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Angry in America: Psychophysiological Responses to Unfair Treatment

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Abstract

Background: African Americans have the highest rates of hypertension-related disease of any ethnic group in the United States. Importantly, racism and discrimination have been linked to these higher rates of morbidity and mortality. Discrimination is deleterious not only to those that are the recipients of this unfair treatment but also to the partners and family members of those affected as well to those that perpetrate this bias.

Purpose: In this paper we identify a unique pattern of physiological response to unfair treatment we have called the "cardiovascular conundrum". This pattern is characterized by greater heart rate variability and greater total peripheral resistance in African Americans compared to their European American counterparts.

Methods and Results: We review the evidence supporting the existence of this pattern and propose several physiological and psychological factors that might underpin it. We also propose a number of factors that might help to mitigate the deleterious effects associated with it.

Conclusions: Whereas the context of the current review is on Black/White disparities the framework we propose may be relevant to others exposed to unfair treatment. Ultimately, the systemic factors that perpetuate these inequalities will require that we first acknowledge and then face the challenges they present if we are to address the wealth and health disparities in our country.

Keywords: racism; unfair treatment; discrimination; anger; total peripheral resistance; heart rate variability

"To be a Negro in this country and to be relatively conscious, is to be in a rage almost all the time. So that the first problem is how to control that rage so that it won't destroy you."

James Baldwin [1]

These words are as relevant now as they were in 1961. As we face an unprecedented time in our country and in fact, the world, it's useful to examine that nature of this statement closely as it is pregnant with meaning. The first sentence lays out the problem. Black and Brown people in this country are exposed to systematic unfair treatment, discrimination, and racism. Whereas these are not the only people exposed to unfair treatment, the unique history of the United States provides a framework for understanding responses to unfair treatment among Black and Brown people that may be broadly applicable. Another issue highlighted in the first sentence is that anger is a natural response to this unfair treatment. Therefore, we need to have some understanding of anger and emotion in general. The second sentence highlights the dilemma. This anger has to be managed in such a way as to not allow it to destroy us. Thus, the issue of emotion regulation, especially, anger regulation requires some understanding. But, if, as it has been said, "anger is the poison you give yourself in the hope that the other person will die", has any validity, it implies that both the giver of the anger as well as the receiver are at risk. What we hope to do in this paper is to try to explicate the nature of the problem and equally if not more importantly, try to understand the costs associated with controlling this rage so that it does not destroy us.

The backdrop

Significant health disparities exist in the US such that African Americans (AAs) have higher rates of mortality and morbidity from a range of disorders including cardiometabolic disease

such as diabetes and hypertension-related cardiovascular disease [2]. In fact, AAs have the highest rates of hypertension-related disease of any ethnic group in the US. Importantly, racism and discrimination have been linked to these higher rates of morbidity and mortality [3,4].

In this context of racism and discrimination being associated with higher rates of negative health outcomes, it is important to note that this discrimination is deleterious not only to those that are the recipients of this unfair treatment but also to those that perpetrate this bias. In a large study examining the effects of European Americans (EAs) explicit and implicit racial bias on both AA and EA health outcomes, it was found that EAs explicit racial bias was associated with increased death from circulatory disease in both AAs and EAs [4]. The effect of EAs explicit racial bias was larger for AAs than for EAs, and when explicit and implicit bias were modeled together only explicit bias was associated with this effect. This is important in light of the current attention given to implicit bias and implicit bias training. The authors speculated that the larger effect for explicit bias may be due to its association with structural as well as interpersonal, behavioral, and emotional factors. For example, with respect to structural factors, AAs compared to EAs receive less pay for the same work, and receive longer prison sentences for the same crime [5,6]. However, the authors left open how these factors translated into biological processes associated with disease risk. In this paper we provide evidence for how such translation might take place.

The effects of racism-related stress are not limited to the immediate individuals directly involved. These effects may extend to others such as partners and families of those involved, as well as persons that have witnessed the transgressions. We recently reviewed the literature on the contagion of social defeat in animals [7]. We showed that rodents that witnessed the social defeat of a conspecific showed physiological stress responses as well as depression- and anxiety-like behaviors [7]. In addition, social interactions with a stressed partner after social defeat were

associated with social avoidance and physiological stress responses in the partner not directly defeated. Emotional contagion in humans has been studied extensively [8] and similar processes as found in the rodents might be involved. Moreover, and particularly relevant in the age of mobile phone cameras and widespread sharing of videos, is the literature on how media exposure to traumatic events is associated with poor health outcomes. In one study on the effects of media coverage of 9/11 and the Iraq wars it was shown that watching 1-3 hours of 9/11 related television coverage was associated with a 20% increase in physician-diagnosed health problems 2 to 3 years later [9]. Moreover, in a follow-up study it was found that high levels of media-based exposure were associated with ongoing trauma-related fears and worry [10]. Relatedly, another study found that racism-related vigilance (i.e., anticipated racial discrimination) was greater in AAs compared to EAs and that this was associated with an increased risk of hypertension [11]. These studies on emotional contagion and media exposure highlight how instances of unfair treatment, and their psychological and physiological sequelae, can propagate throughout the community and society at large thus impacting people far beyond the direct participants. They also highlight how perseverative cognition such as trauma-related worry and racism-related vigilance may contribute to the adverse impact of this exposure.

Research has shown that compared to their EA counterparts, AAs are more likely to perceive interracial hostility, to interpret ambiguous situations as more threatening, to score higher on measures of hostility and anger, and to attribute their hostility and anger to perceived racism [12]. Importantly, compared to EAs, AAs are more likely to use emotional inhibition to cope with their anger or as Baldwin might put it, “control that rage” [13].

In an elegant series of studies in the 1960s, Hokanson and colleagues explored the vascular (blood pressure) responses associated with the recovery from anger instigation [14]. Whereas a

full description of these many studies is beyond the scope of the present paper, briefly what these studies lead Hokanson to propose was a social learning theory account of vascular responses to coping with anger instigation. A number of factors such as the social status of the anger instigator and the gender of the aggrieved individual contributed to the vascular response. However, the primary notion is that the response to the anger instigation that was associated with a higher likelihood of ending the aggression and a lower likelihood of continued aggression was associated with a more rapid recovery of the aggrieved persons vascular responses as indexed by systolic and/or diastolic blood pressure (SBP and DBP, respectively). That is, individuals learn, through conditioning, what coping response to the anger instigation (e.g., counter-aggression versus anger inhibition) leads to a lessening of the aggressive behavior. In the US, AAs learn from an early age to inhibit their anger expression in response to unfair treatment in an effort to reduce or avoid aggression. We experimentally tested this idea in a study in which an EA confederate interacted antagonistically toward AA and EA participants during a debate [12]. The debate topics were either a racially-charged topic such as police use of force against AAs or a non-racially-charged topic such as abortion. The results of the study were largely in line with what would be expected based on the Hokanson studies especially for the EAs. For EAs, when they were able to counter-aggress against the confederate by rating the confederate (express their anger), their systolic and diastolic BP recovered more quickly than AAs that expressed their anger against the confederate. However, when asked to inhibit their anger towards the confederate by rating their best friend, both EAs and AAs showed delayed recovery of total peripheral resistance (TPR) with AAs having the highest total peripheral resistance. Note that there was no good response for the AAs in that those that expressed their anger had delayed blood pressure recovery and those that inhibited their anger had delayed total peripheral

resistance recovery. The EAs that were able to express their anger had faster blood pressure recovery consistent with the social learning theory model of Hokanson. Given that BP elevations due to increased total peripheral resistance are associated with greater risk for cardiovascular events and mortality compared to BP elevations due to increased cardiac output (CO), the AAs were in a situation of "damned if you do, damned if you don't". That is, if one expressed their anger and therefore failed to halt or avoid the aggression, one could die quickly at the hands of police for example. But if one inhibited their anger they could die slowly, as it were, from the increased risk for cardiovascular events associated with elevated total peripheral resistance.

Interestingly in this study we also measured heart rate variability (HRV). HRV refers to the beat to beat variability in the interbeat interval time series and reflects the autonomic neural regulation of cardiac chronotropic activity (heart rate). Under healthy conditions the sympathetic and parasympathetic (vagal) influences are in dynamic balance with vagal influences dominating especially in high-frequency HRV. Greater vagal activity as indexed by higher HRV has been shown to be associated with better psychological and physiological health outcomes [15,16]. And as we detail in the next section, HRV should be inversely related to total peripheral resistance. Thus, the HRV responses in this study were also informative. The AAs that expressed their anger had the lowest HRV during the recovery period whereas the EAs that inhibited their anger had the highest HRV. The HRV of the AAs that inhibited their anger was also higher than that of the AAs that expressed their anger. Whereas these responses to anger instigation were quite informative, we also observed an interesting pattern during the baseline. AAs had both higher total peripheral resistance *and* higher HRV compared to their EA counterparts. As noted above, greater total peripheral resistance is associated with increased risk for cardiovascular events, morbidity, and mortality. However, we have repeatedly shown that greater HRV is

associated with *decreased* risk for poor health outcomes including cardiometabolic disease [16,17]. Thus, AAs show a paradoxical pattern of cardiovascular activity we have termed a "cardiovascular conundrum" [18].

The Cardiovascular Conundrum Phenotype

The Cardiovascular Conundrum phenotype in AAs is characterized by greater HRV *and* greater total peripheral resistance. To understand why this is a "conundrum" one must first understand the regulation of blood pressure. Mean arterial pressure (MAP) equals cardiac output (CO) times total peripheral resistance (TPR) (MAP=CO X TPR). In addition, CO equals stroke volume (SV) times heart rate (HR) (CO=SV X HR). Both short-term and, as is becoming increasingly clear, long-term blood pressure regulation, is achieved via the baroreflex [19]. Whereas a detailed exposition of the baroreflex is beyond the scope of the present paper, a brief description is useful (see [20] for a review). A given level of BP can be reached by the regulation of cardiac output and total peripheral resistance which work in tandem to maintain blood flow to vital organs such as the brain and the heart. When BP increases, pressure sensitive mechanoreceptors in the aorta and carotid sinus "sense" the distension and send signals to the nucleus tractus solitarius via vagal afferent fibers. In a reflex manner, the nucleus tractus solitarius then initiates efferent sympathoinhibition and concomitant vagal activation to reduce total peripheral resistance, stroke volume, and heart rate to return blood pressure to its previous level. The reverse occurs in response to blood pressure decreases. As such, vagal activity as indexed, for example, by HRV, and total peripheral resistance are normally inversely related. Thus, the greater HRV *and* greater total peripheral resistance in AAs represents a "cardiovascular conundrum" [18]. Two recent meta-analyses provide data in support of this pattern in AAs relative to EAs [21,22]. In the first meta-analysis in over 6000 EAs and over 4000 AAs, we showed that AAs had a nearly full

standard deviation greater HRV than EAs [21]. In the second meta-analysis in over 6000 EAs and over 6000 AAs we showed that AAs had a nearly one third of a standard deviation greater total peripheral resistance than EAs [22]. This is depicted in Figure 1. Give that this pattern exists, what are the potential factors that lead to this pattern? We will briefly review several physiological and psychological factors that might help to clarify the nature of this pattern.

Physiological Factors Associated with the Conundrum

The regulation of the cardiovascular system and BP is complex involving many variables including genetics, diet, and lifestyle factors. However, the evidence for the role of genetics and diet as primary drivers of the Cardiovascular Conundrum, particularly BP and HRV, is less than compelling. With respect to genetics, a recent genome wide association study identified 17 single-nucleotide polymorphisms on 8 loci associated with HRV [23]. In addition, the genetic correlations of these HRV genetic variants were associated with systolic and diastolic BP (among other factors) such that lower HRV would be associated with greater BP. This is as would be expected based upon the epidemiological and clinical data in reviews of HRV (e.g., [16]) as well as the role of the baroreflex in BP regulation. Importantly for the present discussion, 6 of the single-nucleotide polymorphisms identified in those of European ancestry generalized to those of African American and Hispanic/Latino ancestry. Thus, in combination with the high degree of genetic admixture in AAs, the evidence for a genetic basis for this health disparity represented by the conundrum pattern is weak [24].

With respect to diet and particularly sodium, the prevailing dogma is that sodium intake is associated with fluid volume and that fluid volume is the major determinant of long-term BP regulation. The data in support of this idea is problematic at best [25]. As noted above, increasing evidence points to the baroreflex as the major determinant of long-term BP [19]. More

specifically to the ethnic difference evident in the conundrum, the greater vascular resistance in AAs is unlikely to be due to sodium as several reviews have noted [22, 26]. In this context, it is interesting to note that in contrast to sodium, potassium may have beneficial effects on BP such that higher levels of potassium independent of sodium levels appear to be associated with lower BP [27].

Another factor that has been less explored but is more promising as a factor leading to greater total peripheral resistance in AAs concerns birth weight. Higher total peripheral resistance is both an antecedent and a consequence of low birth weight [28,29]. Much research has shown that even when matched on many variables such as socioeconomic status, maternal weight, age, and length of gestation, among others, AA women give birth to lower birth weight offspring than EA mothers [30]. Greater total peripheral resistance in AA women can lead to low birth weight offspring via decreased nutrient and blood flow to the fetus. Relatedly, low birth weight has been associated with less vasodilatory response in fetal umbilical cord cells and greater total peripheral resistance in infancy, childhood, and adulthood [29,31]. In addition, we have recently reported that despite the potent endogenous vasodilatory stimulus of pregnancy, AA mothers had greater total peripheral resistance and HRV (the cardiovascular conundrum pattern) compared to EA mothers. Furthermore, even though highly matched on a wide range of factors, the AA mothers gave birth to lower birthweight babies compared to EA mothers [32]. Thus, greater total peripheral resistance can be passed from one generation to the next via a non-genetic pathway.

From the perspective of the cardiovascular conundrum, the greater vagal activity as indexed by HRV may be an attempt to compensate for this greater total peripheral resistance. One piece of evidence in support of this idea is that the greater HRV in AAs seems to appear as early as a few months after birth [21]. Another important piece of evidence is that the greater vascular

resistance in AAs is associated with decreased sensitivity to acetylcholine, which is a potent vasodilator [26,33]. Acetylcholine is the primary neurotransmitter of the vagus nerve. Thus, greater HRV in AAs may be an attempt to compensate for the decreased responsivity to acetylcholine as a vasodilatory agent and therefore help to explain the greater total peripheral resistance in AAs despite their greater HRV.

We have reviewed several potential physiological factors that might account for the cardiovascular conundrum phenotype. Whereas the evidence for genetic or dietary factors is problematic, decreased sensitivity to acetylcholine and low birth weight are more promising as factors that might help to clarify the underlying mechanisms involved in the cardiovascular conundrum. However, these physiological factors likely don't tell the whole story. We next review several psychological factors that might be associated with the "conundrum" pattern of response to racism, discrimination, and unfair treatment.

Psychological Factors Associated with the Conundrum

Successful emotion regulation including anger inhibition has been associated with greater HRV [12, 34,35,36]. In an early study we examined HRV during exposure to alcohol cues in remitted alcoholics [35]. We found that those alcoholics that could successfully regulate their impulse to have a drink showed greater HRV to alcohol cues compared to those that expressed a desire to drink after alcohol cue exposure. Butler and colleagues [34] using the emotion regulation paradigm pioneered by James Gross showed that both reappraisal and suppression were associated with greater HRV. With respect to responses to unfair treatment and the regulation of anger, we showed that after a contentious debate with an EA confederate, AAs that inhibited their anger toward the confederate had greater HRV than AAs that expressed their anger toward the confederate [12]. Furthermore, consistent with the social learning theory model of anger

regulation of Hokanson, AAs that expressed their anger had the lowest HRV. Recently, in a large sample of AAs and EAs we reported that anger-in was associated with greater HRV whereas anger-out was associated with lower HRV in AAs only [36]. In a very large sample in Brazil, where race is defined phenotypically by skin color, we reported that darker skinned Brazilians reported greater HRV, greater rates of hypertension, and greater reported discrimination compared to lighter skinned Brazilians [37]. Importantly, this association between ethnicity (darker skin tone) and HRV was partially mediated by reported discrimination. That is, the greater the reported discrimination the greater the HRV suggesting that greater HRV was associated with inhibited anger about unfair treatment. Taken together, these findings suggest that AAs that have to inhibit their anger in response to unfair treatment may have to regulate their emotions more and as a consequence have greater emotion regulation related HRV. This represents a potential psychological factor that might lead to greater HRV in AAs compared to EAs.

Whereas these results from Brazil examined the relationship between perceived discrimination and HRV *between* ethnic groups we have also investigated the relationship between perceived discrimination and HRV *within* AAs. Within AAs, greater perceived discrimination is associated with *lower* HRV [38] (see Figure 2a). Importantly, this association was mediated by rumination such that only those AAs that had both lower HRV and higher rumination reported greater perceived ethnic discrimination (see Figure 2b). Rumination about ethnic discrimination has been reported to be higher in AAs [11]. In addition, we have provided meta-analytic and experimental evidence that perseverative cognition such as rumination, worry, and angry brooding is associated with both lower HRV and higher vascular responses such as blood pressure and total peripheral resistance [39,40].

A related emotion regulation strategy that might be associated with the greater total peripheral resistance in AAs involves so-called emotional dampening. It has been well documented that greater blood pressure is associated with reduced pain sensitivity (hypoalgesia). A recent meta-analysis suggests that this is associated with an approximately one third of a standard deviation magnitude effect [41]. Interestingly, this effect is seen in neonates suggesting that this effect is potentially due to physiological rather than behavioral factors [42]. Importantly, this effect is not restricted to pain, and greater blood pressure has been associated with decreased responses to both negative and positive emotional stimuli. Of particular relevance for the present discussion, we have reported that this emotional dampening effect is related specifically to total peripheral resistance and extends to both visual and written emotional stimuli in an AA sample [43]. Thus, the greater total peripheral resistance in AAs could be associated with an attempt to attenuate the negative emotions associated with unfair treatment.

In the above we have summarized evidence for potential physiological and psychological factors that might underpin the cardiovascular conundrum pattern of greater HRV and greater total peripheral resistance in AAs relative EAs. However, the pressing question of what can be done to cope with this anger so that, to paraphrase Baldwin's words, "it does not destroy us" remains to be addressed.

Summary and Conclusions

If we are to accelerate the science on health disparities in behavioral medicine, we may need to develop new ways to conceptualize racial and ethnic group differences. Whitfield and colleagues have written cogently on this topic and identified three models that have been used to interpret results from studies on racial group differences [44]. One historically common model has viewed such racial group differences as suggesting that one group is inferior or deviant (the Cultural

Deviant model) thus blaming the victim as it were. Studies attributing these racial group differences to genetics or poor health behaviors may fall into this category. Alternatively, such racial group differences may be framed as adaptations to external forces and emphasizes within ethnic group heterogeneity (the Cultural Variant model). Therefore, in looking for answers to explain these health disparities, we must be extremely careful not to fall into the trap of blaming the victim. As D.L. Hughley notes, “Black people are always the first suspects in their own deaths”. That said, the physiological and psychological factors associated with the cardiovascular conundrum may give some clues as to what can be done to cope with this anger in response to unfair treatment. With respect to the physiological factors, not much can be done with respect to our genetics. Misguided attempts to attribute these health disparities primarily to genetic factors is one example of a blaming the victim strategy. Similarly, with respect to diet, attributing these health disparities to health behaviors such as poor food choices (e.g., eating high salt, high fat foods) also blames the victim. Though beyond the scope of the present paper the structural factors that lead to so-called food deserts and the lack of both resources and access to better food have been well documented [45]. In this context it is interesting to note research adopting the Cultural Variant model comparing Black Americans from the US Virgin Islands to Blacks from the US states and Washington DC. They found that whereas Blacks from the US states had higher rates of cardiovascular disease and associated risk factors, these differences could not be attributed to health behaviors or socioeconomic status [46]. However, better prenatal care to reduce premature and low birth weight babies and increased intake of potassium to lower BP represent things that can be meaningfully advanced to help mitigate the physiological factors associated with the cardiovascular conundrum.

With respect to psychological factors, worry postponement and meditation may help to decrease the rumination and perseverative cognition associated with lower HRV, greater vascular activity, and hypertension [11,47,48]. In addition, it has been reported that AA women that reported using prayer to cope with racism had lower resting blood pressure and total peripheral resistance compared to those that did not use prayer coping [49]. Those that used prayer also reported lower Anger-Out scores (an index of anger expression) and lower Anger-Control scores. In addition, those that reported using prayer coping had lower blood pressure reactivity during recall of an incident of maltreatment or disrespect that participants attributed to racism, and lower blood pressure and greater HRV during the recovery from such racism recall. Thus, there appear to be some things that individuals can do to help mitigate the effects of unfair treatment. However, laying the burden of coping with the unfair treatment solely on the individual is to overlook the structural factors associated with inequality and the stress of exposure to unfair treatment [11]. For example, a report entitled "The Asset Value of Whiteness" documented the great disparity in wealth between Black and Brown people compared to their White counterparts [50]. It was found that commonly asserted causes of these wealth disparities associated with individual behavior such as lack of education, family instability, lack of motivation to work hard, and poor spending habits did not account for these differences in wealth [50].

In this paper we have used the insightful words of one of America's greatest social commentators on matters of race to provide a framework for understanding the challenges associated with coping with unfair treatment. The effects of such unfair treatment are borne not only by the persons directly involved but by those that are indirectly exposed as well. The pattern of psychophysiological response to unfair treatment we identified likely represents an adaptation to external forces consistent with the Cultural Variant model of racial group differences. Clearly,

changes in the structural factors associated with unfair treatment are needed to mitigate the consequences of unfair treatment. Changes such as criminal justice reform and equal pay would certainly help to address the lack of equality present in our society. However, these require changes to deeply embedded political and cultural institutions and will require the dedicated work of all of us. However, in the words of James Baldwin, “Not everything that is faced can be changed, but nothing can be changed until it is faced”.

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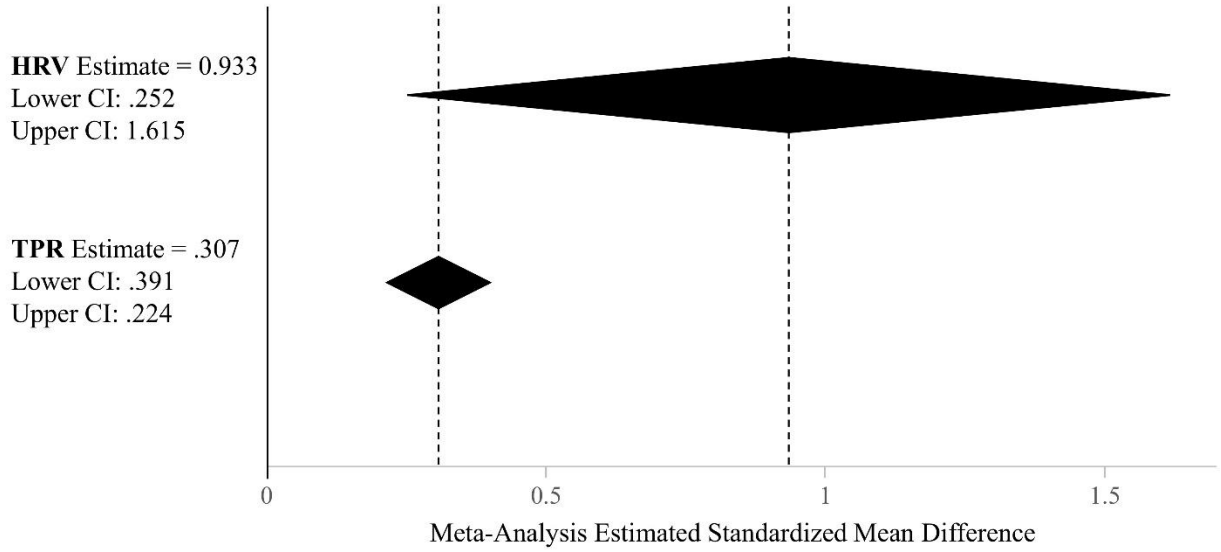
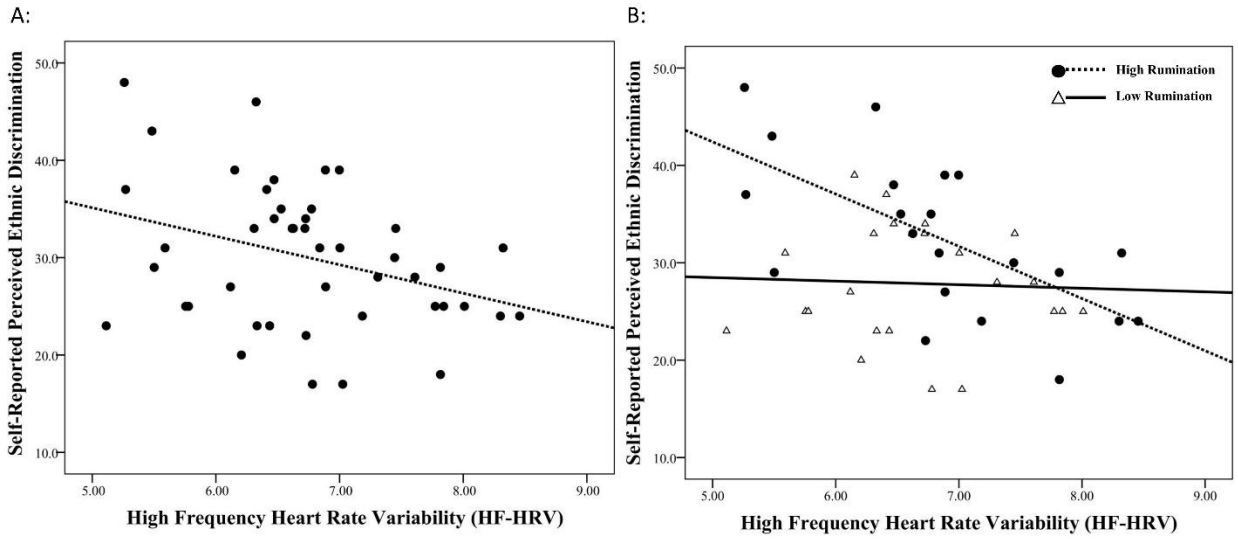
Figure 1. Ethnic Differences in both heart rate variability (HRV) and total peripheral resistance (TPR) Based on Meta-Analysis Findings



Note: Figure 1 represents estimated mean differences in both HRV and TPR based on prior meta-analyses (Hill et al., 2012; Brownlow et al., 2020). Based on our findings, AAs are nearly one standard deviation (Hedge's g = .93) higher in baseline HRV (Hill et al., 2012) and 1/3 standard deviation (Hedge's g = .307) higher in baseline TPR (Brownlow et al., 2020) compared to EAs. Greater mean differences in the above figure reflect a greater mean value (i.e., HRV and TPR) in AAs compared to EAs.

Figure 2. Scatterplots Depicting Relations between Heart Rate Variability, Perceived Ethnic Discrimination, and Rumination in African Americans



Note: Figure 2A is a scatterplot depicting the significant association between high-frequency heart rate variability (HF-HRV) and perceived ethnic discrimination in a sample of African Americans ($r = -.303$, $p < .05$). Figure 2B depicts rumination as significant moderator of this association (*see* Williams et al., 2017 for all statistics), such that in African Americans with higher self-reported rumination (i.e., African American above the median) showed the negative association between HF-HRV and perceived ethnic discrimination, whereas this association was not apparent in African Americans who report lower levels of rumination (i.e., African Americans below the median).