Childhood Violence Exposure Predicts High Blood Pressure in Black American Young Adults

Gaurav Kapur, MD[1,2,*], Anaïs F. Stenson, PhD[3,*], Lisa M. Chiodo, PhD[4], Virginia Delaney-Black, MD, MPH[1], John H. Hannigan, PhD[5,6,7,8,9], James Janisse, PhD[10], and Hilary H. Ratner, PhD[7,9]

Objective To test the impact of childhood adversity, including community violence exposure, on hypertension risk in Black American young adults to understand what risk factors (eg, prenatal factors, later exposures) and ages of adversity exposure increased hypertension risk.

Study design The study included 396 Black American participants with data from prenatal, birth, and age 7-, 14-, and 19-year visits. At age 19 years, individuals with blood pressure (BP) measures >120 mmHg systolic and/or >80 mmHg diastolic were classified as having high blood pressure (HBP), and those with BP <120/80 mmHg were classified as normal. Associations between prenatal and birth risk factors; childhood adversity at age 7, 14, and 19 years; age 19 body mass index (BMI); and both systolic and diastolic BP at age 19 were tested using logistic regression models.

Results Age 19 BMI was positively associated with systolic and diastolic HBP status at age 19. Controlling for all covariates, community violence exposure at age 7 and 19 years was associated with 2.2-fold (95% CI, 1.242-3.859) and 2.0-fold (95% CI, 1.052-3.664) greater odds of systolic HBP, respectively, at age 19 years. Prenatal risk, birth risk, and other dimensions of childhood adversity were not associated with HBP in this cohort.

Conclusion Childhood community violence exposure is a significant risk factor for HBP in young adults. As Black American children typically experience more community violence exposure than other American children, our results suggest that racial disparities in childhood community violence exposure may contribute to racial disparities in adult hypertension burden. *(J Pediatr 2022;248:21-9)*.

Black Americans are disproportionally impacted by hypertension relative to other groups; the prevalence of hypertension is 56.2% in Black Americans versus 48% in White Americans, 46.3% in Asian Americans, and 38.9% in Hispanic Americans.[1] Children with elevated blood pressure (BP) are more likely to have persistently increased BP and/or develop hypertension.[2,3] Elucidating the developmental antecedents of high BP could provide new targets for intervention[4,5] to reduce the prevalence of hypertension and its associated consequences. Epidemiologic studies report significant associations between childhood adversity and cardiovascular risk in adulthood.[6] On average, Black American children experience more childhood adversity than other groups,[6] however, whether this differential exposure may contribute to observed racial disparities in cardiovascular outcomes is unclear.

Primary hypertension, the most common form of elevated BP, results from complex interactions among genetic, environmental, and biological factors.[7] High body mass index (BMI) is a key risk factor for hypertension across demographic groups.[8,9] Childhood adversity increases the risk for adult hypertension, but whether specific types or developmental timing of childhood adversity differentially impact risk remains unclear.[10] Childhood adversity also impacts cardiac function during childhood and adolescence[11,12] and is associated with poor cardiovascular outcomes, including hypertension, stroke, and myocardial infarction, in adulthood.[6,13] However, most studies of childhood adversity and community

ACE	Adverse Childhood Experiences
BP	Blood pressure
BMI	Body mass index
CREV	Children's Report of Exposure to Violence
HBP	High blood pressure
SCECV	Survey of Children's Exposure to Community Violence
SCHOO-BE	School-Based Evaluation
SES	Socioeconomic status
SNS	Sympathetic nervous system
SRSS	Safe and Responsive School Survey
TISH	Things I Have Seen and Heard

From the [1]Department of Pediatrics, Wayne State University, Detroit, MI; [2]Department of Pediatrics, Central Michigan University, Mount Pleasant, MI; [3]Department of Psychiatry and Behavioral Neurosciences, Wayne State University, Detroit, MI; [4]College of Nursing, University of Massachusetts–Amherst, Amherst, MA; [5]Department of Obstetrics & Gynecology, [6]C.S. Mott Center for Human Growth & Development, [7]Merrill-Palmer Skillman Institute for Child & Family Development, [8]Center for Urban Responses to Environmental Stressors, [9]Department of Psychology, and [10]Department of Family Medicine & Public Health Sciences, Wayne State University, Detroit, MI

*Contributed equally.

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violence exposure effects on BP or hypertension are limited by a reliance on the adult participants' retrospective reports of childhood exposures,[10,14-21] cross-sectional designs,[22] small sample size, or examination of BP response to controlled laboratory stressors.[23,24] A meta-analysis found poor agreement between prospectively versus retrospectively collected measures of childhood adversity.[25] This finding highlights the need for studies that conduct prospective assessments of community violence exposure and then examine associations with later cardiovascular health to obtain a more accurate assessment of the association between community violence exposure and cardiovascular risk.

The National Heart, Lung, and Blood Institute Working Group has called for research on environmental, psychosocial, and behavioral factors associated with the higher prevalence of hypertension in Black Americans.[26] The extent to which racial disparities in cardiovascular health may be explained by racial disparities in childhood adversities is unclear. The aim of the present study was to leverage prospectively collected data collected from participants at 3 ages—7, 14, and 19 years—to evaluate how different dimensions of childhood adversity, including prenatal exposures, birth outcomes, poverty, and community violence exposure, impact the risk for hypertension in Black American young adults. We hypothesized that childhood adversity would be positively associated with the risk of hypertension at age 19 years after accounting for BMI, and that this association would be partially or fully driven by childhood community violence exposure.

Methods

This study used data collected between 1989 and 2012 as part of the prospective longitudinal evaluation of the School-Based Evaluation (SCHOO-BE) cohort. The SCHOO-BE study began in 1995, but prenatal and birth data for mothers and children were available from a previous study conducted between 1989 and 1991. The study methodology is described in detail elsewhere.[27] In brief, women from a public, university-based hospital clinic in Detroit, Michigan participated in a pregnancy study beginning at their first prenatal visit. Because Black American women constituted >90% of the prenatal clinic population, participation was limited to this group. Exclusion criteria for the study were known maternal HIV in pregnancy, no prenatal care, children with multiple malformation syndromes, and children from repeat pregnancies in the same participating mother. The inclusion criterion for the study was singleton birth between September 1989 and August 1991. Each caregiver provided written informed consent for herself and her child. Children provided verbal assent. All study procedures were approved by the Wayne State University Institutional Review Board.

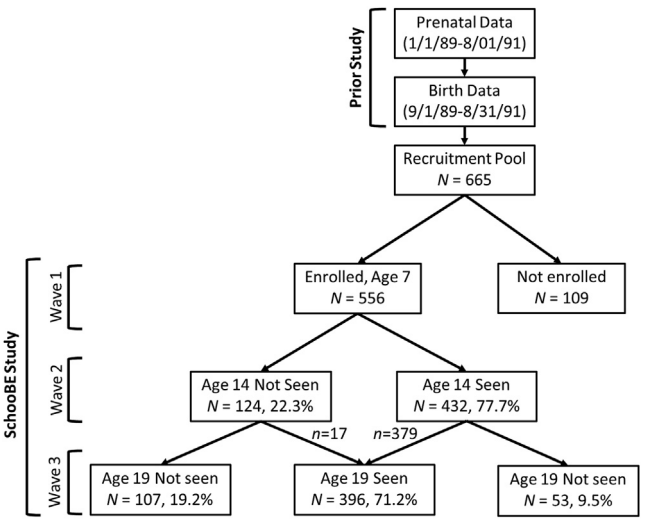
The SCHOO-BE study enrolled children from the hospital study, along with their primary caregiver, who still resided within the Detroit area at age 7. Data were then collected at age 7 years (n = 556), 14 years (n = 432), and 19 years (n = 396) to assess such family and community factors as caregiver education, home environment, and community violence exposure. Retention was strong: 396 participants were assessed at the 19-year visit, which constitutes 92% of the 432 children assessed at age 14 years and 71% of those assessed at age 7 years. Retention details and a study timeline are provided in the **Figure**.

Analyses were performed to evaluate attrition bias. Comparisons of participants included in the sample and those not included in the sample for all predictor risk score variables (see below) revealed that the only significant difference was in the postnatal drug/alcohol risk score (χ^2 = 4.1; P = .042). A higher proportion of participants who completed the age 19 visit than those who did not had a caregiver who used alcohol and/or drugs (27% vs 14.8%).

BP data were available for 388 of the 396 participants (98%) at the age 19 visit. Of these, 52.3% were female, their average age was 20.1 years (SD = 0.7; 2% were age >21 years), and 49.5% were always in the care of their biological mother. At the age 19 visit, caregivers were an average of 48.2 years old (SD = 9.0), 68.9% had a high school education, and <25% were married. **Table I** (available at www.jpeds.com) provides additional information about participant and family characteristics. The Wayne State University Institutional Review Board approved all aspects of this study prior to participant enrollment.

Evaluation of Childhood Adversity

Multiple dimensions of adverse childhood, adolescent, and young adult experiences were assessed. Because substantial developmental change occurred between each study wave (ie, between 7, 14, and 19 year olds), key constructs were



Note. Wave 2 (age 14) and Wave 3 (age 19) boxes report both sample size and percent of the age 7 sample who completed those study waves.

Figure. Study flowchart.

assessed with developmentally appropriate measures at each wave. Summary risk variables were constructed to reduce the number of analyses and decrease the potential for type I and II errors. These variables were defined based on birth outcomes; prenatal substance exposure; postnatal substance exposure; environmental factors at age 7, 14, and 19 years; and community violence exposure at age 7, 14, and 19 years. Cutpoints for risk categories were derived from established standards or from the bottom/top quartile if a standard was not available. The top or bottom quartile was used for this initial examination of what childhood risk factors would be associated with high blood pressure at age 19 years. Our rationale for this approach was that if any of these childhood risk factors impacted later blood pressure, then we would be most likely to detect that effect in the most highly exposed groups.

Birth Risk. Birth risk was determined based on 4 criteria: first prenatal visit at ≥30 weeks of gestation, gestational age at birth <37 weeks, intrauterine growth restriction, and maternal age <18 years.[28,29] Each child received a score of 0 (not at risk) or 1 (at risk) for each item. Total birth risk scores were calculated by summing the 4 items. Scores ranged from 0 to 4, and a score ≥2 constituted at risk (Table II).

Prenatal and Postnatal Substance Risk. Prenatal and postnatal risks were based on maternal (prenatal)[30] and/or caregiver (postnatal) use of alcohol or illicit drugs.[27] Alcohol use was determined from self-reported alcohol consumption prenatally (maternal) and at the age 14 assessment (caregiver).[27] Maternal prenatal drug use was assessed by self-report or biological assessment during pregnancy, including positive maternal and/or infant urine.[28] Meconium testing was used when available.

Prenatal alcohol risk was defined as an average weekly intake of ≥1 standard drink/day.[31] Prenatal substance risk was identified as heavy/persistent prenatal cocaine use,[32] ≥5 cigarettes/day, and/or any self-reported marijuana or opioid use. The total possible prenatal summary score was 5; a score ≥2 was considered to indicate at risk (Table II). Similar cutoffs were used to identify postnatal alcohol (≥1 standard drink/day) and cigarette (≥5 cigarettes/day) risk. Postnatal substance use was identified from caregiver self-report or biomarkers of opioids, cocaine, or marijuana in hair or urine. The total possible postnatal summary score was 5; a score ≥2 was classified as at risk (Table II).

Environmental Risk. An environmental risk metric was constructed for each testing age (7, 14, and 19 years; Table III). Several items were measured using a demographic interview. The Hollingshead Index of Social Position[33] was used to assess socioeconomic status (SES) by ranking occupation and education. Scores ranged from 8 to 66, with lower values indicating lower SES.[34] The Home Observation for Measurement of the Environment (HOME)[35] was used to assess living environment and parenting quality via a 59-item binary checklist completed during caregiver interviews at the age 7 year and 14-year waves. The HOME tool includes subscales for availability and quality of resources for the child in the home and has demonstrated adequate reliability (α = 0.87) and interobserver reliability (κ = .45-1).[36]

Less environmental data were available from the age 19 assessment, because some measures were either no longer age appropriate (eg, HOME, child protective services) or because data were missing due to lack of caregiver report. Only 88% of the caregivers completed the age 19 interview.

Table II. Birth risk, prenatal alcohol and drug exposure risk, and postnatal alcohol and drug exposure variables

Risk summary score	Variables	Risk definition	% risk +
Birth risk	Gestational age	<37 weeks	14.0
	First prenatal screen	≥30 weeks	25.7
	Maternal age	<18 years	8.7
	Growth restriction	<10th percentile	23.2
	Total items	4	–
	Summary score	≥2	13.7
Prenatal alcohol/drug	Alcohol	Average ≥1 drink/day	4.9
	Cocaine	Heavy/persistent	23.2
	Opioids	Any	5.4
	Marijuana	Any	15.7
	Cigarettes	≥5/day	56.2
	Total items	5	–
	Summary score	≥2	31.4
Postnatal alcohol/drug	Alcohol	≥1 drink/day	16.8
	Cocaine	Any	18.8
	Opioids	Any	4.1
	Marijuana	Any	27.6
	Cigarettes	≥5	35.6
	Total items	5	–
	Summary score	≥2	27.8

Table III. Environmental risk variable components and description descriptive at the age 7, 14, and 19 y study waves

Variables	7	14	19	Risk definition	7 y % risk +	14 y % risk +	19 y % risk +
SES	√	√	√	Bottom 25%	25.0	24.5	24.9
Caregiver education	√	√	√	<High school	31.3	32.1	27.4
Mother incarceration	√	√	√	Yes	19.5	25.0	24.4
Father incarceration	√	√	√	Yes	43.5	55.5	48.3
Caregiver incarceration		√		Yes	–	10.3	–
Custody status	√	√	√	Not with mom	23.0	39.5	50.5
Marital status	√	√		Not married	75.0	75.7	–
Spends time with father	√	√		No	57.9	65.7	–
HOME	√	√		Bottom 25%	23.7	22.3	–
Maternal death	√			Yes	1.4	–	–
Paternal death	√			Yes	5.4	–	–
Child protection		√		Yes	–	5.9	–
Total items	10	10	5				
Environmental risk +					32.2	25.6	26.0

The environmental items and risk thresholds included (1) SES (bottom quartile),[37,38] (2) caregiver education (bottom quartile),[37] (3) parent incarceration (yes for mother and/or father),[6,39] (4) custody status (not always with the mother),[6] (5) caregiver marital status (not married),[6] (6) time spent with father (none), (7) parental death (yes for mother and/or father),[6] and (8) living environment and parenting quality[6,40] (bottom quartile). Environmental risk scores were computed at each wave by summing the risk items for each age (**Table III**). Environmental risk cutpoints were identified as standard risk or were approximated by the top or bottom quartile. At each age, environmental at risk was coded as 1 and not at risk was coded as 0.

At age 7 years, the total risk score ranged from 0 to 10. A total risk score ≥5 was seen in 15.6% of the sample, whereas a score ≥4 identified 32.2% as at risk. At age 14, total risk scores ranged from 0 to 10 and a score ≥5 identified 25.6% of the sample considered at risk. At age 19, total risk scores ranged from 0 to 5, and a score ≥3 identified 26% of the sample as at risk.

Violence Exposure Risk. Age 7 community violence exposure was assessed using the child-reported Things I Have Seen and Heard (TISH) tool,[41,42] which includes 17 items about violence exposure ranging from hearing gunshots to witnessing stabbings to personal experience of violence. The frequency of each item is rated on a 5-point scale from "never" to "four or more times".[42] The TISH has adequate test-retest reliability (r = 0.81)[43] and internal consistency (α = 0.80-0.83).[44,45] Detailed information on the theoretically constructed TISH scales (eg, victimization, major witnessing, protection factors) has been published previously.[46]

Age 14 community violence exposure was assessed via both caregiver and child report with the Children's Report of Exposure to Violence (CREV)[47] and Survey of Children's Exposure to Community Violence (SCECV),[41] completed by the teen and caregiver, and the Safe and Responsive School Survey (SRSS).[48] The 29-item CREV asks about reported, witnessed, and experienced violence on a 5-point scale from "never" to "every day".[47] The total score has a stable factor structure (α = 0.75-0.93) and adequate test-retest reliability (r = 0.75).[47,49] The 20-item SCECV also evaluates community violence exposure.[50-52] The summed score indicates lifetime exposures to violence, with adequate test-retest reliability (r = 0.78-0.81).[52] The 45-item SSRS includes items assessing feelings related to safety, frequency of inappropriate behavior, and learning environment at school. Only the questions about safety were used in the present analyses. All items are measured on a 5-point scale from "strongly disagree" to "strongly agree."[48]

Teen (age 14 years) and caregiver-reported data from the CREV, SCECV, and the unsafe subscale from the SRSS were summed to create theoretically constructed summary scores similar to those at age 7 for victimization and major witness and witness minor. Because the age 14 measures do not include protection items, a teen protective scale could not be constructed. Examples of victimization and major witnessing items include "Somebody threatened to shoot me," "Somebody threatened to stab me," and "Grownups in my home threaten to stab or short each other." Examples of witness minor items include "Grownups in my home yell at each other, "I have seen somebody beaten up," and "I have seen somebody arrested."

Age 19 community violence exposure was assessed using a modified My Exposure to Violence survey, which assesses violence exposure in the prior year.[53] The original consisted of 18 items on witnessing and experiencing violence, with adequate test-retest reliability (r = 0.75-0.94) and internal consistency (α = 0.68-0.93). To better compare community violence exposure at age 19 to that at ages 7 and 14, additional items were added to measure "being told about" violence.

Community violence exposure risk scores were computed for each age (7, 14, and 19 years). The total community violence exposure risk score for each age was the sum of all items with risk defined as the top quartile. The number of items included in each risk score, risk definitions for each item, percentage at risk, and community violence exposure risk score cutpoints are provided in **Table IV**. High risk was coded as 1, and low risk was coded as 0.

BP Measurement. BP was measured by trained personnel using an oscillometric device (Dinamap 200x; GE Monitoring Systems) with an appropriate-sized cuff and after the participant had been sitting quietly for ≥10 minutes. Two or more readings were taken, with at least 10 minutes between measurements. Measurements differing by ≥3 mm Hg were followed by a third reading. The 2 closest measurements were averaged to determine a final value. Age 19 BP was categorized as normal (<120/80 mm Hg), elevated (120-129 mm Hg for systolic and <80 mm Hg for diastolic), stage 1 hypertension (130-139 mm Hg for systolic or 80-89 mm Hg for diastolic), or stage 2 hypertension (≥ 140 mm Hg for systolic or ≥ 90 mm Hg for diastolic).[54] For study analyses, systolic BP and diastolic BP readings were dichotomized into normal BP or high BP (HBP; including elevated, stage 1, and stage 2) groups.

Table IV. Violence exposure risk variable descriptions for the age 7, 14, and 19 y study waves

Ages	Variables	Risk definition	% risk +
7 y	Experienced	Top 25%	28.8
	Witnessed	Top 25%	27.2
	Protection	Bottom 25%	27.6
	Total items	3	
	Summary score	≥2	21.2
14 y	Experienced/witnessed major	Top 25%	25.2
	Witnessed minor	Top 25%	25.0
	Total items	2	
	Summary score	≥2	16.0
19 y	Experienced	Top 25%	26.9
	Witnessed	Top 25%	26.4
	Total items	2	
	Summary score	≥2	18.3

Statistical Analyses. All analyses were conducted using SPSS version 26 (IBM). Two logistic regressions were conducted to examine the impact of 8 of the 9 childhood risk factors as well as BMI on age 19 BP. Environmental risk at age 19 was not included in the analyses because of the strong the relationship between environmental risk at age 14 and environmental risk at age 19 ($\chi^2 = 238$; df = 1; $P < .001$). This strong relationship and the higher rate of missing age 19 caregiver data drove the decision to only include age 7 and age 14 environment risk scores. The first regression model evaluated age 19 systolic BP dichotomized as normal (0) or HBP (1); the second model evaluated dichotomized age 19 diastolic BP. For both models, all summary risk variables and age 19 BMI were entered simultaneously. Risk variables significantly associated with BP were examined by χ^2 analysis. This additional analysis was performed to provide proportional HBP information across the risk groups.

Results

At age 19, 45.8% of the participants had normal BMI (<25), 23.8% were overweight (BMI ≥25 and <30), and 30.4% were obese (BMI ≥30). In addition, 28% and 20% of participants were classified as HBP for systolic and diastolic BP, respectively. Mean systolic BP was 113.1 mmHg (SD = 12.3), and mean diastolic BP was 72.0 (9.0) mmHg. Elevated BP findings were also noted at age 14, with 20.1% and 10.6% of participants classified as HBP for systolic and diastolic BP, respectively. Mean systolic BP was 109.2 (11.9) mmHg and mean diastolic BP was 66.5 (9.6) mmHg. At age 7, 14.9% and 30.6% were HBP for systolic and diastolic BP, respectively, mean systolic BP was 99.8 (8.1) mmHg, and mean diastolic BP was 63.9 (9.1) mmHg.

Logistic Regression Analyses

Logistic regression models assessed associations among 8 risk factors (prenatal and postnatal drug/alcohol exposure, birth risk, environmental risk at 7 and 14 years, and community violence exposure at 7, 14, and 19 years), as well as BMI, and age 19 systolic HBP (model $\chi^2 = 29.4$; $P = .001$), and diastolic HBP (model $\chi^2 = 24.8$; $P = .003$). The results indicated positive relationships between BMI and both systolic and diastolic HBP classification (Table V). For every unit increase in BMI, there was 6% (OR, 1.062; 95% CI, 1.029-1.096) increased odds of systolic HBP and 8% (OR, 1.082; 95% CI, 1.047-1.120) increased odds of diastolic HBP.

Controlling for all covariates, age 7 and 19 community violence exposures were associated with increased odds for systolic HBP at age 19. Participants identified as at risk for community violence exposure at age 7 and age 19 had, respectively, 2.2-fold (95% CI, 1.2-3.9) and 2.0-fold (95% CI, 1.1-3.7) greater odds of being identified as HBP at age 19. Age 14 community violence exposure was not associated with higher odds of systolic or diastolic HBP at age 19. Diastolic HBP at age 19 was not predicted by community violence exposure at any age. None of the environmental risk factors, birth risk, or prenatal and/or postnatal alcohol and drug use risk factors were related to the odds of HBP at age 19 (Table V).

Table V. Logistic regressions predicting hypertension as measured by both systolic and diastolic BP

Predictors	B	SE	Wald χ^2	*P* value	OR	95% CI
Systolic BP						
BMI at age 19 y	**0.06**	**0.02**	**13.8**	**<.001**	**1.06**	**1.03-1.10**
Violence risk						
Age 7	**0.78**	**0.29**	**7.4**	**.007**	**2.19**	**1.24-3.86**
Age 14	−0.41	0.36	1.3	.253	0.66	0.33-1.34
Age 19	**0.68**	**0.32**	**4.5**	**.034**	**1.96**	**1.05-3.66**
Birth risk	0.11	0.17	0.4	.524	1.12	0.80-1.57
Prenatal drug/alcohol risk	−0.25	0.29	0.8	.382	0.78	0.44-1.37
Postnatal drug/alcohol risk	0.02	0.28	0.0	.933	1.02	0.59-1.78
Environmental risk						
Age 7	−0.06	0.29	0.0	.845	0.95	0.54-1.66
Age 14	0.22	0.28	0.6	.442	1.24	0.71-2.17
Diastolic BP						
BMI at age 19	**0.08**	**0.02**	**21.2**	**<.001**	**1.08**	**1.05-1.12**
Violence risk						
Age 7	0.18	0.33	0.3	.598	1.19	0.62-2.29
Age 14	−0.03	0.38	0.0	.930	0.97	0.46-2.05
Age 19	−0.11	0.38	0.1	.782	0.90	0.42-1.92
Birth risk	0.20	0.19	1.1	.289	1.22	0.84-1.78
Prenatal drug/alcohol risk	0.17	0.31	0.3	.583	1.19	0.64-2.19
Postnatal drug/alcohol risk	−0.22	0.32	0.5	.486	0.80	0.43-1.50
Environmental risk						
Age 7	−0.08	0.32	0.1	.798	0.92	0.50-1.71
Age 14	0.06	0.31	0.0	.839	1.07	0.58-1.96

Results for independent variables that are statistically significant ($\alpha < 0.05$) predictors of high BP are in bold type.

Risk variables identified as significant in regression analyses (ie, age 7 and 19 community violence exposure) were examined further using χ^2 analysis. At age 19, 43.2% of the age 7 community violence exposure at risk group were categorized as systolic HBP, compared with 24.7% of those not in the at risk group (OR, 2.32; 95% CI, 1.39-3.88), and 36.2% of the age 19 community violence exposure at risk group were in the systolic HBP group, compared with 26.1% of the not at risk group (OR, 1.61; 95% CI, 0.93-2.79).

Discussion

Our results indicate that community violence exposure at age 7 and 19 years, but not at age 14 years, were associated with systolic HBP at age 19 in this cohort of young Black Americans. As in prior studies, BMI significantly predicted age 19 systolic and diastolic HBP.[55,56] Relationships between age 7 and age 19 community violence exposure and systolic HBP were significant after controlling for BMI. Participants in the HBP group at age 19 were twice as likely to have been in the high community violence exposure groups at age 7 and/or 19. Age 19 HBP status was not significantly associated with other risk variables, including prenatal and postnatal

drug/alcohol exposure, birth risk, environmental risk at age 7 and 14, and community violence exposure at age 14. The absence of a significant association between age 14 community violence exposure and age 19 HBP is intriguing given that age 7 and 19 community violence exposure were significantly associated with HBP at age 19. These results may reflect developmental changes in sensitivity to community violence exposure, but future prospective longitudinal studies are needed to assess the replicability of the age effects reported here.

Our finding of a positive association between prospectively assessed childhood community violence exposure and later systolic HBP is consistent with the conclusions of a review of 15 studies by Suglia et al that identified positive associations between retrospectively reported childhood adversity and poor cardiovascular outcomes in adulthood.[13] Of these, 4 studies examined associations between child abuse and/or community violence exposure and adult hypertension, all of which reported positive associations; in contrast, 8 studies examined associations between adult violence exposure and BP, and only 1 study reported a positive association.[13] This pattern of results, in conjunction with the findings of the present study, suggest that childhood and adolescence may be sensitive periods for hypertension risk related to violence exposure.

Su et al examined the impact of retrospectively reported childhood adversity on later BP using the Adverse Childhood Experiences (ACEs) scale, a composite measure which includes adversities including abuse, neglect, and parental divorce.[10] Total ACE scores were not significantly associated with adult BP. However, after age 30, individuals exposed to multiple ACEs had faster increased in systolic and diastolic BP than those with fewer ACEs. Increases in systolic BP were not associated with behavioral risk factors such as physical inactivity, smoking, or illicit drug use, whereas an increase in diastolic BP was associated with drug use.[10] These findings suggest that a positive association between childhood adversity and elevated systolic BP in adulthood may emerge later in adulthood (after age 30), and that this association is not explained by the effects of ACEs on health behaviors.

Our results broadly parallel multiple studies that reported differential effects of threat-type childhood adversities (eg, violence exposure) versus deprivation-type adversities (eg, neglect) on health outcomes[57-59] and shed additional light on the impact of adversity timing and type. Childhood adversity is often studied using broad measures (eg, ACEs) that (1) do not capture differential exposure to threat versus deprivation, (2) have limited temporal resolution (ie, considering <18 years as 1 developmental period), and (3) rely on cross-sectional, retrospective data that might not provide a robust characterization of childhood experiences.[13,17,25] This approach may obscure important associations between specific dimensions of adversity or developmental periods and health outcomes. A more granular approach to examining the impact(s) of adversity and developmental timing may facilitate identification of relevant mechanisms.

We collected data from caregivers and children to characterize children's exposure to various types of community violence exposure, as well as their home, family, neighborhood, and school environments during childhood (age 7), adolescence (age 14), and emerging adulthood (age 19). This decreases the potential for measurement errors due to memory bias, motivation, and measurement features.[25] Our findings of a significant association between age 7 community violence exposure and age 19 systolic HBP suggest that early to middle childhood may be a sensitive developmental period for later hypertension risk. Identification of early risk factors is a critical step toward delineating the etiology of poor cardiovascular outcomes, and our results suggest that childhood community violence exposure is an early risk factor for hypertension in Black Americans.[13]

Interpretation of our results may be facilitated by applying a life-course lens. Life-course theories propose that the impact of experiences depends on their developmental timing because of developmentally specific changes in biology, cognition, and social context.[60] As such, the developmental age of exposure to, for instance, community violence may constitute a moderator or mediator of its effect(s). Here community violence exposure before age 7 and in the year before the age 19 visit was associated with elevated risk for HBP. In contrast, community violence exposure reported at age 14 was not associated with HBP at age 19. These results may indicate that early childhood is a sensitive period for the development of stress-response systems, including the sympathetic-adreno-medullar and hypothalamus-pituitary-adrenal axes,[61] that may mediate the relationship between community violence exposure and cardiovascular risk across the lifespan.[62,63] The significant association between HBP and community violence exposure reported at the age 19 visit may reflect the impact of recent traumatic stressors on BP.[64,65] Alternatively, these results may reflect differences in the measurement of community violence exposure at each age (see Methods for details). In particular, the age 14 community violence exposure assessment included both parent and children report, as well as information about school context, whereas at the age 7 and 19 visits, only self-reported community violence exposure was assessed. Future studies should extend our initial examination of how the developmental timing of community violence exposure impacts later cardiovascular risk.

The next step in elucidating the pathway(s) from childhood violence exposure to adult hypertension is to identify mechanisms that mediate associations between community violence exposure and later cardiovascular health. Threats such as childhood adversity and community violence exposure can impact cardiovascular health by activating the sympathetic nervous system (SNS),[66] affecting adrenocortical and renin-angiotensin systems,[67] and altering modulation of immune system function. The effects of SNS activation on BP are mediated through the renin-angiotensin system: SNS activation triggers the release of renin, renin elevates levels of angiotensin, and increased angiotensin raises BP.[67]

Almuwaqqat et al also reported a positive association between childhood adversity and adverse cardiovascular outcomes following myocardial infarction, mediated by elevated C-reactive protein.[68] These downstream effects of SNS activation may have lasting effects on cardiovascular function.

There are important questions beyond the scope of this study and limitations that should be considered in future research. We could not assess environmental risk factors from the prenatal and birth time points, because those data were not collected. We did not directly assess the mechanisms through which community violence exposure increases the risk of hypertension. We evaluated BP at age 19, and these associations could shift with age. Multiple aspects of environmental risk during childhood were controlled for in the analysis, but health behaviors and other risk factors at age 19 were not evaluated. A family history of hypertension was not evaluated in the SCHOO-BE cohort. There was approximately 28% attrition from the study between the age 7 and age 19 visits, and participants with missing data at age 19 were excluded from the final analyses. Because this is a preliminary examination of how multiple dimensions of risk impact BP, we conducted a logistic regression that did not account for potential interrelationships among these risk factors. Therefore, the present findings leave open the prospect of more complex pathways (eg, mediations) from risk exposures to BP outcomes. Finally, we were not able to consider the duration and sequencing impacts of community violence exposure on BP, tempering any conclusions we can draw about the impact of developmental timing of community violence exposure. Future studies examining potential mechanisms that may link community violence exposure to cardiovascular risk and incorporate familial, genetic, environmental, and behavioral risk in adulthood using causal modeling approaches will be critical for understanding both risk and resilience factors that impact long-term cardiovascular outcomes.

Our study also was not designed to consider potential protective factors that might moderate the association between community violence exposure and cardiovascular risk. Health behaviors that could serve as moderators, such as exercise[69] and nutrition,[70] should be explored in future studies. In addition, familial and social support may buffer children against the negative effects of community violence exposure by decreasing chronic stress and facilitating the development of healthy emotion regulation strategies,[40,71] which in turn may buffer the cardiovascular system against higher or more frequent physiological stress responses.[72] Identification of protective factors that promote resilience following exposure to community violence exposure can inform interventions and may shed light on the mechanisms through which community violence exposure may increase the risk of hypertension.

Our study addresses the National Heart, Lung, and Blood Institute's call for greater understanding of environmental, psychosocial, and behavioral factors affecting cardiovascular health in Black Americans[26] and provides compelling prospective evidence that childhood community violence exposure is linked to elevated BP in young adulthood. The strong evidence obtained through BP tracking from childhood to adulthood implies that early violence exposure contributes to an increased risk of hypertension.[2] Black American children are exposed to more childhood community violence compared with other racial/ethnic groups in the US,[6] and this disparity may contribute to racial disparities in adult hypertension. Future work could estimate how much of the disparate hypertension burden for Black Americans is due to community violence exposure. Our results also highlight the potential impact of interventions to reduce community violence exposure on hypertension. Although community violence exposure is pervasive in many communities, it also is an identifiable target for intervention at the community level.[73] Reducing community violence exposure could be an important step toward reducing the hypertension burden for Black Americans. ■

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Reprint requests: Anaïs F. Stenson, PhD, Wayne State University School of Medicine, Tolan Park Medical Building, Suite 2C, 3901 Chrysler Service Drive, Detroit, MI 48201. E-mail: astenson@wayne.edu

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Table I. Study cohort characteristics at age 19 years (N = 388)

Characteristics	Value
Teen	
Age, y, mean (SD), range	20.1 (0.7), 18.2-23.0
Sex, % male	47.7
BMI at age 19 y, mean (SD), range	27.8 (7.6), 16.8-57.7
Birth mother as primary caregiver, %	81.2
Always with mother, %	49.5
Caregiver	
Education, y, mean (SD), range	12.2 (2.0), 2-21
SES, mean (SD), range	28.5 (11.3), 8-66
Age at 19-y follow-up mean (SD), range	48.2 (9.0), 25.0-81.2
Marital status, % married	21.6