

# Skin Conductance Reactivity and Respiratory Sinus Arrhythmia Among Maltreated and Comparison Youth: Relations with Aggressive Behavior

Elana B. Gordis,<sup>1</sup> Nashla Feres,<sup>2</sup> Christy L. Oleszki,<sup>1</sup> Ari N. Rabkin,<sup>1</sup> and Penelope K. Trickett<sup>2</sup>

<sup>1</sup>University at Albany, SUNY, and <sup>2</sup>University of Southern California

**Objective** The goal of this study was to examine respiratory sinus arrhythmia (RSA), an indicator of parasympathetic nervous system-linked cardiac activity, and skin conductance level (SCL), a sympathetic indicator, as moderators of the link between child maltreatment and adolescent aggression. **Method** Participants were 234 maltreated (48.3% male) and 128 (57.8% male) comparison youth aged 9–16 years participating in wave 2 of a longitudinal study. **Results** Regression analyses suggest that among boys, high RSA may be protective against the effects of maltreatment on aggressive behavior. Among girls, the moderating effect of RSA was further moderated by SCL reactivity such that low levels of both baseline RSA and SCL reactivity, or conversely high levels of both baseline RSA and SCL reactivity, exacerbated the link between maltreatment and aggression. **Conclusions** High RSA may protect against the effects of maltreatment on aggressive behavior, though this effect may be moderated by SCL reactivity among girls.

**Key words** adolescents; behavior problems; child abuse and neglect.

Child maltreatment is associated with numerous behavior and emotional problems, including aggression, delinquency, and depression (Kendall-Tackett, Williams, & Finkelhor, 1993; Margolin & Gordis, 2000; Widom, 1989). However, not all maltreated youth exhibit problems. Individual factors may moderate outcomes (Flores, Cicchetti, & Rogosch, 2005; Margolin & Gordis, 2004). Much remains unknown about why some youth are more affected by maltreatment than are others. Certain lines of research suggest the importance of autonomic nervous system (ANS) variables in moderating the effects of stressful experiences (e.g., Katz & Gottman, 1995; Raine, 2005). The present study examines indicators of sympathetic (SNS) and parasympathetic (PNS) nervous system activity as moderators of the link between maltreatment and youth aggression.

## Child Maltreatment and Aggressive Behavior

The link between maltreatment and aggressive behavior is well-established (e.g., Cullerton-Sen et al., 2008; George & Main, 1979; Margolin & Gordis, 2000; Widom, 1989). Maltreatment may disrupt affect regulation and may

cause children to interpret ambiguous social cues as threatening and to respond to them aggressively (Cullerton-Sen et al., 2008). Traumatic abuse may cause anger and fear in relationships and a need to re-enact trauma with aggression (Haapasalo & Pokela, 1999).

Various types of maltreatment are linked with aggressive behavior. Links between physical abuse and aggressive behavior are well-documented (Dodge, Bates, & Pettit, 1990; Haskett & Kistner, 1991; Trickett, 1993). Physical abuse teaches that aggression is acceptable (Dodge, Pettit, & Bates, 1997) and may cause difficulties with emotion regulation (Repetti, Taylor, & Seeman, 2002). Sexual abuse can lead to hyper-reactivity to abuse-related cues and difficulty managing related emotions (Trickett, McBride-Chang, & Putnam, 1994) and is also linked with aggression (Einbender & Friedrich, 1989; Kendall-Tackett et al., 1993; Trickett & Gordis, 2004; Trickett et al., 1994). Though emotional abuse and neglect have received less attention, both are linked with aggression (Hildyard & Wolfe, 2002; Shaffer, Yates, & Egeland, 2009). Different types of maltreatment often co-occur (Trickett, Mennen, Kim, & Sang, 2009). Here we examine the overall effect of maltreatment on aggression.

All correspondence concerning this article should be addressed to Elana B. Gordis, Department of Psychology, University at Albany, SUNY, Social Sciences 399, 1400 Washington Avenue, Albany, NY 12222, USA.  
E-mail: egordis@albany.edu

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### ***Autonomic Nervous System Activity and Aggressive Behavior***

SNS and PNS activity are related to aggressive behavior. SNS activity leads to excitation of the palmar eccrine sweat glands and thus increased conductivity in the surface of the skin. Low skin conductance level (SCL) is related to aggression, particularly among males (Fung et al., 2005; Lorber, 2004; McBurnett, Harris, Swanson, Pfiffner, & Tamm, 1993; Raine & Venables, 1984; van Goozen, Fairchild, Snoek, & Harold, 2007). Various theories explain this link. Low SCL may indicate fearlessness and poor inhibition of aggressive impulses (Raine, 2005; Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007; van Goozen & Fairchild, 2008; van Goozen et al., 2007). Central nervous system substrates of low tonic SCL activity may be aversive, leading to sensation-seeking behaviors, including aggression (Gatzke-Kopp, Raine, Loeber, Stouthamer-Loeber, & Steinhauer, 2002; van Goozen et al., 2007). Low SCL may also indicate impaired learning to avoid punishment (Raine, Lencz, Bihle, LaCasse, & Colletti, 2000; Shannon, et al., 2007). Aggressive boys exhibit low SNS-linked cardiac activity in response to monetary reward, suggesting low reward sensitivity (Beauchaine, Hong, & Marsh, 2008). However, findings regarding low sympathetic indicators and aggressive behavior have not been completely consistent (Lorber, 2004). Although research with clinical populations suggests that low baseline SCL predicts aggression, high SCL reactivity to challenge has also been linked with aggressive behavior, particularly among non clinic samples (El-Sheikh, Erath, & Keller, 2007; Hubbard et al., 2002). Sex may also affect this relation. Beauchaine et al. (2008) found that higher skin conductance responding related to more aggression among girls. High SCL may reflect downstream effects of CNS processes related to fear, hostility, and aggression (Kagan, Reznick, & Snidman, 1987; van Goozen & Fairchild, 2008).

PNS activity also has been linked to aggression. PNS-linked cardiac activity can be measured by respiratory sinus arrhythmia (RSA), the variability of heart rate across the respiration cycle due to the influence of the vagus nerve on the sinoatrial node (Beauchaine, 2001). RSA relates positively to emotion regulation capacity and responsiveness and negatively to behavior problems (Beauchaine, 2001; Eisenberg et al., 1995; El-Sheikh et al., 2007). However, some findings have been inconsistent. For example, Dietrich, Riese, Sondejker et al. (2007) found higher RSA to relate to externalizing problems. Sex may also affect this relation (e.g., Eisenberg et al., 1995). Beauchaine et al. (2008) report links between low RSA

and aggression among boys but not girls. In addition to baseline RSA, researchers also have examined RSA reactivity to challenge. In response to stress, one would expect vagal withdrawal and lower RSA (Butler, Wilhelm, & Gross, 2006). Butler et al. (2006) report that increased RSA during social interaction relates to emotional suppression. Some studies suggest that higher RSA reactivity to a challenge task relates to lower levels of behavior problems (El-Sheikh, Harger, & Whitson, 2001; Hastings et al., 2008), though findings are inconsistent (El-Sheikh & Whitson, 2006).

### ***ANS Measures as Moderators of the Effects of Maltreatment on Aggressive Behavior***

SNS and PNS activity may moderate effects of maltreatment on aggressive behavior. Raine (2005) suggests that low SNS, as indicated by SCL, combined with abusive parenting may increase the risk of aggression, whereas higher SCL may be protective. Erath, El-Sheikh, and Cummings (2009) found that at values of lower SCL reactivity, the relation between harsh parenting and externalizing problems was heightened; this moderating relation was particularly consistent among boys. The combination of maltreatment with low SCL may be risky for children because low SCL may indicate low trait anxiety and inhibition (e.g., Kagan et al., 1987), which when combined with both traumatic experiences and aggressive parental models, would lead to low inhibition of aggressive behavior (Raine, 2005). On the other hand, high SCL reactivity has also been found to be a vulnerability factor for the effects of marital conflict on externalizing behavior (El-Sheikh et al., 2007). High SCL reactivity may indicate sensitization and thus increased defensiveness to the stress of interpersonal conflict. Drawing from these findings, either very low or very high SCL and SCL reactivity may increase the link between maltreatment and aggression.

PNS-linked cardiac activity, as indicated by RSA, may also interact with psychosocial factors to account for aggression. High RSA appears to buffer against the effects of marital conflict and other parenting problems (e.g., Blandon, Calkins, Keane, & O'Brien, 2008). Katz and Gottman (1995) found that high vagal tone buffers the effects of marital conflict on child behavior problems. El-Sheikh (2005) found that low RSA exacerbates the link between parents' problem drinking and children's behavior problems. Results appear more consistent for baseline RSA than for RSA reactivity to challenge. El-Sheikh et al. (2001) found that high baseline RSA buffers the effects of marital conflict on boys' and girls' problems, but that higher change in RSA to challenge was

protective only for boys. However, El-Sheikh and Whitson (2006) found that RSA suppression during challenge buffers the effects of marital conflict on internalizing problems in boys but increased the effect of marital conflict on girls. If high baseline RSA indexes emotion regulation and empathic ability, then high RSA may protect against the development of aggression in the face of maltreatment. Low RSA combined with maltreatment may carry risk due to the combination of trauma, poor affect regulation and empathy, poor social skills, and rigid and aggressive patterns of emotion expression (Beauchaine, 2001).

### **Multi-systems Approach**

Beyond two-way interactions between environmental stressors and ANS functioning, examining interactions between both branches of the ANS together with maltreatment may shed light on moderating factors. Bauer, Quas, and Boyce (2002) argue that the pattern of activity across systems may be relevant to the development of emotional and behavior problems. To date, few researchers have examined the joint role of PNS and SNS activity in behavior problems among children exposed to family violence and maltreatment. An exception is the work of El-Sheikh et al. (2009), who examined how marital conflict interacts with SCL and RSA to predict behavior problems. Consistent with work by Bernston, Cacioppo, and Quigley (1991) in their conceptualization of autonomic space, these authors note that the SNS and the PNS have generally opposing actions on organ systems, such that the SNS engages the organism for fight or flight and involves increased heart rate and respiration, whereas the PNS corresponds to slower heart rate and respiration and lower overall emotional arousal. Bernston et al. (1991) argue that the PNS and SNS function along two separate dimensions and thus can change in reciprocal fashion such that the effects of sympathetic or parasympathetic activation are maximized. Alternatively, high co-activation of both of ANS and PNS simultaneously would result in a mixture of opposing effects on organ systems. El-Sheikh et al. (2009) found reciprocal, coordinated activation of the SNS and PNS (higher RSA/lower SCL or higher SCL/lower RSA) to buffer the effects of marital conflict on children's problems, whereas co-activation and co-inhibition exacerbated the effects.

Drawing from this work and extrapolating to maltreatment, RSA and SCL may interact jointly with maltreatment to account for aggression. Youth who exhibit relatively high RSA or SCL may be protected against the negative effects of maltreatment on aggressive behavior. These youth may be better able to engage appropriately with stressful situations, to inhibit aggressive responses, to regulate difficult emotions and express them appropriately, and to

experience empathy for the potential target of their own aggression (van Goozen & Fairchild, 2008). Low baseline levels of both RSA and SCL may reflect poor emotion regulation and low inhibition of aggression and may exacerbate negative effects of maltreatment (Beauchaine, 2001; Raine, 2005). Baseline RSA and SCL and RSA and SCL reactivity may combine in different ways to buffer or exacerbate effects of maltreatment. For example low or high reactivity in both SCL and RSA may suggest poor coordination in responses across the systems (El-Sheikh et al., 2009). Low resting RSA and high SCL reactivity may suggest poor ability to regulate emotion combined with hyperreactivity to stressors, potentially leading to behavior problems. Low baseline RSA with low SCL reactivity may suggest low emotion regulation combined with low ability to learn through positive and negative reinforcement.

### **The Present Study: Maltreatment, ANS Measures and Aggressive Behavior**

Here we examine SCL and RSA as moderators of the link between maltreatment and youth aggression. First we examine whether maltreated youth differ from comparison youth in baseline RSA and SCL and RSA and SCL reactivity. Researchers examining the psychobiological effects of trauma have found increased reactivity in SNS indicators to trauma-associated stimuli (Casada, Amdur, Larsen, & Liberzon, 1998), potentially due to increased activity in the HPA axis and catecholamine systems (Charney, Deutch, Krystal, Southwick, & Davis, 1993; De Bellis et al., 1999; De Bellis & Putnam, 1994; Putnam & Trickett, 1997; Southwick & Friedman, 2001). Next we examine main and interactive effects of maltreatment, SCL and RSA in accounting for adolescent aggression.

We hypothesize that maltreatment will be associated with higher aggression scores; that maltreated youth will have lower baseline SCL and RSA and higher SCL and RSA change in response to the conflict clips; and that SCL and RSA will moderate the effect of maltreatment on aggressive behavior. Consistent with work by Beauchaine (2001), Katz and Gottman (1995), and El-Sheikh et al. (2009), we expect that higher RSA will buffer against the link between maltreatment and aggressive behavior. Furthermore, we expect that this effect will be moderated by SCL measures such that high SCL combined with high RSA and low SCL combined with low RSA will exacerbate the link between maltreatment and aggression, whereas high RSA combined with low SCL and low RSA combined with high SCL will be protective. Although we base this hypothesis on the concept of autonomic space (Bernston et al., 1991), we acknowledge that our measures of PNS and SNS activity differ from Bernston et al.'s conceptualization in

that our measures of SNS and PNS are derived from two different organs (electrodermal and cardiac activity) instead of reflecting co-activation or co-inhibition of dually activated target organs such as the heart (Beauchaine, 2009). Based on sex differences observed in the literature (e.g., Eisenberg et al., 1995; El-Sheikh & Whitson, 2006; Erath et al., 2009), we analyze data separately for boys versus girls.

## Methods

### Participants

Participants included 362 youth from a longitudinal study examining effects of child maltreatment. The current study focuses on data from the second of a three-wave longitudinal study, when participants were 9–16 years old. Of the 454 participants in wave 1, 55 youth did not return for wave 2, and 37 were excluded due to equipment malfunction or other issues (e.g., child fell asleep during session). No significant differences on demographics emerged between cases included versus excluded from the analysis, except for total yearly household income before taxes, reported in categories. Youth excluded from analysis were more likely than those included to have a family income of <\$15,000 ( $\chi^2 = 15.050$ ,  $df = 6$ ,  $p < .05$ ).

We recruited maltreated youth from selected zip codes in Los Angeles County, based on having recent reports of child maltreatment to the Los Angeles County Department of Family and Children's Services (DCFS). Participants needed to meet the following inclusion criteria: a report to DCFS within the past two months; age 9–12 years during wave 1; African American, Latino/a or Caucasian due to the aims of the larger study to examine effects of maltreatment in these ethnic groups; and live in 1 of the 11 selected zip codes. We recruited the comparison group from schools and neighborhoods from the same or demographically comparable census blocks. Comparison group youth had no history of involvement with DCFS. We obtained the names and contact information for these families from a local marketing firm. Of families we recruited, 77% of DCFS families and 50% of comparison families agreed.

Average age at wave 2 was 12.1 years (range 9.5 to 16.1;  $SD$  1.21). Ethnic composition was 10.8% Caucasian, 37.6% African American, 39.0% Hispanic or Latino/a, and 12.7% of mixed racial background. Approximately half (51.7%) of the participants were male. Maltreated and comparison youth did not differ on ethnicity. However, maltreated youth were younger than were comparison youth ( $t = 2.25$ ,  $df = 360$ ,  $p < .05$ ). Maltreated youth

were less likely than were comparison youth to have biological or adoptive parents as their caretakers ( $\chi^2 = 52.62$ ,  $df = 1$ ,  $p < .001$ ). Parents/guardians of maltreated youth were less likely than were comparison parents to have a high school diploma ( $\chi^2 = 35.15$ ,  $df = 4$ ,  $p < .001$ ). Parents/guardians of maltreated youth were more likely than comparison parents to earn <\$15,000 per year ( $\chi^2 = 41.69$ ,  $df = 3$ ,  $p < .001$ ).

Based on information in the participants' DCFS case records, we coded the maltreatment experiences of these youth (for a detailed report, see Trickett et al., 2009). Of the maltreated sample, 72% of youth had been neglected, 23% had been sexually abused, 50% had been physically abused, and 50% had been emotionally abused. Forty-seven percent of the maltreated youth had experienced multiple forms of abuse.

### Procedures

All procedures were conducted with the approval of the university IRBs. As part of a larger protocol, we measured SCL, heart rate, and respiration during a baseline period and while participants viewed video clips depicting intense parent-child conflict. The clips appeared in the following sequence; 3 min of baseline, two non-conflict clips (3 min each) and four conflict clips (90 s to 3 min each). All participants viewed the clips in the same order. We examined second minute of the baseline because it allowed 1 min for these measures to stabilize, and because during the third minute of baseline, we measured blood pressure (not examined here), which affected some participants' SCL. Video clips were a mixture of nationally known, studio-created films and films created by students at the University of Southern California. A bioamplifier (James Long Company, Caroga Lake, NY) recorded psychophysiological activity continuously while participants viewed the videos.

After consent procedures, we led the adolescent into a room with the bioamplifier, a TV monitor and DVD player. The adolescent self-applied three disposable ECG electrodes, two axillary (one per side) and one as a ground just above the navel. To collect SCL, we attached Ag/AgCl electrodes, filled with isotonic citrate salt electrode gel with gel contact area limited to a 1 cm diameter circle by double-sided adhesive collars, to the volar surfaces of the distal phalanges of the participant's non dominant hand. We placed a respiration bellows around the participant's torso.

### Measures

Aggression was measured using parent/guardian report on the 23-item Reactive-Proactive Aggression Questionnaire (Raine, et al., 2006). The proactive subscale includes



12 items (e.g., “fight others to show who is on top,” “damage or break things for fun”). The reactive subscale includes 11 items (e.g., “yell at others when they annoy me,” “damage things when I am mad”). Items have three response choices: never (0), sometimes (1) or often (2) and measure a mix of physical and verbal aggression, hostility and anger. Raine et al. (2006) report alpha coefficients of .81–.87 for the subscales, and .89–.91 for the whole scale. Subscales were highly correlated,  $r = .71$ ; we therefore analyzed only the Total Aggression scale ( $\alpha = .91$ ).

SNS activity was indicated by SCL baseline and reactivity (SCL and SCL-R). To collect tonic SCL, the bioamplifier used a 500 mV, 30 Hz sinusoidal excitation waveform. The bioamplifier yielded a skin conductance level output of 10  $\mu\text{S}/\text{V}$ . The A/D converter had a 16-bit resolution and a  $\pm 2.5\text{V}$  input range. Data were digitized at 1 kHz. We measured SCL-R to the conflict clips as average SCL during presentation of the first two conflict clips minus the average SCL during the second minute of baseline.

PNS-linked cardiac activity was measured by RSA measured during second minute of baseline. ECG data were sampled and digitized at 1 kHz. R-wave times were extracted from the ECG channel and edited manually via ECGRWAVE software (James Long Company, Caroga Lake, NY, USA). The strain gauge respiration bellows connected to the bioamplifier for transduction, amplification, and digitization. The RSA program (James Long Company) calculated RSA as the difference between the minimum interbeat interval (IBI) during inspiration and the maximum IBI during expiration (in seconds). This program computes the difference in IBI twice for each respiration cycle, once for inspiration and once for expiration, assigning times of inspiration and expiration as the midpoints of each and calculating the arrhythmia. Grossman, van Beek, and Wientjes (1990) found the peak-to-valley method to be comparable to spectral analysis. We measured RSA reactivity (RSA-R) to the conflict clips as average RSA

during presentation of the first two conflict clips minus the average RSA during second minute of baseline.

## Results

### *Descriptive Information and Effects of Maltreatment on Aggression, RSA and SCL*

Descriptive data for all study variables by maltreatment status appear in Table 1. Because baseline RSA and SCL were substantially skewed, we log transformed these data for all analyses, though Table 1 displays raw means for interpretability. Two-way group (maltreatment vs. comparison) by sex ANCOVAs examined the effects of maltreatment status and sex on aggression, baseline RSA, RSA-R, baseline SCL, and SCL-R, controlling for age, parent’s education, income, and ethnicity (African-American vs. Caucasian and Latino) due to lower SCL reported among African-American samples (Anderson & McNeilly, 1991). SCL-R analyses controlled for baseline SCL, and RSA-R analyses controlled for baseline RSA. A significant main effect of maltreatment emerged on aggression, such that maltreated youth had higher aggression scores than did comparison youth. No sex or maltreatment  $\times$  sex interaction effect was significant on aggression. A main effect of sex emerged on baseline RSA, such that girls’ baseline RSA was higher than was boys’ RSA. Girls also had higher SCL-R than did boys. Repeated measures ANCOVAs controlling for child age revealed that among both boys and girls, SCL increased from baseline to the conflict clips ( $p < .05$ ) but RSA did not increase.

### *Correlations among ANS Variables and Aggression*

Correlations (see Table 2) examined relations among aggression, baseline RSA, RSA-R, baseline SCL, and SCL-R. Correlations between baseline physiological measures and aggression are bivariate, as are correlations between baseline RSA and RSA-R and between baseline SCL and SCL-R.

**Table 1.** Means and SDs of Study Variables by Gender and Maltreatment Status

	Maltreated		Comparison		<i>F</i> (maltreatment)	<i>F</i> (sex)	<i>F</i> (maltreatment $\times$ sex)
	Males ( $n = 113$ )	Females ( $n = 121$ )	Males ( $n = 74$ )	Females ( $n = 54$ )			
Aggression	10.81 (8.43)	10.76 (8.36)	8.65 (5.40)	5.82 (4.77)	14.59**	3.02	3.75
Baseline RSA (s)	.10 (.06)	.11 (.10)	.10 (.09)	.15 (.34)	.07	5.10*	.00
Baseline SCL ( $\mu\text{s}$ )	13.30 (6.21)	13.12 (6.15)	14.2 (6.6)	13.27 (6.04)	.29	.41	.13
RSA reactivity (s)	.03 (.33)	.28 (3.03)	-.01 (.09)	-.01 (.09)	.14 <sup>a</sup>	.47 <sup>a</sup>	.15 <sup>a</sup>
SCL reactivity ( $\mu\text{s}$ )	3.53 (3.81)	1.94 (2.79)	3.46 (4.15)	2.39 (3.13)	.16 <sup>a</sup>	12.49 <sup>a**</sup>	1.77 <sup>a</sup>

Note: Except where indicated with superscript, degrees of freedom for *F*-statistics are (1, 355). SCL = Skin conductance level. RSA = Respiratory sinus arrhythmia.

<sup>a</sup>*df* = (1, 353).

\* $p < .05$ . \*\* $p < .01$ .

**Table II.** Intercorrelations among aggression and psychophysiological measures ( $n = 187$  boys and 175 girls)

	1	2	3	4	5
1. Total aggression	–	–.14	–.14	–.08	.04
2. Baseline RSA	–.19*	–	–.03	–.01	–.19*
3. Baseline SCL	.03	.00	–	–.15	.29**
4. RSA Reactivity	–.04	–.11	–.02	–	.08
5. SCL-Reactivity	.09	–.15*	–.00	–.00	–

Note: Boys' correlations appear below the diagonal. Girls' correlations appear above the diagonal. RSA = Respiratory sinus arrhythmia. SCL = Skin conductance level. With the exception of analyses of correlations between baseline RSA and RSA reactivity and baseline SCL and SCL reactivity, analyses involving SCL Reactivity control for baseline SCL, and analyses involving RSA reactivity control for baseline RSA.

\* $p < .05$ . \*\* $p < .01$ .

**Table III.** Regression equations accounting for aggression from maltreatment, RSA, SCL-R, and their interactions ( $n = 187$  boys and 175 girls)

	Boys				Girls			
	$\beta$	$t$	$F$	$R^2$	$\beta$	$t$	$F$	$R^2$
Baseline RSA and Baseline SCL								
Total Equation <sup>a</sup>			2.60	.10			4.26**	.15
Maltreatment	.14	1.80			.31	3.90**		
Baseline RSA	–.21	–2.87**			–.11	–1.50		
Baseline SCL	.04	.47			–.09	–1.12		
Maltreatment $\times$ Baseline RSA	–.19	–2.47*			–.13	–1.75		
Maltreatment $\times$ Baseline SCL	.05	.65			–.07	–.92		
RSA Baseline $\times$ Baseline SCL	.08	.98			.08	1.12		
Maltreatment $\times$ Baseline RSA $\times$ Baseline SCL	.01	.18			.08	1.13		
Baseline RSA and SCL Reactivity								
Total equation <sup>b</sup>			2.63*	.11			4.59**	.18
Baseline SCL	.03	.43			–.19	–2.41*		
Maltreatment	.14	1.75			.32	4.07**		
Baseline RSA	–.19	–2.57*			–.11	–1.49		
SCL Reactivity	.05	.56			.15	1.93		
Maltreatment $\times$ Baseline RSA	–.16	–2.11*			–.18	–2.40*		
Maltreatment $\times$ SCL Reactivity	.14	1.74			.02	.24		
Baseline RSA $\times$ SCL Reactivity	–.04	–.54			–.01	–.14		
Maltreatment $\times$ Baseline RSA $\times$ SCL Reactivity	–.10	–1.19			.17	2.26*		

Note: RSA = Respiratory sinus arrhythmia. SCL = Skin conductance level.

\* $p < .05$ . \*\* $p < .01$ .

<sup>a</sup>For  $F$ -tests,  $df = 7,170$  boys and 7,159 for girls

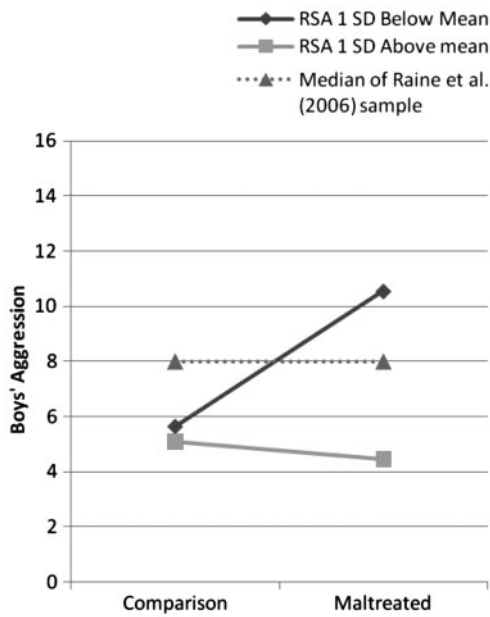
<sup>b</sup>For  $F$ -tests,  $df = 8,169$  boys and 8,156 girls.

All other correlations involving RSA-R control for baseline RSA, and all other correlations involving SCL-R control for baseline SCL. Among boys, baseline RSA was negatively correlated with aggression. Among girls, RSA and SCL measures were not significantly related to aggression. Baseline RSA and SCL-R were negatively correlated with each other among both boys and girls. Among girls, baseline SCL was positively correlated with SCL-R.

### Regression analyses Predicting Aggression

We examined the effects of maltreatment status ( $-1 =$  comparison,  $+1 =$  maltreated), baseline SCL, SCL-R, and baseline RSA in explaining variance in aggression.

We conducted two equations. One examined maltreatment, baseline RSA, baseline SCL, and their two and three way interactions. The other equation examined maltreatment, baseline RSA, SCL-R, and their two and three way interactions. We were unable to examine equations involving RSA reactivity and its interactions with maltreatment and SCL due to high multicollinearity between RSA reactivity and its interactions with maltreatment. We centered all main effect predictors and multiplied centered scores to calculate interaction effects. We conducted analyses separately for boys versus girls. We entered all predictors simultaneously. Regression analyses, which appear in Table 3, controlled for age, ethnicity ( $-1 =$  Caucasian/



**Figure 1.** Plot of the two-way interaction effect between maltreatment and baseline RSA in accounting for boys' aggression.

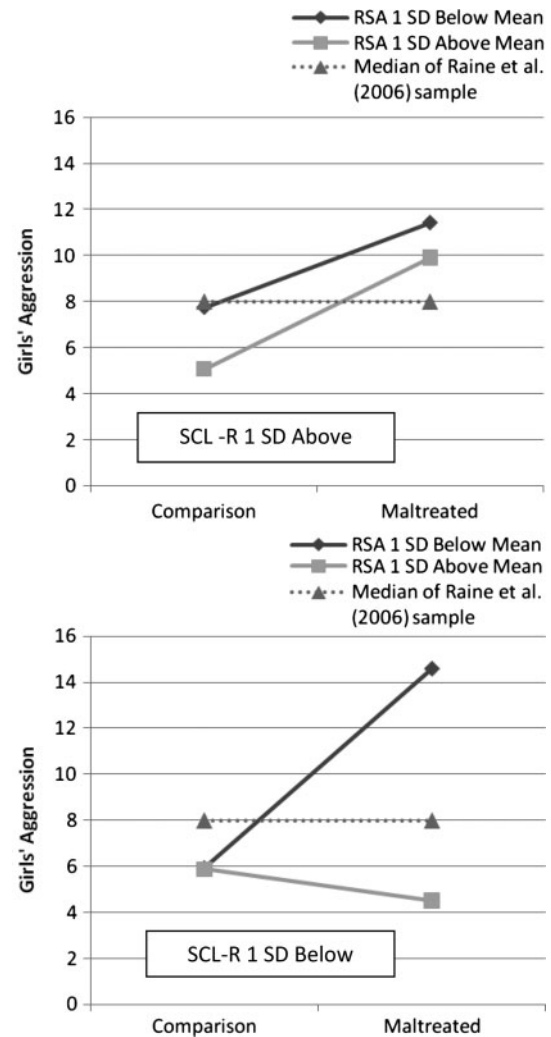
Latino/a, +1 = African-American), parent/guardian education, and income. Covariates were non-significant in accounting for boys' and girls' aggression. Equations examining interactions with SCL-R also included baseline SCL entered simultaneously.

### Boys' Aggression

The first model examined the main and interactive effects of maltreatment, baseline RSA and baseline SCL on aggression. The overall equation was not significant, and thus individual predictors will not be discussed. The second model tested main and interactive effects of maltreatment, baseline RSA and SCL-R. The total equation was significant, accounting for 11% of the variability in aggression. Baseline RSA was negatively related to aggression, and the two-way interaction between RSA and maltreatment was significant. We probed this interaction according to procedures described by Aiken and West (1991). Figure 1 displays the relation between maltreatment and aggression at values of RSA 1 SD above and below the mean. At values of RSA 1 SD below the mean, the relation between maltreatment and aggression was significant and positive ( $b = 2.45$ ,  $t = 4.47$ ,  $p < .001$ ); at values of RSA 1 SD above the mean, the relation was null ( $b = -.32$ ,  $t = -.36$ ,  $p = .72$ ).

### Girls' Aggression

The first model examined maltreatment, baseline RSA, baseline SCL, and their interactions. The overall  $F$ -value



**Figure 2.** Plots of the three-way interaction effect between maltreatment, baseline RSA, and SCL reactivity in accounting for girls' aggression.

was significant, with predictors accounting for 15% of the variance in aggression. The only significant predictor was maltreatment. The second equation examined maltreatment, baseline RSA, SCL-R and their interactions and also included baseline SCL. The total equation was significant, accounting for 18% of the variance. The main effect of maltreatment, the two-way maltreatment  $\times$  RSA interaction, and the three-way maltreatment  $\times$  baseline RSA  $\times$  SCL-R interaction were significant. We probed the three-way interaction as the highest order effect. Tests of simple slopes (see Figure 2) reveal that at values of baseline RSA 1 SD below the mean, the relation between maltreatment and aggression was significant at values of SCL-R 1 SD below the mean ( $b = 5.76$ ,  $t = 3.78$ ,  $p < .001$ ), but at values of baseline RSA 1 SD below the mean and SCL-R 1 SD above the mean, the relation between maltreatment and

aggression was null ( $b = 2.46, t = 1.91, p = .058$ ). In addition, at baseline RSA 1 *SD* above the mean and SCL-R 1 *SD* above the mean, the relation between maltreatment and aggression was also significant ( $b = 3.22, t = 2.29, p = .024$ ), but at values of baseline RSA 1 *SD* above the mean and SCL-R 1 *SD* below the mean the relation between maltreatment and aggression was not significant ( $b = -.90, t = -.67, p = .50$ ).

## Discussion

The present data are consistent with ANS measures as moderators of the effects of maltreatment on youth aggression. Among boys, maltreatment positively related to aggression at lower but not higher values of RSA. Among girls, the moderating effect of baseline RSA on the link between maltreatment and aggression was further moderated by SCL reactivity. The relation between maltreatment and aggression was significant at relatively low RSA only when SCL reactivity was also relatively low. The relation between maltreatment and aggression was also significant at relatively high baseline RSA when SCL reactivity was also relatively high. In other words, at values of both baseline RSA and SCL reactivity being relatively low or relatively high, the relation between maltreatment and aggression was positive and significant. At values of low baseline RSA and high SCL reactivity or high baseline RSA and low SCL reactivity, the relation between maltreatment and aggression was not significant.

Overall, the data from this study suggest that among boys, high RSA may be protective against the effects of maltreatment on aggression. These data are consistent with other studies on the buffering effects of high RSA on psychosocial stress variables. For example, Katz and Gottman (1995) and El-Sheikh et al. (2001), found that high vagal tone buffers against the negative effects of marital conflict on the development of behavior problems. Our findings are also consistent with work by El-Sheikh (2005), who reports that high RSA buffers the effect of parent drinking on children's behavior problems. We did not find three-way interactions between RSA, SCL, and maltreatment among boys. The lack of a significant three-way interaction differs from findings reported by El-Sheikh et al. (2009), though their studies examined boys and girls together and marital conflict rather than child maltreatment.

Among girls, the significant three-way interaction suggests that the moderating effect of RSA was further moderated by SCL reactivity. Low RSA exacerbated the effect of maltreatment on aggression only at low values of SCL

reactivity. At low RSA and high SCL reactivity, the link between maltreatment and aggression was non-significant. Low RSA and SCL reactivity may represent a vulnerability to the effects of maltreatment on aggression, and high RSA and SCL reactivity may also carry some risk. These results are consistent with findings reported by El-Sheikh et al. (2009) and can be understood in terms of the theory of autonomic space (Berntson et al., 1991). Low baseline RSA combined with low SCL reactivity may reflect a lack of engagement of appropriate stress response and emotional regulation systems and may increase the negative effects of child maltreatment. The pattern of relatively high SCL reactivity combined with high baseline RSA may also reflect problematic or contradictory patterning of these systems (El-Sheikh et al., 2009) consisting of high reactivity to negative environmental experiences and thus a stronger link between maltreatment and aggression. In contrast, high baseline RSA coupled with low SCL reactivity and low baseline RSA coupled with high SCL reactivity may buffer against the effects of maltreatment on aggression in girls. El-Sheikh et al. interpret this combination as reciprocal SNS or PNS activation that suggests appropriate coordination between SNS and PNS. However, as we were unable to examine interactions with RSA reactivity due to multicollinearity issues, the terms "co-activation" and "co-inhibition" do not precisely apply to our models, as RSA is at baseline and SCL is in response to the videos. Moreover, we measure SNS and PNS effects on different organ systems.

Our findings are consistent with previous findings regarding sex effects in relations between SCL and RSA with aggression. The protective effect of RSA on boys' aggression is consistent with much of the literature (Beauchaine et al., 2008). Among girls, the link between high SCL reactivity in the context of maltreatment and high RSA is consistent with results of Beauchaine et al. (2008), who found higher electrodermal activity to relate to girls' aggression.

We found no main effect for maltreatment on any SNS or PNS indicators. We were surprised that maltreated youth did not have larger SCL responses to the videos, which would have suggested sensitization to conflict stimuli. One possible explanation is that the videos depicted milder conflict than those experienced by the maltreated youth. Another possibility is that the clips depicted conflict more intense than the comparison youths' experiences, causing a stronger reaction among some of these participants and washing out group effects. Youth in both groups did demonstrate increased SCL from baseline to SCL during the conflict clips.



SCL reactivity did not relate to aggressive behavior as a main effect or in two-way interactions with maltreatment. This result is inconsistent with findings by Erath et al. (2009), who found that low SCL reactivity exacerbated the link between harsh parenting and children's externalizing behaviors. One possible reason for the discrepancy is participants' developmental stage. The relation between aggression and SCL reactivity is unclear among adolescents (Lorber, 2004). Erath et al. (2009) studied a younger sample than the present study. In addition, although SCL reactivity was not significant as a main effect, it interacted with maltreatment and baseline RSA to account for aggressive behavior in girls. RSA reactivity did not relate to aggressive behavior. Some researchers have found higher vagal suppression to be protective against behavior problems (El-Sheikh & Whitson, 2006), whereas others have found higher change in RSA to challenge to be protective (Hastings et al., 2008).

Our study has limitations. Our maltreatment classification is coarse. Further work should examine particular types of maltreatment. We targeted youth from certain ethnic backgrounds due to aims of the larger study and thus cannot generalize to other ethnicities. The cross-sectional design precludes knowing whether the maltreatment truly preceded the aggression. We cannot deduce causality as we cannot manipulate maltreatment experimentally. We were not able to examine interaction effects with RSA reactivity due to multicollinearity. Thus, the significant interactions involved baseline RSA and SCL reactivity, and we have limited information regarding coordination in the two systems to the same events. Moreover, because an electrodermal measure indexed SNS, and a cardiac measure indexed PNS, we did not study co-activation and co-inhibition according to autonomic balance theory as conceptualized by (Berntson et al., 1991), because they refer to organs dually innervated by SNS and PNS (Beauchaine, 2009). Our electrophysiological measures also do not purely index SNS and PNS; they are also influenced by other factors. Participants also may vary in their emotional reactions to the videos. In addition, baselines may have been too short for reliable assessments of RSA and SCL. The physiological measures also have large variability that may not be captured by the means. Some of the effect sizes are small, and these factors may have contributed.

Despite these limitations, this study contributes to knowledge about moderators of the effect of maltreatment on youth aggression. The multi-systems approach sheds light on patterns of PNS and SNS activity that may buffer or exacerbate effects of maltreatment. Understanding the role of these physiological systems in the effects of

maltreatment may help identify youth who are particularly at risk. Research regarding how to maximize adaptive function of these systems may help protect against the negative effects of maltreatment.

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