Effects of Social Stressors on Cardiovascular Reactivity in Black and White Women

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ABSTRACT

Background: *Behavioral scientists have theorized that perceived racism in social interactions may account for some of the observed disparities in coronary heart disease between Black and White Americans.* ***Purpose:*** *The objective was to examine whether racial stress influences cardiovascular reactivity, a risk factor for cardiovascular disease.* ***Methods:*** *We measured cardiovascular responses in Black and White women (n = 80) as they talked about 3 hypothetical scenarios: (a) being accused of shoplifting (racial stressor), (b) experiencing airport delays (nonracial stressor), and (c) giving a campus tour (control).* ***Results:*** *Relative to White women, Black women had significantly greater mean diastolic blood pressure reactivity (3.81 vs. 0.25 mmHg; p < .05) in response to the racial stressor than in response to the nonracial stressor. Black women exhibited significantly lower heart rate during recovery following the racial stressor than during recovery following the nonracial stressor (–0.37 beats/min vs. 0.86 beats/min; p < .001). Among Black women, those who explicitly made race attributions during the racial stressor had greater systolic but not diastolic blood pressure reactivity than those who did not make racial attributions (8.32 mmHg vs. 2.17 mmHg; p < .05).* ***Conclusions:*** *These findings suggest that perceived racism in social interactions may contribute to increased physiological stress for Black women.*

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INTRODUCTION

Approximately 65 million Americans live with one or more forms of coronary heart disease (CHD) and cardiovascular disease (CVD), including heart attack, angina pectoris (chest pain), stroke, hypertension, congestive heart failure, rheumatic heart disease, and congenital heart disease (1). CVD is the leading cause of death among women and even exceeds the rates found in men (1). Like many other major health conditions, there are social and racial disparities in the prevalence, progression, and outcomes of CHD and CVD in the United States. Cardiovascular-related deaths are greater among ethnic minorities than among White Americans (1). Rates of hypertension are especially high among Black Americans (1), and the racial disparities in hypertension appear to exist independent of education level, income level, and other disease risk factors (2).

Behavioral scientists have suggested that social stress, particularly exposure to racism, may account for some of the excess risk for CHD/CVD in Black Americans (2–4). One prominent theory is that racial disparities in CVD can be explained by greater cardiovascular responses to stress in Blacks relative to Whites (5). However, Blacks do not appear to exhibit greater cardiovascular responses than Whites in all stressful situations (6). It also is not clear whether Blacks and Whites respond differently to specific kinds of social stress, such as racial stress (7). For example, Fang and Myers (2001) found that both Black and White men exhibited significant increases in diastolic blood pressure (DBP) in response to anger-provoking and racist film clips, and one group did not respond more negatively than the other group. Based on these findings, Fang and Myers suggested that intensity of blood pressure (BP) response to social stressors, including blatant racism, is not greater in Blacks than Whites. It is important to note, however, that Black and White participants were exposed to different stimuli in this study: Black participants watched films involving racial slurs directed toward Blacks, whereas White participants watched films of a White man being physically abused by Black men. Differences in the stimuli, which may include differences in the overall stressful-

ness and racist content, make it difficult to draw firm conclusions from the results.

In this study, we were interested in whether Black and White women might exhibit differential responses to some classes of social stressors because the stressors may have a different meaning to Blacks and Whites. Because of their history of mistreatment and discrimination, Black women may be more likely than White women to identify negative interpersonal interactions as motivated by discrimination or racial bias and may appraise such events as more stressful (8,9). Studies have shown that stigmatized people perceive more prejudice and discrimination than nonstigmatized people (8). Thus, Black women may perceive racism in some social interactions, whereas a White woman would not (e.g., receiving change from a store clerk who does not make eye contact). In this way, perceived racism in social interactions may create an excess burden of stress in Black women relative to other social groups and may contribute to exaggerated cardiovascular arousal and associated cardiovascular risk (4). People who experience discrimination also tend to become more vigilant for future mistreatment, which can lower their threshold for detecting and responding to challenges and increase physiological responses (10). From this perspective, chronic stressors may sensitize individuals to future stressors and exaggerated reactivity may be a by-product of chronic exposure to stressors (11).

Guyll and his colleagues (12) provided evidence of racial differences in response to a subtle racial stressor, using perceptions of prior experience with discrimination as a moderator variable. Middle-aged Black and White American women completed two stressful tasks: (a) mirror tracing and (b) a speech about being falsely accused of shoplifting. To the extent that the speech task triggered thoughts of racism and social injustice, Black participants were expected to respond more negatively to it than to mirror tracing. Contrary to expectations, Black women were equally reactive to both stressors, and White women had greater DBP reactivity to the speech than to mirror tracing. The investigators did find, however, that a higher level of perceived past discrimination was associated with greater increases in DBP reactivity during the speech among the Black participants. Thus, Black women who reported a history of being discriminated against had greater DBP reactivity to the speech than to mirror tracing, but there was no differential reactivity to the two stressors among the Black women who reported no history of discrimination.

Our study builds on previous literature that examines the effects of racial stress on cardiovascular reactivity among Black people (7,13). We examined BP and heart rate (HR) responses to analogues of two social stressors, one that was race neutral (experiencing an airport delay) and one that could create feelings of racial discrimination in Blacks (being falsely accused of shoplifting), in a sample of relatively young Black and White college women. This sample is appropriate for the study's central research questions, as theorists have suggested that younger Black women may be more likely than older Black women to perceive racism (14), and there is evidence that reactivity in children and young adults is associated with the development of hypertension later in life (15,16). We used a design in which all participants were exposed to identical stimuli to avoid confounds associated with using different stimuli for different groups. In addition, the study included a control speaking condition to adjust for the effects of speaking on reactivity (17–19), presented the speech tasks in counterbalanced order, and included measures of cardiovascular reactivity to the stressors as well as recovery following the stressors.

HYPOTHESES

This study investigated psychological and cardiovascular responses to laboratory analogues of two social stressors among Black and White women. For Black women, we predicted that cardiovascular reactivity would be higher, and recovery slower, in response to a social stressor that could be construed as racist than in response to a nonracial stressor. For White women, we predicted no difference in reactivity or recovery in response to the two different social stressors. Previous research has examined the relation between racial stressors and cardiovascular reactivity during a stressor, but there has been scant attention paid to the relation between racial stress and cardiovascular recovery after a stressor. Persistent activation following a stressor may be as important as the initial magnitude of activation during a stressor in the stress–disease linkage (11,20). Finally, on the basis of recommendations from a recent review of the literature on perceived racism and cardiovascular reactivity (13), we explored whether psychological distress in response to the stressors mediated racial differences in cardiovascular reactivity to the different social stressors. Both theoretical models and empirical evidence suggest that negative emotions may play a role in cardiovascular reactivity (19).

METHOD

Design Overview

We used a mixed, between- and within-participants quasi-experimental design. All participants gave three speeches in a laboratory setting. The control speech scenario (control), in which participants pretended to be giving someone a tour of the college campus, was designed to create little stress and allow us to separate the effects of speaking on BP and HR from the effects of stress. The other two speech scenarios involved discussing stressful social situations: one in which the participant imagines that she is one of many people who experience delays and assorted mishaps at an airport (nonracial stressor) and one in which the participant imagines that she is singled out of a crowd in an upscale store and accused of shoplifting (racial stressor). Participants were randomly assigned to one of six possible task orders to control for order effects. The between-participants factor was racial group (Black/White). The primary dependent variables were BP and HR reactivity (during the speeches) and recovery (after the speeches). Psychological distress was a secondary outcome. We conceptualized psychological distress as a potential mediator variable and a manipulation check.

Participants

The sample consisted of 40 Black and 40 White women, ranging in age from 16 through 41 (M = 23.11, SD = 5.77). All

participants completed informed consent procedures. Participants were either fulfilling a class requirement or were recruited through flyers posted around campus and paid $10 for their participation. Participants were eligible only if they had a negative history of hypertension or other BP problems and were not currently taking any medications that could alter BP. The majority of participants (87.5%) were nonsmokers; 37.5% (20 Blacks, 16 Whites) were born outside of the United States. We do not know the citizenship of foreign-born participants.

Materials and Measures

Cardiovascular reactivity and recovery. We used a Dinamap XL P81T automated BP machine to measure systolic blood pressure (SBP), DBP, and HR. Trained technicians periodically validated the Dinamap readings against a manually operated mercury sphygmomanometer. An occluding BP cuff was placed on the participant's nondominant arm. The experimenter operated the monitor in the control room adjacent to the laboratory. BP and HR were measured at baseline, during each speech task, and after each speech task. Reactivity scores were created by subtracting the mean BP or HR at baseline from the respective mean BP or HR during each of the three speech tasks (e.g., mean SBP during shopping speech – mean SBP during baseline). Recovery scores were calculated by subtracting the mean BP or HR at baseline from the respective mean BP or HR after each speech task (e.g., mean SBP after the racial stressor – mean SBP during baseline).

Psychological distress. Participants used a 7-point scale from 0 (*not at all*) to 6 (*extremely*) to rate their level of stress, anger, calm (reverse scored), and anxiety during each speech. We separately summed and averaged the ratings associate with each speech. Higher scores indicate greater distress. The 4-item scales had adequate reliability across speech tasks (coefficient alphas ranged from .66 to .78).

Body mass index. We measured participants' weight and height to calculate the body mass index (BMI; weight in kilograms / height in meters squared).

Procedure

Participants were brought into the laboratory individually, where they learned about the study and provided their consent to participate. Participants sat upright for the entire experimental session in a comfortable chair. After a 5-min relaxation period, participants completed the reactivity protocol, which included: a baseline period (10 min), Speech Task 1 (~4 min), Rest Period 1 (~4 min), Speech Task Two (~4 min), Rest Period 2 (~4 min), Speech Task Three (~4 min), and a final rest period (~4 min). Four BP and HR readings were taken during the baseline period, and three readings were taken during each of the subsequent periods. During the baseline and resting periods, participants watched a nature video to diminish boredom. At the end of the reactivity protocol, the experimenter administered a questionnaire containing the psychological distress scales and demographic variables.

Speech scenarios. For each of the three speech scenarios—(a) control, (b) racial stressor, and (c) nonracial stressor—participants read a description of a situation they were to talk about while the experimenter read it aloud. They were instructed to talk in front of a video camera about each scenario for 3 min without stopping. The experimenter was not in the room while the participant spoke. Participants were videotaped, and physiologic data were continually recorded. For each speech scenario, participants were asked to imagine themselves in the situation and to talk about the thoughts and feelings they were having in reaction to the situation. They were instructed to act is if the situations were real and to describe what they would tell a friend about the situations. Finally, participants in the stressor conditions were asked to discuss *why* the events might have happened to them.

In the racial stressor speech, participants described their reactions to being singled out of a crowd and accused of shoplifting in an upscale department store. There is no mention of race in the scenario presented to participants. This manipulation is subtly rather than blatantly racist. Blacks living in American society are more likely to experience subtle rather than blatant racism (21), and recent research suggests that subtle racism can lead to heightened cardiovascular reactivity (12,22). We expected that this stressor would evoke perceptions of racism among Black but not White participants, because it resembles the racial profiling that minorities often experience in America. In the nonracial stressor speech, participants were asked to describe their reactions to a flight delay at an airport that resulted in missing part of a vacation. Again, race was not mentioned, but we expected that this scenario would generate racial attributions among Black women. In the control speech, participants imagined that they were giving a friend a campus tour. This scenario was included to estimate and control for the effects of speaking on participants' BP and HR (18).

Speech content. The speeches made in response to the racial and nonracial stressors were transcribed and then coded by two independent raters to determine whether participants had attributed the stressor to their race. The race of the participant was masked before coding. Participants were primed to make attributions in the speech instructions, which specifically asked them to discuss why the stressor might have happened to them.

RESULTS

Data Analysis Plan

For manipulation checks, we examined group differences in psychological distress and attributions of racism using analysis of variance and chi-square techniques, respectively. For the inferential analyses on cardiovascular reactivity, we used analysis of covariance (ANCOVA) techniques to test whether, relative to White women, Black women had higher cardiovascular reactivity during the racial stressor than during the nonracial stressor. The analysis of the difference between reactivity scores (i.e., change from baseline to racial stressor minus change from baseline to nonracial stressor) after adjusting for baseline is an extension of the residualized change score approach to analyz-

ing reactivity data (23). For the inferential analyses on cardiovascular recovery after the speech tasks, we again used ANCOVA techniques to test whether, relative to White women, Black women had slower recovery (i.e., more persistent elevations) in the rest period following the racial stressor than in the rest period following the nonracial stressor. These ANCOVA analyses adjusted for baseline as well as level of reactivity, because rate of recovery is often dependent on degree of reactivity (i.e., higher reactivity generally is associated with longer recovery; 20). Additional covariates in the inferential analyses are described later. Finally, mediation analyses, which were aimed at testing the role of psychological distress in explaining racial differences in reactivity, were tested using a standard ANCOVA strategy (24; i.e., we tested whether psychological distress accounted for the association between race and reactivity or recovery to the different tasks).

Participant Characteristics

There were no statistically significant race differences in age, smoking status, or immigrant status. BMI was higher in Black participants ($M = 23.87$, $SE = 0.66$) than in White participants ($M = 22.12$, $SE = 0.54$), $t(77) = 2.03$, $p < .05$, and was used as a covariate in all analyses. One very overweight White participant (305 lb [138 kg]) was not included in the BMI analysis. When she was included in the analyses, there were no group differences ($p > .05$). To control for the effects of this outlier in subsequent analyses, we Windsorized (25) her data. BMI-adjusted baseline BP and HR data are displayed in Table 1 (column 1). BMI-adjusted baseline DBP was significantly greater among Black than White participants, $F(1, 77) = 6.03$, $p < .05$. BMI-adjusted baseline SBP was greater among Black than White participants, but the difference was statistically marginal, $F(1, 77) = 2.72$, $p < .10$. There was no statistically significant race differences in BMI-adjusted baseline HR, $F(1, 77) = 0.18$, *ns*.

Manipulation Check

As shown in Figure 1, participants were not highly distressed by the control speech, but they were moderately distressed during the other two speeches. A 2 (race) × 3 (scenario) analysis of variance of participants' ratings of psychological distress revealed a significant main effect of scenario, $F(2, 77) = 247.85$, $p < .001$, and a significant Scenario × Race interaction, $F(2, 77) = 5.68$, $p < .01$. Paired t tests revealed that both Black and White participants reported more distress during the racial and nonracial speech stressors than during the control speech (*p*s < .001). Black participants reported equivalent distress in response to the racial and nonracial stressors (*ns*), whereas White participants reported less distress during the racial stressor than during the nonracial stressor ($p < .01$). A similar pattern emerged when we examined the individual items comprising the distress scale.

Analysis of the speech content revealed that more Black participants (43.5%) than White participants (0%) mentioned race as a reason for being singled out for shoplifting, $\chi^2(1, N = 76) = 20.78$, $p < .001$. Neither Black nor White participants mentioned race when talking about the airport stressor.

BP and HR Responses

There were no scenario order effects on cardiovascular outcomes, so we collapsed across order for all subsequent analyses. Table 1 shows the means for the BP and HR during the baseline (column 1), reactivity (columns 2 and 3), and recovery (columns 4 and 5) phases, adjusted for covariates.

We examined whether Black and White participants exhibited differential reactivity to the racial stressor versus the nonracial stressor. The dependent measures in these analyses were the differences in participants' reactivity scores between the two stressors (e.g., column 3 – column 2 in Table 1), adjusting for

TABLE 1
Mean Blood Pressure and Heart Rate Reactivity and Recovery Following Speech Stressors

			Reactivity				*Recovery*			
	Baseline[a]		*Nonracial Stressor Reactivity*[b]		*Racial Stressor Reactivity*[b]		*Nonracial Stressor Recovery*[c]		*Racial Stressor Recovery*[c]	
Racial Group	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*
White[d]										
SBP (mmHg)	103.21	1.59	12.05	1.49	12.21	1.38	–0.12	0.61	0.14	0.57
DBP (mmHg)	61.79	1.08	8.74	1.03	8.99	0.94	–2.74	0.56	–2.26	0.55
HR (bpm)	74.91	1.86	9.97	1.12	10.71	0.92	–0.69	0.33	0.009	0.45
Black[d]										
SBP (mmHg)	106.95	1.59	11.54	1.49	16.10	1.38	–1.35	0.61	0.57	0.57
DBP (mmHg)	65.54	1.08	7.00	1.03	10.80	0.94	–3.39	0.56	–2.59	0.55
HR (bpm)	73.79	1.86	9.24	1.12	10.84	0.92	0.47	0.33	0.18	0.45

Note. $n = 80$. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; mmHg = millimeters of mercury; bpm = beats per minute; BMI = body mass index.

[a]Data reflect mean values adjusted for BMI. [b]Reactivity scores were derived by subtracting baseline BP or HR from corresponding task BP or HR. Data reflect mean values adjusted for BMI, corresponding baseline BP or HR, and corresponding tour reactivity BP or HR. [c]Recovery scores were derived by subtracting baseline from the corresponding postspeech BP or HR. Data reflect mean values adjusted for BMI, corresponding baseline BP or HR, corresponding tour recovery BP or HR, and corresponding stressor BP or HR reactivity scores. [d]$n = 40$.

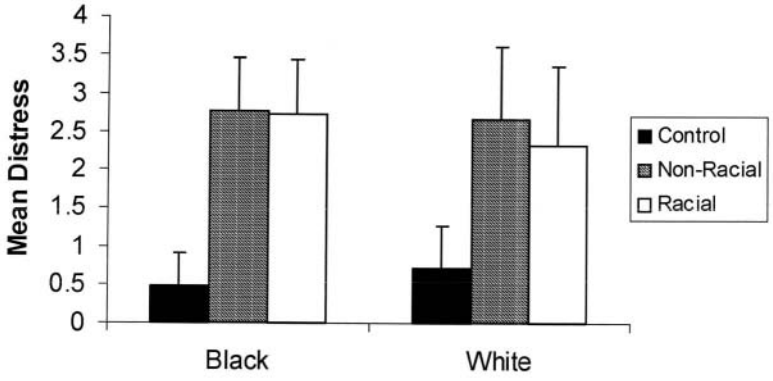

FIGURE 1 Mean level of psychological distress as a function of race and speech condition (n = 80).

covariates. Higher difference scores indicate greater reactivity to the racial stressor than to the nonracial stressor. Separate analyses were conducted for SBP, DBP, and HR. Covariates included BMI and the corresponding SBP, DBP, or HR baseline and reactivity scores during the control speech. Covarying the control speech reactivity scores adjusts for the effects of speaking on arousal (18). Relative to Whites, Blacks exhibited greater DBP reactivity to the racial stressor than to the nonracial stressor (mean DBP reactivity difference score = 3.81, *SE*= 1.15, in Black women vs. 0.25, *SE* = 1.15, in White women), $F(1, 75) = 4.49$, $p < .05$. Black participants also exhibited greater SBP reactivity to the racial stressor than White participants, but the difference was only marginally significant (mean DBP reactivity difference score = 4.56, *SE*= 1.71, in Black women vs. 0.17, *SE* = 1.71, in White women), $F(1, 75) = 3.13$, $p < .10$. There were no race differences in HR reactivity measures

To assess race differences in cardiovascular recovery, we examined whether Black and White participants exhibited differential levels of SBP, DBP, and HR recovery across the two stressors. The dependent measures in these analyses were the differences in participants' recovery scores (i.e., column 5 – column 4 in Table 1), adjusting for covariates. Higher scores suggest greater arousal during the recovery phase following the racial stressor than during the recovery phase following the nonracial stressor. Separate analyses were conducted for SBP, DBP, and HR. Covariates included BMI; the respective baseline SBP, DBP, or HR score; and recovery scores during the control condition (to adjust for the general effects of recovery from speaking on physiological responses). We also covaried participants' respective SBP, DBP, or HR reactivity difference scores (e.g., racial stressor SBP reactivity – nonracial stressor SBP reactivity). Relative to White participants, Black participants exhibited greater SBP in the recovery phase following the racial stressor than in the recovery phase following the nonracial stressor (mean SBP recovery difference score = 1.92, *SE*= 0.60, in Black women vs. 0.26, *SE* = 0.60, in White women), but the difference was marginally significant, $F(1, 74) = 3.59$, $p < .06$. There were no race differences in DBP recovery. Relative to Whites, Blacks exhibited significantly lower HR during the recovery phase following the racial stressor than during the recovery phase following the nonracial stressor (mean HR recovery difference score = –0.37, *SE* = 0.44, in Black women vs. 0.86, *SE* = 0.44, in White women), $F(1, 74) = 11.43$, $p < .001$.

Mediation

We aimed to test whether psychological distress mediated the effects of race on cardiovascular reactivity, but the data did not warrant such an analysis. One of the conditions of mediation is that the predictor is related to the mediator. Because Black women did not exhibit different degrees of distress in response to the racial and nonracial stressors, distress could not explain the racial differences in cardiovascular responses to the two stressors.

We conducted some additional within-race analyses to examine the potential role of perceived racism in the BP reactivity and recovery of Black women in response to the racial versus the nonracial stressor. Attribution to race (yes/no) during the shopping (racial stressor) speech was the independent variable. The analyses examined the difference in the BP reactivity or recovery scores (racial – nonracial reactivity or recovery scores) in Black women who did (n = 17) versus did not (n = 22) make a racial attribution during the speech about the shopping scenario. For the reactivity analyses, we used ANCOVA techniques, controlling for BMI, the relevant baseline BP scores, and the relevant BP reactivity scores from the control scenario. We found a statistically significant difference in SBP reactivity (nonattribution group adjusted mean = 2.17, *SE* = 1.76; attribution group adjusted mean = 8.32, *SE* = 2.00), $F(1, 34) = 5.28$, $p < .05$, but not in DBP. For the recovery analyses, we also used ANCOVA techniques and controlled for BMI, the relevant baseline BP scores, the relevant BP recovery scores from the control scenario, and the relevant BP reactivity score (because reactivity influences recovery). We found no significant effects of racial attributions on recovery.

DISCUSSION

Our findings support the notion that social situations that could be construed as racist by Black women can result in greater and more prolonged physiological stress responses

among Black women than social situations in which race is not an element. Consistent with our expectations, the shoplifting scenario appeared to evoke perceptions of racism only among Black women, and neither Black nor White women appeared to perceive racism in the airport scenario. Although many Black women did not make a verbal attribution to racism when discussing the shoplifting accusation, it is possible that they still perceived it as such. Compared with White women, Black women had significantly higher DBP responses to the racial stressor than to the nonracial stressor. Additional comparisons revealed that, relative to White women, Black women tended to have higher SBP during the recovery phase of the racial stressor than during the recovery phase of the nonracial stressor, but the effect was statistically marginal. These findings suggest that the perceived racism evoked by certain social situations could put Black women at heightened risk for intense and sustained elevations in BP in their daily lives. Over time, the repeated and sustained arousal associated with perceived racism in everyday social situations could contribute to increased risk for CVD and CHD among Black women.

These findings are relevant to the question of whether racial differences in cardiovascular reactivity can contribute to racial differences in CVD and CHD. Consistent with previous research (6), we did not find evidence that Blacks are generally more reactive to stressors than Whites, yet we did find that, relative to White women, Black women tended to get more aroused to a stressor that could be construed as racist than to a nonracial stressor. Previous studies (26–28) also have shown that racist stimuli can generate greater sympathetic arousal than nonracist stimuli among Black responders. However, because these previous studies lacked a comparison group of White participants, one cannot conclude from them that the differential reactivity to diverse social stressors is unique to Black responders. Two studies (12,29) that did compare Black and White responses to racial stressors failed to find racial differences in BP reactivity. In one study, the results are difficult to interpret, because different stimuli were used for Black and White participants (29). Surprisingly, in the other study (12), in which Black and White women were exposed to identical stressors, the White women were more reactive than Black women when exposed to a stressor involving interpersonal mistreatment, which is a common form of racial stress. Thus, our findings may be among the first to show that Black women are more likely than White women to perceive racism in some social situations and experience greater and longer lasting arousal in response to those situations. These findings provide additional evidence that perceived racism in social situations may play a role in racial disparities in cardiovascular health.

The effects of the social stressors on psychological distress only partially confirmed the predictions. Overall, the two social stressors were perceived as more distressing than the control speech. There also were racial differences in psychological distress responses to the social stressors, but not in the same direction as the cardiovascular differences. Among White women, the racial stressor appeared to be less distressing than the nonracial stressor, but among Black women the stressors appeared to be equally distressing. Thus, psychological distress reactions assessed in this study could not account for the observed cardiovascular outcomes in Black women.

The apparent disconnect between emotional and physiological responses to laboratory stressors in our study is not unprecedented (18,19,30). In a meta-analysis of nine studies, Feldman and colleagues (19) found that participants exposed to laboratory stressors (e.g., speech, mirror tracing) responded with both increased cardiovascular response and increased negative emotion, and the increases in negative emotion were associated with increases in cardiovascular response across tasks. However, the associations were quite small, with negative emotions accounting for only 2% to 12% of the variance in cardiovascular response. The nature of the laboratory stress paradigm may contribute to the lack of a strong association between emotional and physiological responses. For example, self-report measures of emotional response may not be sensitive to emotional changes that occur during the stressor, because the emotion measures are administered at the end of the laboratory session (19,31).

It is possible that cognitive variables, rather than level of negative affect, may be important mediators in explaining differential reactivity. For example, a racial stressor could lead to negative thought perseveration (worry, rumination), which in turn could sustain arousal after the stressor terminates (32). Cognitive appraisals also might play a role. Cognitive appraisals reflect one's assessment of a situation (e.g., perceived threat) and feelings of control and ability to cope rather than just the emotional response associated with a particular scenario (31). To Black women, the shopping scenario may have been perceived as threatening, thus requiring increased vigilance and arousal (for fight or flight). To White women, the shopping scenario might have been perceived as a misunderstanding that eventually would be cleared up—leading, perhaps, to embarrassment or indignation rather than alarm. Additional research is needed to tease out the specific cognitive and emotional dynamics that link racial stressors to cardiovascular functioning.

The lack of significant differences in HR reactivity between Black and White women is consistent with the literature on racial differences in hemodynamic responses to stressors (33). Although not specifically addressed by this study, there is evidence that Black individuals typically respond to stressful situations with a vascular pattern of reactivity that is characterized by changes in BP and vascular resistance (33). White individuals, on the other hand, typically exhibit a cardiac pattern in response to stress that is characterized by an increase in HR and cardiac output. As expected, Black women exhibited greater BP reactivity to the racial stressor than to the nonracial stressor, but HR changes did not vary by stressor. Furthermore, relative to White women, Black women exhibited a trend toward higher SBP during the recovery phase following the racial stressor than during the recovery phase following the nonracial stressor. Finally, relative to White women, Black women exhibited lower HR during the recovery phase of the racial stressor than during the recovery phase of the nonracial stressor.

These conclusions are bolstered by several strengths of the experimental design. First, all study participants were exposed to identical stressors, to avoid any confounds associated with using different stimuli for different groups. Second, the study included a control speaking condition to adjust for the effects of speaking on reactivity, and this score was used as a covariate in statistical analyses. Finally, the inclusion of recovery measures expands research in this area by illustrating how racial stressors can produce sustained arousal even after the stressor has terminated.

Despite the strengths of the study, a number of limitations and recommendations for future research warrant consideration. The laboratory-based scenarios for examining reactions to subtle racial discrimination may be a weak analogue to what happens in the real world. Studies including *in vivo* assessments, such as experiential time sampling and ambulatory BP monitoring, have the potential to reveal cardiovascular responses to perceived racism in real social situations. The lack of data on male participants and other ethnic or racial groups limits the generalizability of our findings. Another limitation is the dichotomous grouping of participants as Black or White. The sample of Black women included women of both African and West Indian descent, and some Black and White women were born outside of the United States. Immigrants, particularly those who have recently arrived, may have different expectations for and experiences of discrimination, which could affect reactivity to racial stressors. Because of the small sample size, we were unable to conduct subgroup analyses, but we encourage this for future research.

We recommend that in future research the search for underlying mediators be expanded. Our study focused on a single variable, psychological distress, which proved not to mediate the relationship between race and CVR. Other processes that should be considered in future research include person-level variables, such as sensitivity to racial stress (9), cognitive appraisals, and individual coping strategies, as well as prior history of discrimination (4). Although the evidence is inconclusive that prior experience with discrimination either magnifies reactions to any single event (i.e., a cumulative stress theory), or mitigates reactivity through the use of coping responses such as repression of anger, these are important pathways to explore in future research.

This study adds to the growing literature on cardiovascular reactivity to stress as a contributing factor to existing racial disparities in CVD. The results indicate that Black, but not White, women were likely to perceive racism in a scenario in which they imagined themselves being singled out and accused of shoplifting. Relative to White women, Black women exhibited significantly greater DBP reactivity to this subtle racial stressor than to a stressor with no racial elements. A similar trend was observed for SBP. Relative to White women, Black women also exhibited a trend toward higher SBP during the recovery phase of the racial stressor than during the recovery phase of the nonracial stressor. These findings suggest that perceived racism in interpersonal interactions may contribute to increased physiological stress and associated health risks for Black women.

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