

Contents lists available at ScienceDirect

# Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

# The effects of an acute weight stigma exposure on cardiovascular reactivity among women with obesity and hypertension: A randomized trial



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ARTICLE INFO

Keywords: Weight stigma Blood pressure Heart rate Cardiovascular reactivity

# ABSTRACT

*Objective:* Weight stigma induces cardiovascular health consequences for people with obesity. How stigma affects cardiovascular reactivity in individuals with both obesity *and* hypertension is not known. *Methods:* In a randomized experiment, we assessed the influence of two video exposures, depicting either weight stigmatizing (STIGMA) or non-stigmatizing (NEUTRAL) scenes, on cardiovascular reactivity [resting blood pressure (BP), heart rate (HR), ambulatory BP (ABP), and ambulatory HR (AHR)], among women with obesity and high BP (HBP; n=24) or normal BP (NBP; n=25). Systolic ABP reactivity was the primary outcome. Laboratory BP and HR were measured before/during/following the videos, and ABP and AHR were measured over 19 hours (10 awake hours, 9 sleep hours) upon leaving the laboratory. A repeated measures ANCOVA tested differences in BP and HR changes from baseline in the laboratory and over ambulatory conditions between the two groups after each video, controlling for body mass index, baseline BP and HR. *Results:* Laboratory SBP/DBP increased  $5.5\pm7.3/2.4\pm8.8mmHg$  more in women with HBP than NBP following

the STIGMA versus NEUTRAL video (Ps<0.05). For the primary outcome, ABP increased more in HBP than NBP over sleep (SBP/DBP=4.2+20.6/4.7+14.2mmHg; Ps<0.05) following the STIGMA versus NEUTRAL video, as did HR during sleep (7.5+15.7bpm more in HBP than NBP; P<0.05).

*Conclusions:* Weight stigma increases cardiovascular reactivity among women with obesity and HBP in the laboratory and under ambulatory conditions.

Clinical trial registration: Registered at ClinicalTrials.gov (Identifier: NCT04161638).

### 1. Introduction

Individuals with obesity commonly face stigma and discrimination because of their weight. Approximately 40% of American adults report a history of being stigmatized because of their weight including weight-based teasing, unfair treatment, or discrimination [1,2]. Women typically experience a higher prevalence of weight stigma compared to men [1,3,4], which may be partially attributed to stringent North American ideals of female physical attractiveness which emphasize thinness [5].

Exposure to weight stigma is associated with numerous adverse health consequences [6,7], including negative cardiovascular changes such as increased blood pressure (BP) [8] and cardiovascular stress biomarkers [9–14,40,41]. Unfavorable cardiovascular responses to a psychological stressor (e.g., acute weight stigma exposure) in relation to baseline values is termed *cardiovascular reactivity* [15]. To date, the emerging research on cardiovascular reactivity to weight stigma has focused primarily on blood and saliva biomarkers [9–14,40,41], while only one previous study [8] has examined BP response in the laboratory to an

https://doi.org/10.1016/j.jpsychores.2022.111124

Received 3 June 2022; Received in revised form 16 December 2022; Accepted 18 December 2022 Available online 21 December 2022 0022-3999/© 2022 Elsevier Inc. All rights reserved.

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acute weight stigma stressor.

Stress-induced BP reactivity is also an independent risk factor for cardiovascular disease CVD [16]. Hypertension and obesity are major co-existent CVD risk factors [17]. Indeed, nearly 50% of U.S. adults have hypertension, which accounts for 33% of all cardiovascular deaths [18], and an estimated 60–70% of hypertension in adults is attributable to obesity [19]. Since excess sympathetic nervous system activity also accompanies obesity and hypertension [20], individuals with obesity *and* hypertension may display heightened cardiovascular reactivity to an acute weight stigma stressor compared to individuals with obesity *and* normal BP. To our knowledge, stress-induced cardiovascular reactivity in response to weight stigma among individuals with both obesity *and* hypertension has not been studied. The inclusion of adults with obesity who have high BP along with those with normal BP is critical to determine if weight stigma exacerbates cardiovascular stress among people who already have increased CVD risk due to their obesity *and* high BP.

The current randomized experimental study assessed the influence of two video exposures, one depicting scenes of weight stigma (STIGMA) and the other portraying non-stigmatizing neutral (NEUTRAL) scenes, on cardiovascular reactivity as measured by resting BP and heart rate (HR) and ambulatory BP (ABP) and ambulatory heart rate (AHR), among women with obesity and high BP (HBP) or normal BP (NBP). We hypothesized cardiovascular reactivity would be greater immediately after watching the STIGMA than NEUTRAL video, and persist outside of the laboratory over ambulatory conditions among women with obesity and HBP compared to NBP. The primary outcome was systolic ABP (SABP) reactivity. Secondary outcomes were laboratory systolic BP (SBP) reactivity, diastolic ABP (DABP) reactivity, laboratory diastolic BP (DBP) reactivity, laboratory HR reactivity, AHR reactivity, ambulatory rate pressure product (RPP) reactivity, and laboratory rate pressure product (RPP) reactivity.

### 2. Methods

### 2.1. Participants

Premenopausal women aged 20–50 years with a body mass index (BMI)  $\geq 30 \text{ kg/m}^2$  and no other known chronic cardiovascular or metabolic diseases besides hypertension were enrolled in the present study. Women were excluded from participating if they were: 1) pregnant or planned on becoming pregnant; 2) took medications that may have affected the primary outcome of SABP reactivity (e.g., antihypertensive medications, stimulants for attention deficit hyperactivity disorder, steroids for asthma, sleep aids); 3) were currently using tobacco products; or 4) had been diagnosed with an eating disorder. Fig. 1 depicts a flow diagram for participant enrollment.

### 2.2. Procedures and measures

The study consisted of three visits including a screening visit (Visit 1) and two randomized visits (Visits 2 and 3), which involved watching a 10-min STIGMA or NEUTRAL video exposure. All study visits took place in the morning before 10:00 am to control for hormonal fluctuations that can occur throughout the day and the circadian variation in BP and HR. A detailed timeline of study visit procedures is provided in Fig. 2.

Institutional Review Board approval was obtained at the University



Fig. 1. Participant enrollment flow diagram.

Note. ABP = ambulatory blood pressure; HBP = high blood pressure; NBP = normal blood pressure; WD = withdrawal.



Note. ABP = ambulatory blood pressure; BP = blood pressure; HR = heart rate; NEUTRAL = neutral video exposure; STIGMA = stigma video exposure.

of Connecticut (Storrs, CT, USA) and Hartford Hospital (Hartford, CT, USA). The recruitment of participants began in July 2017 following the ordering of equipment and supplies, and internal pilot testing. The final participant was enrolled in December 2018, resulting in an average enrollment rate of  $\sim$ 3 participants per month. The current study is registered at ClinicalTrials.gov (Identifier: NCT04161638). Of note, there was a clerical error that occurred during initial trial registration which indicated that the study had eight primary outcomes. This error was identified following study completion, and was corrected on ClinicalTrials.gov on May 18, 2022 to indicate one primary outcome, and seven secondary outcomes.

Due to the nature of the experimental manipulation in this study and to maintain validity, mild deception was necessary. Participants were told that the study was examining BP, HR, mood, and behavioral responses to various forms of media, and were not informed of the purpose until after they completed the study. In order to verify that the participants were unaware that their cardiovascular reactivity to a weight stigma exposure was being measured, a manipulation check was implemented, asking participants to report what they believed the study purpose to be. No participants reported the true purpose of the study during the manipulation check. At the conclusion of the study, participants were debriefed about the true purpose of the study.

## 2.2.1. Visit 1 (screening visit) procedures

After providing written informed consent, participants' height and weight were measured to confirm and calculate BMI, and waist circumference was measured at the umbilicus. Participants then completed online questionnaires hosted by Qualtrics, a web-based survey program [21]. Measures included: 1) baseline rumination using the Ruminative Responses Scale ( $\alpha = 0.96$ ) [22]; 2) perceived weight status ('underweight', 'about the right weight', 'overweight', 'obese'); 3) previous history of experienced weight stigma, measured with three yes/no questions about whether participants have been teased, treated unfairly, or discriminated against because of their weight [2,23]; 4) internalization of weight bias using the Weight Bias Internalization Scale-Modified [24] to assess endorsement of weight-based stereotypes toward oneself and self-devaluation due to weight ( $\alpha = 0.84$ ); and 5) depressive symptoms measured using the Beck Depression Inventory (BDI) [25] ( $\alpha = 0.92$ ). Following completion of these questionnaires, participants

remained seated for 5 min after which BP was measured using an automated BPTRU monitor (BPTRU Medical Devices; Coquitlam, Canada) three times, 1 min apart in each arm and averaged as per the standards set forth by the American Heart Association (AHA).<sup>18</sup> Prior to leaving Visit 1, participants were attached to the Oscar 2 automatic noninvasive ABP monitor (Suntech Medical Instruments Inc., Raleigh, NC) for 19 h (10 awake hours, 9 sleep hours) following our laboratory's standard procedures [26,27]. Upon completion of Visit 1, participants were randomized with 1:1 allocation using www.randomization.com to either the STIGMA video at Visit 2 and NEUTRAL video at Visit 3, or vice versa.

### 2.2.2. BP group assignment procedures

The resting laboratory BP from Visit 1 was used to determine the participant's BP status as either HBP (i.e., SBP > 120 to <160 mmHg and/or DBP  $\geq 80$  to  ${<}100$  mmHg) or NBP (i.e., SBP  ${<}$  120 and DBP  ${<}$  80 mmHg) based on the 7th Report of the Joint National Committee (JNC 7) BP classification scheme [28]. However, prior to the start of study enrollment, the original study protocol was amended to use the ABP measurement (i.e., the non-invasive gold standard for diagnosing HBP) to confirm the laboratory BP measurement and for BP group allocation. Of note, the final BP status classification did not differ between the two BP assessment procedures. BP status and group allocation was based on the classification criteria outlined by the European Society of Hypertension [29]. Participants were placed in the HBP group if they met any of the following criteria based on their ABP measurements: 1) 19-h average SBP/DBP >130/80 mmHg, 2) daytime (awake) average SBP/ DBP > 135/85 mmHg, or 3) nighttime (sleep) average SBP/DBP > 120/70 mmHg. Participants were placed in the NBP group if they met all of the following criteria based on their ABP measurements: 1) 19-h average SBP/DBP < 130/80 mmHg, 2) daytime (awake) average SBP/DBP <135/85 mmHg, and 3) night-time (asleep) average SBP/DBP < 120/70 mmHg.

#### 2.2.3. Visits 2 and 3 video exposure procedures and stimuli

Participants were instructed to avoid consuming any food and drink (other than water) for  $\geq$ 12 h and to avoid alcohol and caffeine  $\geq$ 24 h prior to each visit. Participants sat and rested for 20 min prior to the video exposures, during which investigators recorded their BP and HR

every 2 min. BP and HR were averaged over this 20-min period and recorded as baseline BP and HR. Following this baseline period, participants viewed one of the two 10-min videos during which BP and HR were also recorded every 2 min. Both the STIGMA and NEUTRAL videos were previously tested in two published experimental studies [10,30] and showed greater cortisol reactivity, an indicator of stress which accompanies cardiovascular reactivity, following exposure to the stigmatizing video versus the neutral video [10].

The STIGMA video consisted of brief clips from popular television shows that depicted women with overweight and obesity in a stereotypical manner (e.g., lazy, loud, and clumsy). The types of scenes depicted in the video reflected common weight-based stereotypes documented in the literature and included teasing in the workplace, humiliating actions involving individuals with obesity, and interpersonal instances of weight bias. The NEUTRAL video consisted of a series of television clips depicting neutral scenes unrelated to body weight such as insurance commercials. After watching the video, another 20 min of seated rest occurred during which BP and HR were measured every 2 min to assess laboratory BP reactivity (secondary outcome) and laboratory HR reactivity (secondary outcome) following the video. Prior to leaving the laboratory on Visits 2 and 3, participants were attached to the ABP monitor to assess SABP reactivity (primary outcome), DABP reactivity (secondary outcome), and AHR reactivity (secondary outcome) over 19 h following our laboratory's standard procedures [26,27].

# 2.3. Analytic plan

We conducted an a priori sample size calculation based on the existing data on BP changes observed in women with obesity after being exposed to stress and weight stigma [8] as well as previous work by our group examining the ABP response [27]. Based on an estimated BP reactivity difference of 4.5 mmHg and a standard deviation of 5 mmHg between women with HBP versus women with NBP, a minimum of 20 subjects per group was required to achieve a statistical power of 80% with a significance threshold of P = 0.05. To account for an estimated attrition rate of 20%, we aimed to enroll 25 subjects per group.

All data are reported as mean  $\pm$  standard deviation. Shapiro-Wilk tests were used to test for normal distribution of the data. Chi-square examined baseline differences between BP groups for categorical variables and analysis of variance (ANOVA) examined baseline differences between BP groups for continuous variables.

Laboratory BP reactivity (secondary outcome) was calculated as the BP during the STIGMA and NEUTRAL video exposures and post-videos minus baseline BP. ABP reactivity (primary outcome) was calculated as the hourly ABP values over the awake, sleep, and 19 h minus baseline BP following each video. Subsequently, we directly compared BP reactivity to the videos between BP groups by calculating a video BP reactivity difference value (i.e., STIGMA BP reactivity minus NEUTRAL video BP reactivity) within each BP group and compared those values between the BP groups. These same calculations performed for BP reactivity were also performed to determine HR reactivity (secondary outcome) in the laboratory and over ambulatory conditions over the same time intervals. In addition, these same calculations were used for the laboratory rate pressure product (RPP) reactivity (secondary outcome) and ambulatory RPP (ARPP) reactivity (secondary outcome), a noninvasive indicator of myocardial oxygen consumption that reflects the work of the heart [31], calculated as SBP x HR.

An a priori statistical analysis using repeated measures ANCOVA (2 BP groups x 2 video exposures) were performed separately for BP, HR, and RPP in the laboratory (pre, during, and post-video exposure) and ABP, AHR and ARPP over the awake, sleep and 19 h to test for main effects and interactions among the BP groups and video exposures. If main effects and interactions were found to be significant or trending from the a priori analysis, we performed post-hoc analysis with Bonferroni correction by condition (i.e., STIGMA or NEUTRAL), BP group (i. e., NBP and HBP), and for STIGMA minus NEUTRAL, dependent on what effects and interactions emerged as significant. Baseline BP, HR, and BMI were selected as a priori covariates and included in the models. No other moderators were assessed. Statistical analyses were performed using the Statistical Package for the Social Sciences 25.0 for Windows (SPSS Inc., Chicago, IL), and P < 0.05 was established as the level of statistical significance.

### 3. Results

### 3.1. Sample characteristics

The sample consisted of mostly Caucasian (69%) women (N = 49) with obesity (BMI = 35.7 kg•m<sup>-2</sup>), who were  $35.8 \pm 9.1$  years of age. The HBP group (n = 24) had an ambulatory awake SBP/DBP of  $147.2 \pm 12.6/88.7 \pm 9.8$  mmHg, while the NBP group (n = 25) had an ambulatory awake SBP/DBP of  $120.9 \pm 7.6/74.3 \pm 10.3$  mmHg. The HBP group on average had uncontrolled BP since no participants were taking antihypertensive medications. Women in the HBP group had significantly higher weight, BMI, waist circumference, SABP, DABP, total cholesterol, low-density lipoprotein, and high-density lipoprotein than the NBP group (Ps < 0.05; see Table 1). More women perceived themselves as not being 'obese' in the NBP group (40.8%) than in the HBP group (26.5%), but this difference did not achieve statistical significance (P = 0.054). There were no differences between the BP groups for self-

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Participant baseline characteristics (mean  $\pm$  SD).

	Total Sample $(N = 49)$	NBP ( <i>n</i> = 25)	HBP ( <i>n</i> = 24)
Demographics & anthropometrics			
		$34.1 \pm$	
Age (vr)	$35.8 \pm 9.1$	8.9	$37.5 \pm 9.1$
Race. # (%)			
Caucasian	69.0	76.2	61.9
African American	14.3	9.5	4.8
American Indian	2.4	2.4	0.0
Other	14.3	9.5	19.0
		$17.0 \pm$	
Education (vr)	$17.4 \pm 3.7$	3.1	$17.7 \pm 4.3$
		90.3 $\pm$	102.3 $\pm$
Weight (kg)	$96.2 \pm 18.9$	17.9	18.3*
		$33.6 \pm$	$37.8 \pm$
BMI (kg∙m <sup>-2</sup> )	$35.7 \pm 5.9$	4.9	6.1*
		93.8 $\pm$	100.6 $\pm$
Waist circumference (cm)	$97.1 \pm 9.3$	9.0	8.5**
Awake ambulatory systolic blood		120.9 $\pm$	147.2 $\pm$
pressure (mmHg)	$133.8\pm16.8$	7.6	12.6**
Awake ambulatory diastolic		74.3 $\pm$	88.7 $\pm$
blood pressure (mmHg)	$81.4 \pm 12.3$	10.3	9.8**
		11.0 $\pm$	
Nocturnal systolic dipping (%)	$\textbf{9.7} \pm \textbf{6.7}$	6.9	$8.3\pm6.5$
		16.5 $\pm$	
Nocturnal diastolic dipping (%)	$15.3\pm8.3$	9.5	$14.0\pm6.7$
Non-nocturnal dippers, # (%)	22.4	28.0	16.7
		69.0 $\pm$	
Heart rate ( $b \cdot min^{-1}$ )	$67.7 \pm 8.5$	8.4	$\textbf{66.4} \pm \textbf{8.5}$
Questionnaires			
Perceived weight status (%)			
Perceived non-obese	67.3	80.0	54.1
Perceived obese	32.7	20.0	45.9
History of weight stigma (%			
yes)	57.1	56.0	58.3
Weight Bias Internalization			
Scale score	$3.4 \pm 1.5$	$3.1\pm1.6$	$3.6 \pm 1.4$
		$\textbf{39.7}~\pm$	38.3 $\pm$
Ruminative Response Scale score Beck Depression Inventory	$39.0 \pm 13.4$	15.4	11.3
score	$\textbf{9.3} \pm \textbf{8.3}$	$\textbf{9.8} \pm \textbf{9.6}$	$\textbf{8.9} \pm \textbf{6.8}$

\*P < 0.05; \*\*P < 0.01.

*Note.* Nocturnal dipping defined as a minimum of 10% decrease in mmHg from awake to sleep hours.

reported history of weight stigma, weight bias internalization, or depressive symptoms (Table 1; All *Ps* > 0.05). The BDI score for the overall sample was somewhat elevated (mean  $\pm$  SD = 9.3  $\pm$  8.3) but within the minimal range (0–13) [25]. As a result, BDI was checked as a potential covariate for our primary outcome, and was deemed a non-significant covariate.

# 3.2. Ambulatory systolic blood pressure reactivity (primary outcome) and diastolic blood pressure reactivity

SABP increased more in the HBP than the NBP group over awake (8.6  $\pm$  19.6 mmHg, P = 0.037), sleep (15.7  $\pm$  20.6 mmHg, P = 0.001), and 19 h (11.9  $\pm$  17.6 mmHg, P = 0.002) from baseline after viewing the STIGMA video (Figs. 4a and b). Similarly, SABP increased more in the HBP than the NBP group over awake (14.0  $\pm$  19.1 mmHg, P = 0.001), sleep (13.3  $\pm$  16.7 mmHg, P < 0.001) and 19 h (13.7  $\pm$  16.2 mmHg, P < 0.001) from baseline after viewing the NEUTRAL video (Figs. 4a and b). However, when directly comparing BP reactivity following the two video exposures over time (STIGMA minus NEUTRAL), SABP increased 4.2  $\pm$  20.6 mmHg more in the HBP than the NBP group over sleep (P = 0.008, Fig. 5b) from baseline. There were no statistically significant differences in SABP reactivity from baseline between BP groups over awake (Figs. 5a and c) or 19 h for STIGMA minus NEUTRAL (P > 0.05).

DABP increased more in the HBP than the NBP group over sleep (11.2  $\pm$  15.2 mmHg, *P* = 0.001) and 19 h (8.1  $\pm$  12.7 mmHg, *P* = 0.003) after the STIGMA video (Fig. 4d). Similarly, DABP increased more in the HBP than the NBP group over awake (8.1  $\pm$  13.7 mmHg, *P* = 0.007), sleep (7.2  $\pm$  12.7 mmHg, *P* = 0.007), and 19 h (7.7  $\pm$  11.8 mmHg, *P* = 0.003) after the NEUTRAL video (Figs. 4c and d). However, for STIGMA minus NEUTRAL, DABP increased 4.7  $\pm$  14.2 and 0.4  $\pm$  10.8 mmHg more in the HBP than the NBP group over sleep (4.7  $\pm$  14.2 mmHg, *P* = 0.049) and 19 h (0.4  $\pm$  10.8 mmHg, *P* = 0.018; Fig. 5d). There were no statistically significant differences in DABP reactivity from baseline between BP groups for STIGMA minus NEUTRAL over the awake period (*P* > 0.05).

# 3.3. Laboratory systolic blood pressure reactivity and diastolic blood pressure reactivity

SBP increased 5.5  $\pm$  7.3 mmHg more in the HBP than the NBP group after viewing the STIGMA video (P = 0.001), while there was no statistical difference in SBP between the BP groups after the NEUTRAL video (P = 0.972, Fig. 3a). When directly comparing BP reactivity following the two video exposures (STIGMA minus NEUTRAL), SBP increased 5.7  $\pm$  11.3 mmHg more in the HBP than NBP group (P =

### 0.021).

DBP increased 3.8  $\pm$  5.4 mmHg more in the HBP than the NBP group after the STIGMA video (P = 0.002), while there was no statistical difference in DBP between the BP groups after the NEUTRAL video (P =0.297, Fig. 3b). When directly comparing BP reactivity following the two video exposures (STIGMA minus NEUTRAL), DBP increased 2.4  $\pm$ 8.8 mmHg more in the HBP than NBP group, but this difference did not achieve statistical significance (P = 0.206).

### 3.4. Laboratory and ambulatory heart rate reactivity

There were no significant differences in the change in HR from baseline between the BP groups or the type of video exposure in the laboratory (All Ps > 0.05). In contrast, AHR increased 5.0  $\pm$  11.3 bpm more in the HBP than the NBP group over the sleep hours (5.7  $\pm$  7.8 versus 0.7  $\pm$  8.0 bpm, respectively; *P* = 0.040) from baseline following the STIGMA video, while there was no difference in AHR over the sleep hours (4.0  $\pm$  8.3 versus 2.3  $\pm$  8.5 bpm, respectively; P = 0.474) from baseline after the NEUTRAL video. Furthermore, when directly comparing the two video exposures over time (STIGMA minus NEUTRAL), AHR increased more in the HBP than the NBP group over sleep (7.5  $\pm$  15.7 bpm; 4.8  $\pm$  10.3 versus  $-2.7 \pm$  11.0 bpm, respectively; *P* = 0.024) and 19 h (3.9  $\pm$  1.9 bpm; 9.3  $\pm$  6.4 versus  $-2.2 \pm 6.5$ bpm, respectively; P = 0.044) from baseline after the STIGMA versus NEUTRAL video. There were no statistically significant differences in AHR reactivity from baseline between BP groups for STIGMA minus NEUTRAL over the awake period (P > 0.05).

### 3.5. Laboratory and ambulatory rate pressure product reactivity

RPP increased 247.5  $\pm$  119.1 mmHg\*bpm more in the HBP than the NBP group (253.7  $\pm$  80.5 versus 6.1  $\pm$  82.3 mmHg\*bpm, respectively; *P* = 0.043) from baseline after the STIGMA video, while there was no difference in RPP between the BP groups from baseline after the NEUTRAL video. Furthermore, when directly comparing the two video exposures (STIGMA minus NEUTRAL), RPP increased 332.9  $\pm$  154.4 mmHg\*bpm more in the HBP than the NBP group (208.7  $\pm$  110.2 versus  $-124.2 \pm 108.0$  mmHg\*bpm, respectively; *P* = 0.036) from baseline.

ARPP increased more in the HBP than the NBP group over sleep (1205.1  $\pm$  413.3 mmHg\*bpm; 1442.1  $\pm$  324.5 versus 40.8  $\pm$  316.9 mmHg\*bpm, respectively; *P* = 0.006), and 19 h (945.0  $\pm$  446.2 mmHg\*bpm; 2400.9  $\pm$  296.3 versus 1455.9  $\pm$  289.4 mmHg\*bpm, respectively; *P* = 0.040) from baseline after the STIGMA video. Similarly, ARPP increased more in the HBP than the NBP group over sleep (1070.4  $\pm$  409.1 mmHg\*bpm; 1218.2  $\pm$  278.5 versus 147.7  $\pm$  265.6



Fig. 3. Mean laboratory blood pressure change from baseline.

Note. \*\*P < 0.01; HBP = high blood pressure; NBP = normal blood pressure; NEUTRAL = neutral video exposure; STIGMA = stigma video exposure.



**Fig. 4.** Mean ambulatory blood pressure change from baseline during awake and sleep hours following STIGMA and NEUTRAL. *Note.* \*\*\*P < 0.001; \*\*P < 0.01; \*P < 0.05; HBP = high blood pressure; NBP = normal blood pressure; NEUTRAL = neutral video exposure; STIGMA = stigma video exposure.



**Fig. 5.** Mean ambulatory blood pressure change from baseline over awake and sleep hours: STIGMA minus NEUTRAL. *Note.* \*\*\*P < 0.001; \*\*P < 0.01; \*P < 0.05; HBP = high blood pressure; NBP = normal blood pressure.

mmHg\*bpm, respectively; P = 0.012), and 19 h (956.0 ± 415.6 mmHg\*bpm; 2390.1 ± 282.6 versus 1434.1 ± 269.8, respectively; P = 0.026) from baseline after the NEUTRAL video. However, for STIGMA minus NEUTRAL, ARPP increased 471.6 ± 404.5 mmHg\*bpm more in the HBP than the NBP group over sleep (329.1 ± 271.9 versus -142.5 ± 259.1 mmHg\*bpm, respectively; P = 0.049) from baseline. There were no statistically significant differences in ARPP reactivity from baseline between BP groups for STIGMA minus NEUTRAL over the awake period (P > 0.05).

## 4. Discussion

To our knowledge, this is the first rigorously designed, controlled study to test whether an acute weight stigma exposure compared to a neutral exposure leads to heightened cardiovascular reactivity among women with obesity with elevated versus normal BP. Our study offers several novel findings. During and immediately after an acute exposure to weight stigma in the laboratory, SBP/DBP increased from baseline  $\sim$ 6/4 mmHg more in the HBP than NBP group. This heightened BP reactivity in the laboratory for the HBP versus NBP group persisted into the sleep hours under ambulatory conditions as SABP/DABP increased  $\sim$ 4/5 mmHg more from baseline after the STIGMA versus NEUTRAL video during sleep. Furthermore, ambulatory HR increased more in the HBP than NBP group by  $\sim 8$  bpm from baseline during the sleep hours after the STIGMA versus NEUTRAL video. These greater increases in BP and HR from baseline resulted in significantly greater increases in the RPP reactivity in the HBP than NBP group during the laboratory and over sleep hours after the STIGMA video but not the NEUTRAL video, reflecting greater demands on the work of the heart after an acute stigma video exposure. These findings are the first to verify that an acute exposure to weight stigma may be exacerbating adverse cardiovascular health responses among women who have obesity and have high BP.

Our findings indicate that acute exposures to weight stigma may exacerbate cardiovascular stress and disease pathology in women who are at CVD risk due to having obesity and HBP, two major co-existent CVD risk factors. It is well established that elevated BP during the sleep hours is associated with increased risk of cardiovascular events including stroke, myocardial infarction, and death as well as end organ damage in community samples and patients with hypertension [29,32]. It is also established that heightened HR and RPP indicate an increased oxygen demand on the body [33]. Therefore, the heightened cardiovascular reactivity increases from baseline in SABP/DABP of  $\sim$ 4/5 mmHg, AHR of  $\sim$ 8 bpm, and ARPP of  $\sim$ 470 mmHg\*bpm for the HBP compared to the NBP group during the sleep hours after the weight stigma exposure illustrates the possible long-term adverse cardiovascular health outcomes among women with obesity and high BP who are vulnerable to repeated acute weight stigma exposures over time.

The previous literature examining stigma or discrimination and cardiovascular health has focused primarily on the relationship between racial discrimination and BP [34]. To date, only one previous study [8] has examined BP response in the laboratory to an acute weight stigma stressor, which involved a randomized controlled study with 99 women who perceived themselves as overweight (26% with obesity) to examine BP reactivity when presenting a video-recorded speech compared to an audio-recorded speech designed to activate concerns about weight stigma. Although the women with obesity tended to exhibit higher BP during the video than the audio condition, the difference in BP reactivity was not statistically significant. Factors that may help explain why these findings are not entirely consistent with ours include the participants' different weight status (i.e., 26% with obesity versus 100% with obesity), stigma exposures (i.e., video and audio speech versus stigmatizing and neutral video), design (i.e., HBP and NBP in one group versus separate groups), and age of the participants (18.83  $\pm$  1.33 versus 35.8  $\pm$  9.1 yr). We are not aware of any other studies examining the effect of an acute weight stigma exposure on ABP. However, Dolezsar and colleagues [35] conducted a meta-analysis of 44 studies examining the effect of racial discrimination on BP and found that perceived racial discrimination was most strongly associated with an increase in nighttime ABP, which is consistent with our ambulatory findings. This suggests that the persistent BP reactivity observed in our study may not be specific to a particular type of stigma, and therefore ABP reactivity should also be examined in response to multiple types of stigma (e.g., sexual orientation) to explore if this pattern is consistent across other types of stigma.

Although our study did not directly examine mechanisms, there are several possible physiological and psychological mechanisms underlying our findings. Obesity-related hypertension can lead to greater activation of the sympathetic nervous system via increased sympathetic outflow (i.e., norepinephrine spillover and sympathetic nerve activity) to the heart, kidneys and skeletal muscle vasculature, as well as a loss of cardiac sympathetic outflow suppression [20]. In addition, acute stress disrupts BP regulating mechanisms such as nitric oxide release and baroreceptor input in individuals with hypertension [36]. Acute stressors can also lead to increased anxiety which can result in increased BP and HR, especially among those who ruminate about those stressors [37]. Thus, one could hypothesize that the physiological response to the stigma exposure in the laboratory may have persisted during sleep hours as a result of rumination [37], which will be important to assess in future research.

Several limitations of our study should be noted. Our sample consisted only of women; therefore, the results cannot generalize to men. We chose to focus our study on women since the stigma manipulation featured television clips of women (not men) in the video. Future studies should examine how weight stigma may affect cardiovascular health in men. Additionally, the HBP group excluded participants on antihypertensive medication, and therefore, had uncontrolled HBP. Thus, the current results cannot be generalized to women with HBP on antihypertensive medication. Both our sample (69%) and the actresses depicted in the stigmatizing video clips consisted of mostly Caucasian women. African American and Hispanic American women have higher prevalence of obesity than Caucasian women [38,39]. Therefore, it is important that future studies include more racially diverse samples and test diverse portrayals of people with obesity in stigmatizing media exposures. Finally, our findings may underestimate the effects of weight stigma on cardiovascular health because we tested a media exposure to stigma rather than a stigmatizing encounter directly experienced by the participant. While participants may have identified with the individual that they saw being stigmatized in the video, personally experiencing a stigmatizing incident would likely have a stronger adverse impact on cardiovascular reactivity. Future studies should examine the effects of different types of weight stigma exposures on cardiovascular reactivity.

This study has several important strengths. This is the first rigorously designed, controlled study to test the effects of weight stigma on BP and HR among women with obesity and HBP versus NBP both in the laboratory and under ambulatory conditions. All study visits were performed by a single investigator (GAP) at the same time of day, using identical equipment for both exposure visits to minimize technical error. All exposure visits were performed in the same quiet examination room, which was cleared of potential items that could have caused an alerting reaction in this population (e.g., health brochures, weight scale). We also randomized the order of exposure visits to control for any influences that visit order may have on our primary outcome.

### 5. Conclusion

Our experimental study findings show that being exposed to weight stigma increased laboratory SBP/DBP more in women with obesity with HBP than those with NBP. This heightened BP reactivity in the laboratory persisted for those with HBP into the sleep hours as SABP/DABP increased more from baseline after viewing the stigmatizing versus neutral video. Furthermore, AHR and ARPP increased more in the HBP than the NBP group over the sleep hours after watching the stigmatizing versus neutral video, reflecting a greater myocardial demand. These findings suggest the need for health care providers to be educated on the harmful effects of weight stigma on patients' cardiovascular health, and for initiatives to help mitigate stigma-induced cardiovascular health consequences.

### Funding

This work was supported by the UConn Research Excellence Program (OVP-REP), Storrs, CT and Hartford Hospital (RRC), Hartford, CT.

### Disclosure

RMP receives grant funding from WW International, unrelated to this study. The authors report no other financial disclosures.

### **Declaration of Competing Interest**

The authors report no conflicts of interest.

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