



Vagal control moderates the association between endothelial function and PTSD symptoms in women with T2DM

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ABSTRACT

Background: Individuals with posttraumatic stress disorder (PTSD) are more likely to present with metabolic diseases such as type-2 diabetes mellitus (T2DM), and cardiovascular dysfunction has been implicated in this link. These diseases disproportionately affect women and individuals exposed to chronic environmental stressors (e.g., community violence, poverty). We examined associations among PTSD, cardiovascular indices, and metabolic function in highly trauma-exposed Black women with T2DM.

Methods: Participants ($N = 80$) were recruited for a follow-up study of stress and T2DM as part of the Grady Trauma Project. PTSD symptoms were assessed with the Clinician Administered PTSD Scale (CAPS-IV). Cardiovascular indices included heart rate (HR), blood pressure (BP), respiratory sinus arrhythmia (RSA), and endothelial function (assessed via flow-mediated dilation; FMD). An oral glucose tolerance test was used as an indicator of metabolic function.

Results: Of the cardiovascular indices, only FMD was significantly associated with PTSD symptoms (CAPS Avoidance symptoms; $\beta = -0.37, p = .042$), and glucose tolerance ($\beta = -0.44, p = .019$), controlling for age and body mass index. The association between FMD and PTSD Avoidance was moderated by RSA such that the effect of FMD was only significant at low levels of RSA (simple slopes $\beta = -0.87, p = .004$).

Conclusions: Our results indicate that endothelial function is significantly related to PTSD and glucose tolerance, over and above other cardiovascular measures (HR, BP, RSA). Further, our results suggest that low RSA may be a risk factor for the link between poor endothelial function and PTSD in women with T2DM.

1. Introduction

Posttraumatic stress disorder (PTSD) is a heterogeneous psychiatric disorder characterized by chronic re-experiencing symptoms (e.g., intrusive memories), avoidance of trauma reminders, negative mood, and hyperarousal (e.g., hypervigilance) symptoms. Individuals with PTSD also experience chronically elevated sympathetic arousal and

decreased parasympathetic control, as well as increased systemic inflammation due to the dysregulation of the hypothalamic-pituitary (HPA) axis. In addition to impairing the fear response, these physiological dysfunctions may also mediate the high comorbidity of cardiovascular and metabolic diseases, particularly type 2 diabetes mellitus (T2DM), reported among PTSD populations (for reviews, see [Koenen et al., 2017](#) and [O'Donnell et al., 2021](#)). Increased trauma exposure

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among Black women has been linked with their higher risk of both PTSD and T2DM (Dixon et al., 2020; Gillespie et al., 2009; Stojek et al., 2019). Although various causal models have been proposed (Koenen et al., 2017), it remains unclear how cardiovascular function may be associated with PTSD symptoms and metabolic function among this population.

Autonomic and HPA-axis dysfunctions represent hallmark features of PTSD. Indicative of an over-active fear response, individuals with PTSD demonstrate signs of exaggerated sympathetic nervous system activity, such as elevated heart rate (HR) and increased blood pressure (BP), both at rest and when confronted by stress or threat (e.g., Bedi and Arora, 2007; Brudey et al., 2015). Individuals with PTSD also exhibit impairments in other domains of cardiovascular function that may represent downstream or upstream effects of sympathetic arousal, such as baroreflex sensitivity (Park et al., 2017) and impaired endothelial function (Clausen et al., 2016; Sumner et al., 2017, 2018). Further, individuals with PTSD have a dampened ability to regulate this fear response, as indicated by low respiratory sinus arrhythmia (RSA), a common measure of parasympathetic control (e.g., Hauschildt et al., 2011; Jovanovic et al., 2009; Minassian et al., 2014, 2015). Increased sympathetic arousal and reduced parasympathetic control, coupled with heightened HPA axis activation and inflammation, are potential mechanisms through which PTSD leads to increased risk for cardiometabolic disease. One of the most common co-occurring metabolic diseases in PTSD is T2DM.

PTSD disproportionately impacts women (Kilpatrick et al., 2013), particularly Black women exposed to racial discrimination (Mekawi et al., 2021) and trauma-prone, low socioeconomic environments (Gillespie et al., 2009). These inequities also place Black individuals at greater risk for T2DM (Dixon et al., 2020; Rodríguez and Campbell, 2017; Stojek et al., 2019), a disease that is notably twice as common among individuals with PTSD than those of the general US population (Roberts et al., 2015). Given that high BP is a common co-occurrence of T2DM and individuals with T2DM demonstrate low RSA, cardiovascular and autonomic dysfunction have been implicated in the development of T2DM (Benichou et al., 2018; Kim et al., 2015). However, little is known about associations among cardiovascular and metabolic indices in trauma-exposed Black women with T2DM, a high-risk, underserved population. This knowledge may inform detection of individuals susceptible to CVD and future treatment approaches.

The current study tested associations among cardiovascular indices, PTSD symptoms, and metabolic function among Black women with trauma exposure and T2DM. Given that subthreshold PTSD symptoms are associated with significant functional impairment, altered autonomic functioning, and similar treatment response to threshold PTSD, we included individuals with a range of PTSD symptoms and not only those who met diagnostic criteria (Costanzo et al., 2016; Dickstein et al., 2013; Hunnicutt-Ferguson et al., 2018; for a review, see Brancu et al., 2016). Cardiovascular indices included HR, BP, RSA, and endothelial function (assessed as flow-mediated dilation [FMD] of the brachial artery). Metabolic function was assessed with an oral glucose tolerance test. Based on prior research implicating low RSA in PTSD and T2DM, we hypothesized that RSA would moderate the association between cardiovascular dysfunction and PTSD symptoms.

2. Methods

2.1. Participants and procedure

Women aged 18–65 years ($N = 80$) with T2DM were recruited from primary care, diabetes, and gynecology clinics at Grady Memorial Hospital as part of the Grady Trauma Project (Gillespie et al., 2009). All women identified “Black” as their race. To be eligible, individuals needed to have a T2DM diagnosis and current T2DM medication treatment in their medical record, report trauma exposure (described in Measures), speak and comprehend English, and have a working phone

number. Exclusion criteria were current bipolar or psychotic disorder diagnosis, alcohol or substance dependence, treatment for an autoimmune disorder, treatment with nonsteroidal anti-inflammatory, glucocorticoid, or anticonvulsant medications, and current treatment with antipsychotic, benzodiazepine, or antidepressant medications. After obtaining informed consent, participants completed a battery of self-report measures and a brief interview that assessed physical and mental health. Participants with T2DM were invited back to the clinic to participate in a sub-study where metabolic, neuroendocrine, and cardiovascular function were assessed. All study procedures were approved by the Grady Hospital Research Oversight Committee and the Emory University Institutional Review Board.

2.2. Measures

Traumatic Event Inventory (TEI; Schwartz, Bradley, Sexton, Sherry, & Ressler, 2005). The TEI is a 14-item semi-structured screening instrument that was used to assess exposure to and witnessing of traumatic events, such as sexual assault, military combat, life-threatening illness, and natural disasters.

Clinician-Administered PTSD Scale for DSM-IV (CAPS-IV; Blake et al., 1995). The CAPS-IV is a structured clinical interview designed to assess symptomology and severity of PTSD symptoms, as well as determine a binary classification of current/lifetime diagnosis. For each of the 17 diagnostic criteria, the CAPS-IV rates frequency on a scale from 0 = “none of the time” to 4 = “most or all of the time” and rates intensity on a scale from 0 = “none” to 4 = “extreme.” Symptoms were based on the participants’ self-selected “worst trauma.”

2.3. Cardiovascular indices

BP and HR were obtained after 5 min of rest. Systolic and diastolic BP were measured with an automated SphygmoCor device using the oscillometric method (AtCor Medical Pty Ltd, Sidney, Australia). BP was calculated by averaging three repeated measurements separated by 5-min intervals. As reported previously (Powers et al., 2021), HR was measured for 5 min with three Ag/AgCl electrodes using Biopac MP150 for Windows and the BioNomadix wireless ECG amplifier (Biopac Systems, Inc, Goleta, CA). ECG data were sampled at 1000 Hz and analyzed with the heart rate variability (HRV) module of MindWare software (MindWare Technologies, Ltd, Gahanna, OH). The ECG signal was amplified by a gain of 2000, filtered with a Hamming windowing function, and with a 60-Hz notch filter. High frequency HRV (RSA) was sampled from 0.12 to 0.40 Hz and was transformed by natural log during five 1-min intervals by spectral analysis of the time-sampled inter-beat interval series, according to methods recommended by the Society for Psychophysiological Research Committee on HRV (Berntson et al., 1997). To control for circadian rhythm effects, all participants began ECG data acquisition around 11am.

Flow-mediated dilation (FMD) is a non-invasive measurement of endothelium-dependent brachial artery dilation (Corretti et al., 2002). Ultrasound images of the brachial artery were obtained at baseline under standardized conditions and 60 s after induction of reactive hyperemia by 5-min cuff occlusion of the forearm. Image landmarks as well as surface markers were utilized to ensure anatomic consistency between serial imaging studies. All images were digitized online, and arterial diameters were measured with customized software (Medical Imaging Applications, Coralville, IA) by individuals blinded to the clinical and laboratory status of the subjects. Flow-mediated dilatation (FMD) was expressed as the percentage increase in diameter from baseline. Artery dilation is sensitive to room temperature, food and beverage consumption, and body position; thus, participants were asked to fast before the FMD procedure and it was conducted in a temperature-controlled room while they were in the supine position.

2.4. Glucose tolerance

Our measure of metabolic function was glucose tolerance as measured using a 2-h oral glucose challenge test. This test measures the insulinogenic response to glucose by monitoring insulin production emitted by pancreatic β -cells (Matsuda and DeFronzo, 1999). During the 2-h challenge, participants quickly consumed a viscous 75-g glucose solution and blood samples were collected in 15-min intervals via an IV catheter, for a total of six measurements. All blood samples were collected in EDTA tubes, placed immediately on ice, and centrifuged at 4 °C for 10 min at 3,000 rpm. Plasma was aliquoted and stored at -80 °C until assayed for glucose on a Beckman AU480 using reagents from Beckman Coulter (Fullerton, CA) and insulin by immunoturbidometric methods on the Beckman AU480 using reagents from Sekisui Diagnostics (Exton, PA). Area under the curve ground (AUC_g) and area under the curve with respect to increase (AUC_i) were calculated to quantify the total secretion of plasma insulin over the 2-h OGTT, and to assess glucose tolerance and insulin sensitivity (Pruessner et al., 2003).

2.5. Data analysis

Outliers were defined as individuals with scores ≥ 3 standard deviations above the mean for each variable. One outlier was identified for RSA, and one for Glucose AUC_g; these cases were removed from analyses. To minimize the chance of Type-II error and avoid multiple testing, bivariate correlations were first used to determine which PTSD symptom clusters were associated with the cardiovascular indices. Given that CAPS-5 Avoidance symptoms were significantly associated with both HR and BP, we chose this as our dependent variable for PTSD symptoms. The first linear regression was conducted with CAPS-5 Avoidance as the dependent variable and HR, BP, RSA, and FMD as the predictor variables. Age and body mass index (BMI) were included as covariates. Our dependent variable for glucose tolerance was Glucose AUC_g given that it was significantly associated with FMD, whereas Glucose AUC_i was not significantly associated with any of the cardiovascular indices. Thus, our second regression model was conducted with Glucose AUC_g as the dependent variable and HR, BP, RSA, and FMD as the predictor variables. Age and BMI were included as covariates. All variables were continuous and all analyses were conducted using SPSS v.24 with a significance level of $p < .05$.

3. Results

Participant characteristics are presented in Table 1. See Tables 2 and 3 for descriptive and bivariate correlations among study variables. Thirty three participants met criteria for a PTSD diagnosis; given a lack of statistical power, we did not use categorical PTSD diagnosis as a grouping variable in our analyses. The first regression model (Table 4) was significant and accounted for 69% of the variance in CAPS-5 Avoidance, $R^2 = 0.69$, $p = .002$. FMD was the only cardiovascular variable significantly associated with CAPS-5 Avoidance, controlling for age and BMI, $\beta = -0.37$, $p = .042$. The second regression model was not significant and accounted for only 28% of the variance in Glucose AUC_g, $R^2 = 0.28$, $p = .083$ (Table 4). However, FMD was the only cardiovascular variable significantly associated with glucose AUC_g, controlling for age and BMI, $\beta = -0.44$, $p = .019$. These findings suggest that endothelial function as indicated by FMD may be a salient cardiovascular biomarker of PTSD symptoms and T2DM.

To test our hypothesis that RSA would moderate these findings, we conducted additional regression analyses to determine if the effects of FMD on CAPS-5 Avoidance or Glucose AUC_g differed at high versus low levels of RSA. After mean centering RSA and FMD, the RSA \times FMD interaction term was created and entered into each of our two regression models. The interaction was not significant in the model predicting glucose AUC_g. However, it was significant in the model predicting CAPS-5 Avoidance, $\beta = 0.53$, $p = .035$. We then conducted simple slopes

Table 1
Sample Characteristics.

	N	%		
Sex				
Female	80	100%		
Race				
Black/African American	80	100%		
Ethnicity				
Non-Hispanic/Non-Latinx	80	100%		
Highest Level of Education				
Less than 12th Grade	15	18.8%		
12th or High School Graduate	23	28.7%		
GED	4	5.0%		
Some College or Technical School	22	27.5%		
Technical School Graduate	4	5.0%		
College Graduate	10	12.5%		
Graduate School	2	2.5%		
Employment Status				
Currently Employed	23	28.7%		
Not Currently Employed	57	71.3%		
Household Monthly Income				
\$0 - \$249	13	16.3%		
\$250 - \$499	5	6.3%		
\$500 - \$999	19	23.8%		
\$1,000 - \$1,999	27	33.8%		
\$2,000 or more	14	17.5%		
Missing	2	2.5%		
	N	Mean	SD	Range
Age	80	50.6	9.9	21–65
Body Mass Index	80	35.57	8.05	18.9–61.0
LDL Cholesterol	77	97.59	40.10	27.80–246.00
HDL Cholesterol	77	49.20	20.05	26.70–153.00

Note. LDL = low-dose lipoprotein; HDL = high-dose lipoprotein; According to the CDC, desirable cholesterol levels are <100 mg/dL for LDL and ≥ 60 for HDL; According to the CDC, a healthy BMI falls between 18.5 and 24.9.

Table 2
Descriptive Statistics.

	Mean	SD	Min	Max
RSA ^a	4.97	1.54	2	9.79
Heart rate	75.97	11.02	52	106.5
BP – Systolic	130.5	18.69	93	176.5
BP – Diastolic	72.1	10.69	53.5	98
FMD	3.44	0.43	2.51	4.6
Glucose AUC _g ^a	33079.5	11013	13616.3	67545.8
Glucose AUC _i	13520.4	5142.3	2000.25	25923.8
CAPS-5 Total	16.11	12.53	0	50
CAPS-5 Intrusions	3.38	3.85	0	15
CAPS-5 Avoidance	1.91	1.86	0	6
CAPS-5 NACM	5.59	5.11	0	18
CAPS-5 Arousal	5.31	3.91	0	15

Note.

SD = standard deviation; RSA = respiratory sinus arrhythmia; BP = blood pressure; FMD = flow mediated dilation; AUC_g = area under the curve with respect to ground; AUC_i = area under the curve with respect to increase; CAPS-5 = Clinician Administered PTSD Scale for DSM-5; NACM = negative alterations in cognition and mood.

^a Excludes outliers.

analyses to determine the nature of the interaction effect. In simple slopes analyses, high and low values of the moderator variable (RSA) are calculated by adding and subtracting one standard deviation from the mean for each value, and the effect of the independent variable (FMD) on the dependent variable (CAPS-5 Avoidance) is determined at high and low values of the moderator (Aiken and West, 1991). FMD significantly predicted CAPS-5 Avoidance at the level of low but not high RSA, $\beta = -0.87$, $p = .004$ (see Table 5), providing support for moderation. This finding suggests that low RSA may be a risk factor contributing to the association between poor endothelial function and PTSD avoidance symptoms in women with T2DM.

Table 3
Bivariate Correlations among Study Variables.

	1	2	3	4	5	6	7	8	9	10	11	12
1. RSA ^a	–											
2. Heart rate	–.013	–										
3. BP – Systolic	–.040	.161	–									
4. BP – Diastolic	.135	.338**	.705**	–								
5. FMD	–.391**	.038	.248*	.226	–							
6. Glucose AUCg ^a	.202	.009	.166	.174	–.422**	–						
7. Glucose AUCi	.341*	–.097	.138	.180	–.194	.675**	–					
8. CAPS-5 Total	.015	.207	.035	.102	.007	.183	.024	–				
9. CAPS-5 Intrusions	.011	.237	.142	.273*	–.077	.341*	.157	.907**	–			
10. CAPS-5 Avoidance	.022	.322*	.134	.290*	–.104	.296*	.144	.810**	.788*	–		
11. CAPS-5 NACM	.007	.184	.049	.182	–.134	.158	.008	.922**	.758**	.657**	–	
12. CAPS-5 Arousal	.070	.182	.011	.178	.050	.140	–.028	.864**	.677**	.608**	.733**	–

Note.
p* < .05; *p* < .01; RSA = respiratory sinus arrhythmia; BP = blood pressure; FMD = flow mediated dilation; AUCg = area under the curve with respect to ground; AUCi = area under the curve with respect to increase; CAPS-5 = Clinician Administered PTSD Scale for DSM-5; NACM = negative alterations in cognition and mood.
^a Excludes outliers.

Table 4
Primary regression models.

Dependent variable: CAPS-5 Avoidance	β	t	p
RSA ^a	–.348	–2.023	.057
Heart rate	.191	1.018	.321
BP – Systolic	–.098	–.399	.694
BP – Diastolic	.602	2.002	.060
FMD	–.375	–2.230	.038*
Age	–.442	–2.905	.009*
BMI	.196	1.199	.245
<i>R</i> ² = .684			
Dependent variable: Glucose AUCg ^a	β	t	p
RSA ^a	–.073	–.433	.667
Heart rate	–.215	–1.241	.223
BP – Systolic	.027	.120	.905
BP – Diastolic	.344	1.352	.185
FMD	–.436	–2.461	.019*
Age	–.098	–.567	.574
BMI	–.106	–.661	.513
<i>R</i> ² = .279			

Note.
**p* < .05; RSA = respiratory sinus arrhythmia; BP = blood pressure; FMD = flow mediated dilation; AUCg = area under the curve with respect to ground; CAPS-5 = Clinician Administered PTSD Scale for DSM-5.
^a Excludes outliers.

Table 5
RSA moderation and simple slopes.

Dependent variable: CAPS-5 Avoidance	β	t	p
RSA ^a	.058	.244	.810
Heart rate	.208	1.227	.236
BP – Systolic	–.193	–.858	.402
BP – Diastolic	.723	2.608	.018*
FMD	–.426	–2.769	.013*
RSA × FMD interaction	.527	2.287	.035*
Age	–.378	–2.690	.015*
BMI	.231	1.553	.138
<i>R</i> ² = .755			
Simple slopes			
FMD			
Low RSA	–.870	–3.289	.004*
High RSA	.018	.079	.938

Note.
**p* < .05; RSA = respiratory sinus arrhythmia; BP = blood pressure; FMD = flow mediated dilation; CAPS-5 = Clinician Administered PTSD Scale for DSM-5.
^a Excludes outliers.

4. Discussion

The present study investigated which cardiovascular variables were biomarkers of PTSD symptoms and metabolic function in a sample of Black women with trauma exposure and T2DM. Among our sample, endothelial function was the cardiovascular index most associated with both PTSD avoidance symptoms and glucose tolerance, and this relationship was moderated by RSA. This suggests that low FMD may be an early indicator of cardiovascular dysfunction in trauma-exposed Black women with T2DM. Our findings are also consistent with previous reports implicating low RSA as a risk factor for PTSD symptoms, as opposed to high RSA being a protective factor (Kamkwala et al., 2012).

It is well-established that PTSD is associated with poor health outcomes such as T2DM (Boscarino, 2004; Roberts et al., 2015) and cardiovascular dysfunction. While previous studies have demonstrated associations between PTSD diagnosis and cardiovascular indices such as HR and BP (e.g., Bedi and Arora, 2007; Brudey et al., 2015; O'Donnell et al., 2021), we found support specifically for the association between HR, BP, and FMD with CAPS-5 Avoidance symptoms. Avoidance in PTSD refers to the avoidance of internal stimuli (e.g., trying not to think about trauma memories) and the avoidance of external reminders (e.g., not visiting a particular town where trauma occurred). In the emotion regulation literature, avoidance of internal stimuli is conceptualized as experiential avoidance, which is a strategy used to decrease and/or dampen unwanted emotional experiences (Hayes et al., 1996). Given that avoidance requires an individual to “push down” their emotional state (e.g., suppressing anger during a meeting), suppressive emotion regulation strategies place temporary strain on autonomic functioning, and they are associated with heightened sympathetic arousal (Gross and Levenson, 1993). Thus, it is possible that the avoidance cluster of PTSD emerged as significantly related to cardiovascular indices because avoidance increases sympathetic arousal. Further, the associations that we observed between avoidance symptoms and cardiovascular indices may be driven by a combination of changes in cardiovascular physiology directly impacted by hyperarousal and functional impairment that indirectly affects cardiovascular physiology, thus explaining the lack of association between CAPS-5 Arousal and cardiovascular indices. High levels of avoidance may represent adaptations that attempt to mitigate chronically elevated arousal, and worsening of functional impairment in turn may drive adverse lifestyle changes that worsen metabolic and vascular physiology, further amplifying those effects driven by hyperarousal alone.

It is noteworthy that FMD was the cardiovascular index most associated with PTSD avoidance symptoms and glucose tolerance in our sample of women with T2DM. Endothelial function (assessed via FMD) has been less studied in PTSD and T2DM but the evidence for its importance is growing. For example, studies in male Veterans have

demonstrated that those with PTSD demonstrated worse FMD (Grenon et al., 2016), and that FMD was significantly associated with autonomic dysfunction (Clausen et al., 2016). Among women in the Nurses' Health Study II, Sumner and colleagues (2017, 2018) found that women with PTSD had higher levels of vascular cell adhesion molecule-1, a circulating biomarker of endothelial dysfunction. In terms of T2DM, coronary artery disease and alterations in glucose metabolism appear to be highly correlated, raising the hypothesis that atherosclerosis and T2DM may share common genetic and environmental precursors (for a review, see Murea et al., 2012). Given that functional impairment of the vascular endothelium occurs long before the development of visible atherosclerosis (Davignon and Ganz, 2004), evidence is accumulating on the link between endothelial dysfunction and impaired glucose metabolism as a risk for CVD (Jia and Sowers, 2014; Ormazabal et al., 2018). FMD is also a significant predictor of CVD and CVD risk factors, such as hypertension and atherosclerosis (Ras et al., 2013; Yeboah et al., 2009), which are directly linked with autonomic function. Specifically, chronic BP elevation (of which HR is a component) causes shear stress on blood vessel walls that leads to scratching of the endothelium. This damage results in an immune response whereby white blood cells accumulate and form plaques, which can cause atherosclerosis and ultimately myocardial infarctions and ischemia. Given that FMD is a measure of endothelial function and health, this inherently links autonomic function (i.e., BP, HR) with that of the vasculature. Thus, impaired FMD may be considered a downstream indicator of autonomic dysfunction, lending further support for its inclusion in future research in populations with increased cardiovascular risk, such as those with PTSD and T2DM.

With regard to RSA, our study provides evidence of its moderating role on the association between endothelial function and PTSD avoidance symptoms. Specifically, there was only an association between FMD and CAPS-5 Avoidance at low levels of RSA, suggesting that decreased parasympathetic control may be a risk factor that increases the link between PTSD symptoms and cardiovascular dysfunction. This finding is consistent with prior literature implicating low RSA as a risk factor for psychopathology, including PTSD (as opposed to high RSA being protective; Kamkwalala et al., 2012). Given that RSA is an indicator of HR regulation (i.e., the vagus nerve's stimulation of the sinoatrial node), and where HR influences endothelial function via BP, this suggests that parasympathetic control may have influence over FMD and should be considered in addition to sympathetic activity. Indeed, prior research has reported that sympathetic arousal may only be elevated among those with PTSD under conditions of low parasympathetic control. Specifically, Hopper et al. (2006) found that HR was only elevated among individuals with PTSD and low RSA, suggesting that HR alone is an insufficient indicator of overall autonomic function. Future research is needed to understand the relationship between RSA and FMD in trauma-exposed populations, and how parasympathetic control and endothelial function may interact with one another to confer greater PTSD severity and consequently greater CVD risk.

Findings from the current study have clinical implications. Individuals with PTSD may benefit from improvement of parasympathetic control, as it appears to be indicative of both cardiovascular function and PTSD outcomes, and prior studies have demonstrated that it may improve over the course of PTSD psychotherapy (Arditte Hall, Osterberg, Orr and Pineles, 2018). Further, FMD could be a useful tool to assess cardiovascular dysfunction in trauma-exposed samples with T2DM, as poor endothelial function is associated with increased risk of cardiovascular events and has been significantly associated with PTSD (e.g., Grenon et al., 2016). FMD may be a useful indicator of cardiovascular risk in trauma-exposed Black women, particularly because Black women appear to have differences in vascular function compared to White women (e.g., worse microvascular versus macrovascular function; D'Agata et al., 2021). It is critical to note that these differences are not innate, but rather, a likely consequence of longstanding inequities and racial discrimination faced by Black women that place them at higher risk for psychological and physical health problems, including

T2DM (Dixon et al., 2020; Mekawi et al., 2021; Rodríguez and Campbell, 2017; Stojek et al., 2019). These vulnerability factors occur not only at the individual level, but are structural and inherent to socioeconomic systems. Thus, improvement in cardiometabolic health of Black women will require not only greater characterization of the physiological systems underlying disease, but societal change to address health disparities in this population.

There are important limitations to note about the present study. First, while it is a strength that we studied PTSD symptoms and glucose tolerance in an understudied population, this limits generalizability of our findings and requires replication in additional samples. Similarly, our focus on PTSD symptoms and not the categorical diagnosis may be considered a limitation; future research would benefit from comparing associations between cardiovascular indices and glucose tolerance among individuals with versus without PTSD. Second, the current study was cross-sectional and therefore we cannot draw conclusions with regard to the direction of associations among cardiovascular indices, PTSD symptoms, or glucose tolerance. Third, we do not have data on cardiovascular events or CVD in this sample, and future research would benefit from gathering this information at later timepoints in order to determine (prospectively) if low parasympathetic control and poor glucose tolerance confer worse cardiovascular outcomes in trauma-exposed populations.

In summary, results from this study support the association between cardiovascular dysfunction, PTSD symptoms, and glucose tolerance among Black women with T2DM. Endothelial function (indicated by FMD) demonstrated significant associations with CAPS-5 Avoidance and glucose tolerance. With regard to avoidance symptoms, this association was moderated by RSA such that it was only present in the condition of low RSA. Our findings suggest that FMD may be a useful indicator of cardiovascular dysfunction that is highly relevant to PTSD and T2DM, and that parasympathetic control (RSA) may influence these associations.

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Declaration of competing interest

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Data availability

The authors do not have permission to share data.

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