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RESEARCH ARTICLE

Dimensions of childhood adversity differentially affect autonomic nervous system coordination in response to stress

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ABSTRACT

It is well-established that disrupted autonomic nervous system (ANS) reactivity exacerbates risk for long-term maladjustment following childhood adversity (CA). However, few studies have integrated measures of both the sympathetic (SNS) and parasympathetic (PNS) branches of the ANS, resulting in a unidimensional understanding of ANS functioning as a mechanism of risk. Further, past work has primarily measured CA only at the aggregate level (e.g. "total CA"), necessitating further research to accurately characterize this risk pathway. The present study examines how CA, measured cumulatively and dimensionally (i.e. CA characterized by threat versus deprivation), moderates the association between the SNS and PNS at rest and in response to acute social and nonsocial stressors. Participants included 97 adolescents ages 10-15 (M_{age} = 12.22, SD_{age} = 1.68) experiencing a range of CA and one accompanying caregiver. Participants completed questionnaires assessing prior CA exposure. SNS and PNS responses were then continuously measured during rest and two stress tasks. First, results indicate a blunting effect of cumulative CA and CA characterized by threat (e.g. physical abuse) on resting SNS activity. Second, in moderation analyses assessing ANS coordination, threat exposure emerged as a significant moderator of the association between SNS and PNS reactivity to social stress. Results suggest that CA characterized by threat may specifically impact physiologic regulation by disrupting the coordination of the two branches of the ANS. Disentangling the independent and concurrent engagement of biological stress response systems following CA remains an important target for research to identify the etiology of aberrant stress reactivity patterns.

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Childhood adversity (CA), which includes childhood experiences of abuse, neglect, and household dysfunction, represents a particularly detrimental form of stress exposure that has been associated with a variety of psychiatric problems (Green et al., 2010). Research suggests that persistent exposure to stressors throughout development interacts with CA to predict psychopathology and points to the body's physiologic stress response systems as a fundamental risk pathway underlying this link (Doom & Gunnar, 2013). It is well established that CA disrupts the development of the autonomic nervous system (ANS), which plays a crucial role in facilitating effective socio-emotional responses to the environment (Kreibig, 2010), resulting in aberrant stress reactivity patterns that have long-term detrimental effects (Busso et al., 2017; Doom & Gunnar, 2013; McLaughlin et al., 2014). However, findings have been mixed regarding the influence of CA on ANS patterns, with some studies demonstrating heightened reactivity, others demonstrating blunted reactivity, and still others finding no association (Bernard et al., 2017). We propose that greater clarity may be achieved by measuring ANS and CA as multidimensional constructs.

Autonomic nervous system

The ANS facilitates stress reactivity processes through interactions of the sympathetic (SNS) and parasympathetic (PNS) nervous systems. The SNS serves to mobilize physiological resources to respond to environmental demands and the PNS serves to inhibit sympathetic activation and facilitate a return to homeostasis (Porges, 2007). Successful adaptation to stressful situations requires a balance between the operation of the SNS and PNS. Existing frameworks describing ANS functioning emphasize reciprocity between the SNS and PNS whereby withdrawal of the vagal brake (i.e. decreased PNS activity) facilitates SNS activity while application of the vagal brake (i.e. increased PNS activity) inhibits SNS activity. One common framework, the Doctrine of

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Autonomic Space, defines *reciprocal activation* as the PNS and SNS operating together to upregulate (increased SNS and decreased PNS) or downregulate (decreased SNS an increased PNS) target organs and defines *nonreciprocal activation* as the PNS and SNS demonstrating opposing activation patterns (i.e. simultaneous activation [coactivation] or simultaneous inhibition [coinhibition]) that do not exert a clear regulatory effect. Uncoupled activation, though less common, also has been observed (Christensen et al., 2020).

Assessing the pattern of physiological activity across both the sympathetic and parasympathetic arms of the ANS is relevant to the development of psychosocial problems, yet the majority of past research has focused on a single index of the ANS. The failure to measure and analyze both branches of the ANS may contribute to the conflicting findings observed in prior research examining the link between CA and the ANS. Importantly, the few studies that have measured CA and SNS/PNS coordination suggest that patterns of coinhibition or coactivation exacerbate risk for internalizing and externalizing problems following CA (El-Sheikh et al., 2009; Wagner & Abaied, 2015). However, these studies have treated ANS coordination as a moderator rather than an outcome, necessitating an isolated examination of CA and SNS/PNS reactivity to achieve a more nuanced understanding of this link. This study builds on multisystem psychophysiological models (e.g. Beauchaine, 2001; Porges, 2007) and prior approaches (e.g. El-Sheikh et al., 2009, 2013) to address this gap by examining CA as a moderator of the association between the SNS and PNS.

Dimensions of childhood adversity

In contrast to the prevailing cumulative measurement approach in which CA exposures are summed across various exposure types, the Dimensional Model of Adversity and Psychopathology (DMAP) puts forth *threat* (i.e. exposure to actual or threatened violence) and *deprivation* (i.e. the absence of expected cognitive and social inputs) as two dimensions of CA that have shared features which pose unique risk for psychopathology (Sheridan & McLaughlin, 2014). While deprivation exposure has been related to reduced cognitive functioning and language development, threat exposure has been linked to deficits in emotion processing and reactivity (McLaughlin et al., 2019), each of which represents a unique risk pathway. Consistent with this model, research suggests that threat exposure may uniquely impact the ANS (McLaughlin et al., 2014), likely due to the influence of threat on the developing cortico-limbic circuits that underlie fear learning and stress processing. However, further research is needed to understand the links between CA at the dimensional level in relation to SNS and PNS functioning.

Developmental timing

Exposure to stress in adolescence is one of the strongest predictors of psychopathology onset, with risk amplified for adolescents with CA exposure (Starr et al., 2014, 2017). Thus, there is a pressing need to identify intervention targets in this demographic. Investigating the impact of CA on the SNS and PNS in early- to mid-adolescence is optimal, given that (1) biological alterations following CA occur prior to clinical manifestations (do Prado et al., 2017), and (2) the ongoing plasticity of the ANS in adolescence suggests that recalibrating physiological reactivity through intervention is possible (DePasquale et al., 2019). Although increased positive coupling of neurobiological systems is typically observed throughout development (Beauchaine, 2001; Gabard-Durnam et al., 2014), the way in which CA interferes with the development of SNS-PNS coupling during adolescence is currently unknown.

Current study

In this study, we examine how cumulative CA, CA characterized by deprivation, and CA characterized by threat each relate to SNS and PNS activity at rest and in response to acute stress. Skin conductance level (SCL), the electrodermal activity caused by sweat gland activity, was used as an index of SNS. Respiratory sinus arrhythmia (RSA), the variation in heartbeat between respiration frequencies, was used as an index of PNS. Because research suggests that the ANS exhibits different reactivity patterns depending on context (Bush et al., 2011), partly due to the impact of early experiences (Rudd & Yates, 2018), we included two ecologically valid stress tasks: one nonsocial (star-tracing) task and one social (caregiver-child conflict interaction) task. Examining adolescents' responses to both nonsocial (star tracing) and social (conflict interaction) is optimal to characterize physiological reactivity over time and provide information about the specificity versus generality of psychophysiological functioning (El-Sheikh, 2005).

Analyses focused on the associations between CA (cumulative CA, deprivation, threat), the SNS, the PNS, and SNS-PNS coordination in three contexts: (1) at rest; (2) reactivity to nonsocial stress; (3) reactivity to social stress. By including measures of cumulative CA, threat, and deprivation, the study aims to disentangle the differential impacts of these dimensions on ANS activity. This multidimensional approach addresses limitations in previous research that primarily focused on aggregate CA measures and unidimensional ANS assessments. Further, the study design includes multiple stress-induction paradigms and rigorous physiological measurements, enhancing the ecological validity and reliability of the findings. Consistent with recent research suggesting that CA leads to blunted SNS reactivity (Young-Southward et al., 2020) and research suggesting that traumatic stress may lead to non-reciprocal ANS activation patterns (El-Sheikh et al., 2009), we expected CA to be related to lower SNS activity across conditions and coinhibition (i.e. blunted SNS and PNS activation) during stress tasks. Given that this was the first examination, to our knowledge, of the link between dimensions of CA and SNS/PNS coordination, analyses of threat and deprivation were largely exploratory. However, based on preliminary evidence suggesting that threat exposure in childhood may uniquely impact developing physiological systems, we hypothesized that the magnitude of the association between threat and observed ANS patterns would be greater than that of cumulative CA or deprivation.

Method

Participants

Participants included 97 adolescents ages 10-15 (M_{age} = 12.22, SD_{age} = 1.68) experiencing a range of CA and one accompanying caregiver (M_{age} = 42.04, SD_{age} = 6.98). Forty-five adolescents (46.4%) identified as female, 51 (52.6%) identified as male, and 1 (1%) identified as non-binary. Participating caregivers (89.4% female, 10.6% male) included 90 biological parents (92.8%), five adoptive/kinship parents (5.0%), and two stepparents (2%). Seventy-two percent of youth identified as White, 16% identified as Black or African American, 5% identified as Asian, and 7% identified as having a mixed racial/ethnic background or "other." Families reported a range of gross household incomes: 3.1% under $15,000, 6.2% $15,000 to $29,999, 14.4% $30,000 to $44,999, 12.4% $45,000 to $59,999, 15.5% $60,000 to 74,999, 5.2% $75,000 to 89,999, 7.2% $90,000 to 104,999, 6.2% $105,000 to 119,999, 5.2% $120,000 to 134,999, 6.2% $135,000 to 149,999, 17.5% greater than $150,000, and 1% "unknown."

Adolescents were recruited through multiple sources, including email advertisements, adoption service agencies, and psychiatric outpatient clinics. Interested caregivers completed a brief phone screen to determine eligibility. Caregivers who endorsed that adolescents were not 10-15 years old, did not reside with that caregiver at least 50% of the time and for at least six months, or had a preexisting neurodevelopmental condition, pervasive developmental disorder, or psychosis were excluded.

Procedure

Eligible caregivers and adolescents were invited to participate in a laboratory visit which included questionnaires, interviews, and two stress-induction paradigms. First, caregivers and youth completed an adapted version of the Issues Checklist (Robin & Foster, 1988) in which they indicated how often they discussed each of 44 topics (e.g. "cleaning up bedroom") during the past four weeks and rated the intensity of each discussion from 1 (*calm*) to 5 (*angry*). Research assistants identified the most highly rated by the caregiver and their child, which served as the discussion topic for the conflict interaction task.

Second, adolescents and caregivers participated in a physiological assessment to measure RSA and SCL during resting, reactivity, and recovery conditions. Electrodes were placed on the rib cage, chest, back, and hand following standard guidelines (Berntson et al., 1997; Scerbo et al., 1992). Because the focus of this study is on individual differences in interpersonal stress responses rather than differential responding to tasks, and because the star-tracing tends to be frustrating and may serve as a prime for arousing negative affect prior to the caregiver-adolescent conflict task, a fixed task order was used (see El-Sheikh et al., 2013). Dyads first engaged in an acclimation (3 min) and baseline (3 min) period, during which they were instructed to sit quietly and breathe normally. Next, participants completed an adapted version of the star-tracing task (*nonsocial stress task;* Lafayette Instrument Company). In this task, a board was placed across from each participant's chair with a picture of three stars of varying sizes. Stars were only visible through a mirror. Participants were asked to trace the stars in descending size order using only the mirror image as a visual guide (3 min). The star-tracing task is a well-established, cognitively challenging stressor that consistently induces RSA and SCL reactivity, which in turn has been associated with family conflict and internalizing and externalizing problems in youth (El-Sheikh, 2005; El-Sheikh et al., 2013).

After three minutes, a recovery period (3 min) ensued, followed by a 3-minute speaking baseline where participants counted aloud from a list of numbers. The counting baseline was used to account for the effects of speaking on RSA, given that the conflict task is conversation-based and will impact respiration rates. Caregiver-adolescent dyads then engaged in the *social stress task*. Dyads were given their assigned discussion topic and instructed to describe the issue, describe their feelings, discuss why it is a source of conflict, and attempt to resolve the issue (10 min). The caregiver-child conflict interaction task was chosen for its ecological validity and its ability to elicit meaningful physiological and emotional responses within a familiar social context. Conflict tasks using a similar protocol have consistently produced ANS activation (Beijersbergen et al., 2008). This task was followed by a final recovery/baseline period of six minutes.

During the final component of the lab visit, participants completed questionnaires assessing prior CA exposure. In cases where an item of safety concern was endorsed (e.g. physical abuse), staff consulted with clinical supervisors and mandated reporting procedures were followed. All procedures were approved by the University's Institutional Review Board. This study was not preregistered. Data and study materials are available upon request.

Measures

Childhood Adversity

CA was assessed via youth self-report on the Stress and Adversity Inventory (STRAIN; Slavich et al., 2019) and via caregiver- and child-report on the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003). The STRAIN is a NIMH/RDoC-recommended online, interview-based system designed to assess the occurrence of major life stressors over the life course. For each stressor endorsed, participants are asked follow-up questions to determine stressor severity, frequency, timing, and duration (see https://www.strainsetup.com). The CTQ is a 28-item inventory with five subscales assessing exposure to emotional, physical, and sexual abuse and emotional and physical neglect. Each subscale is comprised of five questions using a five-point Likert scale from *never true* (1) to *very often true* (5). Previous studies support the reliability and validity of the CTQ and provide thresholds for assessing maltreatment presence in each category, which were used to code each category as present (1, above threshold) or absent (0, below threshold) (Bernstein et al., 1998, 2003; Spinhoven et al., 2014). Total CA was determined from the STRAIN, which codes stressors as present (1) or absent (0) and provides a

count of *total life stressors*. An a priori selection of items from the CTQ and STRAIN was used to create composites of *threat* and *deprivation*. Items comprising each CA dimension are listed in Supplementary Table 1

Physiological reactivity

RSA and SCL were assessed using the MindWare MW100A Acquisition System and scored using MindWare analysis software (V3.2). ANS data was collected continuously during resting and reactivity conditions and synced with the MindWare BioNex system to examine simultaneous readings.

Mean levels of SCL at rest and during stress tasks were determined by averaging SCL responses during 1-minute increments. Mean SCL was calculated for the initial baseline (SCL: B), counting baseline (SCL: CB), star tracing task (SCL: ST), and conflict task (SCL: CT). SCL reactivity (SCL-R) to the nonsocial stress task was computed as a residualized change score by regressing SCL: B on SCL: ST (SCL-R: ST). SCL reactivity to the social stress task was computed by regressing SCL: CB on SCL: CT (SCL-R: CT). SCL is expressed in microSiemens (μS).

RSA was assessed via rhythmic fluctuations in heart rate accompanied by phases of the respiratory cycle (Grossman et al., 1991) using the peak-to-valley method (Berntson et al., 1997). Resting RSA (RSA-B) was computed by averaging the 1-minute increments of differences in interbeat interval readings from inspiration to expiration onset in the initial baseline (RSA: B) and counting baseline (RSA: CB) assessments. RSA reactivity (RSA-R) was computed as residualized change by regressing RSA: B and RSA: CB on the mean RSA level during the star tracing (RSA: ST) and conflict discussion tasks (RSA: CB), respectively. Low RSA-R values indicate greater RSA withdrawal in response to the tasks. Eleven participants had missing ANS data barring SCL/RSA change score calculations, resulting in complete data for 86 participants.

Data analysis

In preliminary analyses, paired-sample *t*-tests were used to determine whether RSA and SCL at baseline significantly differed from RSA and SCL during stress tasks. Bivariate correlations were then conducted as a first step in examining relations among variables. Third, we tested the main and interaction effects of total CA, threat, and deprivation on ANS coordination via moderated regression analyses performed in PROCESS for SPSS (Hayes, 2013). RSA served as the dependent variable, and interactions between SCL and adversity (i.e. total CA, threat, or deprivation) were calculated and entered into each model. Age was controlled for in all models to account for development-related changes in ANS functioning. Additionally, consistent with prior research assessing the unique impact of threat and deprivation on developmental risk pathways (Busso et al., 2017; Machlin et al., 2019; Milojevich et al., 2019), deprivation exposure (present/absent) was included as a covariate when threat served as the moderator, and threat exposure (present/absent) was included as a covariate when deprivation served as the moderator.

Variables were mean-centered and independent variables and interaction effects were entered simultaneously. Unstandardized regression coefficients (*b*), standard errors of unstandardized regression coefficients (*SE*), 95% confidence intervals (CI), and *p*-values were obtained. Bootstrapping of the CIs of the indirect effects was used to determine significance. Significant interactions probed at low (-1 SD) and high (+1 SD) levels of the moderator via simple slopes using PROCESS and graphically plotted.

Results

Means, standard deviations, and bivariate correlations are presented in Table 1. Compared to baseline, paired-sample *t*-tests indicated increased SNS activation during both stress conditions (*nonsocial stress task*: $t(91) = -6.66$, $p < .001$; *social stress task*: $t(91) = -4.59$, $p < .001$), decreased RSA activation during the nonsocial stress task ($t(91) = 4.99$, $p < .001$), and increased RSA activation during the social stress task ($t(91) = -8.43$, $p < .001$). When examining associations between the two branches of the ANS, no significant correlations emerged between RSA and SCL at rest, during stress tasks, or when utilizing measures of reactivity (*ps* > .10).

Bivariate correlations of CA and ANS indices revealed that SCL at rest (SCL: B) was negatively correlated with total CA ($r = -.22$, $p = .042$) and threat ($r = -.23$, $p = .029$). The association between SCL: B and deprivation ($r = -.18$) approached

Table 1. Descriptive statistics and bivariate correlation matrix.

	Mean	SD	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. Total CA	20.71	13.88	–															
2. Deprivation	0.87	1.12	0.65**	–														
3. Threat	1.81	1.62	0.72**	0.59**	–													
4. RSA: B	6.65	0.96	0.01	0.00	0.05	–												
5. SCL: B	11.91	8.17	−0.22*	−0.18+	−0.23*	−0.08	–											
6. RSA: ST	6.33	0.92	0.11	0.08	0.10	0.66**	−0.04	–										
7. SCL: ST	14.52	9.01	−0.15	−0.14	−0.15	−0.09	0.90**	−0.04	–									
8. RSA: CB	6.28	0.97	−0.16	−0.11	−0.11	0.72**	0.06	0.70**	0.01	–								
9. SCL: CB	14.61	8.96	−0.18+	−0.15	−0.18+	−0.10	0.89**	−0.06	0.96**	0.03	–							
10. RSA: CT	16.85	9.71	−0.11	−0.09	−0.03	0.75**	0.03	0.76**	0.01	0.83**	−0.01	–						
11. SCL: CT	6.77	0.81	−0.19+	−0.22*	−0.16	−0.06	0.78**	−0.02	0.89**	0.05	0.89**	0.03	–					
12. RSA-R: ST	0.01	0.65	0.07	−0.07	0.04	−0.02	0.01	0.74**	0.07	0.27*	0.04	0.35**	0.08	–				
13. SCL-R: ST	0.02	3.92	0.12	0.14	0.16	−0.12	0.00	−0.01	0.44**	−0.15	0.36**	−0.09	0.39**	0.13	–			
14. RSA-R: CT	−0.41	2.72	0.05	−0.04	0.08	0.27*	−0.07	0.35**	−0.05+	0.02	−0.12	0.58**	−0.05	0.25*	0.02	–		
15. SCL-R:CT	0.00	0.46	−0.13	−0.15	−0.11	0.16	0.11	0.10	0.20+	0.15	0.13	0.14	0.41**	0.04	0.19+	0.07	–	
16. Age	12.22	1.68	0.27*	0.09	0.24*	−0.14	−0.25*	−0.04	−0.19+	−0.15	−0.24*	−0.03	−0.21*	0.10	0.04	0.19+	−0.05	–

Note. +*p* < .10, **p* < .05, ***p* < .01, ****p* < .001. CA = Childhood adversity; RSA = Respiratory sinus arrythmia; SCL = Skin Conductance Level; B = Baseline; ST = Star tracing; CB = Counting baseline; CT = Conflict task; RSA-R = RSA reactivity; SCL-R = SCL reactivity.

significance at p = .079. Mean SCL during the conflict task was negatively correlated with deprivation (r = -.22, p = .036), and the association between SCL: CT and total CA also approached significance (r = -.19, p = .082). No other significant correlations emerged.

Models testing the moderating effect of total CA, threat, or deprivation on the association between SCL-R and RSA-R to the nonsocial and social stress tasks are presented in Tables 2 and 3, respectively. No significant interaction effects emerged in models assessing total CA, threat, or deprivation exposure as moderators of SNS and PNS coordination during the nonsocial stress task (Table 2). In models focused on the social stress task (Table 3), threat significantly moderated the association between SCL reactivity and RSA reactivity to the conflict discussion task (b = .03, p = .044) when controlling for child age and deprivation exposure, as hypothesized. Specifically, individuals with high threat exposure demonstrated a significant positive association (i.e. coactivation pattern) between SCL-R and RSA-R (b = .07, p = .035), while no significant association between SCL-R and RSA-R emerged for individuals with low threat exposure (b = -.04, p = .253) (Figure 1).

Discussion

The impact of traumatic stress on developing physiologic regulatory systems is a well-established mechanism of risk linking CA to psychopathology. However, the tendency of prior research to utilize broad measures of CA and unidimensional

Table 2. Childhood adversity as a moderator of ANS reactivity to nonsocial stress (Star-Tracing).

	Coefficient	SE	95% CI	*p*
Total CA				
Age	0.04	0.04	[-0.05, 0.13]	0.366
SCL-R	0.02	0.02	[-0.02, 0.06]	0.252
Total CA	0.00	0.01	[-0.01, 0.01]	0.792
SCL-R X CA	−0.00	0.00	[-0.00, 0.00]	0.963
Deprivation				
Age	0.04	0.04	[-0.05, 0.12]	0.397
Threat	0.05	0.19	[-0.33, 0.42]	0.810
SCL-R	0.03	0.02	[-0.01, 0.07]	0.131
Deprivation	−0.06	0.07	[-0.20, 0.08]	0.384
SCL-R X Deprivation	−0.02	0.02	[-0.06, 0.01]	0.218
Threat				
Age	0.03	0.04	[-0.05, 0.12]	0.435
Deprivation	−0.10	0.16	[-0.42, 0.22]	0.544
SCL-R	0.02	0.02	[-0.01, 0.06]	0.188
Threat	0.01	0.05	[-0.09, 0.11]	0.844
SCL-R X Threat	−0.01	0.01	[-0.04, 0.02]	0.447

Note. Respiratory sinus arrythmia reactivity in the start tracing task (RSA-R: ST) serves as the dependent variable in all models. CA = Childhood adversity; SCL-R = Skin conductance level reactivity to the star tracing task.

Table 3. Childhood adversity as a moderator of ANS reactivity to social stress (Conflict Discussion).

	Coefficient	SE	95% CI	*p*
Total CA				
Age	0.04	0.03	[-0.02, 0.11]	0.177
SCL-R	0.02	0.02	[-0.02, 0.06]	0.389
Total CA	0.01	0.00	[-0.01, 0.01]	0.852
SCL-R X CA	0.00	0.00	[-0.00, 0.00]	0.508
Deprivation				
Age	0.04	0.03	[-0.03, 0.10]	0.274
Threat	0.13	0.13	[-0.13, 0.38]	0.330
SCL-R	0.01	0.02	[-0.03, 0.06]	0.493
Deprivation	−0.02	0.05	[-0.13, 0.08]	0.645
SCL-R X Deprivation	0.01	0.03	[-0.04, 0.06]	0.799
Threat				
Age	0.02	0.03	[-0.04, 0.09]	0.447
Deprivation	0.06	0.12	[-0.17, 0.30]	0.587
SCL-R	0.02	0.02	[-0.02, 0.06]	0.296
Threat	0.02	0.04	[-0.05, 0.09]	0.597
SCL-R X Threat	0.03	0.02	[0.00, 0.06]	0.044

Note. Respiratory sinus arrythmia reactivity in the conflict task (RSA-R: CT) serves as the dependent variable in all models. CA = Childhood adversity; SCL-R = Skin conductance level reactivity to the conflict task.

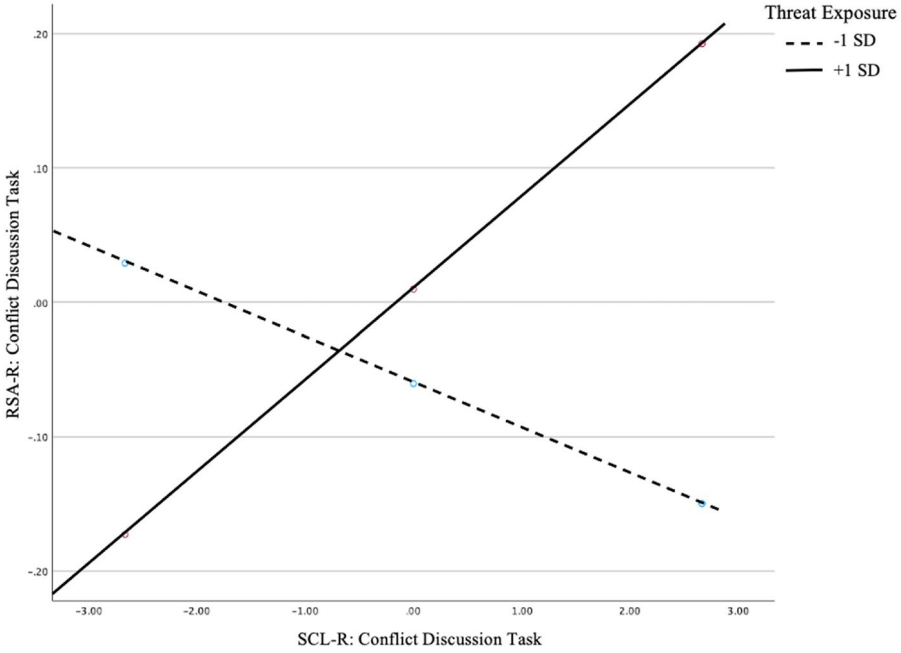

Figure 1. Threat exposure moderates the association between SCL and RSA reactivity to social stress (Conflict Discussion).

assessments of ANS functioning has limited our ability to accurately characterize this risk pathway. The present study extends prior work by investigating the impact of CA, measured cumulatively (i.e. total count of stressors) and dimensionally (i.e. threat, deprivation), on SNS and PNS activity at rest and in response to acute social and nonsocial stressors during a critical period in development (adolescence). Results highlight the utility of examining CA dimensionally, suggesting that CA characterized by threat may uniquely disrupt the coordination of the two branches of the ANS.

In initial examinations of ANS activity, paired-sample *t*-tests demonstrated a significant increase in SCL during stress conditions compared to baseline, suggesting that the star tracing and conflict interaction tasks successfully elicited an SNS response. RSA activation decreased during the nonsocial stress task, as expected, but increased during the social stress task. The increase in RSA during the conflict interaction task could reflect a regulatory mechanism where the PNS becomes more active alongside the SNS response in an effort to help individuals navigate social challenges more effectively (Butler et al., 2006; Porges, 2007; Skowron et al., 2014); however, further research is necessary to examine this possibility. In bivariate correlation analyses, no significant correlations emerged between SCL and RSA across conditions. One potential explanation for the lack of direct correlations arises from the continued development of ANS coordination during adolescence. Research has suggested that physiological systems may display reduced coordination in childhood, such that a robust response may be observed in one system without a response in another (Alkon et al., 2011). It is possible that the age range of 10-15 years captures a developmental period during which reciprocal coordination patterns have not yet been fully achieved.

Consistent with our hypotheses, total CA and threat were each negatively correlated with baseline SCL, indicating that CA exposures in general, as well as adversity characterized by threat, are both linked to lower SNS arousal. Deprivation was negatively correlated with SCL levels during the conflict task, raising the possibility that this form of CA may exert a unique effect on sympathetic activity during social interactions. Interestingly, no significant correlations emerged between measures of CA and RSA. Although some studies have observed lower baseline RSA in association with adversity (e.g. Dale et al., 2009; Miskovic et al., 2009), links between CA and ANS activity tend to emerge more consistently in examinations of SNS activity, while associations between CA and the PNS have been more mixed (Young-Southward et al., 2020). Our findings are consistent with other studies that have not observed associations between parasympathetic activity and CA (e.g. van Ockenburg et al., 2015; Winzeler et al., 2017).

The lack of significant correlations between any indices of CA and SNS or PNS *reactivity* to the lab stress tasks was unexpected. To date, most research on CA and physiological reactivity has focused on other physiologic systems (e.g. HPA axis), and the impact of CA on ANS activation patterns has not been well-established in adolescents (Chiang et al., 2015). Findings could, therefore, indicate a true absence of a direct link between CA and each individual ANS branch. Alternatively, findings could be attributable to the relatively simple indices of exposure to CA employed in this study, as we did not collect data on factors like perceived severity of CA that may influence these associations. A third possibility is that the link between CA and ANS functioning may be complex and only emerge when additional stress response systems (e.g. HPA axis) are considered, which should be explored in future work.

In moderation analyses, threat emerged as the only significant moderator between SNS and PNS reactivity, such that threat explained significant variability in link between SCL-R and RSA-R during the social stress task, even when controlling for participant age and deprivation exposure. This finding is consistent with evidence that threat uniquely impacts emotion reactivity and regulation processes, including the modulation of the SNS and PNS (Slavich, 2020, 2022), while deprivation impacts other neurobiological developmental pathways (e.g. cognitive functioning; McLaughlin et al., 2014; Young-Southward et al., 2020). Specifically, our results demonstrated a positive link between SCL-R and RSA-R for individuals with threat exposure only, indicating patterns of coinhibition (low SCL-R and low RSA-R) and coactivation (high SCL-R and high RSA-R). Coinhibition may impede individuals' ability to respond to environmental demands due to a failure to activate the SNS resources necessary to organize and mobilize a response, while coactivation has been tied to dysregulated emotional reactivity and increased externalizing problems (El-Sheikh et al., 2009). Given that these functional implications of ANS coordination are not fully understood in this age range, future studies should replicate analyses testing the moderating impact of CA dimensions on SNS-PNS activation patterns and investigate the links between SNS/PNS coordination and indicators of adjustment (e.g. emotion regulation) and psychopathology symptoms.

Limitations

These findings should be interpreted in light of several study limitations. First, we intentionally recruited a typically developing sample of young adolescents with a range of adverse experiences. This may be considered a strength, as findings are more likely to generalize to the broader population. However, results may differ in a sample of adolescents with more severe CA exposures or psychopathology, underscoring the importance of replication. Second, our measurement of CA only provides information on the presence or absence of events and the total number of these experiences. The impact of CA severity or timing in relation to SNS/PNS activation patterns is currently unknown. Third, the utilization of mean-level reactivity scores to measure sympathetic and parasympathetic activity, although a standard in the field, does not capture the nuance of moment-to-moment analyses of RSA and SCL patterns, which could elucidate periods of ANS branch coordination not observable via aggregate measures. Fourth, future studies would benefit from analyzing different aspects of the stress-reactivity tasks (e.g. assessing post-task recovery) including additional indices of the SNS and PNS (e.g. pre-ejection period) and additional physiological systems (e.g. HPA-axis [cortisol]), as there are likely more complex CA-related disturbances in the central and peripheral physiological feedback

loops that were outside of the scope of this study (McEwen, 2007). Fifth, although in line with our study goals, we did not include measures of psychopathology in this study. As such, conclusions on how observed ANS patterns impact psychological functioning cannot be drawn. Finally, although fixed task order was intentionally chosen in order to focus on individual differences in ANS stress responses, the possibility of task sequencing influencing physiological reactivity results should be acknowledged and addressed in future studies.

Conclusion

Given that the two branches of the ANS have dynamic and complementary responses, measuring the SNS and PNS within a single paradigm offers valuable insights into how CA may impact physiological stress reactivity. Findings from this study advance our knowledge of how CA impacts physiological functioning, highlighting possible specificity in the association between dimensions of CA and each branch of the ANS. Specifically, results suggest that adverse experiences may have a general blunting effect on resting SNS activity, whereas threat exposure may impact physiologic regulation by disrupting the coordination of the two branches of the ANS, particularly in response to social stress. Disentangling the independent and interrelated engagement of biological stress response systems following CA remains an important target for research to identify the etiology of aberrant stress reactivity patterns and, ultimately, to inform preventive intervention efforts for this high-risk population.

Ethical approval

Procedures were approved by the Vanderbilt University Institutional Review Board (#181531).

Disclosure statement

No potential conflict of interest was reported by the author(s).

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