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Blood Pressure, HIV, and Cocaine Use Among Ethnically and Racially Diverse Individuals

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Abstract

Objectives: Racial minorities are at greater risk of cardiovascular disease (CVD), and CVD is the primary cause of mortality among human immunodeficiency virus (HIV)–infected individuals. Cocaine use also has been associated with hypertension. This study examined the contribution of lifestyle factors to systolic, diastolic, and mean arterial pressure (MAP) among people living with HIV and cocaine users from racially and ethnically diverse backgrounds.

Methods: Participants (N = 401: 213 men, 188 women) aged 18 to 50 years with no history of CVD were recruited from South Florida. A total of 200 participants were HIV-cocaine-infected, 100 were HIV-infected individuals with no history of cocaine use, and 101 were HIV-uninfected individuals with cocaine abuse or dependence. Carotid intima-media thickness and plaque, blood pressure (BP), and lifestyle risk were assessed.

Results: Mean age was 36 years (standard deviation 9.33); the majority (62%) were African American. Carotid plaques were identified in 23% of participants; 42% were obese, 68% engaged in ≥150 minutes of weekly exercise, and 68% were smokers. Sex, body mass index (BMI), and diet were associated with systolic blood pressure. Age, BMI, cannabis use, and diet were associated with diastolic BP and MAP.

Conclusions: Age, BMI, cannabis use, and diet were associated with increased diastolic BP and MAP. Cocaine did not emerge as a significant predictor of CVD after controlling for cannabis dependence. Cocaine and HIV lacked significant association with CVD, possibly because the majority of the sample was younger than age 40. Lifestyle modifications and substance abuse counseling may be important in preventing CVD among those without a history of CVD.

Keywords

cardiovascular disease; carotid plaque; intima-media thickness; ultrasonography

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Hypertension has been associated with obesity, smoking, and alcohol consumption.[1] In the United States, the association between cardiovascular disease (CVD) and obesity and weight gain emerges as early as adolescence.[2] Elevated body mass index (BMI) has been associated with increased systolic blood pressure (SBP), independent of smoking and alcohol consumption,[1] whereas controlled hypertension has been associated with lower low-density lipoprotein-cholesterol (LDL-C) levels.[3]

Health disparities in CVD outcomes among African Americans have been recognized for decades. African American men in particular are more likely to be prehypertensive in comparison to white men (58.6% and 47.4%, respectively), and prehypertension is more common among African Americans, even after accounting for common risk factors such as age, sex, and smoking status.[4] Greater proportions of African Americans are diagnosed and treated for hypertension than whites and Hispanics, but African Americans are less likely to respond to treatment as compared with whites.[5] In addition, African Americans are at greater risk of myocardial infarction, arterial stiffness, and microcirculation than whites, despite similar levels of glucose and LDL-C.[6] A large proportion of the variance in these CVD outcomes have been partially accounted for by a greater prevalence of smoking and obesity among African Americans. Human immunodeficiency virus (HIV) infection and comorbid substance use, including cocaine use, are most prevalent among ethnic and racial minorities and may contribute to racial and ethnic differences in CVD outcomes.[7,8] Mean arterial pressure (MAP) values indicate blood vessel pressure and help to determine the efficacy of the cardiovascular system. Arterial stiffness worsens with increased MAP, highlighting the crucial role of MAP as a cardiovascular risk indicator.[9] Overall, African Americans had been found to have higher MAP values in comparison with whites, also indicating decreased efficacy of the endothelial lining of blood vessels.[6]

CVD is the primary cause of mortality among HIV-infected individuals; in this group, smoking and having a detectable viral load have been found to increase risk of CVD.[10–12] Blood vessel decay, kidney problems, and microbial translocation also contribute to a greater risk of hypertension among individuals living with HIV.[11,13]

This study examined the relative contribution of lifestyle factors to SBP, diastolic blood pressure (DBP), and MAP among minority individuals from ethnically and racially diverse backgrounds without a history of CVD. We contrasted predictors of BP among HIV-infected individuals and cocaine-using individuals without a history of CVD with the aim of identifying factors associated with SBP, DBP, and MAP before CVD is diagnosed or treated. Based on past research and theory,[8,10–12] it was hypothesized that cocaine users and HIV-infected individuals would have higher arterial pressures than their uninfected non-cocaine-using counterparts, after controlling for risk factors associated with increased BP such as smoking, BMI, substance dependence and depression, and sex. In contrast with previous studies,[8,10–12] the participants included in this study were young and had not been diagnosed as having or been treated for CVD. This study addressed younger substance-using individuals with no previous CVD diagnosis, to identify novel factors that may be associated with BP elevation. It was anticipated that results could provide evidence to guide preventive strategies targeting this population, such as smoking cessation, exercise, and diet, to reduce CVD risk in a potentially at-risk population.

Method

Participants and Procedures

All of the participants provided written informed consent before study participation. Participant data were drawn from a larger study examining CVD in HIV, and deidentified data underlying the present study can be obtained from the corresponding author. Recruitment began in December 2014 and continued through August 2017. Inclusion criteria included living in South Florida, aged 18 to 50 years, being HIV-infected or HIV-uninfected, and having or not having a history of cocaine abuse. Exclusion criteria included a history of hepatitis C, diabetes mellitus, hypertension, myocardial infarction, transient ischemic attacks, bypass surgery or angioplasty, and currently taking statins. Overall, 213 men and 188 women (N = 401) were enrolled and recruited into groups of HIV-uninfected individuals without a history of cocaine abuse or dependence (HIV− cocaine− n = 200), HIV-infected individuals without a history of cocaine abuse or dependence (HIV+ cocaine− n = 100), and HIV-uninfected individuals who met the criteria for cocaine abuse or dependence (HIV− cocaine+ n = 101).

Study Design

The study used a prospective cross-sectional design. Participants were recruited using intact group sampling. Further details on study recruitment, participants, and procedures has been published previously.[14] We obtained approval from the University of Miami Miller School of Medicine institutional review board for this study.

Measures

BP and BMI—Participants underwent a physical assessment that included measuring weight and height and three measurements of SBP and DBP. From these values, MAP was calculated using MAP = SBP + 2 (DBP)/3 and average values for DBP and SBP. The MAP and average SBP and DBP values were used as the outcome variables for this investigation.

Demographic and Lifestyle Assessment—Information assessed included basic demographics and lifestyle, including age, sex, race, and current or past cigarette smoking status. Cigarette smoking status was assessed dichotomously (yes/no).

Exercise—Weekly exercise was calculated using the International Physical Activity Questionnaire,[15] which assesses how many days the respondent exercised for ≥10 minutes in the past week and how many minutes of physical activity they were engaged in every day. The final value of weekly exercise was computed by calculating the product of the number of days of physical activity and the number of daily minutes of physical activity. Scores were dichotomized into one of two categories according to recommended guidelines: those who exercised <150 minutes/week and those who exercised >150 minutes/week.[16]

Eating Habits—The Rapid Eating Assessment for Participants (REAP) was used to evaluate participants' eating habits,[17] and the individual REAP score represented consumption of different types of food. A higher REAP score reflected a healthier diet. Reliability for this scale in this sample was acceptable ($\alpha = 0.75$).

Depressive Symptoms—The Center for Epidemiological Studies-Depression scale was used to assess depression.[18] The values were computed based on the number and type of depressive symptoms experienced during the course of the previous week. The scores range from 0 to 60, with higher scores reflecting greater levels of depression. In this sample, internal consistency was excellent ($\alpha = 0.91$).

Substance Dependence—The Structured Clinical Interview for the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, nonpatient version was used to measure cocaine, cannabis, stimulant, and alcohol use, abuse, and dependence.[19] The interview evaluates the duration and frequency of substance use. From these responses, a dichotomous variable was used to represent any cannabis, stimulant, and alcohol dependence, such that those individuals meeting the criteria for dependence were assigned one (1); a zero (0) was assigned for nondependence.

Data Analysis

Univariate analyses were used to describe demographic characteristics and lifestyle risk factors. Bivariate analyses (t or Mann-Whitney tests and χ^2 or Fisher exact tests) were used to compare the study outcomes, SBP, DBP, and MAP, by demographic characteristics and lifestyle risk factors among participants. If variables were associated with these outcomes at $P < 0.10$ in bivariate analyses, then they were included in three subsequent linear regression models. The final analyses were three separate multivariable linear regression models using SBP, DBP, and MAP as the outcome. Group membership (HIV− cocaine−, HIV+ cocaine−, and HIV− cocaine+) was included as a predictor regardless of association with the outcomes in the bivariate analyses. All of the analyses were conducted using SPSS version 24,[20] and statistical significance was defined as $P < 0.05$.

Results

Demographic and Psychosocial Characteristics of Participants

Participants were a median age of 36 years (standard deviation 9.33). A little more than half (52%) were men and one-third (62%) were African American. Nearly half (42%) were obese. Two-thirds of participants (68%) reported ≥150 minutes of weekly exercise, and 68% were current or past smokers. The prevalence of alcohol, cannabis, and stimulant dependence was 11%, 10%, and 3%, respectively. HIV-infected individuals had higher DBP levels than other groups and were less likely to report exercising ≥150 minutes/week. Cocaine users were more likely to report current or past smoking and endorse alcohol, stimulant, and/or cannabis dependence. Cocaine users also reported higher levels of depression. Comparisons between HIV− cocaine−, HIV+ cocaine−, and HIV− cocaine+ groups are detailed in Table 1.

Multivariable Associations with SBP and DBP and MAP

Age, HIV+ cocaine− group status, HIV− cocaine+ group status, BMI, stimulant dependence, and eating habits were associated with DBP in bivariate analyses. In multiple linear regression analyses, sex ($P < 0.001$), BMI ($P < 0.001$), and diet ($P = 0.048$) were associated

with SBP. Age, BMI, cannabis use, and diet were associated with DBP and MAP ($P < 0.05$). Summaries of the final multiple linear regression models are presented in Table 2.

Discussion

This study examined the relative contribution of lifestyle factors to MAP and SBP and DBP among individuals from ethnically and racially diverse backgrounds younger than age 50 years. Predictors of MAP and SBP and DBP were contrasted among HIV-infected individuals, cocaine-using individuals and HIV-uninfected noncocaine users. Although differences were apparent between cocaine users and HIV-infected individuals (ie, cocaine use was associated with less eating, smoking, substance dependence, and depression, and HIV infection was associated with higher DBP), neither HIV infection nor cocaine use was associated with MAP when controlling for other factors associated with BP (eg, smoking, BMI, substance use, depression, sex).

Cocaine and stimulant use have been associated with weight loss, appetite reduction, dysregulation of fat, and leptin interference,[21,22] and HIV infection has been associated with weight loss and metabolic disturbances, increased resting energy expenditure, and wasting.[23,24] Having a healthy weight and specifically having a BMI score in the normal range is associated with normal BP levels. In contrast, cannabis use often is related to a high-calorie diet because of increased appetite during periods of use, periods that would be expected to be more common among those meeting the criteria for cannabis dependence.[25] These periods of increased appetite among cannabis-dependent individuals may result in weight gain, which may have contributed to the association between cannabis dependence and DBP and MAP. In a previous study, the association between cannabis dependence and hypertension disappeared after accounting for alcohol use; the opposite pattern was seen in the present study.[25] In other words, the association between alcohol consumption and BP disappeared after accounting for cannabis dependence. It is possible that marijuana use, which has been associated with a reduction in inflammation, may have affected results. Further research is needed to estimate the impact of marijuana use on negative outcomes associated with substance use.

In contrast with previous studies that identified associations between cocaine use and CVD,[26–30] our study did not find an association after controlling for other CVD risk factors. The impact of cocaine use and HIV infection may have been diminished by the relative youth of the study sample, most of whom were younger than age 40, as well as the fact that cocaine users in this study reported lower levels of unhealthy eating habits compared to their non-drug-using counterparts, including those in both the HIV-infected and -uninfected groups. Lower levels of unhealthy eating habits may have led to lower BMI scores, thereby contributing to reduced risk or a nonsignificant association with increased blood pressure. CVD is highest among older populations, specifically those older than age 60.[31] In addition, cardiovascular complications are rare among cocaine users, in part because of the young age of many cocaine users,[32] as well as less common among younger HIV-infected individuals. The effect of cocaine use at younger ages may not be reflected in CVD risk, or its effect may be cumulative, such that its effect would not have been captured by the cross-sectional design of this study.[33,34] Overall, most individuals living with HIV experience CVD

symptoms with advanced age,[35,36] and as found in this study, increased age was associated with increased BP.

Results support previous studies identifying high BMI and poor diet as risk factors for hypertension and CVD,[37–43] which highlights the value of interventions that target weight control, diet, and exercise to reduce CVD risk. The results of this study are limited by self-reported drug use and lack of data regarding the amount of cocaine or other drug use. Studies suggest that the effects of cocaine on cardiovascular health are largely dose dependent or the result of long-term use of the drug.[44] Similarly, immune function status (eg, CD4 counts) and time since diagnosis were not available, which could have contributed to cardiovascular outcomes. Lastly, the cross-sectional design of the study limits causal interpretation of the study findings. Longitudinal studies may reveal causal mechanisms associated with substance dependence, HIV infection, and cardiovascular outcomes, and should further investigate factors that may protect high-risk HIV-infected patients from CVD.

This study identified predictors of BP among younger cocaine users and HIV-infected individuals. Overall, lifestyle factors, including weight reduction, diet, and exercise, remain the most influential factors on healthy BP. These findings underscore the importance of established clinical directives for cardiovascular health in these high-risk groups to limit the growing burden of CVD. As a result of increased access to care associated with the specific needs of those living with HIV, incorporating prevention and intervention strategies into routine HIV care may represent an opportunity to reduce CVD risk in this vulnerable group. Treatment for substance use disorders also may benefit from the inclusion of psychoeducational components addressing CVD risk reduction.

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Key Points

- This study examined the relative contribution of lifestyle factors to mean arterial pressure (MAP) and systolic and diastolic blood pressure among individuals younger than age 50 years from ethnically and racially diverse backgrounds.
- Predictors of MAP and systolic and diastolic blood pressure were contrasted among individuals infected with the human immunodeficiency virus (HIV), cocaine-using individuals, and HIV-uninfected noncocaine users.
- Although differences were apparent between cocaine users and HIV-infected individuals, neither HIV infection nor cocaine use were associated with MAP when controlling for other factors associated with blood pressure.
- These findings underscore the importance of established clinical directives for cardiovascular health in these high-risk groups to limit the growing burden of cardiovascular disease.

Table 1.

Comparison of HIV− cocaine−, HIV+, and cocaine+ participants

	All, N = 401 Mean (SD) n (%)	HIV− cocaine−, n = 200 Mean (SD) n (%)	HIV+ cocaine−, n = 100 Mean (SD) n (%)	HIV− cocaine+, n = 101 Mean (SD) n (%)	F/χ^2, P
Age y	35.03 (9.33)	34.32 (8.98)	34.93 (10.54)	36.55 (8.60)	1.95, 0.143
Sex					**64.1, <0.001**[a,b,c]
Male	213 (53.1)	102 (51.0)	36 (36.0)	75 (74.3)	
Female	188 (46.9)	98 (49.0)	64 (64.0)	26 (25.7)	
Race					**19.88, 0.001**[a,c]
White	45 (11.0)	22 (11.0)	5 (5.0)	18 (18.0)	
African American	250 (62.0)	123 (62.0)	78 (78.0)	49 (49.0)	
Hispanic	106 (26.0)	55 (28.0)	17 (17.0)	34 (34.0)	
SBP	118.95 (13.89)	118.35 (13.82)	119.48 (16.17)	119.60 (11.46)	0.37, 0.693
DBP	74.60 (9.62)	73.91 (9.55)	76.75 (10.55)	73.87 (8.51)	**3.34, 0.036**[a]
MAP	89.38 (10.58)	88.72 (10.52)	90.98 (11.98)	89.11 (9.06)	1.58, 0.207
BMI					
Normal	111 (28.0)	54 (27.0)	30 (30.0)	27 (27.0)	
Overweight	123 (31.0)	64 (32.0)	24 (24.0)	35 (35.0)	
Obese	167 (42.0)	82 (41.0)	46 (46.0)	39 (39.0)	3.05, 0.550
Weekly exercise, min					
≤149	127 (32.0)	55 (28.0)	44 (44.0)	28 (28.0)	
≥150	272 (68.0)	144 (72.0)	55 (56.0)	73 (72.0)	**9.66, 0.008**[a,c]
Smoking					**63.74, <0.001**[b,c]
Nonsmoker	167 (42.0)	109 (55.0)	50 (50.0)	8 (8.0)	
Current/past	234 (58.0)	91 (46.0)	50 (50.0)	93 (92.0)	
Alcohol dependence					
No	356 (89.0)	194 (97.0)	93 (93.0)	69 (68.0)	
Yes	45 (11.0)	6 (3.0)	7 (7.0)	32 (32.0)	**57.80, <0.001**[b,c]
Cannabis dependence					
No	337 (90.0)	179 (98.0)	89 (92.0)	69 (73.0)	**46.72, <0.001**[a,b]
Yes	37 (10.0)	3 (1.0)	8 (8.0)	26 (27.0)	
Stimulant dependence					**22.89, <0.001**[b,c]
No	364 (97.0)	182 (100.0)	96 (99.0)	86 (91.0)	
Yes	10 (3.0)	0 (0.0)	1 (1.0)	9 (10.0)	
Depression (CES-D)	13.19 (11.81)	8.76 (9.41)	13.54 (10.79)	21.64 (12.42)	**49.83, <0.001**[a,b,c]
Eating habits (REAP)	51.33 (8.22)	53.09 (8.47)	50.51 (7.43)	48.68 (7.69)	**10.79, <0.001**[a,b]

Bold values indicate significance at $P < 0.05$. BMI, body mass index; CES-D, Center for Epidemiologic Studies-Depression Scale; DBP, diastolic blood pressure; HIV, human immunodeficiency virus; MAP, mean arterial pressure; REAP, Rapid Eating Assessment for Patients; SBP, systolic blood pressure; SD, standard deviation.

[a]Significant difference at $P < 0.05$ between HIV− cocaine− and HIV+ cocaine−.

[b] Significant difference at $P < 0.05$ between HIV− cocaine− and HIV− cocaine+.

[c] Significant difference at $P < 0.05$ between HIV+ cocaine− and HIV− cocaine+.

Table 2.

Summary of multivariable models predicting BP and MAP

	SBP			DBP			MAP		
	B	**β (SE)**	***P***	**B**	**β (SE)**	***P***	**B**	**β (SE)**	***P***
Constant	105.724	(5.890)	<0.001	60.958	(4.105)	<0.001	73.159	(4.585)	<0.001
Age	0.218	0.143 (0.074)	0.003	0.231	0.221 (0.049)	**<0.001**	0.232	0.201 (0.057)	**<0.001**
Sex	−5.560	−0.197 (1.402)	**<0.001**	−0.150	−0.008 (0.973)	0.877	—	—	—
HIV+	0.961	0.029 (1.590)	0.565	1.810	0.082 (1.810)	0.101	1.108	0.045 (1.221)	0.365
Cocaine+	−2.331	−0.072 (1.857)	0.210	−1.334	−0.060 (1.221)	0.275	−1.417	−0.058 (1.476)	0.338
BMI	1.788	0.419 (0.090)	**<0.001**	0.440	0.342 (0.062)	**<0.001**	0.523	0.368 (0.067)	**<0.001**
Weekly exercise	—	—	—	−1.259	−0.061 (1.008)	0.212	−0.684	−0.030 (1.093)	0.532
Smoking status	−0.209	−0.007 (1.531)	0.891	—	—	—	0.032	0.001 (1.156)	0.978
Alcohol status	2.102	0.046 (2.276)	0.356	—	—	—	—	—	—
Cannabis	5.858	0.124 (2.345)	0.013	4.181	0.130 (1.574)	**0.008**	4.712	0.132 (1.748)	**0.007**
Stimulant	—	—	—	−4.128	−0.069 (2.828)	0.145	−4.823	−0.073 (3.125)	0.124
Depression	—	—	—	—	—	—	0.046	0.051 (0.046)	0.320
Eating habits	−0.198	−0.115 (0.083)	**0.018**	−0.136	−0.115 (0.056)	**0.016**	−0.156	−0.120 (0.064)	**0.015**
F		12.400			8.461			9.340	
R^2		0.245			0.252			0.255	

BMI, body mass index; BP, blood pressure; DBP, diastolic blood pressure; HIV, human immunodeficiency virus; MAP, mean arterial pressure; SBP, systolic blood pressure; SE, standard error.