Exposure to Violence and Cardiovascular and Neuroendocrine Measures in Adolescents

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ABSTRACT

Background: *Exposure to violence has clear, detrimental psychological consequences, but the physiological effects are less well understood.* ***Purpose:*** *This study examined the influence of exposure to violence on biological basal and reactivity measures in adolescents.* ***Methods:*** *There were 115 high school student participants. Systolic and diastolic blood pressure (SBP, DBP), heart rate (HR), HR variability (HRV), and cortisol levels were recorded during baseline and a laboratory stressor. The Exposure to Violence interview was administered and assessed two dimensions: total observed violence and total personally experienced violence. These were then divided into component parts: lifetime frequency, proximity, and severity.* ***Results:*** *Greater total experienced violence was associated with increased basal SBP ($r = .19$, $p < .05$) and decreased acute stress reactivity in terms of SBP ($\beta = -.13$, $p = .05$), HR ($\beta = -.21$, $p = .00$), and HRV ($\beta = .13$, $p = .05$). Lifetime frequency of experienced violence was associated with higher basal DBP ($r = .33$, $p < .05$), HR ($r = .33$, $p < .05$), and cortisol ($r = .53$, $p < .00$), and decreased SBP ($\beta = -.27$, $p < .05$) and DBP ($\beta = -.31$, $p < .05$) reactivity. Exposure to violence is associated with increased biological basal levels in adolescents, supporting allostatic-load research and decreased cardiovascular reactivity, supporting the inoculation effect.* ***Conclusions:*** *The findings illustrate that being a victim of violence has more pervasive biological consequences than witnessing violence and that the accumulation of stressful experiences has the greatest effect on biological markers.*

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INTRODUCTION

Exposure to violence is a psychological stressor that has gained increasing recognition in the fields of public health, community psychology, and child health and psychopathology. Recent studies report that approximately 4 million adolescents have been the victims of violence and 9 million have witnessed violence during their lifetimes, in a nationally representative sample of U.S. adolescents. Further, 70% of U.S. inner-city youth have been victims of violence, and 85% of this population has witnessed violence during their lifetimes (1,2). Previous literature has documented a number of psychological consequences of exposure to violence, including heightened risk of posttraumatic stress disorder (PTSD) and higher levels of anxiety, depression, and aggressive and antisocial behaviors (3–8).

The pervasive psychological effects of exposure to violence have been well demonstrated. Recently, the question has emerged of whether exposure to violence, in addition to taking a psychological toll, also has physiological consequences. This literature remains preliminary and inconclusive. Thus, the goal of the study presented here was to obtain psychophysiological profiles of exposure to violence among adolescents. In particular, we focused on the effects of exposure to violence on laboratory assessments of cardiovascular and neuroendocrine measures, both at baseline and in response to an acute stressor.

Exposure to Violence and Basal Cardiovascular and Neuroendocrine Levels

Understanding alterations in basal biological profiles in younger populations can reveal early precursors to health problems later in life. Poor basal physiological profiles, such as elevated basal blood pressure and heart rate (HR), decreased HR variability (HRV, a measure of the parasympathetic nervous system), and dysregulated stress hormones, have been associated with the development of diseases such as hypertension and coronary artery disease (2,9–13).

These physiological profiles, in turn, have been linked to psychological characteristics such as stress. For example, greater reports of chronic or background stress are associated with increased basal blood pressure, increased cortisol, and decreased HRV (14–18). However, some studies have found dysregulated basal cortisol profiles in adults, such as a blunted cortisol profile (19–21). Overall, these adult studies suggest that as background or chronic stress increases, basal cardiovascular levels increase, HRV decreases, and cortisol levels become dysregulated. However, there is sparse literature regarding the formative stages of childhood and adolescence. In one such study (22), basal cardiovascular measures of total peripheral resistance were greater for adolescents reporting resolved stressors than for those reporting ongoing stressors.

These previous studies have often relied on general measures of background stress. Thus, it remains unclear what the cardiovascular and neuroendocrine profile of a specific life stressor might be. We focused on exposure to violence as one type of stressor that is salient to adolescents and that has been increasingly experienced in recent years (23). Only a few previous studies have been conducted in this area. Wilson et al. (2) monitored exposure to violence and ambulatory physiological measures during adolescents' daily lives. The authors found significant positive correlations between daytime systolic blood pressure (SBP) and nighttime diastolic blood pressure (DBP)

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and exposure to violence (2). Another study by Wilson, Kliewer, Teasley, Plybon, and Sica (24) found that adolescents who experienced violence victimization were more likely to be classified as nondippers in terms of blood pressure at night. In contrast, one laboratory study of adolescents found that increased exposure to violence was associated with lower basal pulse rates (25). However, this study included media violence in its measure of exposure to violence, which may have resulted in different patterns of effects. Finally, one study found that increased violence exposure, along with a harsh parenting style, predicted lower levels of SBP and DBP in children (26).

Overall, the chronic stress literature suggests that stress is associated with elevated basal physiological profiles and with dysregulated cortisol profiles. Furthermore, these physiological patterns are thought to place individuals at heightened risk for negative health outcomes. In focusing on the effects of one specific type of negative life event—exposure to violence—we hypothesized that similar to chronic stress patterns, adolescents with greater exposure to violence in their lives would exhibit basal physiological profiles including greater SBP, DBP, HR, lower HRV, and dysregulated cortisol levels at baseline.

Exposure to Violence and Cardiovascular and Neuroendocrine Reactivity

Background life experiences that individuals bring into the laboratory setting may influence the way they respond to novel stressors. Reactivity patterns to novel stressors are important indicators of global alterations or dysfunction in biological systems (27,28). Although there are very few studies that investigate the relationship between the specific life event of exposure to violence and cardiovascular reactivity, there is more general literature on background stressors and biological reactivity.

This literature has been mixed, finding support for both heightened acute stress reactivity and reduced reactivity. Gump and Matthews (28) reviewed 19 studies that investigated the effects of background stressors on reactivity to acute stressors and reported that the majority of the studies found greater acute stressor reactivity with heightened background stressors. This supported the notion of a sensitization effect, or heightened reactivity to a novel stressor. However, a considerable minority of the studies found reduced acute stressor reactivity under conditions of heightened background stress, supporting the idea of a habituation effect, or reduced reactivity to a novel stressor (28).

Several studies of adolescents found decreased physiological reactivity in response to acute laboratory stressors, which supports the habituation theory (29,30). Boyce and Chesterman (30) found that increased number of life events in adolescent boys was related to decreased cardiovascular reactivity. In contrast, Matthews et al. (22) found a heightened cardiovascular response to acute stress in the presence of background stress in adolescents; however, these stressors were reported as ongoing. Gump and Matthews (28) suggested that ongoing stressors are associated with increased reactivity to acute stressors, whereas resolved or past stressors can dilute or change the direction of reactivity to an acute stressor. With regard to neuroendocrine measures, several studies have demonstrated that when presented with acute stress situations, adults who have prolonged background stress in their lives exhibit a blunted cortisol response (19–21,31). Given that exposures to violence can be conceptualized as past stressors, we hypothesized that increased exposure to violence will be associated with decreased cardiovascular reactivity and blunted cortisol response to acute stressors in adolescents.

Dimensions of Exposure to Violence and Biological Measures

A secondary goal of this study was to better understand the dimensions of exposure to violence that are most relevant to biological markers. Exposure to violence is a complex stressor that has multiple components, such as whether the violence was personally experienced versus observed, in addition to characteristics such as the frequency, severity, and proximity of experienced or observed violence. In the few studies mentioned that utilized an exposure to violence scale, only Wilson et al. (2) distinguished between witnessing violence and being the victim of violence in their reporting of physiological correlates of exposure to violence. They found significant positive correlations between daytime SBP and being a victim of violence and nighttime DBP and witnessing violence in an ambulatory study (2). In addition, in the field of child psychopathology researchers have argued that understanding the dimensions of exposure to violence can help in understanding psychological responses to violence. For example, both the frequency of previous experiences with violence or trauma and the severity of exposure have been linked to PTSD in children (32,33). In our study, we explored which components of exposure to violence are most strongly related to changes in cardiovascular and neuroendocrine markers in adolescents in a controlled laboratory setting.

In sum, the primary goal of the study was to examine the influence of exposure to violence on cardiovascular (HR, SBP, DBP, HRV) and neuroendocrine (cortisol) basal and reactivity levels in adolescents. We hypothesized that (a) greater exposure to violence will be associated with greater HR, SBP, and DBP; less HRV; and dysregulated cortisol secretion at baseline, and (b) greater exposure to violence will be associated with decreased cardiovascular reactivity and a blunted cortisol response to acute stressors. The secondary goal of this study was to better understand the associations of specific dimensions of violence exposure with biological markers.

METHODS

Participants and Recruitment

One hundred fifteen participants were recruited from Kirkwood High School, a public high school in St. Louis, Missouri, with a diverse student body (approximately 25% African American). Permission was obtained from the school board to recruit students from Kirkwood High School via flyers and school announcements. We oversampled minority students to obtain a diverse sample with a range of exposure to violence experiences. Ages ranged from 16 to 19 years (M age = 16.85), with 62% female, 42% White, 55% African American, and 3% other (see

TABLE 1
Demographics and Summary of Violence Exposure

Group	*%*	
Gender		
Female	62	
Male	38	
Race		
White	42	
African American	55	
Other	3	
	M	*SD*
Age (years)	16.85	0.83
Parent education (years)	14.81	2.23
Observed exposure to violence		
Lifetime frequency	11.00	138.15
Proximity	2.77	1.20
Severity	2.97	1.49
Experienced exposure to violence		
Lifetime frequency	4.50	19.61
Proximity	3.03	1.62
Severity	1.76	1.69
Subjective exposure to violence	3.46	2.46

Note. Lifetime frequency refers to the median number of times violent events were observed or experienced during one's lifetime. Proximity refers to the average physical or relational proximity of the participant to the violent event, rated on a 1-to-7 scale, with higher numbers indicating closer proximity. Severity refers to how serious the violent event was on average, rated on a 1 (*no injury*) to 7 (*emergency care needed*) scale for experienced violence, and a 1 (*no injury*) to 8 (*death*) scale for observed violence. Subjective violence refers to the total number of *yes/no* questions endorsed (out of 17) regarding concern about violence in one's community.

Table 1). Eligibility criteria included that participants be medically healthy and not be taking medications that influence the cardiovascular system.

Materials

Exposure to Violence Interview

The Exposure to Violence measure is an interview that assesses adolescents' experience with witnessing or being the victims of violent acts in children (34). This Exposure to Violence measure has been demonstrated to have good reliability and validity in children as young as 8 years (34). Adolescents were questioned as to whether they witnessed or experienced acts of violence during their lifetimes. If they endorsed any of the mentioned violent acts, they were asked follow-up questions regarding the specifics of those acts.

We distinguished between three primary dimensions of the Exposure to Violence scale: observed (whether they had witnessed acts of violence), experienced (whether they had been the victim of violence), and subjective (how concerned they were about violence in their lives) violence. We then divided the observed and experienced dimensions into subcomponents: frequency, proximity, and severity.

Violence subcomponents. We assessed the objective subcomponents of frequency, proximity, and severity of exposure to violence. The idea behind cumulative frequency of life events is that the accumulation of multiple negative life events can contribute to alterations in biological profiles. Frequency was obtained by summing violence questions that asked the total number of times in one's lifetime violence had been witnessed or experienced (e.g., "How many times in your life have you seen or been present when someone was attacked with a knife?"). Violent acts probed included physical attacks (punching or kicking), assault with a knife, hearing gunshots, and seeing someone get shot. Lifetime frequency for each of these acts was assessed as open-ended questions. The frequency questions were scored by summing the number of lifetime occurrences of witnessing or experiencing violence that adolescents reported; a greater sum indicated greater cumulative frequency of lifetime exposure.

The idea behind the proximity component is that the closeness of an individual to a violent event will determine the magnitude of the impact of that event on the individual. Proximity can be conceptualized in two ways, one related to the physical closeness of an individual to the event that occurred and the other related to the relational (or emotional) closeness of the individual to the victim of the event. Proximity was assessed by summing violence questions that regarded how physically close someone was to the witnessed event (e.g., "Where did this event happen?") and how well he or she knew the victim of violence (e.g., "Who was the person this happened to?"). Close-ended response options for proximity questions were presented and scored on a 7-point scale ranging from 1 (*event happened at school*) to 7 (*event happened in home/apartment*). Lower values indicated less close to violence, and higher scores illustrated a closer proximity. These questions applied to proximity for observed violence. For experienced violence, participants were asked how well they knew the person who attacked them and how physically close they were on a daily basis to the location of the attack, scored on a 1-to-7 scale.

The idea behind severity is that the more severe a violent event, the greater the magnitude of the impact of that event on the adolescent. A severity of violence exposure score was obtained by summing the violence questions that addressed severity of violence (e.g., "How badly was the victim injured?") for each type of attack (beating, knife attack, gunshot). The questions were scored on a 7-point scale, from 1 (*no injury*) to 7 (*emergency medical care was obtained*), with the intermediate values increasing with increasing severity; a greater sum of severity questions indicated greater observed severity of violence. Severity was assessed for both experienced and observed events.

Primary dimensions. Three exposure to violence scores were calculated to distinguish between observed violence, experienced violence, and subjective perceptions of violence.

Observed violence refers to whether the adolescent witnessed any violent events but was not a direct victim. Total observed violence scores were obtained by summing the fre-

quency, proximity, and severity questions for each act of violence (witnessing a physical attack, witnessing a knife attack, hearing gunshots, and witnessing shootings), for a total of 12 items. Greater scores were illustrative of greater observed violence.

Experienced violence refers to whether the adolescent personally experienced or was the victim of a violent event. Total experienced violence scores were obtained by summing the frequency, proximity, and severity questions for each act of directly experienced violence (experiencing a physical attack, experiencing a knife attack, experiencing a shooting), for a total of 9 items. Greater total scores were indicative of greater experienced violence.

The subjective exposure to violence score refers to respondents' concern about violence in their lives. This subjective score, scored as 1 (*yes*) or 0 (*no*) responses, was obtained by summing 17 subjective questions (e.g., "Have you ever been worried about safety in your neighborhood?"). Greater scores meant a greater report of subjective exposure to violence.

Acute Stressor

Adolescents participated in an acute stress task in the laboratory. The acute stressor was either a debate with the experimenter or a verbal puzzle that required working with the experimenter. In the debate task, the adolescent was given 8 min to debate a controversial topic (e.g., whether school officials should have the right to search students' lockers and book bags for illegal possessions). In the verbal puzzle task, the adolescents were given 8 min to verbally instruct the experimenter on how to solve a series of three-dimensional puzzles, with regular prompts about keeping up with the expected pace for solving each puzzle. Both stressors involved verbal interactions, and both stressors were overtly videotaped to increase the evaluation component of both tasks. In both tasks, experimenters read from a script in order to present the task in a standardized way across participants. In the debate task, experimenters were given a standard set of arguments that they presented across all participants. In the puzzle task, experimenters were given a standard set of prompts about the participant's pace that were presented across all participants. Laboratory-type interpersonal stressors have been shown to elicit physiological responses that are equivalent to or surpass those produced by traditional physical or nonsocial (i.e., cold pressor) laboratory stressors (35). This study was conducted in the context of a larger study that involved an experimental manipulation during one of the tasks that was irrelevant to exposure to violence. Thus, in our study, we used only reactivity to the no-manipulation task (for 57 adolescents, this was the debate task; for 58 adolescents, this was the verbal puzzle task) and controlled for the type of task performed.

Biological Measures

HR was measured using an EKG machine. An EKG signal was transduced using two active Meditrace SF450 disposable silver/silver chloride electrodes (Kendall-LTP, Chicopee, MA). The EKG signal was filtered and amplified by the Biopac MP100 system (Biopac Systems, Santa Barbara, CA). HRV data were obtained using customized software that extracted interbeat interval (IBI) data from the EKG program used. SBP and DBP were monitored using a Dinamap Pro 100 automated blood pressure monitor (Critikon, Tampa, FL) with a standard occluding cuff on the participant's nondominant arm. Salivary cortisol samples were obtained from cotton rolls that were chewed on by the participants, spun at 3,000 rpm for 5 min, and then frozen at –70 °C until assayed. The assay involved time-resolved immunoassay with fluorescence detection using a biotin-CORT conjugate as a tracer and a streptavidin europium label. This assay has a sensitivity of 0.43 nM and assay coefficents of variance of less than 10% (36).

Procedures

Parents and adolescents were required to sign a consent form prior to participation in the study. The Institutional Review Board at Washington University approved this study. All testing was done during late afternoon hours. Once consent was obtained, the participant was seated in an individual testing room, and a blood pressure cuff was placed on the participant's nondominant arm. Three EKG electrodes were placed in the abdominal area. The adolescent was seated for a 10-min rest period, during which he or she watched a video depicting serene nature scenes. Resting HR and blood pressure measurements were obtained during these 10 min. At the end of the rest period, a baseline salivary cortisol sample was obtained.

After the rest period, the adolescent was asked to participate in a stress task. SBP, DBP, HR, and HRV were measured during the task. Twenty min after the task, another salivary cortisol sample was obtained, as an indicator of acute cortisol reactivity to the stress task. The delay in salivary cortisol sample collection ensured that the sample adequately reflects the reactivity period, as salivary cortisol reaches it peak in circulation at least 20 to 40 min after the onset of the acute stressor (37). Following collection of study cardiovascular measures, the blood pressure cuff and electrodes were removed. The participant then completed the Exposure to Violence interview with an interviewer trained on sensitivity and confidentiality issues related to this interview. Each participant was debriefed and reimbursed $40 for time and travel.

Data Analysis

Data Reduction

SBP and DBP readings that were taken every other minute during the last 5 min of baseline were to calculate baseline SBP and DBP. SBP/DBP measures were taken every minute during each acute stressor task and averaged for each task period. HR was recorded continuously during the last 5 min of baseline and averaged to calculate the baseline HR. HR was continuously recorded during each acute stress task and averaged. For the HRV measures, we edited EKG waveforms for artifacts and computed the mean successive difference (MSD) statistic for the baseline and acute stress task time intervals. The MSD is an average of the difference between consecutive IBIs for a certain time interval and has been shown in previous studies to track cardiac vagal control as well as more involved techniques (38,39). Reactivity scores for all cardiovascular and neuroendocrine measures were

obtained by subtracting baseline mean levels from the task means for each measure. As mentioned earlier, HRV is a reflection of parasympathetic activity. It is typically interpreted in an opposite manner to blood pressure and HR, which are measures of sympathetic activity. Thus, if blood pressure and HR increase during a task, HRV is expected to decrease (see Table 2).

Analyses

Analyses were conducted using regression analyses controlling for baseline values and task in reactivity analyses. Physiological measures were related first to summary exposure to violence variables, and significant associations were followed by additional testing to determine the relevant subcomponents of exposure to violence that contributed to physiological profiles.

RESULTS

Preliminary Analyses

There were no gender differences in exposure to violence scores. There were no significant associations between total parent education and exposure to violence scores. There were marginal race differences in exposure to violence. It was revealed by *t* tests that White adolescents reported less observed proximity and frequency of exposure to violence than African American adolescents: observed proximity to violence, $t(109) = -1.79$, $p < .1$; observed frequency of violence, $t(92) = -1.81$, $p < .1$.

There were gender differences in physiological measures. Male participants had lower basal DBP and higher SBP than female participants—DBP, $t(113) = -2.57$, $p \leq .05$, and SBP, $t(113) = 5.45$, $p \leq .01$—and increased DBP reactivity but decreased cortisol reactivity—DBP, $t(113) = 2.61$, $p \leq .01$, and cortisol, $t(109) = -2.29$, $p \leq .05$. There were also race differences in physiological measures. White adolescents had lower basal HRV and higher cortisol levels than African American adolescents: HRV, $t(107) = -2.59$, $p \leq .05$, and cortisol, $t(106) = 2.46$, $p \leq .05$. All analyses reported subsequently were conducted controlling for race and gender.

TABLE 2
Basal and Reactivity Physiological Measures

Group	*M*	*SD*	*Range*
Basal			
SBP (mmHg)	111.34	10.24	83 – 140
DBP (mmHg)	61.84	6.68	46 – 78
HR (beats per min)	72.26	10.34	48 – 105
HRV (msec)	52.26	30.12	10 – 202
Cortisol (nmol/L)	6.95	9.91	0.8 – 100
Reactivity[a]			
SBP (mm/Hg)	7.56	10.57	–15 – +38
DBP (mm/Hg)	7.96	6.96	–9 – +28
HR (beats per min)	6.21	7.68	–10 – +31
HRV (msec)	–8.23	17.73	–59 – +48
Cortisol (nmol/L)	–2.72	3.34	–13 – +10

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; HRV = heart rate variability.
[a]Change scores (average during lab stressor – average basal measure).

There were some associations among the different types of Exposure to Violence measures. Total observed and experienced exposure to violence were significantly correlated ($r = .46$, $p < .001$), but neither was associated with subjective violence (ps > .15). In addition, experienced proximity and frequency of violence were significantly correlated ($r = .36$, $p < .05$); however, neither was associated with severity of violence (ps > .2). Observed frequency and proximity of violence were highly correlated ($r = .98$, $p < .05$); however, neither was associated with severity of violence (ps > .5).

Exposure to Violence and Basal Physiological–Neuroendocrine Levels

To test our first hypothesis, SBP, DBP, HR, HRV, and cortisol levels were correlated with Exposure to Violence scores. As presented in Table 3, greater reports of total experienced violence were significantly associated with higher basal DBP ($r = .22$, $p < .05$). Greater experienced violence was marginally significantly associated with higher basal SBP ($r = .15$, $p = .1$) and higher basal HR ($r = .18$, $p = .06$). Given that total experienced violence was associated with physiological variables, we divided it into subcomponents to determine which aspects were associated with basal levels. Frequency of experienced violence was the only subcomponent significantly associated with higher basal cardiovascular and neuroendocrine measures (see Table 3). Higher experienced frequency of violence was associated with elevated basal DBP ($r = .31$, $p < .05$), HR ($r = .30$, $p < .05$), and cortisol levels ($r = .57$, $p < .001$) and marginally associated with basal SBP ($r = .26$, $p < .10$).

There were no significant associations between total observed violence and basal cardiovascular and neuroendocrine levels. There was a significant association of subjective exposure to violence with basal SBP ($r = -.24$, $p < .01$).

Exposure to Violence and Physiological/Neuroendocrine Reactivity

To test the second hypothesis, each acute stress reactivity score was regressed onto the exposure to violence scores, controlling for basal levels and type of task. With respect to type of

TABLE 3
Personally Experienced Exposure to Violence and Basal Physiological and Neuroendocrine Levels

	Cardiovascular and Neuroendocrine Measures (r)				
Stress Measure	*SBP*	*DBP*	*HR*	*HRV*	*Cortisol*
Experienced violence	.15*	.22**	.18*	–.11	.07
Frequency	.26*	.31**	.30**	–.15	.57***
Proximity	.10	.13	–.04	.01	.07
Severity	–.11	.08	–.05	.03	–.06

Note. These analyses control for the effects of race and gender. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; HRV = heart rate variability.
*$p \leq .1$. **$p \leq .05$. ***$p \leq .001$.

task, there were no significant differences in violence exposure levels or biological measures for participants who received one task versus the other.

As total experienced violence increased, there was a significant association with decreased SBP reactivity ($\beta = -.14$, $p < .05$), HR reactivity ($\beta = -.21$, $p < .001$), and marginally increased HRV ($\beta = .11$, $p = .1$). Given that total experienced violence was associated with physiological variables, we divided experienced violence into subcomponents to determine which of its aspects were associated with reactivity. Greater frequency of experienced violence was significantly associated with decreased SBP reactivity ($\beta = -.27$, $p < .05$), HR reactivity ($\beta = -.21$, $p < .05$), and marginally with DBP reactivity ($\beta = -.27$, $p = .06$). Closer proximity to experienced violence was significantly associated with decreased HR reactivity ($\beta = -.14$, $p < .05$), and increased HRV ($\beta = .13$, $p < .05$). (See Table 4.)

As total observed violence increased, there was a significant association with decreased SBP reactivity ($\beta = -.14$, $p < .05$). Specifically, as frequency of observed violence ($\beta = -.13$, $p < .1$) and proximity to observed violence ($\beta = -.11$, $p < .1$) increased, there were marginally significant associations with decreased SBP reactivity. There were no significant associations between cardiovascular and neuroendocrine reactivity and subjective exposure to violence.

In addition, we found that gender moderated the effect of exposure to violence on HRV reactivity, as indicated by a significant Gender × Total Experienced Violence effect on HRV reactivity ($\beta = -.84$, $p < .01$). Among male participants, greater exposure to violence was associated with greater HRV reactivity ($\beta = .31$, $p < .01$), whereas among female participants, there was no association of exposure to violence with HRV reactivity ($\beta = .03$, $p > .5$).

Exposure to Violence as a Past Stressor

Finally, to strengthen our conceptualization of exposure to violence as a past stressor, we restricted our exposure to violence frequency analyses to include only events that occurred over 1 year ago. Higher frequency of personally experienced violent events remained associated with basal SBP ($r = .25$, $p < .1$), DBP ($r = .31$, $p < .05$), HR ($r = .29$, $p < .06$), and cortisol levels ($r = .59$, $p < .001$). In addition, higher frequency of personally experienced violent events remained associated with decreased SBP reactivity ($\beta = -.26$, $p < .05$), DBP reactivity ($\beta = -.27$, $p < .1$), and HR reactivity ($\beta = -.20$, $p = .06$).

Alternative Explanations

One possible explanation for the reactivity patterns is that adolescents who have experienced high levels of violence find a lab stressor to be much less stressful than do adolescents who have not experienced violence. To test this hypothesis, we correlated task stressor appraisal with the experienced exposure to violence dimension. There were no significant relationships between laboratory task stress appraisal and total experienced violence or any of its subcomponents (all ps > .1). That is, adolescents with greater experiences with violence did not perceive the lab task to be less stressful than those who had not experienced violence.

DISCUSSION

Exposure to Violence and Cardiovascular–Neuroendocrine Basal Levels

We found that exposure to violence is related to increased cardiovascular and neuroendocrine basal levels in adolescents. Specifically, experienced violence is associated with increased basal SBP, DBP, HR, and cortisol. Further, of the subcomponents of exposure to violence, we found that frequency of experienced violence was most strongly associated with basal cardiovascular and neuroendocrine measures. In contrast, proximity and severity of violence were not associated with basal physiological measures.

These findings are consistent with the literature regarding increased chronic or background stress and elevated basal physiological profiles (2,28). Our results suggest that exposure to violence can be conceptualized as a chronic stressor that is internalized and has lasting effects on basal neuroendocrine and cardiovascular systems of adolescents. This alteration in basal levels may be best understood by the concept of allostatic load

TABLE 4
Personally Experienced Exposure to Violence and Physiological and Neuroendocrine Reactivity

	Cardiovascular and Neuroendocrine Measures				
Stress Measure	*SBP*	*DBP*	*HR*	*HRV*	*Cortisol*
Experienced violence (β)	–.14**	–.09	–.21***	.11*	–.05
Overall model *R*	.74***	.66***	.80***	.77***	.95***
Variance accounted for (%)	54.7	43.2	64.3	59.3	89.7
Frequency (β)	–.27**	–.27*	–.21**	.07	–.10
Proximity (β)	–.05	.01	–.14**	.13**	–.01
Severity (β)	.00	–.02	–.08	.10	–.02

Note. β represents the unique contribution of the exposure to violence measure (after controlling for all other factors in the overall model). The overall model includes basal level of each biological measure, type of acute stressor task, race, gender, and total exposure to violence. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; HRV = heart rate variability.

*$p \leq .1$. **$p \leq .05$. ***$p \leq .001$.

(10). Allostasis, an adaptive mechanism referring to the idea of maintaining physiological stability through changing environmental states, can shift into the maladaptive state of allostatic load if there is prolonged wear and tear on the body resulting from repeated adaptive efforts (40). Increased exposure to violence in one's lifetime may act as a prolonged, chronic stress that causes allostatic-load wear and tear and leads to sustained, elevated basal physiological levels.

In addition, this profile of elevated basal cardiovascular levels follows the trauma and PTSD literature, wherein individuals who develop PTSD due to a traumatic event have elevated basal cardiovascular levels (41). Specifically, those who have the most chronic PTSD have the largest and most robust elevations in basal levels (41). This is further support for an allostatic-load model of the effects of stress, trauma, and exposure to violence on biological systems, as repeated and sustained responses to chronic stress or trauma may lead to maladaptive elevations in basal cardiovascular levels.

Our basal level findings of associations with experienced violence are consistent with Wilson et al.'s (2) finding that exposure to experienced violence was associated with higher ambulatory daytime SBP. Our findings contrast with some other work on exposure to violence where increased violence exposure was associated with decreased basal pulse rates (25). However, this previous study used an exposure to violence measure that included media exposure and hearing about violent events from others. In contrast, the Wilson et al. (2) study and our study included only situations that occurred in real life. Taken together with the Wilson et al. (2) study, results demonstrate that across two contexts—both in adolescents' daily lives and in a controlled laboratory setting—the personal experience of being a victim of violence is associated with increased daytime cardiovascular and neuroendocrine basal profiles. The elevated cardiovascular and neuroendocrine basal levels we found present a profile of potential health risks for outcomes such as hypertension and cardiovascular disease and are congruent with risk factors found in urban youth (2,9). Thus, these basal profiles both support a theory of sustained arousal and suggest a possible trajectory of health risk starting in adolescence.

Exposure to Violence and Cardiovascular Reactivity

We found that exposure to violence is related to decreased cardiovascular reactivity. Total observed violence and total experienced violence were associated with decreased cardiovascular reactivity. Also, both the frequency of experienced violence and the proximity to experienced violence were significantly associated with decreased cardiovascular reactivity.

These results support the inoculation or habituation effect, wherein consistent exposure to stressful events can dampen physiological responsivity to novel stressors over time (29,42). Boyce and Chesterman (30) explained that the inoculation effect can result in decreased cardiovascular reactivity as repeated life events aid in the development of coping strategies. These coping strategies can thereby serve to dampen responses to laboratory stressors. In addition, the pattern of decreased reactivity corresponds with previous findings that described decreased cardiovascular reactivity in participants that reported past (as opposed to ongoing) stressors (22,28). Adolescents may cope with repeated exposure to violent life events by regarding violence as discrete events in the past, which then may result in an inoculation-effect pattern of cardiovascular reactivity to new acute stressors.

In addition, it is important to note that the decreased reactivity to a novel stressor in adolescents who reported greater exposure to violence is not attributable to those adolescents' perceiving the lab stressor as less threatening. All adolescents, regardless of degree of violence exposure, reported the lab task to be similarly stressful. Thus, we can rule out the alternative hypothesis that the blunted reactivity is due to those adolescents who have been exposed to violence finding our lab stressor to be nonstressful.

Dimensions of Exposure to Violence

We found that objective components of violence exposure were associated with biological markers more consistently than subjective concern about violence. These findings suggest the possibility that exposure to violence is such a salient life event that if it occurs, there are physiological consequences, regardless of an adolescent's reported subjective reaction to the violence event.

Among the different subcomponents of exposure to violence, cumulative frequency of lifetime violence exposure was associated most robustly with both basal and reactivity measures. This importance of cumulative frequency of lifetime exposure is in accordance with much of the literature discussing accumulation of stressors as having the greatest effect on the body (37,43). This idea of cumulative stressor exposure also fits with allostatic load. As the body's systems fluctuate and adapt to multiple stressors over one's life, the cumulative effect of this background stress can be a buildup of allostatic load (10,44). Thus, our findings that frequency of experienced violence over the lifetime has pervasive effects on cardiovascular and neuroendocrine systems are illustrative of allostatic-load-induced biological alterations.

Limitations, Implications, and Future Studies

Limitations of this study include the fact that measurements of basal neuroendocrine and cardiovascular levels were taken on one day during a laboratory study. In addition, this study was cross-sectional, so it is difficult to infer that exposure to violence causes altered biological basal and reactivity levels. Third, future studies could improve on existing exposure to violence measures. For example, we were not able to directly assess whether participants perceived that exposure to violence was a past stressor or an ongoing stressor. Including parental interviews to corroborate adolescents' reports of violence also will be important in future studies. Finally, it will be important to investigate coping methods in future studies to ascertain if the continual exposure to violent events elicits an inoculation-effect type of coping response.

This research is unique on several levels. It adds to the existing literature that has documented effects of exposure to violence on blood pressure in the daily lives of adolescents by conducting a controlled laboratory study. The laboratory environment allowed us to test whether adolescents who are exposed to controlled stimuli in the lab respond differently depending on their background experience with violence. It also allowed us to conduct more comprehensive cardiovascular and neuroendocrine assessments than can be done in the field. This work also was unique in examining how adolescents with a background of exposure to violence would respond biologically to a new acute stressor. Finally, it was unique in assessing both objective and subjective components of violence, as well as probing dimensions such as frequency, proximity, and severity of violence.

Overall, our finding that greater exposure to violence is associated with elevated biological basal levels suggests that exposure to violence could lead to cardiovascular or neuroendocrine mediated health problems later in life. The blunted cardiovascular response to acute stress in the presence of greater exposure to violence alludes to wear and tear on physiological systems that can lead to inadequate responses to novel stressors. Our findings also suggest that objective measures of exposure to violence may be better than subjective reports as indicators of stress-related effects on psychophysiological functioning. Also, we found that being the victim of violence has pervasive effects on biological markers and that cumulative frequency of experiencing violent events has the most pervasive biological effects. This illustrates the importance of focusing on the health and well-being of populations that are exposed to multiple violent events over time and of developing interventions and policies that can alleviate the wear and tear associated with cumulative violent event exposure.

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