work focused on physical forms of aggression (e.g., Guerra et al., 2003; Thomson et al., 2021). Further, the use of physiological measures to assess both SNS-R and PNS-R to fear affords a comprehensive understanding of how ANS-R shapes aggression subtypes in the context of adversity. Finally, investigating these pathways from middle childhood to early adolescence extends prior research on adults (e.g., Thomson et al., 2021) to a salient time for the development of relational forms of aggression (i.e., when peer relationships take on heightened significance and complexity; Flynn et al., 2017).

Despite these strengths, it is important to evaluate the current findings in the context of several limitations. First, with respect to measurement of aggression, although significant overlap between the forms and functions of aggression is consistent with prior research (e.g., Card & Little, 2006; Card et al., 2008), future research in this area may benefit from employing statistical approaches (see Little et al., 2003) or measures (see Polman et al., 2009) focused on distinguishing between proactive and reactive functions of aggression. Additionally, although caregiver reports are known to have strong reliability for assessing subtypes of aggression (e.g., Baker, 2022), caregivers may not observe the full range of aggressive behaviors across varied social contexts, such as in peer and school settings (Kuppens et al., 2009). In particular, regardless of their proactive or reactive functions, relationally aggressive behaviors may be harder for caregivers to notice because they may be subtle and indirect (e.g., gossiping, social exclusion; Brandes et al., 2021). The use of multiple informants, including teacher, peer, and self-reports, would provide a more comprehensive picture of aggressive subtypes. Relatedly, our aggression measures did not assess some developmentally advanced manifestations of relational aggression that become increasingly salient during adolescence, such as gossip and rumor spreading (Voulgaridou & Kokkinos, 2015), and instead primarily assessed behaviors such as social exclusion and threats to withdraw friendship. As these more sophisticated behaviors can be proactive (e.g., threatening to share a peer's secret to control their behavior) or reactive (e.g., spreading gossip against a peer in response to provocation) in function, this study may underestimate the prevalence of proactive and reactive relational aggression during this developmental stage. Nevertheless, the assessed behaviors continue to be socially meaningful and are linked to important developmental adaptation in adolescence (e.g., Merrell et al., 2006). Future research will benefit from statistical and assessment advances to capture varied behavioral manifestations of proactive and reactive physical and relational aggression during adolescence.

Second, although questionnaires have been widely used in studies of CVE (J. E. Richters & Saltzman, 1990), future research should consider qualitative CVE reports and/or administrative data sources, such as Federal Bureau of Investigation crime statistics (e.g., P. Sharkey, 2010; P. T. Sharkey et al., 2012). Likewise, future research may benefit from investigating distinct components of CVE (e.g., witnessing vs. victimization, violence between strangers vs. familiar people) as they may exhibit unique associations with subtypes of aggression (Calvete & Orue, 2011; Chaux et al., 2012). Finally, because the effects of CVE on youth outcomes may be misattributed or overestimated when other neighborhood factors are not considered (e.g., sexual harassment; Mora et al., 2022), future research in this area should consider controlling for other types of adversity.

Third, when measuring ANS-R to fear, participants observed other children in a fearful situation rather than experiencing a direct threat. Prior work has demonstrated that exposure to film stimuli effectively elicits both subjective and physiological fear responses (Fernández-Aguilar et al., 2019). Moreover, this approach provided a standardized method to evoke children's fear without the ethical concerns introduced by first-hand emotion induction (Fernández-Aguilar et al., 2019). Indeed, this witnessing paradigm is well-aligned with the current focus on CVE, which primarily involves observed fear experiences (e.g., witnessing a knife being pulled on

someone else) rather than direct ones (e.g., being stabbed with a knife). Nevertheless, study findings may depend on fear induction techniques, and future research should consider using alternative methods, such as direct exposure to fear-inducing stimuli.

Fourth, future research would benefit from a focus on additional developmental processes that may help elucidate the effects observed in the present study. For instance, given bidirectional associations between CVE and aggressive or delinquent behaviors (i.e., Esposito et al., 2017; Farrell et al., 2022), future research should examine bidirectional effects and potential mediators (e.g., effortful control; Esposito et al., 2017) that may underlie longitudinal associations between CVE and aggression. Similarly, future research should investigate associations between CVE and ANS-R in the development of aggression across longer developmental periods, given developmental changes in forms (Card et al., 2008) and functions (Card & Little, 2006) of aggression, as well as potential pubertal effects on both functioning of ANS functioning and aggression (Viau, 2002).

Finally, some researchers have assessed trajectories of ANS activity across tasks, which may afford novel insights, particularly regarding ANS recovery (Obradović & Finch, 2017). However, theoretical and practical consideration informed our a priori decision not to estimate trajectory-based models (e.g., latent growth curve or latent basis models) based on physiological epochs of 30 s. Theoretically, we sought to align our analytic approach with prior research examining interactions between PNS and SNS reactivity in relation to aggression (e.g., Thomson et al., 2021), which has predominantly relied on residualized change score approaches to operationalize ANS reactivity. This facilitated comparability with existing findings in this emerging area of research. Practically, modeling within-task trajectories of SNS and PNS activity requires highly complex analytic models (e.g., incorporating three-way interactions including latent variables) that could not be supported by the limited sample size within the physiological subsample ($N =$ 137). Although our residualized change score approach to model ANS reactivity allowed us to limit the complexity of our analytic models, the inclusion of higher order interactions and high levels of missing ANS data may have rendered some analyses underpowered. Thus, we conducted additional sensitivity analyses to estimate the minimum detectable effect size for the physiological subsample. These analyses were sufficiently powered to detect small-to-moderate effects ($f^2 = .06$), indicating that smaller but potentially meaningful effects may have gone undetected. Despite our statistical accommodation of missing data through maximum likelihood estimation with robust standard errors (Little et al., 2014), missingness within the physiological data may still have reduced power and compromised the stability of higher order interaction estimates. Consequently, these findings should be interpreted with caution, and replications with both larger samples and trajectory-based modeling approaches are warranted.

Conclusions

This study provides unique and valuable insights regarding how individual and interactive relations between CVE and ANS-R shape the development of aggression forms and functions across the transition to early adolescence. Consistent with diathesis–stress models, these findings suggesting that environmental stressors (e.g., CVE) interact with physiological factors (e.g., ANS-R) to shape the development of specific externalizing behaviors. Evidence for a distinct set of physiological vulnerability factors by gender highlights the need for further research that explores the unique pathways through which CVE and ANS-R influence aggression in girls. These findings also have important implications for improving prevention and intervention strategies aimed at reducing aggressive behavior in adolescents. Specifically, tailoring interventions to account for individual differences in ANS-R, aggression subtypes, and gender may enhance their effectiveness. For example, interventions that help youth recognize and regulate dysregulated physiological responses to stress or fear, particularly in high-adversity environments, could mitigate the development of aggression. Moving forward, considering both physiological and environmental factors will enhance prevention and intervention efforts.

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