EXPOSURE TO VIOLENCE AND CARDIOVASCULAR AND NEUROENDOCRINE
MEASURES IN ADOLESCENTS

by

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B.A. New York University, 2000

A THESIS SUBMITTED IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF ARTS

in

THE FACULTY OF GRADUATE STUDIES
(DEPARTMENT OF PSYCHOLOGY)

We accept this thesis conforming
to the required standards

THE UNIVERSITY OF BRITISH COLUMBIA

August 2004

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Title of Thesis: Exposure to Violence and Cardiovascular and Neuroendocrine Measures in Adolescents

Degree:

MA

Year: 2004

Department of Psychology

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Abstract

This study examined the influence of multiple dimensions of exposure to violence (EXPV) on biological basal and reactivity measures in adolescents. 115 high school students participated. Systolic and diastolic blood pressure (SBP, DBP), heart rate (HR), heart rate variability (HRV) and cortisol levels were recorded during baseline and in response to an acute stressor. The EXPV 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. 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 ($\beta = .13, p = .05$). The lifetime frequency of experienced violence subcomponent was associated with higher basal DBP ($r = .33, p < .05$), HR ($r = .33, p < .05$), and cortisol ($r = .53, p < .001$), and decreased SBP ($\beta = -.27, p < .05$), DBP ($\beta = -.31, p < .05$) reactivity. EXPV is associated with increased biological basal levels in adolescents, supporting allostatic load research. EXPV is associated with decreased cardiovascular reactivity, supporting the inoculation effect. The findings with experienced violence illustrate that being a victim of violence has more pervasive biological consequences than observation. The associations with frequency of experienced violence illustrate that 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 lifetime, 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 lifetime (1-2). Previous literature has documented a number of psychological consequences of exposure to violence, including heightened risk of post-traumatic stress disorder, and higher levels of anxiety, depression, 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 present study 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 cardiovascular and neuroendocrine 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, decreased heart rate 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 decreased basal cortisol levels in adults, alluding to a hypoactive stress system (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 study by Matthews et al. (22), basal cardiovascular measures of total peripheral resistance were greater for adolescents reporting resolved background stressors than for those reporting ongoing stressors.

However, these previous studies 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. In the present study, we focused on exposure to violence as one type of stressor that is very salient to the lives of adolescents, and that has been increasingly experienced in recent years (23). A few previous studies have been conducted, but have yielded mixed results. Wilson et al. (2) monitored exposure to violence and ambulatory physiological measures during adolescents' daily lives. They found significant positive correlations between daytime systolic blood pressure (SBP) and nighttime diastolic blood pressure (DBP) and exposure to violence (2). In contrast, however, one laboratory study of adolescents found that increased exposure to violence (including media violence) was associated with lower basal pulse rates (24). One study of adults found that exposure to violence was not associated with basal levels of salivary cortisol, heart rate (HR), and HRV (25).

Our conceptualization of violence was closer to Wilson et al's (2), in focusing on directly experienced violence (not hearsay or media violence). Thus based on the patterns found in that study, and the background stress literature generally, we hypothesized that increased exposure to violence in the lives of adolescents would be associated with 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 (26-27). 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 (27) 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 (27).

Several studies of adolescents found decreased physiological reactivity in response to acute laboratory stressors, which supports the habituation theory (28-29). 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 (27) suggest 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. In 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 (31; 19-21). Given that exposure 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 the present study, we explore which components of exposure to violence are most strongly related to changes in cardiovascular or neuroendocrine markers in a controlled laboratory setting.

In sum, the primary goal of the study is to examine the influence of exposure to violence

on cardiovascular (HR, SBP, DBP, HRV) and neuroendocrine (cortisol) basal and reactivity levels in adolescents. We hypothesize that (1) greater exposure to violence will be associated with greater HR, SBP, and DBP, less HRV and dysregulated cortisol secretion at baseline, and (2) 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 is to gain a greater understanding of the associations of specific dimensions of exposure to violence with biological markers.

MATERIALS AND METHODS

Subjects and Recruitment

115 subjects were recruited from Kirkwood High School (KHS), 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 KHS via flyers and school announcements. Student ages ranged from 16-19 years (mean 16.85), with 62% female, 42 % Caucasian, 55% African American, and 3% other. Eligibility criteria included participants being medically healthy and not taking any medication that influences the cardiovascular system. (See Table 1 for demographic information.)

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 and has been validated in children as young as 8 (32). The exposure to violence measure parallels an exposure to violence measure that has good internal consistency, test-retest reliability, and validity in a sample of youth and young adults (33). Adolescents were questioned as to whether they witnessed or experienced acts of

violence during their lifetime. 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 their subcomponents: frequency, proximity, and severity.

Violence subcomponents: We assessed the objective subcomponents of frequency, proximity, and severity of exposure to violence. The idea that cumulative frequency of specific life events could be associated with alterations in biological profiles was assessed via the cumulative lifetime frequency component of the exposure to violence measure (frequency). Frequency was obtained by summing violence questions that addressed cumulative lifetime violence occurrence (e.g. "How many times in your life have you seen or been present when someone was attacked with a knife"). The frequency questions were scored by summing the number of occurrences of witnessing incidences of physical abuse, assault with a knife, hearing a gun shot, or seeing someone getting shot; a greater sum indicated greater cumulative frequency of lifetime exposure.

In order to determine whether physical or relationship proximity to the event and victim were associated with biological parameters, we calculated the proximity component of the exposure to violence measure (proximity). 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 they knew the victim of violence (e.g. "who was the person this happened to?"). The questions were scored on an ascending scale where lower

values were less close (“event happened at school”) than higher values (“event happened in home/apartment”), and greater sums illustrated closer proximity to violence. These questions applied to proximity for observed violence. For experienced violence, subjects were asked how well they knew the person that attacked them and how physically close they are on a daily basis to the location of the attack.

In order to determine if the severity of a violent event is associated with biological parameters, we assessed severity of violent event exposure (severity). The 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?”). The questions were scored on a 1-7 scale, with “1” indicating no injury and “7” indicating that emergency medical care was obtained, with the intermediate values increasing with increasing severity; a greater sum of severity questions indicated greater severity of observed violence.

Primary dimensions. Three exposure to violence scores were calculated in order 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 all the questions in the frequency, proximity and severity subcomponents for observed violence; 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 all questions in the frequency, proximity and severity subcomponents for directly experienced violence. Greater total scores were indicative of greater experienced violence.

The subjective exposure to violence score refers to the adolescent's concern about violence in their lives. The subjective exposure to violence score was obtained by summing subjective questions that were scored according to "yes" (1) or "no" (0) responses (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 task was either a debate with the experimenter or a puzzle completed verbally with the experimenter. In the debate task, the adolescent was given a total of 8 minutes to debate a controversial topic presented to them by the experimenter (e.g. whether school officials should have the right to search students' lockers and book bags for illegal possessions). In the puzzle task, the adolescents were given a total of 8 minutes to verbally instruct the experimenter in solving a series of 3-D puzzles. Both stressors involved interactions with the experimenter that were overtly videotaped in order to induce a challenging social stressor situation. Laboratory-type interpersonal stressors have been shown to elicit physiological responses that are equivalent to or surpass those produced by traditional physical or non-social (i.e. cold-pressor, mental arithmetic) laboratory stressors (34). 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 the present study, we utilized only reactivity to the no manipulation 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 was 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 subjects and spun at 3000 rpm for 5 minutes, 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 CVs of less than 10%. See Table 2.

Procedures

Parents and adolescents were required to sign a consent form prior to participation in the study. The Institutional Review Board at Washington University in St. Louis 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 adolescents were seated for a 10-minute rest period, during which they watched a video depicting serene nature scenes. Resting heart rate and blood pressure measurements were obtained during this 10-minute period. At the end of the rest period, a baseline salivary cortisol sample was obtained.

After the rest period, adolescents were asked to participate in a stress task. SBP, DBP, HR and HRV were measured during the task. 20 minutes 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 assured that the sample adequately reflects the reactivity

period, as salivary cortisol reaches its peak in circulation at least 20-40 minutes after the onset of the acute stressor (35). Following collection of study cardiovascular measures, the blood pressure cuff and electrodes were removed. Subjects then completed the Exposure to Violence interview. Subjects were 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 minutes of baseline were used 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 minutes 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 inter-beat intervals (IBIs) for a certain time interval, and has been shown in previous studies to track cardiac vagal control as well as more involved techniques (36-37). 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. As a result, it is typically interpreted in an opposite manner as blood pressure and heart rate, which are measures of sympathetic activity. Thus, if blood pressure and heart rate increase during a task, HRV is expected to decrease.

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. T-tests revealed a marginally significant difference in race difference in the observed proximity to violence ($t(109) = -1.79; p < .1$) and observed frequency of violence ($t(92) = -1.81; p < .1$) subcomponents of total observed exposure to violence. Analyses reported below were repeated controlling for race, however, patterns of significance and non-significance did not change. The three types of exposure to violence measures were moderately associated with one another. Total observed and experienced exposure to violence were significantly correlated ($r = .46, p < .001$); however neither was correlated with subjective exposure to violence.

Exposure to Violence and Basal Cardiovascular/Neuroendocrine Levels

In order 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 increased basal SBP ($r(65) = .19, p < .05$). Greater experienced violence was marginally significantly associated with higher basal DBP ($r = .18, p < .1$). Given that total experienced violence was associated with physiological variables, we divided it into subcomponents in order 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 ($r = .33, p < .05$), HR ($r = .33, p < .05$), and cortisol levels ($r = .53, p < .001$).

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 ($r = -.21, p < .05$).

Exposure to Violence and Cardiovascular/Neuroendocrine Reactivity

In order to test the second hypothesis regarding the relationship of cardiovascular and neuroendocrine reactivity with exposure to violence scores, each acute stress reactivity score was regressed onto the exposure to violence scores, controlling for basal levels and type of task.

As total experienced violence increased, there was a significant association with decreased SBP reactivity ($\beta = -.13, p = .05$), HR ($\beta = -.21, p = .001$), and increased HRV ($\beta = .13, p = .05$) (Table 4). Given that total experienced violence was associated with physiological variables, we divided experienced violence into subcomponents to determine which aspects of it were associated with reactivity. Greater frequency of experienced violence was significantly associated with decreased SBP reactivity ($\beta = -.27, p < .05$), DBP ($\beta = -.31, p < .05$); and marginally with HR reactivity ($\beta = -.19, p < .1$) (Table 4). Closer proximity to experienced violence was significantly associated with decreased HR reactivity ($\beta = -.15, p < .01$), and increased HRV ($\beta = .15, p < .05$) (Table 4).

With respect to observed violence, 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 ($\beta = -.12, p < .1$) 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.

Alternative Explanations

One possible explanation for the reactivity patterns is that adolescents who have experienced high levels of violence in their lives find an acute lab stressor to be much less stressful than adolescents who have not experienced violence. To test the hypothesis that decreased reactivity was due to decreased subjective appraisal of the laboratory tasks as stressful by those adolescents that reported greater exposure to violence, we correlated task stressor appraisal with the experienced exposure to violence dimension. There were no significant relationships between laboratory stressor task stress appraisal and total experienced violence or any of its subcomponents (all p 's $> .1$). That is, adolescents with greater experiences with violence did not perceive the lab task to be less stressful than adolescents 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, 27). 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 (10, 38). 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 (39). Increased exposure to violence in one's lifetime may act as a prolonged, chronic stress that causes allostatic load wear and tear, and ultimately leads to sustained, elevated basal physiological levels.

In addition, this profile of elevated basal cardiovascular levels follows the trauma and PTSD (post-traumatic stress disorder) literature, wherein individuals who develop PTSD due to a traumatic event have elevated basal cardiovascular levels (40). Specifically, those who have the most chronic PTSD have the largest and most robust elevations in basal levels (40). 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 but not observed violence are consistent with those of Wilson et al. (2) where increased exposure to experienced violence was associated with increased ambulatory daytime SBP. Interestingly, our findings contrast to some other work on exposure to violence where increased violence exposure was associated with decreased basal pulse rates (24). However, this previous study utilized 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. study (2), 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. Both total observed violence and total experienced violence were associated with decreased cardiovascular reactivity. In addition, 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 (28,41). Boyce et al. (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. Thus, adolescents experiencing greater exposure to violence may develop strategies to cope with the consistent presence of violent events, which may blunt their cardiovascular responses to novel stressors. In addition, the pattern of decreased reactivity corresponds with previous findings that described decreased cardiovascular reactivity in subjects that reported past (as opposed to ongoing) stressors (22, 27). 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 adolescent 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 non-stressful.

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. This idea is consistent with literature that reveals that urban and African American populations that are exposed to stressful events such as violence or discrimination may not report subjective distress or may accept and internalize these incidents (42-43). For example, Krieger et al. (43) found that African Americans that internalized their responses to situations of racial discrimination had elevated basal blood pressure levels compared to those that reported or challenged the incidents of discrimination. That is, those who responded to discrimination by accepting it as an unchangeable fact and kept the "fact" to themselves seemed to internalize the unfair events and manifest them in elevated basal physiological levels. Our findings are similar to these as there were few relationships between subjectively reported concern about violence and biological measures; however, there were multiple significant associations between objective exposure to violence measures and both basal and reactivity biological measures.

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 (39; 44-45). 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 build up of allostatic load (10, 45). 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, including parental interviews to corroborate adolescents' reports of violence 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 controlled environment allowed us to test whether adolescents who are exposed to identical 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. Lastly, 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 suggest 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. In addition, we found that being the victim of violence has pervasive effects on cardiovascular and neuroendocrine 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 developing interventions and policies that can alleviate the wear and tear associated with cumulative violent event exposure.

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Table 1. Demographics and Summary of Violence Exposure

Group	Percentage	
Gender		
Female	62%	
Male	38%	
Race		
Caucasian	42%	
African American	55%	
Other	3%	
	Mean	Standard Deviation
Age (years)	16.85	0.83
Parent education (years)	14.81	2.23
Observed Exposure to Violence		
Lifetime Frequency ^a	11.00 [*]	138.15
Proximity ^b	2.77	1.20
Severity ^c	2.97	1.49
Experienced Exposure to Violence		
Lifetime Frequency ^d	4.50 [*]	19.61
Proximity ^e	3.03	1.62
Severity ^f	1.76	1.69
Subjective Exposure to Violence ^g	3.46	2.46

^amedian number of times violent events observed during lifetime; * median values are reported as the range of number of observed violent events was large

^bphysical proximity to place where violent event was observed and emotional proximity to victim and offender on 1-7 scale for place, victim, and offender; higher numbers are closer to home and closer to person (victim and offender)

^cseverity of outcome of observed violent event on 1-8 scale, where 1 is "no injury" and 8 is "death"

^dmedian number of times violent events experienced during lifetime; ^emedian values are reported as the range of number of experienced violent events was large

^fphysical proximity to place where violent event was experienced and emotional proximity to offender on 1-7 scale for place and offender; higher numbers are closer to home and closer to person (offender)

^fseverity of outcome of experienced violent event on 1-7 scale, where 1 is "no injury" and 7 is "emergency care needed"

^gtotal across 17 yes/no questions regarding concern about violence in community; higher numbers indicate higher concern about violence

Table 2. Basal and Reactivity Physiological Measures

Group	Mean	Standard Deviation
Basal		
SBP (Systolic Blood Pressure; mm/Hg)	111.34	10.24
DBP (Diastolic Blood Pressure; mm/Hg)	61.84	6.68
HR (Heart Rate; beats/min)	72.26	10.34
HRV (Heart Rate Variability; msec)	52.26	30.12
Cortisol (nmol/liter)	6.95	9.91
Reactivity*		
SBP (mm/Hg)	7.56	10.57
DBP (mm/Hg)	7.96	6.96
HR (mm/Hg)	6.21	7.68
HRV (msec)	-8.23	17.73
Cortisol (nmol/Liter)	-2.72	3.34

* Change scores (Average during lab stressor – Average Basal Measure)

Table 3. Personally Experienced Exposure to Violence and Basal Cardiovascular and Neuroendocrine Levels

Stress measure	Cardiovascular and neuroendocrine measures				
	SBP	DBP	HR	HRV	Cortisol
Experienced Violence	r: .19**	r: .18*	r: .15	r: -.09	r: .06
frequency	r: .07	r: .33**	r: .33**	r: -.20	r: .53***
proximity	r: .14	r: .10	r: -.06	r: .03	r: .06
severity	r: .05	r: .05	r: -.07	r: .02	r: -.09

* $p \leq .1$

** $p \leq .05$

*** $p \leq .001$

Table 4. Personally Experienced Exposure to Violence and Cardiovascular and Neuroendocrine Reactivity

Stress measure	Cardiovascular and neuroendocrine measures				
	SBP	DBP	HR	HRV	Cortisol
Experienced Violence	$\beta: -.13^{**}$	$\beta: -.06$	$\beta: -.21^{***}$	$\beta: .13^{**}$	$\beta: -.04$
frequency	$\beta: -.27^{**}$	$\beta: -.31^{**}$	$\beta: -.19^{*}$	$\beta: -.02$	$\beta: -.21$
proximity	$\beta: -.04$	$\beta: -.03$	$\beta: -.15^{**}$	$\beta: .15^{**}$	$\beta: -.00$
severity	$\beta: .01$	$\beta: .03$	$\beta: -.08$	$\beta: .12$	$\beta: -.01$

Note: All regression analyses controlling for basal levels and acute stressor task

* $p \leq .1$

** $p \leq .05$

*** $p \leq .001$