

Victimization and Biological Stress Responses in Urban Adolescents: Emotion Regulation as a Moderator

Wendy Kliewer¹

Received: 19 October 2015 / Accepted: 7 December 2015 / Published online: 16 December 2015
© Springer Science+Business Media New York 2015

Abstract Associations between urban adolescents' victimization experiences and biological stress responses were examined, as well as emotion regulation as a moderator of these associations. Data from a 4-wave longitudinal study with a low-income, community-based sample ($n = 242$; 91 % African American; 57 % female; $M = 11.98$, $SD = 1.56$ years at baseline) revealed that victimization, assessed over 3 study waves, was associated with an attenuated cortisol response to a stress interview at the final study wave, indicating that responses of the Hypothalamus–Pituitary–Adrenal (HPA) axis were dysregulated. Cortisol responses were moderated by caregiver-reported adolescent emotion regulation, suggesting that this modifiable protective factor that is taught in many school-based prevention programs could help reduce harm associated with HPA axis dysregulation linked to victimization.

Keywords Victimization · Cortisol · Alpha amylase · Emotion regulation · Adolescents

Introduction

Numerous studies have linked victimization experiences to dysregulated hypothalamic–pituitary–adrenal (HPA) axis responses (Aiyer et al. 2014; Alink et al. 2012; Griffin et al. 2005; Kim et al. 2015; Negriff et al. 2015). Studies also have examined social moderators (e.g., support) of the

relationships between exposure to violence and HPA axis responses (Aiyer et al. 2014). These and other studies are useful in that they can help us understand why, how, and for whom exposure to risks are related to later health problems given that HPA axis dysregulation is linked to a range of poor mental and physical health outcomes (Cohen et al. 2000; Herbert 2013).

The vast majority of the extant literature on exposure to violence or conflict and biological stress responses, however, has focused on the HPA axis. Victimization experiences, however, may activate more than one biological system. Victimization experiences may activate the HPA axis because of their socially evaluative qualities (Dickerson and Kemeny 2004) as well as the sympathetic branch (SNS) of the autonomic nervous system (ANS) due to evoking a “fight or flight” response (Berntson et al. 1991). The present study builds on the body of literature to date by examining associations between adolescents' experiences of victimization and physiological stress responses in two biological systems—the HPA axis and the ANS. Additionally, when moderators have been examined, they have tended to be social moderators such as social support or relationship quality. The current study examines the role of emotion regulation—a modifiable skill taught in many school-based prevention programs—as a potential protective factor in the association between victimization experiences and biological stress responses.

Psychobiology of the Stress Response

Historically, research to elucidate the mechanisms linking stress and disease has focused on the HPA axis and the SNS branch of the ANS (Rohleder 2014). When activated, the HPA axis triggers the release of glucocorticoids, including cortisol, and the SNS releases catecholamines

✉ Wendy Kliewer
wkliewer@vcu.edu

¹ Department of Psychology, Virginia Commonwealth University, PO Box 842018, Richmond, VA 23284-2018, USA

into circulation. Within the ANS, the SNS and parasympathetic nervous system (PNS) work in concert to regulate physiological arousal to stressors (Lucas-Thompson and Granger 2014). The ANS is responsible for “fight or flight” reactions and responds more quickly to acute stressors than the HPA axis. While short-term activation of these systems is adaptive and promotes allostasis, or the dynamic interplay of physiological systems focused on adjusting the body’s operating range in response to physical, psychological, and environmental demands, prolonged and/or repetitive stress results in dysregulation of these systems through disruption of diurnal patterns and inefficient turning on and off of glucocorticoids and catecholamines, thus contributing to allostatic load and increased disease risk (McEwen and Seeman 2003). In this study, HPA axis activation was indexed by cortisol; SNS functioning was indexed by salivary α -amylase (sAA). Salivary alpha amylase (sAA) is a sensitive, reliable and valid biomarker of stress-related changes in the sympathetic nervous system (Nater and Rohleder 2009).

Exposure to Violence, Cortisol, and Salivary Alpha Amylase Responses

A number of studies over the past decade have reported data linking adolescents’ direct or indirect experiences with violence to a blunted cortisol response (e.g., Aiyer et al. 2014; Alink et al. 2012; Dulin-Keita et al. 2010; Kliewer 2006; Negriff et al. 2015; Peckins et al. 2012). For example, Aiyer and colleagues followed 266 high-risk youth (80 % African American) over middle and late adolescence. Cumulative exposure to direct and indirect community violence, assessed over 7 waves of data collection, was associated with an attenuated cortisol response in early adulthood, assessed with three saliva samples collected during the young adult interview at wave 7. This response was most pronounced in males. Peckins et al. (2012) also examined both direct and indirect exposure to community violence, but in association with cortisol reactivity to a laboratory task among 124 youth (90 % white, non Hispanic) recruited from a rural community. Exposure to violence predicted cortisol reactivity in males (but not females) longitudinally, even after accounting for prior exposure to violence and mental health symptoms. In a longitudinal study of 454 youth aged 9–13 at baseline, Negriff et al. (2015) found that child maltreatment status was associated with an attenuated cortisol response to the Trier Social Stressor Test for Children (TSST-C) for males, but not for females. Overall, these and other studies reveal a tendency for victimized youth to show a blunted cortisol response, perhaps as an adaptation to chronic stress and hypersecretion of cortisol over a period of time (Blair 2010; Trickett et al. 2010).

In contrast to studies examining linkages between exposure to violence and cortisol, research linking adolescents’ direct and indirect exposure to violence and ANS responses is less common. Gordis et al. (2010) studied family members’ biological stress responses to a family conflict discussion task. Adolescents from families with a history of interparental aggression showed a reactive response to the task, indicated by elevations in sAA, compared with adolescents from families with no family history of interparental aggression. In another study of responses to marital conflict, Lucas-Thompson and Granger (2014) also found that youth exposed to high levels of interparental conflict showed high levels of sAA reactivity to a stress task. Based on these data, it might be reasonable to hypothesize a positive association between youth’s victimization experiences and their sAA responses.

Emotion Regulation

Thompson (1994) defined emotion regulation as “...the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals” (pp. 27–28). Shields and Cicchetti (1997) emphasized the importance of socially appropriate emotional displays as well as the adaptive nature of regulation in their assessment instrument. One way to conceptualize emotion regulation skill is as an asset, a perspective consonant with a focus on resources that can buffer youth from experiencing the full extent of the risks to which they are exposed. Several studies have demonstrated the buffering effects of emotion regulation on the relationship between exposure to community violence and various adjustment outcomes, including aggressive behavior and internalizing symptoms (Kliewer et al. 2004; Shahinfar 1998). For example, Shahinfar found, in a study of 155 African American preschool children enrolled in Head Start, that child emotion regulation skill moderated the effect of children’s victimization by community violence and externalizing problems. Kliewer et al. (2004), in a short-term longitudinal study with 101 low-income African American families, found that caregivers’ ratings of youth emotion regulation skill attenuated the association between exposure to community violence and youth- and caregiver-rated internalizing symptoms, although emotion regulation was not protective at the highest levels of exposure to violence. Researchers, however, have not extended this work to health outcomes. That is, researchers have not investigated the extent to which emotion regulation skill buffers adolescent victimization experiences on health outcomes or physiological stress responses such as dysregulated cortisol that predict future health. However, a related construct termed shift-and-persist (Chen 2012;

Chen and Miller 2012), which has been conceptualized as a form of emotion regulation, has health benefits for children low in socio-economic status (Chen et al. 2011; Kalleem et al. 2013). Thus, a second aim of the present study was to investigate emotion regulation—a modifiable skill taught in many school-based prevention programs—as a potential protective factor in the association between victimization experiences and biological stress responses.

Hypotheses

The sample focused on youth recruited from neighborhoods with moderate to high levels of violence, based on police statistics and census data. Families were followed annually for four waves; data were collected via in-home interviews conducted with youth and their maternal caregivers. It was hypothesized, based on prior literature (Aiyer et al. 2014; Kliewer 2006), that victimization experiences assessed over the first three waves of the study would be associated with a hyporesponsive cortisol pattern and with an amplified salivary alpha amylase response at Wave 4 of the study. That is, it was expected that as youth's victimization experiences increased their cortisol responses would decrease, and their salivary alpha amylase responses would increase. It also was hypothesized that good emotion regulation skills would buffer the relationship between victimization experiences and biological stress responses. Specifically, it was predicted that the relationship between victimization and cortisol would be less attenuated for youth with good emotion regulation skills compared to youth with poor emotion regulation skills, and the relationship between victimization and salivary alpha amylase would be diminished for youth with good emotion regulation skills compared to youth with poor emotion regulation skills.

Methods

Participants

Youth and their maternal caregivers ($N = 358$ families) were recruited for a 4-wave longitudinal study of the consequences of exposure to violence. The analytic sample for the present study included 242 urban adolescents who had biological data at Wave 4. Youth were either in the 5th or 8th grade at baseline ($M = 11.98$ years, $SD = 1.56$, range = 9–16 years; 43 % male; 91.3 % African American/black; 3.4 % white; 2.5 % American Indian; 0.6 % Asian; 2.2 % multiracial). Most caregivers were the youth's biological mother (88.0 %), but the sample also included grandmothers (6.2 %), adopted mothers (2.1 %),

stepmothers (0.8 %), and other female relatives (2.9 %). Many caregivers had never married (41.3 %) although a variety of family structures were represented in the sample including families who were married/cohabitating (32.2 %), separated (14.9 %), divorced (9.5 %), or widowed (2.1 %). Caregiver education also was diverse. A quarter of the sample had not completed high school (26.1 %), another quarter had completed high school or had earned a General Education Diploma (GED) (24.9 %), a third quarter had completed some college but had not earned a degree (24.1 %), 5.8 % had earned an Associate's degree, 9.1 % had earned a Vocational degree, 8.3 % had earned a bachelor's degree, and 1.6 % had earned advanced degrees. Median household income was between \$301–400 a week at baseline, and 54 % of the sample had household incomes below the poverty line based on Federal guidelines.

Measures

Victimization

At waves 1, 2 and 3, youth reported on their past year victimization experiences using the *Survey of Children's Exposure to Violence* (Richters and Saltzman 1990). Youth indicated on a scale from 0 (*never*) to 4 (*almost every day*) how often they had experienced 10 types of victimization ranging from being slapped, hit, or punched by someone to being shot. Items began with the stem, "How many times in the past year have you yourself been ..." All items used in the study are listed on Table 2. Correlations across the 3 waves ranged from .47 to .56, $ps < .001$. At baseline our assessment included follow-up questions asking who perpetrated the victimization for five of the ten items (see Table 3). These follow-up questions were part of the original Richters and Saltzman measure. A recent meta-analysis reported that this measure is the most frequently used tool to assess youth's exposure to community violence, and it appears to have good predictive validity (Fowler et al. 2009).

Emotion Regulation

The 8-item emotion regulation subscale from the Emotion Regulation Checklist (ERC; Shields and Cicchetti 1997), reported by caregivers at Waves 1, 2, and 3, was used to assess youth's emotion regulatory skills. The emotion regulation subscale consists of items tapping adaptive regulation, including equanimity, emotion understanding, and empathy, such as "Is empathic towards others; shows concern when others are upset or distressed." Each item on the ERC is rated on a four-point Likert scale from (1) *never* to (4) *always*. The ERC has excellent reliability and validity. Cronbach alphas in the present study were .70,

.72, and .69 at Waves 1–3, respectively. Assessments at these three waves were correlated .55 to .59 ($ps < .001$) and were combined to form an aggregate index of emotion regulation skill.

Biological Stress Responses

Cortisol and salivary alpha amylase (sAA) in response to a stress task, the *Social Competence Interview* (SCI; Ewart et al. 2002) was assessed at Wave 4. The SCI is a 15–20 min audiotaped interview designed to promote physiological arousal and has been correlated repeatedly with changes in blood pressure and heart rate (Chen et al. 2002). The SCI has two phases: a hot phase and a cool phase. During the hot phase, the interviewer asks the adolescent to re-experience a stressful event and asks questions about the participant's thoughts and feelings during the event. The cool phase follows with the interviewer asking the participant to describe how the situation would have ideally ended and what could be done to achieve that outcome. Saliva samples were collected from the adolescents directly before the start of the SCI (2 samples), at the end of the "hot" phase, and 10, 10, and 20 min later, yielding 6 samples. Participants were instructed not to exercise, eat, or drink caffeinated beverages 2 h prior to the SCI. Saliva was collected using salivettes. Adolescents were asked by the interviewer to place a cotton swab in their mouth and chew for about 1 min. The adolescent spit the swab into the salivette tube and the samples were frozen at a 270 °C or below until the samples were taken to the laboratory for analysis. The saliva samples were assayed at the [location removed for blind review] for using enzyme immunoassays specifically designed for saliva analysis. Saliva samples were thawed, spun and refrozen for batch processing. On the day of the assay, samples were thawed and assayed directly with no further centrifugation. The assay for sAA employed a chromagenic substrate, 2-chloro-*p*-nitrophenol, linked to maltotriose. The enzymatic action of sAA on this substrate yields 2-chloro-*p*-nitrophenol, which can be spectrophotometrically measured at 405 nm using a standard laboratory plate reader. The amount of sAA activity present in the sample is directly proportional to the increase (over a 2-min period) in absorbance at 405 nm. Results are computed in units/milliliter of sAA using the following formula: (absorbance difference per minute \times total assay volume [328 ml] \times dilution factor [200])/(millimolar absorptivity of 2-chloro-*p*-nitrophenol [12.9] \times sample volume [0.008 ml] \times light path [0.97]). The assay for cortisol used 25 μ l of saliva, which has a lower sensitivity of .007 μ g/dl. Staff determined method accuracy by spike recovery, and staff determined linearity by serial dilution of 105–95 %. Following Granger et al. (2007), all samples

were assayed in singlet. Area under the curve with respect to ground (AUC_G), which computes total output, was calculated for the cortisol and sAA responses using Pruessner et al.'s (2003) formula.

Control Variables

Adolescent age, adolescent sex, interview start time, and pubertal status at baseline were included as control variables as these variables are known correlates of either cortisol or alpha amylase. The Pubertal Development Scale developed by Petersen et al. (1988) was used to measure pubertal status. This 5-item non-verbal assessment of pubertal status requires an adolescent to answer questions pertaining to the degree of his or her own pubertal status. Regardless of sex, all adolescents are asked to answer items on growth spurt, pubic hair, and skin change. Boys have additional questions about facial hair and girls have additional questions about menarche and breast development. The four-item response scale provides responses that allow the adolescent to tell where they are in pubertal development: 1 (has not yet begun), 2 (has barely started), 3 (is definitely underway), and 4 (growth or development is complete). The reliability of the items ranges from .68 to .83 (Petersen et al. 1988). Cronbach alpha in the current study was .84 for males and .74 for females. Adolescent age and sex were used as controls for the predictor, victimization, as well as the outcomes, biological stress responses.

Procedure

The Virginia Commonwealth University Institutional Review Board approved the project. Participants were recruited from Richmond, VA, a community with a large population of African-American adolescents. U.S. Census data (2000) indicated that 61 % of 15 to 24-year-olds in Richmond were African-American, and 61 % of children lived in neighborhoods classified as high in poverty (Kids Count 2004). Participants were recruited from neighborhoods in the Richmond area that had high levels of violence and/or poverty based on police statistics and census data. Participants were recruited through community agencies and events, and by canvassing qualifying neighborhoods via flyers posted door-to-door. Respondents were screened for eligibility over the telephone. To be eligible, participants had to have a fifth or eighth grader living in the home, and an adult female caregiver needed to participate in the interview. Only English-speaking participants were recruited into the study. Consistent with studies using similar designs and populations, 63 % of eligible respondents chose to participate and were scheduled for interviews, which were conducted in participants' homes unless a family requested to be interviewed elsewhere.

Interviewers thoroughly reviewed the parental consent and adolescent assent forms with the family, and answered any questions. The caregiver received a copy of the signed consent form. After the maternal caregiver provided written consent, the caregiver and adolescent separated for the interviews. Assent was provided by the adolescent before initiating the interview. Participants agreed to participate in series of four annual interviews. A Certificate of Confidentiality was obtained from the National Institutes of Health (NIH) to protect families' responses since adolescents were reporting on illegal behavior (i.e., substance use) as part of the study. Information about discounted health insurance for children and other community resources were distributed to caregivers at the time of the interview. Face-to-face interviews using visual aids were used to collect the data, and all questions were read aloud, with the exception of a small portion of the adolescent interview. Adolescents who had passed a reading-screening test answered several (primarily sensitive) questions in a booklet without interviewer assistance. Tests for interviewer race and sex effects revealed no systematic biases, $ps > .10$. Interviews with the caregiver and adolescent lasted approximately 2.5 h and participants received \$50 in gift cards per family at each wave.

Analytic Strategy

Descriptive information on and correlations among the study variables is presented first. Next, structural equation modeling was utilized to test the primary study hypotheses using Mplus version 7.31 (Muthén and Muthén 2015), which allowed missing data to be handled with full information maximum likelihood (FIML). FIML uses all information in the data for analyses, allows for less biased estimates, and is an efficient missing data technique (Wang and Wang 2012). The fit for each model was assessed using the χ^2 value, the Comparative Fit Index (CFI), the Tucker Lewis Index (TLI), the Root Mean Square Error of Approximation (RMSEA), and the Standardized Root Mean Square Residual (SRMR). Values of 0.90 or above for the CFI (Bentler 1990) and 0.08 or below for the RMSEA (Browne and Cudeck 1993) indicated that the model adequately fit the data. Separate models were run for cortisol and sAA. Youth reports of victimization at Waves 1, 2, and 3 were used as manifest indicators of the latent construct victimization. Victimization was regressed on adolescent sex and age to control for these influences on the likelihood of exposure; the marker of biological stress response (cortisol or sAA) was regressed on adolescent sex and age, pubertal status at wave 1, and time of day of data collection to control for these influences on the biological stress response.

Hierarchical linear regression analyses were used to test the hypothesis that youth higher in caregiver-rated emotion regulation capacity would show a weaker association between victimization and biological stress responses than youth lower in caregiver-rated emotion regulation capacity. These models allowed for the full range of the emotion regulation variable to be modeled in the analyses. Regression models controlled for adolescent sex, age, pubertal status at wave 1, and time of day. A composite victimization score was computed from waves 1, 2, and 3 using parameter estimates from the structural equation model. This composite variable, as well as the composite measure of emotion regulation, was centered and a victimization \times emotion regulation interaction term was computed from the centered variables (Aiken and West 1991). Cook's Distance measure (Cook and Weisberg 1982) was examined to test for possible multivariate outliers that could be affecting the regression results. Interactions between victimization and adolescent sex also were computed to examine the possibility that the association between victimization and cortisol differed by adolescent sex. When significant, interactions were plotted to facilitate interpretation of the findings.

Results

Attrition Analyses

Youth who completed all four waves of interviews and who provided physiological data ($N = 242$) were compared with youth who provided data at earlier waves of the study ($N = 116$) on adolescent sex, baseline levels of victimization, and pubertal status using t -tests and χ^2 tests. There were no differences between youth who attrited versus youth who remained in the study on either baseline levels of victimization, $t(354) = 1.26$, $p > .10$ or pubertal status, $t(347) = 1.05$, $p > .10$. However, youth who attrited were more likely to be male, $\chi^2(1) = 4.35$, $p < .05$, Phi $\phi = .11$, which is considered a small effect. At Wave 1, 54 % of the sample was female; at Wave 4, 57 % of the sample who provided physiological data was female. (Five families completed Wave 4 interviews where the adolescent did not provide physiological data.)

Data Cleaning and Transformation

The SCI for each participant was reviewed prior to beginning analysis. Participants were excluded from analyses if the SCI was incomplete, if a stressful event was not recalled, if the participant was not engaged in the process based on the interviewer's impression, or if the times for the hot and cool phases of the SCI were anomalous.

Descriptive statistics for cortisol and sAA were calculated and outliers were truncated to 3 SD from the mean (Granger et al. 2012). AUC_{Ground} was calculated for cortisol and sAA; their distributions were skewed and kurtotic and therefore they were log transformed.

Descriptive Information and Correlations Among Study Variables

Descriptive information on and correlations among the study variables is presented in Table 1. Percentages of youth reporting any victimization at waves 1, 2, 3 for each of the 10 types of victimization assessed in the study are reported in Table 2. As seen in the table, being slapped, hit, or punched was endorsed most frequently, followed by being threatened with serious physical harm. Being attacked with a knife or shot with a gun were extremely rare. Table 3 presents information on the source of perpetration for five of the ten victimization events at baseline, the only wave for which these data were obtained. As seen in Table 3, over two-thirds of the slapping, hitting, and punching; over 80 % of the physical threats; nearly three-quarters of the beatings; and half to three-fourths of the victimization involving drugs was perpetrated by same-age mates that were friends, acquaintances, or strangers of the adolescent, with the majority of these events perpetrated by same age friends and acquaintances.

Associations Between Victimization and Biological Stress Responses

Outcome: Cortisol

Results of the structural equation model for cortisol revealed that the model was a good fit to the data ($N = 237$, $\chi^2(12) = 13.60$, $p > .05$; RMSEA = .024 CI [0, .073]; CFI = .994; TLI = .989; SRMR = .027). As hypothesized, victimization was negatively associated with total cortisol output ($b = -.15$, $p < .05$), after accounting for time of day of data collection, adolescent sex, and pubertal status at baseline, each of which were significant, and adolescent age, which was not significant. This model also accounted for effects of adolescent age and sex on victimization, both of which were significant. See Fig. 1 for a depiction of the findings.

Table 4 presents the hierarchical regression results of the moderator analysis. As seen in the table, this model was significant, accounting for 43.9 % of the variance in cortisol AUC_G, $F(7, 212) = 23.71$, $p < .001$. Inspection of outlier statistics revealed no multivariate outliers. After accounting for adolescent age, sex, pubertal status, and time of day of data collection, as well as the main effects of victimization and emotion regulation, there was a significant victimization \times emotion regulation interaction. This interaction is plotted in Fig. 2. As illustrated in the figure, there was a strong, negative association between

Table 1 Descriptive information on and correlations among the study variables

	1	2	3	4	5	6	7	8	9	10
1. Victimization (W1) ^a	–	.47***	.50***	-.17**	.09	.25***	0	-.05	-.04	.08
2. Victimization (W2) ^a		–	.56***	-.13*	.17**	.22**	.17**	-.04	-.04	-.05
3. Victimization (W3) ^a			–	-.19**	.11	.29***	.11	-.07	.02	-.02
4. Emotion regulation ^b				–	-.01	-.10	-.08	.01	-.01	-.05
5. Pubertal status (W1) ^a					–	.04	.55***	-.13*	.25***	0
6. Sex						–	-.03	-.04	.19**	.05
7. Age							–	-.16*	.15*	-.04
8. Time of day								–	-.60***	.09
9. Cortisol AUC _G (W4)									–	-.06
10. sAA AUC _G (W4)										–
<i>M</i>	1.53	1.71	1.54	75.46	11.04		11.98		.80	3.08
<i>SD</i>	1.77	2.15	1.98	9.42	3.47		1.56		.28	.37

Sex was coded 0 = female, 1 = male. Biological stress measures were log transformed. Emotion Regulation was a composite of reports across Waves 1–3

^a Reported by youth

^b Reported by caregiver

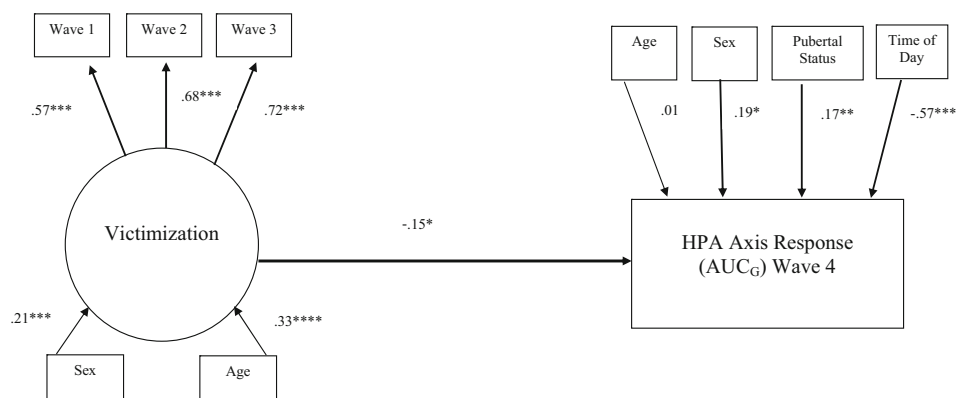
* $p < .05$; ** $p < .01$; *** $p < .001$

Table 2 List of forms of victimization assessed and percentage of youth reporting any exposure in the previous year

Item	Wave 1	Wave 2	Wave 3
<i>How many times have you yourself been ...</i>			
Chased by gangs or older kids?	15.9	12.7	7.3
Asked to get involved in any aspect of selling or distributing illegal drugs?	3.5	15.9	15.1
Asked to use illegal drugs?	7.2	18.4	18.1
At home when someone has broken into or tried to force their way into your home?	3.1	8.3	7.0
Threatened with serious physical harm by someone?	16.7	20.0	21.4
Slapped, punched, or hit by someone?	50.4	36.5	37.3
Beaten up or mugged?	8.3	15.0	7.7
Attacked or stabbed with a knife?	.01	1.9	2.2
Shot with a gun?	.004	1.6	1.1
In any kind of situation not already described where you were extremely frightened or thought that you would get hurt very badly or die?	15.3	18.7	13.5

Table 3 Percentage of perpetrators from different categories across differing types of victimization experiences at Wave 1

	Slapped, hit, punched	Physically threatened	Beaten up, mugged	Asked to use drugs	Asked to sell drugs
Young friends	29.6	26.3	31.6	43.8	75.0
Young acquaintances	29.6	39.5	15.8	6.3	
Young strangers	7.0	15.8	26.3		
Siblings	13.0		5.3	6.3	
Parents	12.2				
Other relatives	3.5	2.6		12.5	
Adult acquaintances	1.7	5.3			
Adult strangers		5.3	15.8		12.5
Adult friends	0.9				12.5
Don't know/missing	2.6	5.2	5.3	31.3	

**Fig. 1** Relationship between victimization and HPA axis response controlling for the contributions of youth sex and age on victimization, and the contributions of youth sex, age, pubertal status at Wave 1, and time of day of data collection on cortisol responses.

Standardized parameter estimates are presented in the figure. $N = 237$, $\chi^2(12) = 13.60$, $p > .05$; RMSEA = .024 CI [0, .073]; CFI = .994; TLI = .989; SRMR = .027

Table 4 Hierarchical linear regression predicting cortisol AUC_G at Wave 4 from victimization, emotion regulation, their interaction, and controls

Step		1	2	3
1	Sex	.14**	.17**	.16**
	Age	-.01	-.01	.01
	Pubertal status	.17**	.17**	.15**
	Time of day	-.57***	-.58***	-.59***
2	Victimization		-.12*	-.10
	Emotion regulation		0	.02
3	Victimization × emotion regulation			.11*
	Model <i>F</i>	37.90***	26.47***	23.71***
	Model <i>R</i> ²	.414***	.427***	.439***
	Change in <i>R</i> ²		.014	.012*

F(7, 212) = 23.71 at the final step of the model

* *p* < .05; ** *p* < .01; *** *p* < .001

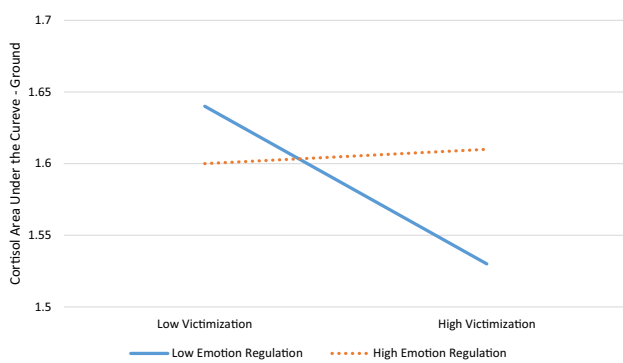


Fig. 2 Relationship between victimization and cortisol AUC_G at high and low levels of emotion regulation

victimization and cortisol when emotion regulation was low, and no association when emotion regulation was high.

Addition analyses were conducted to determine if there were victimization × sex or victimization × emotion regulation × sex interactions. There were none, indicating that the relationship between victimization and cortisol was operating similarly for both males and females, and that emotion regulation was having a similar protective effect on the relationship between victimization and cortisol for both males and females.

Outcome: Salivary Alpha Amylase

Results of the structural equation model for salivary alpha amylase revealed that the model was an adequate fit to the data (*N* = 237, χ^2 (12) = 15.79, *p* > .05; RMSEA = .037 CI [0, .080]; CFI = .974; TLI = .952; SRMR = .029). However, in contrast to the model with cortisol and contrary to hypotheses, victimization was not associated with salivary alpha amylase (*b* = -.01, *p* > .05). Additionally, none of the control variables were significantly associated

with sAA. The hierarchical regression analysis predicting sAA from victimization experiences, emotion regulation capacities, and their interaction was not significant, *F*(7, 207) = 0.60, *p* > .10.

Discussion

Numerous studies have linked victimization experiences to dysregulated hypothalamic–pituitary–adrenal (HPA) axis responses (Aiyer et al. 2014; Alink et al. 2012; Griffin et al. 2005; Kim et al. 2015; Negri et al. 2015), but largely have ignored responses in other biological systems. However, victimization experiences likely activate more than one biological stress response system, given the threats that they evoke and the potential to heighten concerns regarding social evaluation (Berntson et al. 1991; Dickerson and Kemeny 2004). Further, the protective factors that have been studied in relationship to victimization and stress responses tend to be social rather than skill-based. In response to these gaps in the literature, the present study examined prospective associations between adolescents’ victimization experiences, which were aggregated over three waves, and biological stress responses in the HPA axis and the autonomic nervous system (ANS), assessed at the fourth and final study wave. Additionally, caregiver-rated adolescent emotion regulation was examined as a protective factor in the relationship between victimization and biological stress responses.

Structural equation modeling revealed that youth’s victimization experiences were negatively associated with total cortisol output in response to a stress task. However, no such association was observed between victimization and salivary alpha amylase. Our findings contribute to a growing literature supporting the attenuation hypothesis (Trickett et al. 2010), which is particularly applicable for

youth experiencing chronic stress. The negative association between victimization and cortisol output observed in our data may reflect the down regulation of the HPA axis in an attempt to minimize the physiological damage caused by repeated arousal of the stress response system. As noted by Miller et al. (2007), this cycle of repeated activation followed by a blunting of the stress response may increase youth's vulnerability to mental as well as physical health problems. A comprehensive approach to health assessment is important, as some youth experiencing chronic stress show good adaptation in some areas, and poor functioning in other areas (c.f., Brody et al. 2013).

One reason the victimization reported by the youth in this study may be threatening and, therefore, lead to activation of the HPA axis, followed by downregulation, is that the majority of victimization acts reported by youth were perpetrated by same-age friends and acquaintances—individuals who likely are a part of the adolescents' social circles. Social comparison intensifies during adolescence, and ongoing brain development during this developmental period makes some adolescents especially sensitive to social evaluation and rejection (Somerville 2013). Further, the urban context places considerable importance on reputation and maintaining “respect” (Farrell et al. 2010; Jäggi and Kliwer 2015). The extent to which these victimization experiences made youth feel disrespected or feel that their standing in the community was diminished likely contributed to activation of the biological stress response system.

Several distinct areas for future research emerge from the above findings. First, the data on perpetrators suggests that more systematic assessment of the source of exposure to violence should occur, even when using measures designed to assess exposure to “community” violence. The violence literature rightly has been criticized for being “siloeed” (Hamby and Grych 2013), and scholars have argued that greater recognition of the connection between different forms of violence will improve approaches to research and prevention (Hamby and Grych 2013). By systematically assessing who has perpetrated violence in different settings or of different types (e.g., hitting; threatening) researchers will be able to get a more accurate view of the unique and common types of victimization to which youth are exposed. Second, consistent with stress and coping theory (Lazarus and Folkman 1984), a cognitive appraisal process likely mediated the pathway from victimization experiences to HPA axis dysregulation. Future research should attempt to measure this appraisal process, perhaps by incorporating assessments of the appraisals of specific events, and evaluate the extent to which the appraisal process accounts for the observed association between victimization and dysregulation.

Results of the analyses yielded several implications for interventions. The fact that the most commonly reported victimization experienced were perpetrated by young friends and acquaintances suggests that these experiences were very impactful to youth. Prior research has shown that victimization by peers affects physical and emotional well-being (Hawker and Boulton 2000) and academic performance (Nakamoto and Schwartz 2010). Thus, adolescents who are targets of peer victimization may need special attention from interventionists.

Emotion Regulation as a Protective Factor

The second major finding in the article was that adolescents' emotion regulation skill, as rated by caregivers, moderated the victimization—cortisol association: There was significant negative association for youth with low levels of emotion regulation, and no association for youth with high levels of emotion regulation. This finding is consistent with prior research focused on exposure to community violence and mental health outcomes using similar samples and measures (e.g., Kliwer et al. 2004).

This finding is particularly good news for prevention scientists because emotion regulation is a modifiable skill and it is taught in many school-based prevention programs. For example, the PATHS curriculum (Greenberg et al. 1995), the Resolving Conflict Creatively Program (Brown et al. 2004), and Second Step (Frey et al. 2000) all include modules on emotion regulation. Thus, many youth may have access to these resources in their local communities. In addition to school-based prevention programs, emphasizing this skill to parents and training parents as emotion coaches is a strategy that could be used to complement school programming. Researchers have shown, for example, that parents' philosophy about emotion, that is, their awareness, acceptance, and coaching of emotions (Gottman et al. 1997) is associated with low-income African American boys' emotion regulation (Cunningham et al. 2009). The fact that the data from this study indicate that emotion regulation appears to be equally helpful in terms of physiological outcomes for male and female youth following victimization suggests that programs do not need to be designed specifically for one sex.

The current study did not address the intersection of different forms of aggression (e.g., proactive and reactive aggression) with victimization, emotion regulation, and subsequent stress responses. An area for future research might be to evaluate the role different forms of aggression play in the promotion of emotion regulation (or alternatively, dysregulation) following victimization, and the impact on health or re-victimization, as proactive aggression may actually promote emotion regulation (Ostrov

et al. 2013) and or buffer against peer victimization (Ostrov et al. 2014).

Study Strengths and Limitations

Strengths of the study include a hard to recruit sample, data from multiple sources (youth, caregivers), use of multiple methods (questionnaires, physiological assessments), tests of stress responses in two biological systems, and evaluation of a modifiable, skill-based protective factor widely linked to both physical and mental health outcomes. Well-validated measures of both victimization and emotion regulation were used and aggregated over time which provided stable estimates of these constructs. Unique among most reports of exposure to violence in youth, the source of exposure was documented for the items for which it made sense to do so. Thus, this study used a measure of victimization that likely captured exposures occurring in several microsystems in an adolescent's life. Lastly, structural equation modeling was used to test the main hypothesis.

Despite these strengths, a number of limitations to the study can be noted. Across the four waves of the study, there was 31 % attrition, with males more likely to attrit than females, although this effect was small. Although this is not unusual in studies located in under resourced communities, and although the analytic sample did not differ from the sample who attrited on key variables other than adolescent sex, there may be subtle differences that were undetectable given the loss of participants. Second, due to resource and access issues, youth and maternal caregivers were recruited into the study. Thus, the data on emotion regulation does not have the perspective of social or biological fathers. Third, the study focused on emotion regulation because this is a modifiable protective factor that has tremendous practical significance for intervention programming. There likely are other viable protective factors that were not evaluated that are equally important to examine. Finally, the study methodology assumed that the victimization youth were experiencing and reported was stressful, but appraisals were not measured in such a way as to isolate youth's assessments of victimization experiences versus other types of exposure to violence. However, symptoms of post-traumatic stress that were measured directly following assessments of exposure at each wave were correlated between .31 and .42 (all $ps < .001$), suggesting that the experience of the event, or the memory of it, was distressing.

Conclusion

The present study contributes to our understanding of adaptation in the face of stressors, specifically victimization experiences. The study demonstrated a robust link between being victimized and producing a blunted cortisol

response, which may increase the likelihood of youth developing health or mental health disorders (Miller et al. 2007). This now-classic attenuation response likely is the result of the down-regulation of the HPA axis in an attempt to minimize the physiological damage caused by repeated arousal of the stress response system. This study found that the most common victimization experiences encountered by urban adolescents, including slapping, hitting, and punching and being threatened with physical harm were perpetrated by young friends and young acquaintances, which may explain why these experiences are particularly threatening to youth. Further, the study showed that victimization was not associated with an HPA axis response for youth with above average emotion regulation skill, as rated by caregivers, while victimization was negatively associated with an HPA axis response for youth with below average emotion regulation skill. The finding with emotion regulation suggests a promising avenue to reduce potential negative physical and mental health consequences associated with a blunted HPA axis response, namely bolstering youth's emotion regulation skills. School-based prevention programming augmented with parent training as emotion coaches are some potential avenues for intervention suggested by these data.

Acknowledgements This research was supported by National Institute on Drug Abuse Grants K01 DA015442 01A1 and R21 DA 020086-02 awarded to Wendy Kliever and by Clinical and Translational Science Award UL1TR000058 from the National Center for Advancing Translational Sciences of the National Institutes of Health awarded to Virginia Commonwealth University. I thank the families who participated in this study and the research staff who supported this work.

Conflict of interest The author reports no conflict of interests.

Ethical Approval The study was approved by the Institutional Review Board at Virginia Commonwealth University (VCU). All procedures performed in this study were in accordance with the ethical standards of Virginia Commonwealth University and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Written informed consent was provided by the maternal caregiver and assent was provided by the adolescent prior to initiating the data collection.

References

- Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.
- Aiyer, S. M., Heinze, J. E., Miller, A. L., Stoddard, S. A., & Zimmerman, M. A. (2014). Exposure to violence predicting cortisol response during adolescence and early adulthood: Understanding moderating factors. *Journal of Youth and Adolescence*, 43, 1066–1079. doi:10.1007/s10964-014-0097-8.
- Alink, L. R. A., Cicchetti, D., Kim, J., & Rogosch, F. A. (2012). Longitudinal associations among child maltreatment, social functioning, and cortisol regulation. *Developmental Psychology*, 48, 224–236. doi:10.1037/a0024892.

- Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, 107(238–246), 1037. doi:10.1037/0033-2909.107.2.238.
- Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review*, 98, 459–487. doi:10.1037/0033-295X.98.4.459.
- Blair, C. (2010). Stress and the development of self-regulation in context. *Child Development Perspectives*, 4, 181–188.
- Brody, G. H., Yu, T., Chen Y. F., Kogan, S. M., Evans, G. W., Beach, S. R. H. et al. (2013). Cumulative socioeconomic status risk, allostatic load, and adjustment: A prospective latent profile analysis with contextual and genetic protective factors. *Developmental Psychology*, 49, 913–927. doi:10.1037/a0028847.
- Brown, J. L., Roderick, T., Lantieri, L., & Aber, J. L. (2004). The Resolving Conflict Creatively Program: A school-based social and emotion learning program. In J. E. Zins, R. P. Weissberg, M. C. Wang, & H. J. Walberg (Eds.), *Building academic success of social and emotional learning. What does the research say?*. New York, NY: Teachers College Press.
- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Beverly Hills, CA: Sage.
- Chen, E. (2012). Protective factors for health among low socioeconomic status individuals. *Current Directions in Psychological Science*, 21, 189–193.
- Chen, E., Matthews, K. A., Salomon, K., & Ewart, C. K. (2002). Cardiovascular reactivity during social and nonsocial stressors: Do children's personal goals and expressive skills matter? *Health Psychology*, 21, 16–24. doi:10.1037/0278-6133.21.1.16.
- Chen, E., & Miller, G. E. (2012). "Shift-and-persist" strategies: Why being low in socioeconomic status isn't always bad for health. *Perspectives in Psychological Sciences*, 7, 135–158.
- Chen, E., Strunk, R. C., Trethewey, A., Schreier, H. M., Maharaj, N., & Miller, G. E. (2011). Resilience in low-socioeconomic-status children with asthma: Adaptations to stress. *Journal of Allergy and Clinical Immunology*, 128, 970–976.
- Cohen, S., Hamrick, N. M., Rodriguez, M. S., Feldman, P. J., Rabin, B. S., & Manuck, S. B. (2000). The stability of and intercorrelations among cardiovascular, immune, endocrine, and psychological reactivity. *Annals of Behavioral Medicine*, 22, 171–179. doi:10.1007/BF02895111.
- Cook, R. D., & Weisberg, S. (1982). *Residuals and influence in regression*. London: Chapman & Hall.
- Cunningham, J. N., Kliewer, W., & Garner, P. (2009). Emotion socialization, child emotion understanding and regulation, and adjustment in urban African American families: Differential associations across child gender. *Development and Psychopathology*, 21, 261–283. doi:10.1017/S0954579409000157.
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130, 355–391. doi:10.1037/0033-2909.130.3.355.
- Dulin-Keita, A., Casazza, K., Fernandez, J. R., Goran, M. I., & Gower, B. (2010). Do neighborhoods matter? Neighborhood disorder and long-term trends in serum cortisol levels. *Journal of Epidemiology and Community Health*, 66, 24–29. doi:10.1136/jech.2009.092676.
- Ewart, C. K., Jorgensen, R. S., Suchday, S., Chen, E., & Matthews, K. A. (2002). Measuring stress resilience and coping in vulnerable youth: The social competence interview. *Psychological Assessment*, 14, 339–352. doi:10.1037/1040-3590.14.3.339.
- Farrell, A. D., Mays, S., Bettencourt, A., Erwin, E. H., Vulin-Reynolds, M., & Allison, K. W. (2010). Environmental influences on fighting versus nonviolent behavior in peer situations: A Qualitative Study with Urban African American Adolescents. *American Journal of Community Psychology*, 46(1–2), 19–35. doi:10.1007/s10464-010-9331-z.
- Fowler, P. J., Tompsett, C. J., Braciszewski, J. M., Jacques-Tiura, J. J., & Baltes, B. B. (2009). Community violence: A meta-analysis on the effect of exposure and mental health outcomes of children and adolescents. *Development and Psychopathology*, 21, 227–259. doi:10.1017/S0954579409000145.
- Frey, K. S., Hirschstein, M. K., & Guzzo, B. A. (2000). Second step: Preventing aggression by promoting social competence. *Journal of Emotional and Behavioral Disorders*, 8, 102–112. doi:10.1177/10634266000800206.
- Gordis, E. B., Margolin, G., Spies, L. A., Susman, E. J., & Granger, D. A. (2010). Interparental aggression and parent-adolescent salivary alpha amylase symmetry. *Physiology & Behavior*, 100, 225–233. doi:10.1016/j.physbeh.2010.01.006.
- Gottman, J. M., Katz, L. F., & Hooven, C. (1997). *Metaemotion: How families communicate emotionally*. Mahwah, NJ: Erlbaum.
- Granger, D. A., Johnson, S. B., Szanton, S. L., Out, D., & Schumann, L. L. (2012). Incorporating salivary biomarkers into nursing research: An overview and review of best practices. *Biological Research in Nursing*, 14, 347–356. doi:10.1177/109980041243892.
- Granger, D. A., Kivlighan, K. T., Fortunato, C., Harmon, A. G., Hibel, L. C., Schwartz, E. B., & Whembolua, G. (2007). Integration of salivary biomarkers into developmental and behaviorally-oriented research: Problems and solutions for collecting specimens. *Physiology & Behavior*, 92, 583–590. doi:10.1016/j.physbeh.2007.05.004.
- Greenberg, M. T., Kusche, C. A., Cook, E. T., & Quamma, J. P. (1995). Promoting emotional competence in school-aged children: The effects of the PATHS curriculum. *Development and Psychopathology*, 7, 117–136. doi:10.1017/S0954579400006374.
- Griffin, M. G., Resick, P. A., & Yehuda, R. (2005). Enhanced cortisol suppression following dexamethasone administration in domestic violence survivors. *The American Journal of Psychiatry*, 162, 1192–1199. doi:10.1176/appi.ajp.162.6.1192.
- Hamby, S., & Grych, J. (2013). *The web of violence: Exploring connections among different forms of interpersonal violence and abuse*. Springer briefs in sociology. New York, NY: Springer.
- Hawker, D. S. J., & Boulton, M. J. (2000). Twenty years' research on peer victimization and psychosocial maladjustment: A meta-analytic review of cross-sectional studies. *Journal of Child Psychology and Psychiatry*, 41, 441–455.
- Herbert, J. (2013). Cortisol and depression: Three questions for psychiatry. *Psychological Medicine*, 43, 449–469. doi:10.1017/S0033291712000955.
- Jäggi, L., & Kliewer, W. (2015). "Cause That's the Only Skills in School You Need" a qualitative analysis of revenge goals in poor urban youth. *Journal of Adolescent Research*. doi:10.1177/0743558415569728
- Kallem, S., Carroll-Scott, A., Rosenthal, L., Chen, E., Peters, S. M., McCaslin, C., & Ickovics, J. R. (2013). Shift-and-persist: A protective factor for elevated BMI among low socioeconomic status children. *Obesity*, 21, 759–763.
- Kids Count. (2004). *Annie E. Baltimore*, MD: Casey Foundation.
- Kim, H. K., Tiberio, S. S., Capaldi, D. M., Shortt, J. W., Squires, E. C., & Snodgrass, J. J. (2015). Intimate partner violence and diurnal cortisol patterns in couples. *Psychoneuroendocrinology*, 51, 35–46. doi:10.1016/j.psychoneu.2014.09.013.
- Kliewer, W. (2006). Exposure to violence and cortisol responses in urban youth. *International Journal of Behavioral Medicine*, 13, 109–120. doi:10.1207/s15327558ijbm1302_2.
- Kliewer, W., Nelson Cunningham, J., Diehl, R., Adams Parrish, K., Walker, J. M., Atiyeh, C., et al. (2004). Violence exposure and adjustment in inner-city youth: Child and caregiver emotion

- regulation skill, caregiver-child relationship quality, and neighborhood cohesion as protective factors. *Journal of Clinical Child and Adolescent Psychology*, 33, 477–487. doi:10.1027/s15374424jccp3303_5.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York, NY: Springer.
- Lucas-Thompson, R. G., & Granger, D. A. (2014). Parent-child relationship quality moderates the link between marital conflict and adolescents' physiological responses to social evaluative threat. *Journal of Family Psychology*, 28, 538–548. doi:10.1037/a0037328.
- McEwen, B. S., & Seeman, T. E. (2003). Stress and affect: Applicability of the concepts of allostasis and allostatic load. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences. Series in affective science* (pp. 1117–1137). New York, NY: Oxford University Press.
- Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, 133, 25. doi:10.1037/0033-2909.133.1.25.
- Muthén, L. K., & Muthén, B. O. (2015). *Mplus user's guide*. Los Angeles, CA: Muthén & Muthén.
- Nakamoto, J., & Schwartz, D. (2010). Is peer victimization associated with academic achievement? A meta-analytic review. *Social Development*, 19, 221–242. doi:10.1111/j.1467-9507.2009.00539.x.
- Nater, U. M., & Rohleder, N. (2009). Salivary alpha amylase as a non-invasive biomarker for the sympathetic nervous system: Current state of research. *Psychoneuroendocrinology*, 34, 486–496. doi:10.1016/j.psyneuen.2009.01.014.
- Negriff, S., Saxbe, D. E., & Trickett, P. K. (2015). Childhood maltreatment, pubertal development, HPA axis functioning, and psychosocial outcomes: An integrative biopsychosocial model. *Developmental Psychobiology*, 57, 984–993. doi:10.1002/dev.21340.
- Ostrov, J. M., Kamper, K. E., Hart, E. J., Godleski, S. A., & Blakely-McClure, S. J. (2014). A gender-balanced approach to the study of peer victimization and aggression subtypes in early childhood. *Development and Psychopathology*, 26, 575–587. doi:10.1017/S0954579414000248.
- Ostrov, J. M., Murray-Close, D., Godleski, S. A., & Hart, E. J. (2013). Prospective associations between forms and functions of aggression and social and affective processes during early childhood. *Journal of Experimental Child Psychology*, 116, 19–36. doi:10.1016/j.jecp.2012.12.009.
- Peckins, M. K., Dockray, S., Eckenrode, J. L., Heaton, J., & Susman, E. J. (2012). The longitudinal impact of exposure to violence on cortisol reactivity in adolescents. *Journal of Adolescent Health*, 51, 366–372. doi:10.1016/j.jadohealth.2012.01.005.
- Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, 17, 117–133.
- Pruessner, J., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology*, 28, 916–931. doi:10.1016/S0306-4530(02)00108-7.
- Richters, J. E., & Saltzman, W. (1990). *Survey of exposure to community violence: Self-report version*. Rockville, MD: National Institute of Mental Health.
- Rohleder, N. (2014). Stimulation of systemic low-grade inflammation by psychosocial stress. *Psychosomatic Medicine*, 76, 181–189. doi:10.1007/s00213-010-1879-7.
- Shahinfar, A. (1998). *Preschool children's exposure to community violence: Prevalence, correlates, and moderating factors* (Unpublished doctoral dissertation). Chapel Hill, NC: University of North Carolina.
- Shields, A., & Cicchetti, D. (1997). Emotion regulation in school-age children: The development of a new criterion Q-sort scale. *Developmental Psychology*, 33, 906–916.
- Somerville, L. H. (2013). The teenage brain: Sensitivity to social evaluation. *Current Directions in Psychological Science*, 22, 121–127. doi:10.1177/0963721413476512.
- Thompson, R. A. (1994). Emotion regulation: A theme in search of a definition. *Monographs of the Society for Research in Child Development*, 59(2/3), 25–52.
- Trickett, P. K., Noll, J. G., Susman, E. J., Shenk, C. E., & Putnam, F. W. (2010). Attenuation of cortisol across development for victims of sexual abuse. *Development and Psychopathology*, 22, 165–175. doi:10.1017/S0954579409990332.
- Wang, J., & Wang, X. (2012). *Structural equation modeling*. West Sussex: Wiley.

Wendy Kliever is Professor and Chair of the Department of Psychology at Virginia Commonwealth University. Dr. Kliever's research centers on the broad theme of risk and resilience, with specific attention to cumulative stressors, their impacts on a broad array of functioning, and protective factors that mitigate risk. She has long-standing interests in interdisciplinary, cross-cultural research, and is committed to training the next generation of scholars to continue to do research that matters. Dr. Kliever earned her Ph.D. in Social Ecology from the University of California, Irvine, and completed post doctoral training in prevention science at Arizona State University.