Title

Testing State and Trait Influences of Anxiety, Anger, and Sadness on Ambulatory Blood Pressure and Whether Race Impacts These Relationships

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Publication Date

2020

Peer reviewed|Thesis/dissertation

UNIVERSITY OF CALIFORNIA, MERCED

Testing State and Trait Influences of Anxiety, Anger, and Sadness on Ambulatory Blood Pressure and Whether Race Impacts These Relationships

A Thesis submitted in partial satisfaction of the requirements for the degree of Master of Arts

in

Psychological Sciences

by

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2021

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2021

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Acknowledgments

Thank you to my faculty committee, friends, and family for your constant encouragement and support. Thank you for encouraging me to work toward my dreams and believing in me.

Abstract

Testing State and Trait Influences of Anxiety, Anger, and Sadness on Ambulatory Blood Pressure and Whether Race Impacts These Relationships

by Larisa Gavrilova for the partial satisfaction of the requirements for the degree of Master of Arts in Psychological Sciences
University of California, Merced 2021
Dr. Matthew Zawadzki, Chair

Anxiety, anger, and sadness are related to elevated ambulatory blood pressure (ABP), yet it is unclear whether each emotion exerts unique effects. Moreover, an understanding of who might be most susceptible to the negative effects of these emotions is limited, with the trait tendency to experience them or one's race as potential moderators. Participants (*n* = 153) completed trait anxiety, anger, and depressive symptoms measures at baseline. ABP was collected over two 24-hour periods 3-4 months apart. Momentary measures of anxiety, anger, and sadness were assessed via ecological momentary assessment (EMA) after each ABP reading. Results revealed that momentary anxiety consistently predicted DBP, but momentary anger and sadness did not. Conditional effects were found with momentary anxiety and anger predicting elevated BP in those individuals with trait anxiety/anger at its mean. Trait anxiety and depression consistently predicted heightened BP in Black participants. Findings suggest that momentary anxiety and anger should be given attention as a potential risk factors for hypertension and highlight the unique perspective of EMA methods. Black participants who were more anxious and depressed experienced heightened BP, with anxiety and depression providing possible intervention targets in improving racial disparities in cardiovascular health.

Introduction

Considering the high prevalence of hypertension (Benjamin et al., 2019) and its life-threatening consequences (WHO, 2013), it is critical for researchers to investigate factors contributing to high BP. One factor might be negative emotional experiences, as abundant evidence suggests that anxiety, anger, and sadness relate to high blood pressure and its downstream consequences, including hypertension (e.g., Suls, 2018). Yet, due to an overlap among anxiety, anger, and sadness, researchers tend to either evaluate the effects of a single negative emotion at a time or average across these negative emotions to create a composite score of negative affect. These approaches either assume that a single type of negative emotion may be sufficient to contribute to harmful effects on health (Suls & Bunde, 2005; Suls, 2018), or disregard the possibility that a particular emotion has an independent relationship with BP and thus may present a unique target for an intervention. Furthermore, much of previous work has focused on trait measures of anxiety, anger, and sadness that help stratify who might be most at risk for hypertension. However, how these trait tendencies contribute to the development of hypertension in people's everyday life remains unclear. Possibilities may range from the trait tendencies leading to increased experiences of negative emotion to functioning as a moderator in which the trait tendency sensitizes people to the momentary experience of the negative emotion causing an exaggerated response.

Ecological momentary assessment (EMA; Stone & Shiffman, 1994) makes it possible to measure emotions repeatedly within an individual and examine the relationship between one's emotions and physiology as it unfolds over time. This approach may be combined with more traditional approaches to test whether these effects are amplified for those individuals with a trait tendency to experience certain negative emotions (e.g., a state by trait interaction). In addition, it is possible to test whether certain groups of people may be at an increased risk for the deleterious effects of negative emotions. Notably, some evidence suggests that Black participants respond to negative emotions and stress differently than non-Black participants (Barnes et al., 2000; Wilcox et al., 2005), yet these associations have had limited testing in the context of people's everyday lives. Better understanding whether the experience of negative emotions in daily life contributes to racial disparities in cardiovascular disease is important; this knowledge may inform interventions aimed at reducing racial health disparities in cardiovascular health. The present study investigated the potential for differential effects of momentary anxiety, anger, and sadness on ambulatory blood pressure (ABP). It then assessed whether a trait tendency to experience these negative emotions and/or race (Black versus non-Black) would moderate the relationships between negative emotions and BP.

BP and Negative Emotions

Type of Emotion. Most research examining the importance of negative emotional experiences has focused on trait tendencies and their relation to the prevalence of hypertension. For example, more trait anxiety (Pan et al., 2015), trait anger (e.g., Gentry et al., 1982; Schum et al., 2003), and depression (Patten et al., 2009) each predict higher rates of hypertension. However, there are limitations to this work. Due to an appreciable construct and measurement overlap among the dispositions of anxiety, anger, and sadness and shared pathways by which they contribute to cardiovascular disease, much of this work has focused on evaluating the effects of a single affective disposition at a time (Suls & Bunde, 2005; Suls, 2018). This approach assumes that a general tendency to experience negative emotions, rather than any specific negative emotion may explain the harmful effects of negative emotions on health. A common alternative approach involves researchers averaging the negative emotional states together to create a composite score of negative affect (Crawford & Henry, 2004). Yet, this approach may be problematic as the overlap among anger, anxiety, and sadness opens the possibility that these emotions act synergistically and substantially increase the risk of cardiovascular disease (Rozanski et al., 1999; Suls & Bunde, 2005). Furthermore, this approach ignores the prognostic importance of the different negative emotions within the same individual (Frasure-Smith & Lespérance, 2003; Suls, 2018; Suls & Bunde, 2005).

Despite the overlap in valence, negative emotions have distinctive features that may further justify examining them separately. Sadness is characterized by low physiological arousal, whereas anxiety and anger are both characterized by high physiological arousal (Russel, 1980). In addition, anxiety, anger, and sadness are characterized by different cognitive appraisals (Roseman, 2013), with anxiety being characterized as anticipation of a perceived future stressor, anger as an emotional reaction to injustice, and sadness being characterized by perception of loss and withdrawal (Leventhal, 2008). Demonstrating the uniqueness of these negative emotions, Kubzansky and colleagues (2006) found that when shared general distress was accounted for, tendency to experience anxiety and anger were differentially predictive of coronary heart disease outcomes. Findings like this highlight the need for research testing the unique effects of anxiety, anger, and sadness simultaneously within the same person if we are to better understand whether these emotions impact outcomes differently. Thus, one goal of this paper is to systematically examine whether anxiety, anger, and sadness uniquely predict ambulatory BP.

Method of Assessment. In addition, previous work provides limited insight into how the risk for hypertension develops. For example, it remains unclear whether the effects of trait anxiety on cardiovascular health are driven by repeated instances of feeling anxious or, whether the trait measure taps into a different construct that uniquely contributes to the risk of developing hypertension. Previous work highlights the uniqueness of the state and trait measure. For example, research reveals that state anxiety is more commonly predicted by a specific situation, rather than individual's trait anxiety (Mothersill et al., 1986). In a similar vein, one recent study found that trait anxiety does not positively correlate to state anxiety across all threatening situations (Leal et al., 2017). Together, these studies highlight that assessing the relationship between emotions and BP as a dynamic (within-person) process may complement the traditional trait approach.

EMA approach allows researchers to assess the relationships between emotions and BP in the real world as it unfolds over time within a person. In addition to testing these within-person associations, researchers are able to gain insight into whether trait tendencies to experience negative emotions moderate the relationships between the momentary experiences of an emotion and BP. For example, an individual with high trait anxiety may experience a stronger physiological response during a high anxiety moment than a person with low trait anxiety. Understanding trait as a potential moderator provides a more nuanced understanding of the relationship between emotions and BP and can inform the development of interventions delivered during moments of high vulnerability for those individuals most prone to experiencing those vulnerable moments or those most sensitive to them.

The second goal of the present study was to assess trait as a potential moderator of the association between momentary anxiety, anger, and sadness and BP in daily life. Due to trait sadness not being typically studied in the way that trait anxiety and anger have been examined, the study used a measure of depressive symptoms over the past two weeks as a stand-in for trait sadness, as we believe there is enough conceptual overlap to make comparisons (or at least as much overlap as between any trait measure and characteristic that persists for two weeks). Similarly, other researchers proposed that recurrent nature of depression suggests that some individuals have a depressogenic disposition and therefore, depressive symptomatology can be conceptually treated like a disposition (Judd & Akiskal, 2000; Suls, 2018; Suls & Bunde, 2005).

Race and Negative Emotions

Research suggests that there may be racial differences in the relationships between negative emotions and BP. Notably, individuals who identify as Black are likely to be exposed to more psychosocial stressors such as discrimination, unemployment, and lower socioeconomic status (e.g., Krieger & Sidney, 1996). These chronic stressors can produce a persistent experience of negative emotions, leading to sustained increased in ABP. Additionally, many studies indicate that Black individuals exhibit greater BP reactivity than do White individuals across a range of stressors (Barnes et al., 2000; Wilcox et al., 2005). Thus, persistent experience of negative emotions as well as greater BP reactivity may underly the observed race differences in cardiovascular health. Studies indeed suggest that Black participants overall have higher BP levels during negative mood states, and BP increases to a greater extent when they experienced more negative emotions compared to White participants (e.g., McGrath et al., 2006).

Given racial disparities in cardiovascular health (e.g., Lackland et al., 1998; Mensah et al., 2005) and research indicating that individual-level risk factors (e.g., smoking, obesity, dietary factors) and even socioeconomic status do not fully explain these racial disparities (e.g., Dressler et al., 2005, Lackland, 2014), it is necessary for research to consider the role of negative emotions. Understanding how negative emotions impact cardiovascular health in Black individuals as they navigate their daily lives is critical as this knowledge may provide insights into ways to reduce health disparities in this population. Thus, the third goal of this paper is to examine whether race moderates the relationship between emotions and BP.

Current Project

The current study aimed to examine the relationship between anxiety, anger, and sadness assessed in daily life via EMA and ABP. Research Question 1 investigated whether each negative emotion had a unique relationship with ABP. Due to the link with high physiological arousal, we expected that anxiety and anger would produce a stronger effect on BP compared to sadness, with stronger effects for anxiety given its association with hyperarousal. Research Question 2 assessed whether a trait tendency to experience a certain emotion assessed at baseline would moderate these effects. We hypothesized that individuals with high trait anxiety would experience a stronger BP response during moments with high anxiety than those with low trait anxiety. Similar predictions were made for trait anger moderating momentary anger, and depressive symptoms moderating momentary sadness. Research Question 3 tested whether race moderates the effects of negative emotions on BP response. We hypothesized that effect of negative emotions on BP would be stronger among Black participants than non-Black participants.

Method

Participants

Participants were recruited by advertisement at Columbia University Medical Center. The study included 282 participants. See Table 1 for demographics. Participants were excluded if they were under 18 years old, were unable to read and write in English, were diagnosed with diabetes, irregular heartbeat, or cerebrovascular disease (including stroke), were currently taking mood altering medication (including antidepressants, antianxiety medications or prescription sleep aids), or were currently pregnant, as these impact the interpretation of ABP readings. Out of 282 participants who completed the baseline survey, 153 participants completed the ambulatory part of the study.

Procedure

At an initial baseline session, informed consent was obtained from all participants who agreed to participate in the study. A research assistant explained study procedures and confirmed eligibility. Participants completed demographics and were scheduled for the next session 1-2 weeks later. At this follow-up, participants were fitted with a SpaceLabs ABP monitor to undergo a 24-hour protocol. The monitor took ABP readings every 20 minutes during daytime, and every 30 minutes during nighttime. Daytime readings were preprogrammed to occur between 6 am and 10 pm; nighttime readings occurred between 10 pm and 6 am. This timing coincided relatively well with most participants' sleep and awake schedules. Participants were also given a PalmPilot handheld computer and were asked to complete surveys after each daytime ABP reading. After this session, the procedure was repeated once more after 3-4 months to increase the number of measurements per person but to minimize burden by having consecutive days of measurement. ABP data had a total of 8528 readings for SBP and 8517 for DBP, with an average of 32.3 SBP and 32.3 DBP readings per person. The EMA resulted in a total of 8528 assessments, with an average of 32.3 assessments of per person.

Measures

Baseline measures. During baseline, participants completed demographic information about participants' age, gender (coded as 0 = male, 1 = female), and race (coded as 0 = non-Black, 1 = Black). Next, trait anxiety was assessed with the Taylor Manifest Anxiety scale (Taylor, 1953), which consists of 50 true-false questions assessing the tendency to experience anxiety in a variety of situations (e.g., "I feel anxiety about something or someone almost all of the time."). The total score is calculated by adding up the number of chosen responses that correspond to the responses in the answer key. The total score ranges from 0 to 38, with higher scores indicating higher tendency to experience anxiety ($\alpha = .73$).

Trait anger was assessed with the State-Trait Anger Scale (STAS-T; Spielberger et al., 1983), which consists of 15 items assessing individuals' proneness to anger as a personality trait (e.g., "I am quick tempered."). Participants rated items on a 1 (*almost never*) to 4 (*almost always*). The total score is calculated by summing the ratings for each of the questions. The total score ranges from 15 to 60, with higher scores indicating higher tendency to experience anger ($\alpha = .94$).

Finally, experience of depressive symptoms in the past two weeks was assessed with the Beck Depression Inventory-Second Edition (Beck et al., 1996) which consisted of 21 items being rated on a 4-point scale ranging from 0 to 3 assessing self-reported symptoms of depression based on a severity of each item (e.g., "I am sad all the time."). The total score is calculated by summing the ratings for each of the questions, with scores ranging from 0 to 63. Higher scores indicate greater depressive symptoms ($\alpha = .91$).

EMA measures. Participants were asked to separately rate their anxious, angry, or sad emotional states on a 0 to 100 sliding scale, with higher scores indicating higher experience of anxiety, anger, and sadness. In addition, participants reported on their position ranging from laying down (1) to standing (2) to active (3), to control for how physical movement can influence ABP.

Data Analysis

We tested a two-level structure with momentary observations (Level 1) nested within individuals (Level 2). The PROC MIXED procedure in SAS 9.4 was used to conduct a hierarchical mixed effects analysis. Time of day (number of minutes since midnight), age, gender, race, and body position were included in all subsequent analyses as control variables. Session was modeled as all of the EMA assessments within session 1 versus EMA observations within session 2 that occurred 3-4 months later to control for potential differences across the two measurement periods.

Research Question 1 tested for differential effects of momentary anxiety, anger, and sadness on ABP. For these analyses, momentary anxiety, anger, and sadness were entered in the same model. Momentary level variables (Level 1) were person-mean centered around the individual's overall mean to examine the within-person effect of anxiety, anger, and sadness on ABP separately from the between-person effect (Bolger & Laurenceau, 2013).

Research Question 2 tested whether these effects were moderated by the general tendency to experience these negative emotions. Specifically, we assessed whether momentary anxiety (Level 1) and trait anxiety (Level 2) levels interacted in such a way that individuals with high trait anxiety will experience a stronger BP response to momentary anxiety, compared to those with low trait anxiety. Similar models were run for anger and sadness. In addition, the average across momentary assessments of negative emotions was included in the models as an additional control variable.

In order to interpret results of the interactions in a more meaningful way, we centered the predictors. As recommended by Bolger and Laurenceau (2013), momentary data were first grand-mean centered by subtracting the sample mean from every momentary score. These grand mean-centered variables were subsequently used to calculate the person-mean centered and person-mean variables. In these analyses, an autoregressive covariance structure was modeled to account for the likelihood that BP measurements closer in time to each other are more strongly related than those further apart. Random intercepts were included to account for individual differences in initial SBP and DBP. We tested fixed effects for all predictors besides the intercept.

Finally, Research Question 3 tested whether race moderates the relationship between momentary negative emotions and BP. The average across momentary assessments of negative emotions as well as trait measure were included in the models as additional control variables. In the resulting models, momentary (Level 1), the average across momentary assessments (Level 2), trait measure (Level 2), as well as interaction terms between each of these variables and race were entered in the same model. Separate models were run for anxiety, anger, and sadness. In these analyses, negative emotion variables were grand-mean centered.

Results

Overall, participants had an average SBP of 125.73 (*SD* = 12.71) and DBP of 79.86 (*SD* = 9.37). Means, standard deviations, and correlations for emotional states of anxiety, anger, and sadness (that in this context represent an average of each emotion state across all observations for the participant) and traits anxiety, anger, and depression for the full sample are shown in Table 2. Furthermore, descriptive statistics for the predictor variables were calculated for Black and non-Black participants. Independent samples t-test indicated that trait anxiety, anger, and depression were significantly different among the two groups; nonequal variances were assumed. Black participants reported significantly lower trait anxiety (*M* = 5.03, SD = 3.45) compared to non-Black participants (*M* = 5.81, *SD* = 3.99), *t*(5385.6) = 7.83, *p* < .001. Black participants also reported significantly lower trait anger (*M* = 16.23, SD = 4.50) compared to non-Black participants (*M* = 16.79, *SD* = 4.29), *t*(5602.5) = 4.78, *p* < .001. Black participants reported significantly higher trait depression (*M* = 5.82, *SD* = 5.36) compared to non-Black participants (*M* = 4.64, *SD* = 5.52), *t*(5562.9) = -8.10, *p* < .001. For momentary experiences of emotions, Black participants reported significantly higher experience of anxiety (*M* = 21.15, *SD* = 24.07) compared to non-Black participants (*M* = 15.69, *SD* = 19.82), *t*(5546.2) = -9.33, *p* < .001. Black participants also reported significantly higher experience of anger (*M* = 7.21, *SD* = 13.12) compared to non-Black participants (*M* = 5.86, *SD* = 11.83), *t*(5626.6) = -4.04, *p* < .001. There were no significant differences in sadness, *t*(5595.5) = -0.98, *p* = .33, despite Black participants reporting slightly higher scores on sadness (*M* = 6.32, *SD* = 11.45) compared to non-Black participants (*M* = 6.02, *SD* = 11.63).

Research Question 1 tested differential relationships between momentary anxiety, anger, sadness, and BP. As Table 3 shows, when all negative emotions were included in the same model, momentary anxiety uniquely predicted elevated DBP (*p* = .032), while anger and sadness did not predict BP (*ps* > .147).

Research Question 2 tested the presence of a moderation effect between the momentary and trait levels; three separate models were run for anxiety, anger and sadness (see Tables 4, 5, 6). No significant effects were found for the interaction between the state and trait emotion for either anxiety, anger, or sadness (*ps* > .081). There were conditional effects for the momentary effect of anxiety on both SBP (*p* = .038) and DBP (*p* = .014), such that momentary anxiety predicted elevated BP (with this term representing the effect of momentary anxiety for those individuals with trait anxiety at its mean). In addition, the conditional effect of momentary anger (*p* = .037) was found, with momentary anger predicting elevated DBP in those individuals with trait anger at its mean.

Research Question 3 tested the presence of a moderation effect between negative emotions and race. When testing whether race moderates the relationship between anger and SBP, the covariance matrix did not estimate for this model. As a result, this model was rerun without this restriction permitting responses to be independent. A moderation effect was found between trait anxiety and race (see Figure 1); Black participants with high trait anxiety had higher SBP (*p* = .013) and DBP (*p* = .040) compared to non-Black participants with high trait anxiety, see Table 7. No moderation or conditional effects were found for anger and race on SBP (*ps* > .068), see Table 8. Interaction effect was found between momentary anger and race (*p* = .031) on DBP; both low and high momentary anger predicted higher DBP in Black participants compared to non-Black participants suggesting harmful effects of anger, both low and high, for Black participants. Finally, a moderation effect was found between trait depression and race (see

Figure 2); high trait depression predicted higher SBP (*p* = .012) and DBP (*p* = .020) in Black participants compared to non-Black participants with high trait depression, see Table 9.

Discussion

Research Question 1 investigated whether momentary anxiety, anger, and sadness differentially predict BP. Momentary anxiety emerged as a robust predictor of DBP across all models suggesting a unique link between anxiety and DBP. We interpret these findings in line with many studies indicating that anxiety is associated with physiological hyperarousal and symptoms such as pounding heart, tense muscles, and exaggerated startle. As an illustration, previous studies reported higher resting heart rate in individuals with high trait anxiety (Kelly et al., 1970). Given these findings, along with other researchers (Thurston et al., 2013), we recommend that special attention should be placed on anxiety in-the-moment as a potential risk factor for hypertension although more work is needed in this area. In the present data, participants reported more anxiety across moments than anger and sadness suggesting it is more prevalent as a risk factor in this sample (and that this greater range and variability enables more power to detect relationships with ABP). It may be that individuals who experience elevated BP and intense feelings of anxiety are at an increased risk for developing hypertension and cardiovascular disease and thus, may benefit from additional screening.

Unexpectedly, the study did not find an effect of momentary anger and sadness on BP. We offer several possible interpretations of these results. Much work tested the relationships between these negative emotions and BP cross-sectionally and thus, used a between-person approach. Less is known about the momentary experience of anger and sadness that normally vary throughout the day for an individual. For sadness, these results may be, in part, explained by the fact that while living with depression over time may predict hypertension, the study measured BP over a short period of time – although the two 24-hour periods of ABP data 3-4 months apart suggests a robustness of the findings. Furthermore, most work has focused on depression, a complex mood disorder that is the result of a persistent set of symptoms, including feelings of both sadness and anxiety (APA, 2013). It may be that the link between depression and hypertension exists at least in part, due to the anxiety component, not solely sadness. Lastly, these results may be attributed to low anger and sadness scores across our sample. Further empirical work is needed to test these assumptions.

Research Question 2 tested whether these effects were moderated by the general tendency to experience these negative emotions. Trait level did not significantly predict BP in the models, and no interaction effect was found between momentary (Level 1) and trait (Level 2) levels. These findings are in contrast to previous work linking trait anxiety, anger, and depressive symptoms to elevated BP (e.g., Pan et al., 2015; Schum et al., 2003). The lack of direct associations between traits and BP may be explained by low trait levels of anxiety, anger, and depressive symptoms in our sample which reduced the potential to detect effects. The lack of interaction effects appears to indicate that the momentary negative experience of emotion relates similarly to BP for all individuals highlighting the need to focus on in-the-moment experiences of negative emotions than measures of the trait tendencies to experience those emotions.

Research Question 3 tested whether Black participants versus non-Black participants exhibit different relationships between the experience of negative emotions and BP in everyday life. Results indicated that trait anxiety was associated with higher BP in Black participants compared to non-Black participants. The findings about trait anxiety are worth noting; Black participants reported significantly lower trait anxiety compared to non-Black participants and yet, trait anxiety was associated with heightened BP for Black participants but not for non-Black participants. These results could at least in part, be explained by research suggesting that Black individuals have a higher cardiovascular reactivity to a variety of stressors (Wilcox et al., 2005);

it is possible that the experience of a negative emotion and heightened reactivity act synergistically to produce harmful effects in Black individuals.

The experience of both low and high momentary anger predicted higher DBP in Black participants, suggesting that anger is detrimental for Black participants. Although speculative, one possible explanation of this finding is that the experience of anger may be different for Black and non-Black individuals, with Black individuals experiencing more discrimination-related anger. Studies indeed consistently link perceived racism with exaggerated blood pressure reactivity (Clark, 2000; Fang & Myers, 2001). Future studies are needed to differentiate between the effects of discrimination-related and non-discrimination-related anger on BP in Black individuals to better understand these disparities.

Results also indicated that depressive symptoms were associated with higher BP in Black participants compared to non-Black participants. These results may be interpreted in line with literature suggesting that racial discrimination is related to both increased levels of depressive symptoms and increased odds of depression (e.g., Hudson et al., 2012). As a potent stressor, discrimination may induce stress-related changes in BP. Furthermore, research suggests that living in economically and socially challenging environments which many Black individuals are exposed to can be psychologically challenging (Taylor et al., 1997) and can be another factor to further contribute to cardiovascular functioning. In our sample, Black participants indeed reported significantly higher trait depression compared to non-Black participants. Overall, the results indicate that dispositions toward anxiety and depression are more harmful for Black individuals, providing a plausible explanation to racial disparities in cardiovascular health. Our findings suggest that when looking at cardiovascular health, emotional health needs to be factored in as an important risk factor of cardiovascular disease, especially in Black individuals. Finally, it is important to note that state and trait measures appeared to produce non-redundant information. Notably, Black participants reported significantly lower trait anxiety and anger than non-Black participants, but significantly higher experience of anxiety and anger. These results highlight the unique perspective of the between-person (trait) and within-person (momentary experience of emotion) approaches which may explain the conflicting results that often occur for studies examining the relationships between affective states and physiology (Feldman et al., 1999). In their recent work on disentangling this issue, Zawadzki and colleagues (2017) argue that the between-person and within-person approaches ask fundamentally different questions. For example, in this context, within-person analyses hold a great potential to help understand the relationship between affect and physiology and be particularly important in interventions delivered in time of vulnerability. In contrast, the between-person trait approach may reflect the repeated patterns of BP reactivity and recovery that occur within a given time period (Smyth et al., 2018).

Limitations and Future Directions

While anxiety, anger, and sadness were used to compare the effect of high arousal versus low arousal emotions, other emotions that differ on the arousal component should be investigated to gain a more nuanced insight on the effect of negative emotions on physiology. For example, while such negative emotional states as boredom and tiredness are characterized by a low arousal, they may have a different effect on BP compared to sadness. Similarly, fear and frustration may produce different effects compared to anxiety and anger despite of these states being characterized by a high arousal.

Future work should test positive emotions as they also differ on the arousal dimension. In accord with the circumplex model, Posner and colleagues (2005) posit that both positive and negative emotions arise from common, overlapping neurophysiological systems which may explain comparable physiological response to different emotions. In regard to cardiovascular function, Dockray and Steptoe (2010) conclude that the pattern of cardiovascular response and recovery may be the same for high arousal positive and negative emotions, with positive emotions being of lesser magnitude. Empirical evidence comes from the study conducted by Jacob and colleagues (Jacob et al., 1999); it was found that emotions labeled as “anxious/annoyed” and “elated/happy” were associated with comparable levels of BP reactivity. One important explanation of the differences between the physiological effects of negative and positive emotions is that negative emotions may imply some difficulties that may require time to be resolved. In addition, this may imply worrying or ruminating about these difficulties which may in turn prolong the duration of the stressor (Smyth et al., 2013).

The study did not assess whether participants were taking antihypertensive medication. It is known that antihypertensive medication may impact the interpretation of ABP readings, but it is unclear if these medications would alter the relationship between emotions and BP compared to just raising and lowering BP. Lastly, due to the most variance being found in the momentary EMA and between-person level, these two levels were the main focus of the study and variance due to the day level was not explored. It is important to note that day variation may be an important factor impacting BP. For example, previous work suggested higher levels of physiological stress (i.e., cortisol levels) on workdays compared to non-workdays (Myers et al., 2012). Thus, future work should further investigate the relationship between BP and affective states on the day level ensuring a large enough sample of days, including both workdays and non-workdays.

Summary and Implications

Overall, the results align with cross-sectional evidence linking anxiety and anger to elevated BP. The results emphasize the importance of investigating in-the-moment anxiety and anger as a potential risk factor for hypertension. Along with other researchers (Myers et al., 2012; Zawadzki et al., 2017), we recommend the use of within-person analyses in examining the relationship between affect and BP, as it provides a unique perspective into the relationship between affective states and physiology. Trait anxiety and depression appeared to be particularly harmful for Black than non-Black individuals providing a possible intervention target in improving racial disparities in cardiovascular health.

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Table 1

Demographics of Participants (n = 153)

Characteristic	Statistic
Age	
Range	20-70
Mean	40.59
Sex	
Female (*n*, %)	81, 52.94%
Male (*n*, %)	71, 46.41%
Missing (*n*)	1
Race	
White	34, 22.82%
Black (*n*, %)	84, 56.38%
Asian (*n*, %)	15, 10.07%
Native American (*n*, %)	2, 1.34%
Other (*n*, %)	13, 8.72%
Missing (*n*)	5
Ethnicity	
Hispanic/Latino	22, 14.77%
Non-Hispanic/non-Latino	125, 83.89%
Missing (*n*)	6

Note. n = sample size

Table 2

Pearson Correlations Among State and Trait Measures

Measure	M	SD	1	2	3	4	5
Average anxiety	22.00	18.26	-				
Average anger	9.59	12.62	.57***	-			
Average sadness	9.55	13.96	.53***	.85***	-		
Trait anxiety	5.55	3.67	.16$^+$	.16*	.15$^+$	-	
Trait anger	16.69	5.21	.17*	.25**	.23**	.49***	-
Trait depression	5.93	6.76	.19*	.26**	.33***	.51***	.44***

Note. ***p < .001; **p < .01; *p < .05; ^+p < .10. Average is the grand mean of states across all participants (n = 263). Trait is the grand mean of traits across all participants (n = 151).

Table 3

Parameter Estimates (Standard Errors) for Systolic and Diastolic Blood Pressure with Momentary Anxiety, Anger, and Sadness as Predictors

	SBP	DBP
Fixed effects		
Intercept	135.64***	89.86***
	(4.80)	(3.50)
Time	-.003**	-.004***
	(.001)	(.001)
Session	1.52**	1.26**
	(.52)	(.42)
Age	.20*	.13*
	(.08)	(.06)
Gender	-8.91***	-6.09***
	(1.88)	(1.36)
Race	3.48^{+}	3.86**
	(1.96)	(1.42)
Position	-3.24***	-3.63***
	(.23)	(.20)
Momentary anxiety	.01	.02*
	(.01)	(.01)
Momentary anger	.01	.01
	(.01)	(.01)
Momentary sadness	.003	.01
	(.02)	(.01)
Random effects		
UN (1,1)	107.73***	54.87***
	(14.66)	(7.72)
Residual	51.19***	38.63***
	(2.27)	(1.91)
Model statistics		
AIC	39646.1	37995.5
BIC	39658.0	38007.3
Pseudo R^2	.19	.20

Note. ***$p < .001$; **$p < .01$; *$p < .05$; $^{+}p < .10$. Time is the number of minutes that elapsed since midnight. Session is entered as a continuous variable. Gender is coded as 0 = male and 1 = female. Race is coded as 0 = non-Black and 1 = Black. Momentary level is the momentary reports that are person-mean centered around the individual’s overall mean.

Table 4

Parameter Estimates (Standard Errors) for Systolic and Diastolic Blood Pressure with Anxiety as a Predictor and Trait Anxiety as a Moderator

	SBP	DBP
Fixed effects		
Intercept	136.37***	89.83***
	(4.87)	(3.55)
Time	-.003**	-.003***
	(.001)	(.001)
Day	1.54**	1.24**
	(.52)	(.42)
Age	.17*	.12*
	(.08)	(.06)
Gender	-8.72***	-5.79***
	(1.90)	(1.36)
Race	3.33$^+$	3.69*
	(2.01)	(1.45)
Position	-3.28***	-3.67***
	(.23)	(.20)
Momentary anxiety	.02*	.02*
	(.01)	(.01)
Average anxiety	.09	.07$^+$
	(.06)	(.04)
Trait anxiety	.15	.06
	(.27)	(.19)
Momentary X trait anxiety	-.002 (.002)	.003 (.002)
Average anxiety X trait anxiety	.001 (.02)	.01 (.01)
Random effects		
UN (1,1)	108.83***	54.67***
	(14.98)	(7.79)
Residual	51.08***	38.86***
	(2.28)	(1.87)
Model statistics		
AIC	39411.3	37761.0
BIC	39423.2	37772.8
Pseudo R^2	.19	.20

Note. ***p < .001; **p < .01; *p < .05; ^+p < .10. Time is the number of minutes that elapsed since midnight. Day is entered as a continuous variable. Gender is coded as 0 = male and 1 =

female. Race is coded as 0 = non-Black and 1 = Black. Momentary anxiety, average anxiety, and trait anxiety are grand-mean centered.

Table 5

Parameter Estimates (Standard Errors) for Systolic and Diastolic Blood Pressure with Anger as a Predictor and Trait Anger as a Moderator

	SBP	DBP
Fixed effects		
Intercept	136.55***	90.29***
	(4.87)	(3.56)
Time	-.003**	-.004***
	(.001)	(.001)
Day	1.54**	1.29**
	(.52)	(.42)
Age	.18*	.13*
	(.08)	(.06)
Gender	-8.85***	-5.94***
	(1.91)	(1.38)
Race	3.47^{+}	3.76**
	(2.00)	(1.45)
Position	-3.30***	-3.67***
	(.23)	(.20)
Momentary anger	.02	.02*
	(.01)	(.01)
Average anger	.10	.06
	(.12)	(.09)
Trait anger	.12	.09
	(.21)	(.15)
Momentary X trait anger	-.003	$-.004^{+}$
	(.003)	(.002)
Average anger X trait anger	.0002	-.01
	(.02)	(.01)
Random effects		
UN (1,1)	110.00***	55.88***
	(15.12)	(7.97)
Residual	51.09***	39.01***
	(2.28)	(1.86)
Model statistics		
AIC	39412.8	37764.9
BIC	39424.6	37776.7
Pseudo R^2	.19	.20

Note. ****p* < .001; ***p* < .01; **p* < .05; $^{+}p < .10$. Time is the number of minutes that elapsed since midnight. Day is entered as a continuous variable. Gender is coded as 0 = male and 1 =

female. Race is coded as 0 = non-Black and 1 = Black. Momentary anger, average anger, and trait anger are grand-mean centered.

Table 6

Parameter Estimates (Standard Errors) for Systolic and Diastolic Blood Pressure with Sadness as a Predictor and Trait Depression as a Moderator

	SBP	DBP
Fixed effects		
Intercept	137.16***	90.81***
	(4.80)	(3.51)
Time	-.003**	-.004***
	(.001)	(.001)
Day	1.53**	1.29**
	(.52)	(.42)
Age	.17*	.11^{+}
	(.08)	(.06)
Gender	-8.76***	-5.88***
	(1.88)	(1.36)
Race	3.73^{+}	4.00**
	(1.98)	(1.43)
Position	-3.33***	-3.71***
	(.23)	(.20)
Momentary sadness	.01	.02
	(.02)	(.01)
Average sadness	.19	.13^{+}
	(.10)	(.07)
Trait depression	.18	.13
	(.26)	(.19)
Momentary X trait depression	-.0003 (.003)	-.003 (.003)
Average X trait depression	-.027 (.02)	-.03 (.02)
Random effects		
UN (1,1)	106.83***	54.22***
	(14.73)	(7.73)
Residual	51.11***	38.97***
	(2.28)	(1.87)
Model statistics		
AIC	39409.4	37762.7
BIC	39421.2	37774.5
Pseudo R^2	.20	.20

Note. ***p < .001; **p < .01; *p < .05; ^{+}p < .10. Time is the number of minutes that elapsed since midnight. Day is entered as a continuous variable. Gender is coded as 0 = male and 1 =

female. Race is coded as 0 = non-Black and 1 = Black. Momentary sadness, average sadness, and trait depression are grand-mean centered.

Table 7

Parameter Estimates (Standard Errors) for Systolic and Diastolic Blood Pressure with Anxiety as a Predictor and Black as a Moderator

	SBP	DBP
Fixed effects		
Intercept	136.02***	89.90***
	(4.76)	(3.50)
Time	-.003**	-.003***
	(.001)	(.001)
Day	1.52**	1.27**
	(.52)	(.42)
Age	.19*	.12*
	(.08)	(.06)
Gender	-8.50***	-5.72***
	(1.86)	(1.35)
Race	3.24	3.61*
	(1.98)	(1.44)
Position	-3.29***	-3.66***
	(.23)	(.20)
Momentary anxiety	.02	.02^{+}
	(.01)	(.01)
Average anxiety	.18^{+}	.11
	(.10)	(.07)
Trait anxiety	- .50	- .29
	(.37)	(.27)
Momentary anxiety	-.001	-.002
X Race	(.02)	(.02)
Average anxiety X	- .13	-.06
Race	(.12)	(.09)
Trait anxiety X	1.30*	.78*
Race	(.52)	(.38)
Random effects		
UN (1,1)	104.17***	53.54***
	(14.45)	(7.68)
Residual	51.07***	38.91***
	(2.28)	(1.87)
Model statistics		
AIC	39396.9	37751.7
BIC	39408.7	37763.5
Pseudo R^2	.21	.20

Note. ***$p < .001$; **$p < .01$; *$p < .05$; $^{+}p < .10$. Time is the number of minutes that elapsed since midnight. Day is entered as a continuous variable. Gender is coded as 0 = male and 1 =

female. Race is coded as 0 = non-Black and 1 = Black. Momentary anxiety, average anxiety, and trait anxiety are grand-mean centered.

Table 8

Parameter Estimates (Standard Errors) for Systolic and Diastolic Blood Pressure with Anger as
a Predictor and Black as a Moderator

	SBP	DBP
Fixed effects		
Intercept	135.26***	89.32***
	(4.80)	(3.59)
Time	-.002*	-.004***
	(.001)	(.001)
Day	1.11***	1.26**
	(.29)	(.42)
Age	.20*	.14*
	(.08)	(.06)
Gender	-8.34***	-5.54***
	(1.95)	(1.40)
Race	3.11	3.58*
	(2.04)	(1.47)
Position	-3.61***	-3.68***
	(.24)	(.20)
Momentary anger	.03	.05**
	(.02)	(.02)
Average anger	.30	.16
	(.24)	(.17)
Trait anger	- .43	- .28
	(.35)	(.25)
Momentary anger X Race	-.04 (.03)	-.05* (.02)
Average anger X Race	- .21 (.28)	-.12 (.20)
Trait anger X Race	.78+	.51
	(.43)	(.31)
Random effects		
UN (1,1)	120.64***	55.51***
	(15.21)	(7.93)
Residual	95.01***	38.66***
	(1.86)	(1.89)
Model statistics		
AIC	40232.2	37751.7
BIC	40238.2	37763.5
Pseudo R^2	.19	.20

Note. ***p < .001; **p < .01; *p < .05; ^{+}p < .10. Time is the number of minutes that elapsed since midnight. Day is entered as a continuous variable. Gender is coded as 0 = male and 1 =

female. Race is coded as 0 = non-Black and 1 = Black. Momentary anger, average anger, and trait anger are grand-mean centered.

Table 9

Parameter Estimates (Standard Errors) for Systolic and Diastolic Blood Pressure with Sadness
as a Predictor and Black as a Moderator

	SBP	DBP
Fixed effects		
Intercept	135.63***	89.63***
	(4.73)	(3.44)
Time	-.003**	-.004***
	(.001)	(.001)
Day	1.48**	1.23**
	(.52)	(.42)
Age	.19*	.13*
	(.08)	(.06)
Gender	-8.33***	-5.50***
	(1.85)	(1.33)
Race	4.03*	4.13**
	(1.95)	(1.40)
Position	-3.34***	-3.72***
	(.23)	(.20)
Momentary sadness	.04^{+}	.04*
	(.02)	(.02)
Average sadness	.37*	.33**
	(.15)	(.11)
Trait depression	- .45	- .30
	(.35)	(.25)
Momentary sadness	-.05^{+}	-.04
X Race	(.03)	(.03)
Average sadness X	- .36^{+}	- .37**
Race	(.19)	(.14)
Trait depression X	1.28*	.85*
Race	(.51)	(.37)
Random effects		
UN (1,1)	101.99***	51.04***
	(14.14)	(7.34)
Residual	51.11***	38.92***
	(2.28)	(1.87)
Model statistics		
AIC	39390.6	37744.7
BIC	39402.5	37756.5
Pseudo R^2	.21	.21

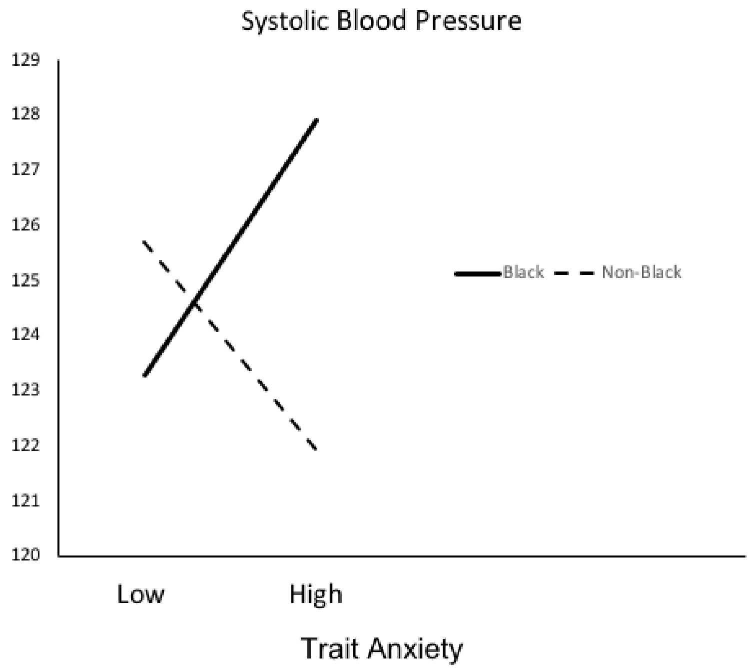
Note. ***p < .001; **p < .01; *p < .05; ^{+}p < .10. Time is the number of minutes that elapsed since midnight. Day is entered as a continuous variable. Gender is coded as 0 = male and 1 =

female. Race is coded as 0 = non-Black and 1 = Black. Momentary sadness, average sadness, and trait depression are grand-mean centered.

Figure 1

Panel A

Systolic Blood Pressure Estimates Grouped by Trait Anxiety and Race



Panel B

Diastolic Blood Pressure Estimates Grouped by Trait Anxiety and Race

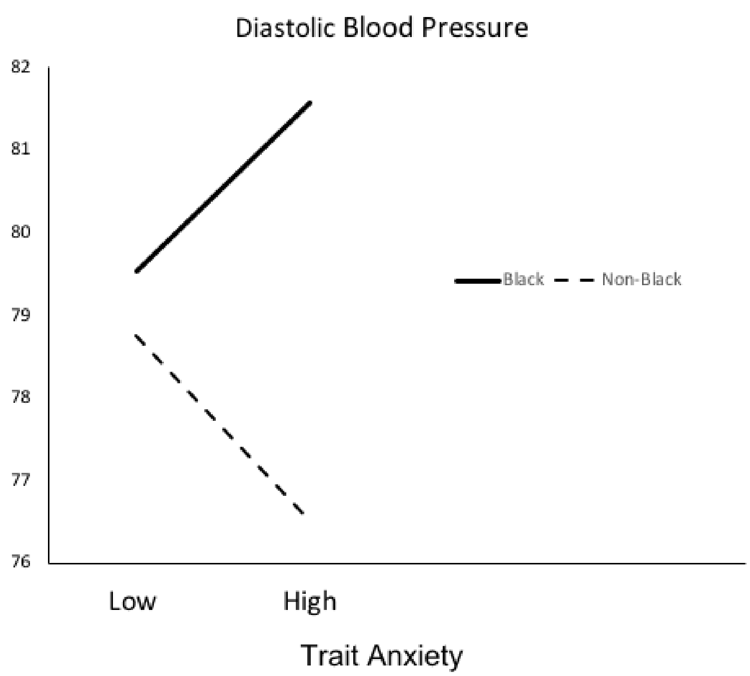
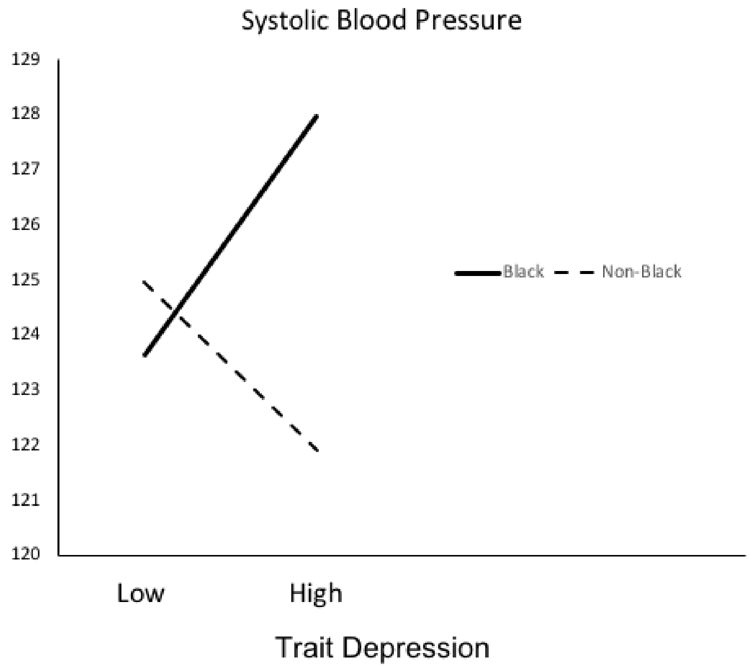


Figure 2

Panel A

Systolic Blood Pressure Estimates Grouped by Trait Depression and Race



Panel B

Diastolic Blood Pressure Estimates Grouped by Trait Depression and Race