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Hemodynamic profile and compensation deficit in African and European Americans during physical and mental stress

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Abstract

ABSTRACT

Increased vascular reactivity to stress has been suggested to contribute to the greater risk for developing hypertension in African Americans. Here, we examined the way (hemodynamic profile) and the extent to which (compensation deficit) cardiac output and total peripheral resistance compensate for each other in determining blood pressure responses to a physical (orthostasis) and a mental (anger recall) stress task, in normotensive African American (AA, $n = 30$) and European American (EA, $n = 48$) college students. Blood pressure stress reactivity did not differ as a function of race. However, AAs showed a prominent vascular hemodynamic profile and a significant compensation deficit in response to both tasks, while EAs showed no hemodynamic response to orthostasis and a mixed profile in response to anger recall. The present findings demonstrate a more prominent vascular hemodynamic reactivity to stress in AAs, which could contribute to the pathogenesis of hypertension in this ethnic group.

Keywords

Compensation deficit; Ethnic differences; Hemodynamic profile; Hypertension; Stress

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1. Introduction

The prevalence of hypertension in African Americans (AAs) is the highest in the world (Go et al., 2014; Musemwa & Gadegbeku, 2017), thus representing a main reason for the high cardiovascular (e.g., stroke and coronary heart disease) and noncardiovascular (e.g., end-stage kidney disease) mortality rates attributable to hypertension in this ethnic group (Keenan & Shaw, 2011; Lloyd-Jones et al., 2010). Compared with European Americans (EAs), AAs develop hypertension at an earlier age, experience a greater impairment (i.e., their average blood pressure (BP) is much higher), and have a worse prognosis (Mensah, Mokdad, Ford, Greenlund, & Croft, 2005). While several studies demonstrate that these disparities persist after adjustment for a wide range of socioeconomic, behavioral, and biomedical risk factors (e.g., smoking, obesity for men, lipid profile) (Hicken, Lee, Morenoff, House, & Williams, 2014; Mensah et al., 2005; Redmond, Baer, & Hicks, 2011), substantial research efforts have been directed at unveiling the biological underpinnings of elevated tonic BP in AAs. For example, findings of enhanced vascular reactivity to sympathetic stimulation (Stein, Lang, Singh, He, & Wood, 2000), attenuated response to vasodilator agents (Kahn et al., 2002), and a relatively narrow vascular lumen diameter (Taherzadeh, Brewster, van Montfrans, & VanBavel, 2010) may explain the presence of persistent or excessive vasoconstriction, a pathognomonic indicator of hypertension, in this ethnic group (Musemwa & Gadegbeku, 2017; Snieder, Harshfield, & Treiber, 2003). Several studies indicate that heightened hemodynamic reactions to stress confer a modest but reasonably consistent risk for developing hypertension (Carroll et al., 2001; Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Chida & Steptoe, 2010; Phillips & Hughes, 2011). Racial differences in physiological reactivity to stress may therefore be implicated as a potential mechanism for the higher rates of hypertension in AAs (Anderson, McNeilly, & Myers, 1991; Clark, Anderson, Clark, & Williams, 1999; Taherzadeh et al., 2010). Yet, data are conflicting with regard to racial differences in BP responses to stress since several factors, including family history of hypertension (Barnes, Schneider, Alexander, & Staggers, 1997; Goldstein & Shapiro, 1995), stereotype threat (Blascovich, Spencer, Quinn, & Steele, 2001) and the type of stressor (Reimann, Hamer, Schlaich, Malan, Rudiger et al., 2012, 2012b), may account for these differences. However, one important limitation of these studies is their focus on BP reactivity per se and not on the underlying hemodynamic determinants. For example, it has long been established that BP changes of similar magnitude may come about as a result of different underlying adjustments in cardiac output (CO) and total peripheral resistance (TPR) (e.g., (Eliot, Buell, & Dembroski, 1982)). Therefore, BP reactivity to laboratory stressors can be distinguished by the extent to which cardiac (i.e., increases in CO) or vascular (i.e., increases in TPR) mechanisms are predominant (Gregg, Matyas, & James, 2002; Kline et al., 2002). Evidence suggests that AAs demonstrate greater regulation of BP through increased TPR than EAs (Anderson, Lane, Monou, Williams, & Houseworth, 1988; Harrell & Floyd, 2000; Stein et al., 2000; Trieber et al., 1993). Moreover, sustained increases in TPR, but not CO, have been related to prolonged elevations in BP following a mental stressor in normotensive individuals (Steptoe, Willemsen, Kunz-Ebrecht, & Owen, 2003). Notably, elevated BP driven by TPR has been linked to a higher incidence of hypertension, vital organ damage, and increased risk for other forms of cardiovascular disease (Cooper & Waldstein, 2004; Dorr, Brosschot, Sollers, & Thayer, 2007; Kahn et al.,

2002; Mayet & Hughes, 2003). The tendency of an individual to exhibit cardiac or vascular responses appears to be consistent across different types of tasks (Ottaviani, Shapiro, Goldstein, James, & Weiss, 2006). However, such dichotomous categorization fails to capture the continuous nature of the relationship between CO and TPR (Gregg et al., 2002). Moreover, the same hemodynamic profile may differ in the extent to which CO and TPR compensate for each other, which is considered to be a critical factor in the prediction of BP (Gregg et al., 2002). In order to overcome the limitations and inconsistencies inherent in previous attempts to interpret hemodynamic reactivity by the use of categorical distinctions (see for a review (James, Gregg, Matyas, Hughes, & Howard, 2012)), Gregg and colleagues proposed a quantitative model of hemodynamics which relates BP reactivity to two continuous and independent measures: hemodynamic profile (HP; i.e., the way in which individuals compensate) and compensation deficit (CD; i.e., the extent to which individuals compensate) (Gregg et al., 2002). Previous application of the HP-CD model in different laboratory studies, all of which involved healthy normotensive participants, has provided empirical confirmation of its utility for characterizing hemodynamic response patterns that are the result of multiple sources of influence, including stressors of diverse physical, cognitive, and emotional content, and personality differences (reviewed in James et al., 2012; see also Ottaviani et al., 2017). Further, the HP-CD model has demonstrated superior laboratory-to-life generalizability than typical reactivity measures for the prediction of everyday life BP (Ottaviani et al., 2006; Ottaviani, Shapiro, Goldstein, & Mills, 2007). Therefore, the study of ethnic differences in these hemodynamic parameters could shed new light on the pathophysiological mechanisms underlying the greater risk for developing hypertension in AAs.

The current study sought to explore the hemodynamic determinants of BP change in a sample of normotensive AAs and EAs college students. We hypothesized that AAs would be characterized by a more prominent vascular hemodynamic profile and a higher deficit in compensating than EAs in response to a physical (i.e., orthostatic challenge) and a mental (i.e., anger recall) stress task.

2. Materials and methods

2.1. Participants

The present study is based on a secondary analysis of data from a subsample of undergraduate students, recruited from an introductory psychology research participant pool, to participate in a larger study ($n = 135$; Hill, 2009). For the aim of this study, we considered only participants belonging to the AA and EA ethnicities ($n = 89$). After exclusion of 11 participants because of missing data or severe artifacts in peripheral physiology data, the final sample comprised 78 undergraduate students (40 females; mean age = 19.82 ± 2.58), 30 of which were AAs and 48 of which were EAs. The only exclusionary criterion was age less than 18 years old. The study was approved by the Ohio State University Institutional Review Board. All participants provided written informed consent and received partial course credit for their participation.

2.2. Procedure

Participants were asked to refrain from physical activity, caffeine consumption or smoking at least 2 h prior to the experimental session, as these variables may have transient effects on cardiovascular measurements (e.g., Zimmermann-Viehoff et al., 2015). After signing the consent form, participants were fitted with 7 surface electrodes for ECG recordings. Then, they were seated in a reclining chair and instrumented with the Finometer sensor around their index or middle finger. Subsequently, participants completed an orthostasis task and an anger recall task, in a random and counterbalanced order. During the orthostasis task, participants were asked to stand up for 5 min. During the anger recall task, participants were asked to verbally describe *“a time in your past when you have been angry with another person”* (Ottaviani, Shapiro, & Fitzgerald, 2011). After a 1-min period for preparation, participants were asked to visualize as many details about the situation as they could and instructed that they would need to talk about the event or situation for at least 3 min. To assist the participant in recalling the event, the experimenter used prompts, such as *“What physical sensations were you feeling at the time? What aspect of the situation made you the maddest?”*. Each task was preceded (baseline) and followed (recovery) by a 5-min recording period during which participants sat quietly. At the end of the recovery period of the second task, the BP monitoring apparatus was removed to allow participants to complete the questionnaires. Lastly, participants were debriefed and received research credit for their participation.

2.3. Questionnaires

Participants completed questionnaires assessing sociodemographic information, physical activity (University of Houston Non-Exercise Questionnaire; (Jackson et al., 1990)), the tendency to worry (Penn State Worry Questionnaire; PSWQ; (Meyer, Miller, Metzger, & Borkovec, 1990)), state and trait anxiety (State-Trait Anxiety Inventory, STAI; (Spielberg, Gorsuch, Lushene, Vagg, & Jacobs, 1983)), and depression (Beck Depression Inventory-II, BDI-II; (Beck, Steer, Ball, & Ranieri, 1996)).

The University of Houston Non-Exercise Questionnaire assesses physical activity over the previous month with scores ranging from 0 (“Avoid walking or exertion”) to 7 (“Run over 10 miles/Spend more than 3 h a week in comparable physical activity”). Higher scores on the Houston indicate a greater level of physical fitness (Rossy & Thayer, 1998). The median split (3.3) was used to divide the sample into “high” and “low” on physical activity.

The PSWQ is a 16-item self-report questionnaire commonly used to assess the tendency to engage in worrisome thoughts. Each item (e.g., ‘Once I start worrying, I cannot stop’) is rated on a scale from 1 (“not at all typical of me”) to 5 (“very typical of me”). Higher PSWQ scores reflect greater levels of pathological worry. The PSWQ showed satisfactory reliability in the examined sample (Cronbach’s alpha = .74).

The STAI consists of 20 items assessing levels of state and trait anxiety. Respondents indicate how they feel right now in the state version (“I feel tense”) or generally in the trait version (“I am a steady person”) using a four-point Likert scale, from 1 (Not at all/Almost

never) to 4 (Very much/Almost always). In the present study, Cronbach's alphas were 0.92 and 0.91 for the state and trait forms, respectively.

The BDI-II consists of 21 items requiring participants to respond how they have felt during the previous 2 weeks ("I am so sad and unhappy that I can't stand it") on a 4-point scale ranging from 0 to 3. A total score of 0–13 is considered minimal, 14–19 mild, 20–28 moderate, and 29–63 severe depression. Cronbach's alpha was 0.90 in the present study.

2.4. Cardiovascular and hemodynamic assessment

Hemodynamic parameters and beat-to-beat BP were measured noninvasively using the Finometer Midi® and derived using the Beatscope analysis package (FMS Medical Systems 1.1a), which corrects for age, height, and weight, to obtain mean arterial pressure (MAP), systolic BP (SBP), diastolic BP (DBP), CO, and TPR. MAP, SBP, DBP, CO, and TPR reactivity values were computed by subtracting the initial baseline from task values; MAP, SBP, DBP, CO, and TPR recovery values were computed by subtracting task values from post-task values. HP and CD were computed as detailed in the following section.

2.5. Assessment of hemodynamic profile and compensation deficit

HP and CD were assessed following the orthogonal, physiologically grounded model proposed by Gregg et al. (Gregg et al., 2002). The HP- CD model draws on physiological theory that explains BP regulation as a dynamic compensatory relation between CO and TPR. Following Gregg et al. (Gregg et al., 2002), the equation used to address the concept of hemodynamic profile was $\log(\text{CO})_r + \log(\text{TPR})_r = \log(\text{MAP})_r$, where r indicates:

- a) a ratio of task to baseline values for reactivity periods (2017, Gregg et al., 2002; Ottaviani et al., 2006);
- b) a ratio of recovery to task values for recovery periods (2017, Ottaviani et al., 2006).

According to the model, the failure to compensate is a critical issue in the prediction of BP. The concept of compensation deficit is achieved by a 45° rotation of the two-dimensional space formed by the CO and TPR dimensions. In this two-dimensional space, participants are described as more vascular reactors when the algebraic increase in $\log(\text{TPR})_r$ exceeds that in $\log(\text{CO})_r$, and more myocardial when the algebraic increase in $\log(\text{CO})_r$ exceeds that in $\log(\text{TPR})_r$. CD increases as the algebraic sum of the $\log(\text{CO})_r$ and $\log(\text{TPR})_r$ values increases. Greater CD values indicate that increased TPR is not compensated by a commensurate decrease in CO.

2.6. Data analysis

All data are expressed as means (SEM). Differences at $p < .05$ were regarded as significant. Data processing was performed with the software modules of SPSS 23 (IBM). Age, BMI, MAP, SBP, DBP, CO, TPR, HP, CD, and scores on personality questionnaires (STAI, BDI-II) were treated as continuous variables. Sex and ethnicity were treated as dichotomous variables. Assumptions for normality were tested for all continuous variables using the Shapiro-Wilk test. Differences between the two groups (AAs vs EAs) in age, sex distribution, BMI, and levels of state and trait anxiety and depression were analyzed by t -

tests and χ^2 tests. The variables that differed significantly between groups were included as covariates in all the subsequent analyses.

To test for differences between AAs and EAs in resting state cardiovascular variables, a series of univariate general linear models (GLMs) were performed on SBP, DBP, CO, and TPR. To test for differences between AAs and EAs in BP reactivity and recovery in the three experimental conditions, three 2 (task: orthostasis, anger recall) $\times$ 2 (time: reactivity, recovery) $\times$ 2 (ethnicity: AAs, EAs) repeated measures GLMs were performed on MAP, SBP, DBP, CO, and TPR. Reactivity (task minus baseline) and recovery (recovery minus task) change scores were used in these GLMs.

It has to be noted that the model by Gregg and colleagues challenges the categorical nature of earlier methods of classification, such as arbitrary cut-off taxonomies for classifying individuals as cardiac or vascular responders to stress (Gregg et al., 2002). As such, the model only allows to describe participants as more vascular or myocardial along a continuum. However, consistent with the approach adopted by James and Gregg (James & Gregg, 2004), one-sample *t*-tests can be used to test the difference from zero of HP and CD scores for each condition and subsequent recovery periods. A significant *t*-test result for HP is taken to indicate either a vascular (positive *t* value) or a myocardial profile (negative *t* value). A nonsignificant HP result coupled with a significant CD result means that the response was mixed (i.e., neither vascular nor myocardial). No hemodynamic response at all is deemed to have occurred when both HP and CD results are not significant.

Then, to test for ethnic differences in HP and CD in the two experimental conditions, three 2 (task: orthostasis, anger recall) $\times$ 2 (time: reactivity, recovery) $\times$ 2 (ethnicity: AAs, EAs) GLMs were performed.

3. Results

3.1. Demographic, anthropometric and psychological characteristics

Table 1 illustrates differences between AAs and EAs for the demographic, anthropometric and psychological variables of the study. The two groups were significantly different in terms of BMI and sex distribution, with AAs having a higher BMI ($t = 2.17$, $p = .033$), and a higher female to male ratio ($\chi^2 = 12.57$, $p < .001$) compared to EAs. Differences in physical activity were not significant ($\chi^2 = 3.25$, $p = .071$). Because BP and hemodynamic parameters obtained from the Finometer Midi® are already corrected for height and weight, only sex was controlled for in all the subsequent analyses of BP and hemodynamic data.

3.2. Cardiovascular and hemodynamic parameters at rest

Resting cardiovascular and hemodynamic parameters in AAs and EAs are reported in Table 2.

In resting conditions, AAs showed higher values of DBP compared to EAs ($F(1,74) = 4.24$, $p = .04$; $\eta_p^2 = .054$), and a tendency for higher values of MAP compared to EAs ($F(1,74) = 3.26$, $p = .075$; $\eta_p^2 = .042$). No ethnic differences emerged for resting state values of SPB, HR, CO and TPR. A significant effect of sex was found for MAP ($F(1,75) = 7.42$, $p < .01$;

$\eta_p^2 = .091$), SBP ($F(1,75) = 12.28, p < .01; \eta_p^2 = .141$), and DBP ($F(1,74) = 12.87, p < .01; \eta_p^2 = .148$). Specifically, males showed higher values of MAP ($88.1 \pm 1.8 \text{ mmHg}$ vs $82.6 \pm 1.8 \text{ mmHg}$, $p < .01$), SBP ($126.7 \pm 2.9 \text{ mmHg}$ vs $116.7 \pm 2.2 \text{ mmHg}$, $p < .01$) and DBP ($69.0 \pm 2.0 \text{ mmHg}$ vs $62.5 \pm 1.25 \text{ mmHg}$, $p = .010$) compared to females.

3.3. Cardiovascular and hemodynamic reactivity and recovery

Fig. 1 shows reactivity and recovery response patterns for MAP, SBP, DBP, CO, and TPR for AAs and EAs during the orthostasis and anger recall tasks.

The GLM having MAP as the dependent variable yielded a significant task x time interaction ($F(1,72) = 8.14, p < .01; \eta_p^2 = .102$). Specifically, the anger recall task elicited a significantly larger increase in MAP than the orthostasis task ($+ 17.46 \pm 1.10 \text{ mmHg}$ vs $+ 5.12 \pm 0.93 \text{ mmHg}$, $p < .001$). Moreover, MAP recovery values differed significantly between the two tasks (anger recall: $-10.61 \pm 0.92 \text{ mmHg}$ vs orthostasis: $+0.15 \pm 0.67 \text{ mmHg}$, $p < .001$). No ethnic differences emerged for MAP reactivity and recovery patterns (Fig. 1A).

The GLM having SBP as the dependent variable yielded a significant task x time interaction ($F(1,73) = 5.26, p = .025; \eta_p^2 = .07$). Specifically, the anger recall task elicited a significantly larger increase in SBP than the orthostasis task ($+ 22.47 \pm 1.40 \text{ mmHg}$ vs $+ 2.93 \pm 1.4 \text{ mmHg}$, $p < .001$). Moreover, SBP recovery values differed significantly between the two tasks (anger recall: $-15.09 \pm 1.36 \text{ mmHg}$ vs orthostasis: $+2.68 \pm 1.05 \text{ mmHg}$, $p < .001$). The GLM also yielded a task x time x ethnicity interaction ($F(1,73) = 7.26, p < .01; \eta_p^2 = .09$). Specifically, AAs showed a significantly smaller decrease of SBP following the anger recall task than EAs ($p = .021$) (Fig. 1B). No ethnic differences emerged for SBP reactivity to the orthostasis and anger recall tasks, and for SBP recovery from the orthostasis task (Fig. 1B).

The GLM having DBP as the dependent variable yielded a significant task x time interaction ($F(1,72) = 8.25, p < .01; \eta_p^2 = .10$). Specifically, the anger recall task elicited a significantly larger increase in DBP than the orthostasis task ($+ 15.44 \pm 0.98 \text{ mmHg}$ vs $+ 6.11 \pm 0.90 \text{ mmHg}$, $p < .001$). Moreover, recovery values differed significantly between the two tasks (anger recall: $-9.45 \pm 0.80 \text{ mmHg}$ vs orthostasis: $-1.37 \pm 0.66 \text{ mmHg}$, $p < .001$). No ethnic differences emerged for DBP reactivity and recovery patterns (Fig. 1A).

The GLM having CO as the dependent variable yielded a significant task x time interaction ($F(1,74) = 5.84, p = .018; \eta_p^2 = .073$). Specifically, the anger recall task elicited a significantly larger increase in CO than the orthostasis task ($+ 0.339 \pm 0.095 \text{ l/min}$ vs $-0.139 \pm 0.101 \text{ l/min}$, $p < .001$). Moreover, recovery values differed significantly between the two tasks (anger recall: $-0.397 \pm 0.079 \text{ l/min}$ vs orthostasis: $-0.044 \pm 0.072 \text{ l/min}$, $p < .001$). The GLM also yielded a significant time x ethnicity interaction ($F(1,74) = 6.24, p = .015; \eta_p^2 = .08$). Specifically, AAs showed a significantly smaller decrease of CO following the anger recall task than EAs ($p < .01$) (Fig. 1D). No ethnic differences emerged for CO reactivity to the orthostasis and anger recall tasks, and for CO recovery from the orthostasis task (Fig. 1D).

The GLM having TPR as the dependent variable did not reveal any significant effect of ethnicity. The anger recall task elicited a significantly larger increase in TPR than the orthostasis task ($+0.146 \pm 0.023 \text{ mmHg/ml*s}^{-1}$ vs $+0.047 \pm 0.025 \text{ mmHg/ml*s}^{-1}$, $p < .001$), with no ethnic differences (Fig. 1E). Moreover, recovery values differed significantly between the two tasks (anger recall: $-0.075 \pm 0.21 \text{ mmHg/ml*s}^{-1}$ vs orthostasis: $+0.021 \pm 0.024 \text{ mmHg/ml*s}^{-1}$, $p < .001$), with no ethnic differences (Fig. 1E).

3.4. Hemodynamic profile and compensation deficit

Fig. 2 illustrates the scatterplots for hemodynamic profile and compensation deficit during the two tasks.

The GLM having hemodynamic profile as outcome, yielded a main effect of ethnicity ($F(1,70) = 6.48$, $p = .013$; $\eta_p^2 = .085$), with AAs being characterized by a more vascular profile compared to EAs (0.10 ± 0.02 vs 0.03 ± 0.02 ; $p = .01$). A significant time x ethnicity interaction also emerged ($F(1,70) = 11.43$, $p = .001$; $\eta_p^2 = .14$).

One group *t*-tests for hemodynamic profile indicated that in AAs a vascular profile was induced by both tasks ($t(28) = 5.50$, $p < .001$ for orthostasis; $t(28) = 3.96$, $p < .001$ for anger recall) and persisted during recovery ($t(28) = 4.34$, $p < .001$ for orthostasis; $t(27) = 4.38$, $p < .001$ for anger recall). In EAs, no hemodynamic response occurred during the orthostasis task, whereas recovery from that task evoked a mixed profile ($t(47) = 1.67$, $p = .10$ for hemodynamic profile and $t(47) = 2.34$, $p = .02$ for compensation deficit) (Fig. 2). A mixed profile was also evoked by the anger recall task ($t(47) = 0.12$, $p = .91$ for hemodynamic profile and $t(47) = 7.66$, $p < .001$ for compensation deficit), while recovery from that task was characterized by a vascular profile ($t(47) = 4.49$, $p < .001$) (Fig. 2).

The GLM having compensation deficit as outcome yielded a marginally significant task x time x ethnicity interaction ($F(1,69) = 3.65$, $p = .060$; $\eta_p^2 = .05$). As shown by Fig. 2, AAs were characterized by compensation deficit during reactivity to ($t(28) = 2.67$, $p = .01$ for orthostasis; $t(28) = 7.87$, $p < .001$ for anger recall) and recovery from ($t(27) = 3.57$, $p = .001$ for orthostasis; $t(28) = 8.19$, $p < .001$ for anger recall) both tasks, whereas EAs did not show any compensation deficit during the orthostasis task, but a significant compensation deficit during both reactivity to ($t(47) = 7.66$, $p < .001$) and recovery from ($t(47) = 3.33$, $p = .002$) the anger recall task.

4. Discussion

The major and novel finding of the present study is that BP responses of the same magnitude are characterized by different hemodynamic patterns in a sample of AA and EA college students. Specifically, AAs exhibit a more prominent vascular hemodynamic profile in response to both orthostasis and anger recall tasks than EAs, which may be implicated in the pathogenesis of hypertension in this ethnic group.

The main objective of this study was to extend previous research by exploring potential ethnic differences in the underlying hemodynamics of BP change that are characterized as a dynamic compensatory relationship between cardiac output and total peripheral resistance.

We addressed this issue by using the model of HP and CD suggested by Gregg et al. (Gregg et al., 2002), which has been successfully applied in previous studies to characterize hemodynamic reactivity in healthy normotensive individuals (reviewed in James et al., 2012) as well as for the prediction of everyday life BP (Ottaviani et al., 2006, 2007), suggesting high laboratory-to-life generalizability. In line with our hypothesis, AAs show a prominent vascular HP during both reactivity to and recovery from a physical (i.e., orthostasis) and a mental (i.e., anger recall) task. On the other hand, EAs showed no hemodynamic response to orthostasis and a mixed profile in response to anger recall. Interestingly, recovery from anger recall is the only condition that elicits a vascular profile in both groups, which is in line with previous results showing that angry rumination is more vascular in nature (2017, Ottaviani et al., 2006). Moreover, AAs show a higher deficit in compensating than EAs, particularly during orthostasis. Notably, individuals who typically respond with a vascular profile might be at risk of suffering persistent vascular contractility due to repeated and/or prolonged episodes of increased vascular reactivity, which in the long run may contribute to elevated tonic blood pressure (Gregg et al., 2002). This is of particular relevance for those populations, including AAs, being subjected to chronic stress resulting from discrimination (Williams & Neighbors, 2001). Therefore, the prominent vascular HP described here in healthy normotensive AAs may be among the earliest precursors to the development of hypertension in this ethnic group. Recent studies have suggested that the greater prevalence of hypertension observed in AAs may reflect a larger underlying pattern of vascular remodelling, by demonstrating (i) that total peripheral resistance was positively associated with minimal forearm vascular resistance, an index of vascular hypertrophy, among AAs, but not Whites, with elevated blood pressure (Hill, Sherwood, Blumenthal, & Hinderliter, 2016), and (ii) that in a sample of individuals with undiagnosed, untreated hypertension, AAs exhibited a blunted night-time BP dipping which was accompanied by an attenuated fall in total peripheral resistance compared with Whites (Sherwood, Hill, Blumenthal, & Hinderliter, 2018). The present findings in healthy normotensive young adults indicate statistically reliable higher values of resting diastolic BP in AAs, while no statistically reliable ethnic differences are observed in resting measures of MAP, systolic BP, cardiac output and total peripheral resistance although they were in the expected direction. This is partly inconsistent with a previous study showing higher resting levels of systolic BP and total peripheral resistance in AA male college students compared with their EA matching counterparts (Dorr et al., 2007). A potential explanation for this discrepancy lies in the substantial sex imbalance across the two ethnic groups of this study. Given the higher BP found in men compared to women, which is consistent with the literature (Stoney, Davis, & Matthews, 1987), it is plausible that the relatively small number of males in the AA group and the greater proportion of males in the EA group might have masked other potential sex-specific ethnic differences in baseline cardiovascular and hemodynamic measurements.

Reactivity studies have previously shown greater cardiovascular responses to a variety of physical (e.g., orthostasis (Goldstein & Shapiro, 1995) and cold pressor test (Reimann, Hamer, Schlaich, Malan, Rudiger et al., 2012, 2012b)) and mental (e.g., remote associate test (Blascovich et al., 2001) and anger provoking manipulations (Armstead, Lawler, Gorden, Cross, & Gibbons, 1989; Goldstein & Shapiro, 1995; Johnson, 1989) challenge tasks in AAs compared with EAs, even though a variety of factors such as family history of

hypertension (Barnes et al., 1997; Johnson, 1989), a higher pain perception (Reimann, Hamer, Schlaich, Malan, Rudiger et al., 2012, 2012b), and stereotype threat (Blascovich et al., 2001), among others, seem to contribute significantly to the ex-aggerated cardiovascular reactivity observed in these populations of AAs. Here, healthy normotensive young adults do not display differences in their cardiovascular responses to orthostasis and anger recall tasks as a function of race. We must acknowledge, however, that the influence of family history and personality characteristics on cardiovascular reactivity and recovery patterns was not controlled for in the present investigation.

The present findings are in line with the idea that hemodynamic responses to acute and chronic environmental stressors represent a biobehavioral marker in individuals at risk of developing essential hypertension, and that HP and CD patterns may be superior to traditional BP and hemodynamic reactivity measures in the prediction of daily-life BP in normotensive subjects (Ottaviani et al., 2006). One should note, however, that the current results are merely suggestive, and their implications for hypertension should be interpreted within the context of their limitations. First, the above discussed sex imbalance across the two ethnic groups. However, given the lack of significant sex effects on cardiovascular and hemodynamic reactivity and recovery patterns, it seems likely that this factor had minimal influence on the main study findings. Second, these results were obtained from a convenience sample of young, healthy, college students. This suggests that ethnic differences in some of the outcome measures of this study might potentially be more pronounced in a community-based sample, where greater variability in health status and lifestyle factors is expected. Third, the short recovery period between the two tasks. Despite having adopted a counterbalancing order, they may have still influenced each other, for example by enhancing or flattening the response to the other. Fourth, while the HP- CD model holds promise for the study of the hemodynamics underlying BP change, it has been suggested that it should be used cautiously when examining the role of psychological factors in biological outcomes (Why & Chen, 2013). However, this would not be an issue for the orthostatic challenge used in the present study in which largely similar results were found. Lastly, the physiological mechanisms responsible for the prominent vascular hemodynamic profile observed in AAs in response to the experimental tasks were not examined. Previous research has shown that normotensive Black and White Africans do not differ regarding common indices associated with sympathetic outflow to peripheral vasculature (Ray & Monahan, 2002; Reimann, Hamer, Schlaich, Malan, Rudiger et al., 2012, 2012b). Moreover, a meta-analysis indicated that AAs have greater resting state vagally-mediated heart rate variability (HRV, a surrogate index of vagal modulation to the heart), after controlling for several covariates and subgroup stratification by age and sex (Hill et al., 2015). Notably, greater resting vagally-mediated HRV is typically associated with lower total peripheral resistance and BP via the baroreflex - a physiological mechanism adjusting cardiovascular activity in response to changes in BP and vice versa (Reyes del Paso, Hernandez, & Gonzalez, 2006). However, AAs have been found to exhibit both higher tonic vagally-mediated HRV and elevated total peripheral resistance at rest (Dorr et al., 2007), a pattern that may represent something of a “Cardiovascular Conundrum” and may hint at the existence of ethnic differences in baroreflex sensitivity. However, to date, research exploring baroreflex sensitivity as a function of race is inconsistent (Reimann, Hamer, Schlaich, Malan, Rudiger et al., 2012,

2012b; Sanderson, Billingham, & Floras, 1983; Thomas, Nelesen, Ziegler, Bardwell, & Dimsdale, 2004). This may be due in part to the fact that only the cardiac limb of the baroreflex is routinely examined. In fact, cardiac baroreflex is one of the main sources of vagal cardiac influences (Reyes del Paso, Langewitz, Robles, & Perez, 1996). Recent approaches that have estimated the myocardial, and especially the vascular limbs, of the baroreflex may be particularly informative with respect to ethnic differences (Reyes Del Paso, de la Coba, Martín-Vázquez, & Thayer, 2017). In examining possible asymmetries in the vasomotor and myocardial baroreceptor loops during BP rises versus BP falls, Reyes del Paso and colleagues found that greater baroreflex-induced changes in TPR were associated with greater TPR, with evident implications for the current findings (Reyes Del Paso et al., 2017).

In conclusion, the results of this study extend previous research on ethnic differences in the hemodynamic determinants of BP change by demonstrating a more prominent vascular hemodynamic reactivity to stress in AAs, using a model of hemodynamics that takes into account the dynamic compensatory relationship between cardiac output and total peripheral resistance and overcomes previous classification approaches (Gregg et al., 2002; James et al., 2012). Clearly, future prospective studies are needed in order to understand the physiological mechanisms underlying the increased vascular reactivity to stress observed in AAs and the trajectory of its contribution to the pathogenesis of hypertension in this ethnic group.

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Fig. 1.

Reactivity (REACT) and recovery (RECOV) response patterns of mean arterial pressure (MAP), systolic and diastolic blood pressure (SBP, DBP), cardiac output (CO) and total peripheral resistance (TPR) during orthostasis and anger recall tasks, in African Americans (AAs) and European Americans (EAs). Data are reported as sex-adjusted means $\pm$ SEM. * significantly different from corresponding EA values ($p < 0.05$).

Fig. 2. Scatterplots for hemodynamic profile and compensation deficit during each task in African Americans (AAs) and European Americans (EAs). A “more vascular” profile is associated with more positive values along the hemodynamic profile axis and a “more myocardial” profile is associated with more negative values along the hemodynamic profile axis. A “higher deficit” in compensating is associated with more positive values on the

compensation deficit axis and a “lower deficit” in compensating is associated with more negative values on the compensation deficit axis.

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Table 1

Differences in demographic, anthropometric and psychological characteristics between African Americans (AAs) and European Americans (EAs).

	AAs (n = 30)	EAs (n = 48)	t/ χ^2	p
Age (years)	20.3 $\pm$ 0.7	19.5 $\pm$ 0.2	1.21	.229
Sex (F/M)	23/7	17/31	12.57**	< .001
BMI (kg/m ²)	26.1 $\pm$ 0.9	24.0 $\pm$ 0.5	2.17*	.033
Physical activity (L/H)	17/12	12/30	3.25	.071
PSWQ	53 $\pm$ 13.1	49.6 $\pm$ 12.9	1.11	.269
STAI state score	36.1 $\pm$ 1.5	38.9 $\pm$ 1.6	1.20	.233
STAI trait score	37.8 $\pm$ 1.6	39.9 $\pm$ 1.6	0.87	.390
BDI score	7.5 $\pm$ 0.9	8.9 $\pm$ 1.2	0.82	.413

Data are reported as means $\pm$ SEM. Abbreviations: F = Females; M = Males; BMI = Body Mass Index; L = Low; H = High; PSWQ = Penn State Worry Questionnaire; STAI = State Trait Anxiety Inventory; BDI = Beck Depression Inventory.

Table 2

Resting cardiovascular and hemodynamic parameters in African Americans (AAs) and European Americans (EAs).

	AAs (n = 30)	EAs (n = 48)	<i>F</i>	<i>p</i>
MAP (mmHg)	88.5 ± 2.1	83.3 ± 1.7	3.26	.075
SBP (mmHg)	123.6 ± 2.6	121.4 ± 2.4	0.44	.509
DBP (mmHg)	69.4 ± 1.9	64.3 ± 1.5	4.24	.043
CO (l/min)	6.12 ± 0.23	6.09 ± 0.18	0.01	.923
TPR (mmHg/ml [*] s ⁻¹)	0.92 ± 0.05	0.87 ± 0.04	0.60	.439

Data are reported as ± SEM. Blood pressure and hemodynamic data are corrected for sex. Abbreviations: MAP = Mean Arterial Pressure; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; CO = Cardiac Output; TPR = Total Peripheral Resistance.