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ORIGINAL ARTICLE

Effect of passive heat stress and exercise in the heat on arterial stiffness

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Abstract
Purpose Prior evidence indicates that acute heat stress and aerobic exercise independently reduce arterial stiffness. The combined effects of exercise and heat stress on PWV are unknown. The purpose of this study was to determine the effects of heat stress with passive heating and exercise in the heat on arterial stiffness.
Methods Nine participants ($n = 3$ females, 47 ± 11 years old; 24.1 ± 2.8 kg/m^2) completed four trials. In a control trial, participants rested supine (CON). In a passive heating trial (PH), participants were heated with a water-perfusion suit. In two other trials, participants cycled at ~50% of $\dot{V}O_{2peak}$ in a hot (~40 °C; HC trial) or cool (~15 °C; CC trial) environment. Arterial stiffness, measured by PWV, was obtained at baseline and after each intervention (immediately, 15, 30, 45, and 60 min post). Central PWV (C_{PWV}) was assessed between the carotid/femoral artery sites. Upper and lower peripheral PWV was assessed using the radial/carotid (U_{PWV}) and dorsalis pedis/femoral (L_{PWV}) artery sites. The mean body temperature (T_B) was calculated from the skin and rectal temperatures.
Results No significant changes in T_B were observed during the CON and CC trials. As expected, the PH and HC trials elevated T_B 2.69 ± 0.23 °C and 1.67 ± 0.27 °C, respectively ($p < 0.01$). PWV did not change in CON, CC, or HC ($p > 0.05$). However, in the PH trial, U_{PWV} was reduced immediately (−107 ± 81 cm/s) and 15 min (−93 ± 82 cm/s) post-heating ($p < 0.05$).
Conclusions Heat stress via exercise in the heat does not acutely change arterial stiffness. However, passive heating reduces U_{PWV}, indicating that heat stress has an independent effect on PWV.

Keywords Pulse wave velocity · Passive heating · Thermoregulation · Hypotension

Abbreviations

CC	Cool cycling trial
CON	Control trial
C_{PWV}	Central pulse wave velocity
HC	Heat cycling trial
L_{PWV}	Lower peripheral pulse wave velocity
MAP	Mean arterial pressure
PH	Passive heating trial
PWV	Pulse wave velocity
T_B	Mean body temperature
T_{rec}	Rectal temperature
T_{sk}	Mean skin temperature
U_{PWV}	Upper peripheral pulse wave velocity
$\dot{V}O_{2peak}$	Peak oxygen consumption

Introduction

The leading cause of death in the USA, accounting for one out of every four deaths, is cardiovascular disease (Mozaffarian et al. 2016). One of the most prevalent and costly risk factors for cardiovascular disease is hypertension. Interestingly, hypertension is often preceded by increased arterial stiffness (Weisbrod et al. 2013; O'Rourke 1990). Therefore, arterial stiffness may be a more sensitive predictor of

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cardiac events compared to blood pressure alone (Duprez and Cohn 2007).

Lower arterial stiffness is strongly associated with aerobic fitness (Vaitkevicius et al. 1993), suggesting that the cardiovascular protective effect of exercise may be partially explained by lower arterial stiffness (Blair et al. 1995; McDonnell et al. 2013). Further, chronic aerobic exercise can reduce arterial stiffness within 1 week of training (Cameron and Dart 1994). Chronic changes are likely a cumulative effect of acute bouts of exercise. Acute bouts of moderate intensity (65% $\dot{V}O_{2max}$) cycling in young adults causes reductions in both central (~4% reduction) and peripheral (~10% reduction) arterial stiffness (Kingwell et al. 1997). The same response has also been observed in older adults (Nickel et al. 2011) even with a lower intensity of exercise (cycling at ~50% heart rate reserve). However, not all acute exercise bouts reduce arterial stiffness (Heffernan et al. 2007), and this response may differ by arterial branch (Naka et al. 2003). This poses the question as to what factors influence acute changes in arterial stiffness with acute aerobic exercise.

Aerobic exercise increases metabolic heat production, such that body temperature can increase as much as 1.0 °C in as little as 30 min (Gregson et al. 2002; Saltin and Hermansen 1966). In vitro research suggests that heating vessels improves arterial compliance (Mitchel et al. 1994) and recent in vivo work (Ganio et al. 2011; Moyen et al. 2016) indicates that passive heat stress may decrease arterial stiffness, as measured by pulse wave velocity (PWV). Specifically, increasing mean body temperature ~2.5 °C with a water-perfused suit in about ~90 min led to decreases in arterial stiffness in young smokers and non-smokers (Ganio et al. 2011; Moyen et al. 2016). Patients with peripheral artery disease also have other beneficial hemodynamic changes with acute heat exposure (Neff et al. 2016; Thomas et al. 2016). The exact mechanisms for these improvements are unknown, but may be related to the increased shear stress secondary to increased cardiac output during heat stress (Ganio et al. 2011; Moyen et al. 2016).

Despite evidence regarding the independent acute effects of exercise and heat stress on arterial stiffness, the interaction of heat stress and exercise on arterial stiffness is largely unexplored. It is possible that increased metabolic heat production during exercise is partially responsible for beneficial changes in arterial stiffness with acute exercise. Thus, manipulating body temperature during exercise using different environmental temperatures will provide insight into the influence of body temperature on arterial stiffness during exercise.

Therefore, the purpose of this study was to examine the acute effects of passive heating and exercise in the heat on arterial stiffness in otherwise healthy men and women who were screened for baseline arterial stiffness. This population was selected because prior evidence suggests that the improvement in arterial stiffness with heat stress is greatest in individuals with high, normothermic arterial stiffness (Ganio et al. 2011; Moyen et al. 2016). We hypothesized that both exercise and passive heat stress would reduce arterial stiffness, but greater reductions would be observed with both exercise and heat stress, due to the additive effects of both stressors.

Methods

Written informed consent was obtained from all participants in this study. Study procedures and informed consent documents were approved by the Institutional Review Board at the University of Arkansas and were in accordance with the current guidelines of the Declaration of Helsinki. Participant characteristics can be found in Table 1. Participants included six men and three women ($n = 9$) ranging from 35 to 65 years of age, who had no reported medical illnesses. Participants were excluded if they were smokers, taking medications, hypertensive (resting systolic blood pressure >139 mmHg), or had any cardiovascular, renal, metabolic, or neurological diseases. To avoid the confounding effects of the menstrual cycle (Robb et al. 2009) and increase the likelihood of meeting our arterial stiffness criteria (see below) (Zaydun et al. 2006), only post-menopausal women were included. All participants had their body composition measured using dual energy X-ray absorptiometry (GE Lunar Prodigy, Madison, WI).

Measures of pulse wave velocity (PWV; an index of arterial stiffness) via Doppler ultrasound were taken at a familiarization visit to determine eligibility for the study. Previous research indicates that those with greater baseline stiffness are more likely to have greater reductions in stiffness with passive heating (Ganio et al. 2011; Moyen et al. 2016). Therefore, only those with measured central PWV (carotid–femoral; direct distance multiplied by 0.80) of 600 cm/s or greater were included in the study. This cutoff was determined from previously published normative values (Collaboration RVfAS 2010) which indicates that those with central PWV greater than 600 cm/s have a higher than normal PWV relative to young healthy adults.

Table 1 Participant characteristics, mean ± SD

Sex (males/females)	6/3
Age (years)	47 ± 11
Height (cm)	170.2 ± 24.1
Body mass (kg)	70.4 ± 11.9
Body mass index (kg/m^2)	24.1 ± 2.80
Adiposity (% body fat)	26.0 ± 10.0
Screening C_{PWV} (cm/s)	700 ± 110

Qualified participants performed a graded exercise test to determine maximal oxygen consumption ($\dot{V}O_{2peak}$). This was performed on an electronically braked cycle ergometer (Velotron, RacerMate Inc., Seattle, WA) with oxygen uptake measured by indirect calorimetry (Parvo Medics' TrueOne® 2400, Sandy, UT). Exercise started at ~50 W and increased 25 W every 2 min until volitional exhaustion. Every 2 min and at exhaustion, heart rate and rating of perceived exertion were recorded.

Experimental protocol

The overall protocol is displayed in Fig. 1. Participants took part in four separate experimental trials: control (CON), passive heating (PH), cool cycling (CC), and heat cycling (HC). Each trial took place in a randomized order separated by a minimum of 48 h. Participants refrained from alcohol and exercise 24 h, caffeine 12 h, and food 4 h before each trial. Compliance with this protocol was verified with a 24 h history questionnaire. Prior to each trial, euhydration was encouraged by having participants consume an additional 500 mL of water the night before testing and 2–3 h prior to arrival.

In all trials, upon arrival at the laboratory, participants voided their bladder to provide a small urine sample, and a nude body mass was obtained. To classify hydration status, a urine specific gravity of $\leq$1.020 was designated as euhydrated, and a urine specific gravity > 1.020 as dehydrated (Armstrong et al. 2010). If participants reported to a trial with a urine specific gravity >1.020, they were given ~500 mL of water to drink. The trial was not started until they produced a urine sample with a specific gravity $\leq$1.020.

In the CON trial, participants were dressed in a water-perfused, tube-lined suit that covered the entire body, except the head, face, hands, and feet (Allen-Vanguard Technologies, Ottawa, Canada). The suit permits control of skin, and thus core temperature by changing the temperature of the water perfusing the suit. Participants remained supine in a thermo-neutral environment while skin temperature water (34 °C) perfused through the suit for 50 min, which is the approximate time length of the PH trial.

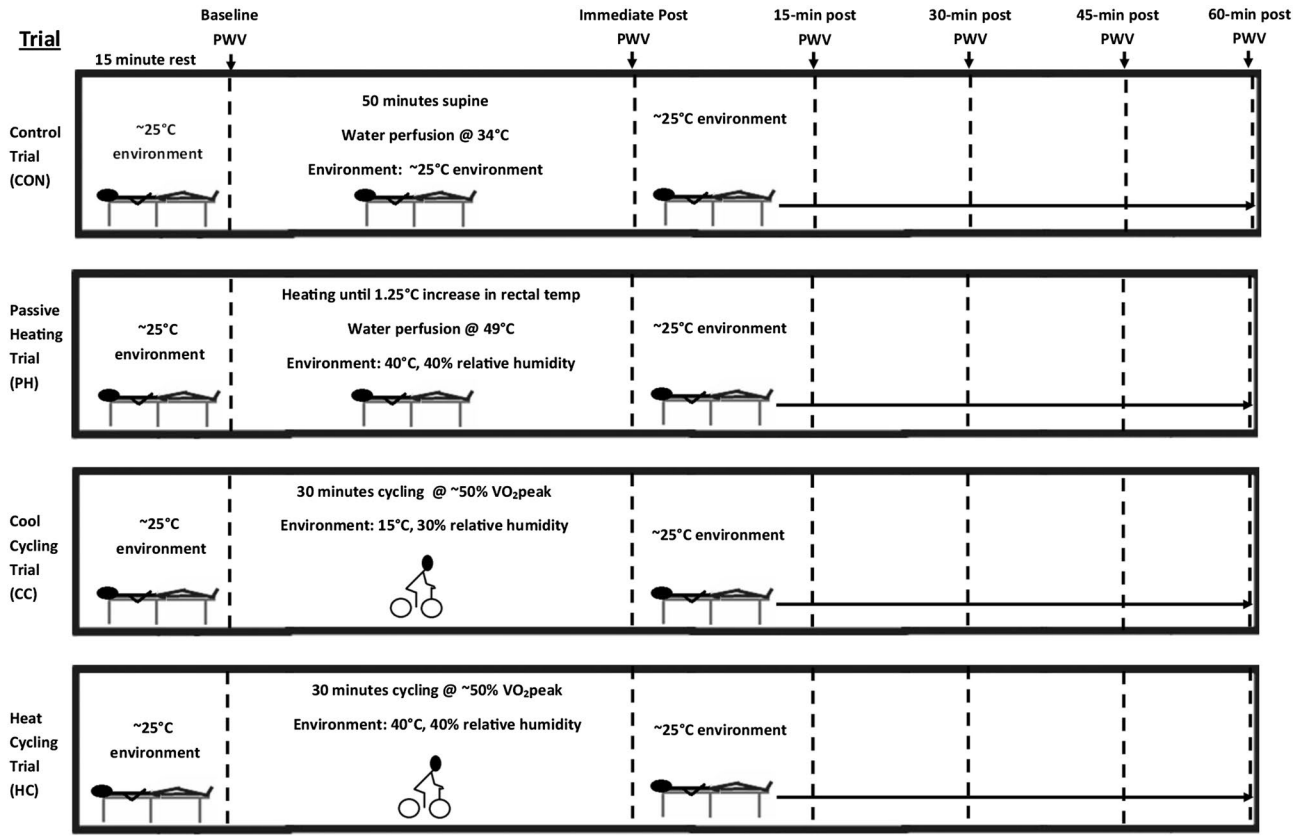

Fig. 1 Experimental protocol. *Dashed lines* represent when pulse wave velocity (PWV) was obtained. The middle portion, immediately following baseline PWV, indicates the intervention during each experimental trial. After each intervention, participants were supine for 1 h while PWV was measured

In the PH trial, participants donned the same tube-lined suit and laid in a supine position. After baseline measures, participants were moved into an environmental chamber (40 °C, 40% relative humidity) on a movable gurney, thus remaining supine. Hot water (49 °C) then perfused through the suit until a 1.25 °C elevation in rectal temperature (T_{rec}) was achieved.

For the exercise trials (i.e., HC and CC), participants laid in a supine position for ~15 min prior to baseline PWV measures. Participants then proceeded to exercise on an electronically braked cycle ergometer for 30 min (Velotron, RacerMate Inc., Seattle, WA). In the CC and HC trials, participants cycled at 51.7 ± 2.6 and 52.6 ± 3.5% of $\dot{V}O_{2peak}$, respectively ($p = 0.58$). The environmental chamber was maintained at 40 °C, 40% relative humidity for the HC trial, and 15 °C, 30% relative humidity for the CC trial.

After each respective intervention, participants immediately left the environmental chamber, and/or had the water-perfused suit removed. All experimental measures were obtained at this time and every 15 min for the 60 min following each intervention. This timing was based on similar protocols examining the acute effect of exercise on arterial stiffness (Nickel et al. 2011; Kingwell et al. 1997). After the last PWV measure, participants voided their bladder into a collection container and a nude body mass was obtained.

Experimental measures

Participants were instrumented with an automated sphygmomanometer (Tango+; SunTech Medical, Inc., Morrisville, NC, USA) for the measurement of blood pressure. Mean arterial pressure (MAP) was determined using one-third systolic and two-thirds diastolic blood pressure. Mean skin temperature (T_{sk}) was calculated as previously validated (Ramanathan 1964) by weighting skin temperature from the right anterior thigh, chest, lateral calf, and triceps using four, small, wireless Thermochron thermocouples that recorded skin temperature every minute (iButtons, Maxim Integrated, San Jose, CA). T_{rec} was recorded during the trial via a rectal thermistor inserted 15 cm beyond the anal sphincter (RET-1, Physitemp, Clinton, NJ). Mean body temperature (T_B) was calculated as $T_B = 0.64T_{rec} + 0.36T_{sk}$ (Lenhardt and Sessler 2006).

Arterial stiffness was indexed with PWV, which is the preferred method (Laurent et al. 2006; Townsend et al. 2015). PWV was measured with Doppler ultrasound (GE GoldSeal LOGIQ eBT08) using the foot-to-foot method (Calabia et al. 2011). Specifically, PWV was calculated as the distance between measurement sites divided by the time delay between the two waveforms. A three-lead ECG was utilized to calculate the time delay from the R-wave to the foot of the pulse wave. The time delay was averaged from a minimum of ten cardiac cycles wherein the standard deviation between the ten measurements was less than 5 cm/s. Central PWV (C_{PWV}) was calculated from the carotid and femoral arteries, while upper peripheral PWV (U_{PWV}) was calculated from the carotid and radial arteries. Lower peripheral PWV (L_{PWV}) was calculated from the femoral and dorsalis pedis arteries. The distance between arterial measurement sites for C_{PWV} was calculated as the combined distance from the suprasternal notch site to the umbilicus and from the umbilicus to the femoral site minus the distance from the carotid to the suprasternal notch. The distance between arterial sites for U_{PWV} was calculated as the distance between the suprasternal notch and the radial site minus the distance from the carotid to the suprasternal notch. The distance between arterial sites for L_{PWV} was calculated as the direct distance between the femoral and dorsalis pedis sites. Distances between sites were calculated for each individual trial by measuring from the distal edge of the ultrasound probe with a retractable cloth tape measure (Bentonville, AR). To minimize the effects of body contours over the central section of the body, custom-made calipers were utilized to measure the distance between the umbilicus and the suprasternal notch (Townsend et al. 2015). All PWV measures were performed on the left side of the body with consistent probe location being assured by marking the skin with a surgical marker. Measurement site order was randomized between participants, but consistent within participants for each trial.

Statistical analysis

Data were analyzed using IBM SPSS version 23 (Chicago, IL, USA). A two-way repeated-measures analysis of variance was used to compare trials and to examine PWV, HR, and MAP differences throughout the trial (time) and between conditions (CON vs PH vs CC vs HC). When Mauchly's test of sphericity was violated, the Greenhouse–Geisser correction was utilized. If there was a significant main effect or interaction, pairwise comparisons were made with a Bonferroni correction. To assess changes in PWV that were independent of MAP, a separate analysis was run with MAP as a time-varying covariate (Winer et al. 1991). All data were reported as mean ± SD. Significance was set at $p < 0.05$.

Results

There were differences in T_B over time between all trials (i.e., interaction; $p < 0.01$, Fig. 2). Immediately post-intervention, T_B was higher in PH and HC compared to CON and CC ($p < 0.01$, Fig. 2). Moreover, PH led

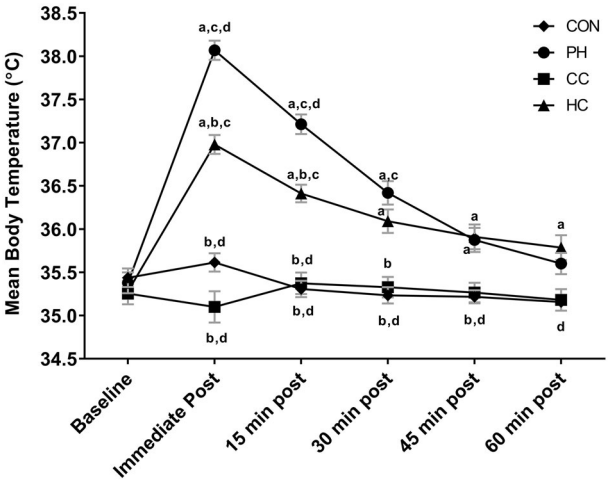

Fig. 2 Mean body temperature (mean ± SEM) over the course of each trial. *a* Indicates a difference ($p < 0.05$) from control (CON), *b* a difference ($p < 0.05$) from passive heating (PH) trial, *c* a difference ($p < 0.05$) from cool cycling (CC) trial, *d* a difference ($p < 0.05$) from the heat cycling (HC) trial

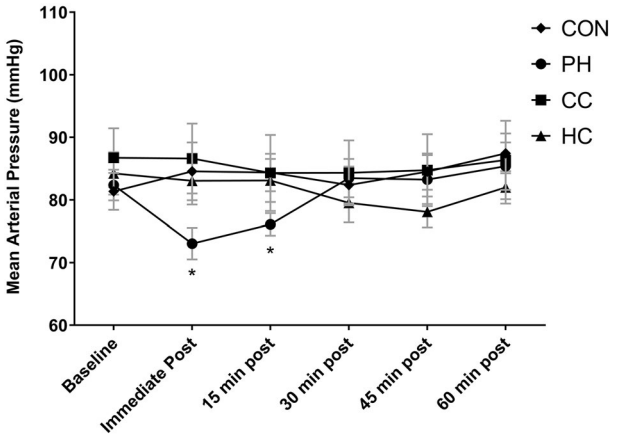

Fig. 3 Mean arterial pressure (mean ± SEM) over the course of each trial. *Significantly different from CON ($p < 0.05$)

to higher T_B compared to HC ($p < 0.05$, Fig. 2). After 60 min, T_B remained elevated in the HC trial only compared to CON ($p < 0.05$, Fig. 2).

Changes in T_{rec} over time were dependent on the trial (i.e., significant interaction, $p < 0.01$). There were no differences between trials at baseline (grand mean: 36.67 ± 0.11 °C; all $p > 0.60$). However, T_{rec} was elevated in all trials immediately post-intervention compared to CON (36.52 ± 0.27 vs 38.16 ± 0.51 vs 37.48 ± 0.19 vs 37.31 ± 0.13 °C, for CON, PH, CC, and HC, respectively; $p < 0.05$). After 15 min, T_{rec} was still elevated in all trials compared to CON (36.52 ± 0.23 vs 38.43 ± 0.31 vs 36.99 ± 0.19 vs 37.66 ± 0.11 °C; $p < 0.01$). At 30 and 45 min post, T_{rec} was still elevated in the PH and HC trials, but not in the CC trial compared to CON (36.55 ± 0.19 vs 37.94 ± 0.23 vs 36.78 ± 0.21 vs 37.47 ± 0.14 °C and 36.59 ± 0.20 vs 37.94 ± 0.23 vs 36.78 ± 0.21 vs 37.47 ± 0.14 °C, for 30 and 45 min, respectively; $p < 0.05$). There were no differences at 60 min post between CON and CC, but T_{rec} was still elevated in the PH and HC trials (36.61 ± 0.17 vs 37.43 ± 0.21 vs 36.62 ± 0.24 vs 37.12 ± 0.15 °C; $p < 0.01$).

T_{sk} changes over time were dependent on the trial (i.e., significant interaction, $p < 0.01$). There were no differences between trials at baseline (grand mean: 32.97 ± 0.14 °C; all $p > 0.05$). Immediately post-intervention, T_{sk} was elevated in the HC and PH trials, but reduced in the CC trials compared to CON (34.04 ± 0.44 vs 37.90 ± 0.38 vs 30.88 ± 1.30 vs 36.39 ± 0.46 °C, for CON, PH, CC, and HC, respectively; $p < 0.01$). However, PH led to greater elevations in T_{sk} compared to HC ($p < 0.01$). At 15 min post-intervention, T_{sk} was only elevated in the PH trial compared to CON ($p < 0.01$; 33.15 ± 0.49 vs 35.04 ± 0.31 vs 32.49 ± 0.88 vs 34.19 ± 0.79 °C). There were no differences between trials at 30, 45, or 60 min post-intervention (all $p > 0.10$; grand means 33.34 ± 0.10 to 32.94 ± 0.15 to 32.75 ± 0.15 °C for 30, 45, and 60 min, respectively).

There was an effect of trial and time on MAP (i.e., significant interaction; $p < 0.05$). MAP did not differ from CON in either the HC or CC trials. In the PH trial, MAP was lower, in comparison to CON, immediately post and at 15 min post ($p < 0.05$; Fig. 3).

Neither C_{PWV} nor L_{PWV} differed over time between conditions (i.e., no interaction or main effects; both $p > 0.10$; Fig. 3a, c). The change in U_{PWV} over time depended on condition (i.e., significant interaction; $p < 0.05$), such that at immediate and 15 min post, U_{PWV} was lower in PH compared to all other trials (all $p < 0.05$; Fig. 4b). Even when adjusting for changes in MAP, there was still a reduction in U_{PWV} immediate post (−106 ± 33 cm/s; $p < 0.05$) and at 15 min (−100 ± 29 cm/s $p < 0.05$) in PH.

Discussion

This study examined the effects of active and passive heat stress on arterial stiffness in men and women. This was accomplished by individuals being heat stressed in non-exercise and exercise settings (i.e., PH and HC, respectively) along with non-heat stress conditions during non-exercise and exercise settings (i.e., CON and CC, respectively). Our data indicate that the acute exercise bout imposed does not result in any acute changes in PWV; regardless of heat stress. However, similar to prior data (Ganio et al. 2011), we demonstrate passive

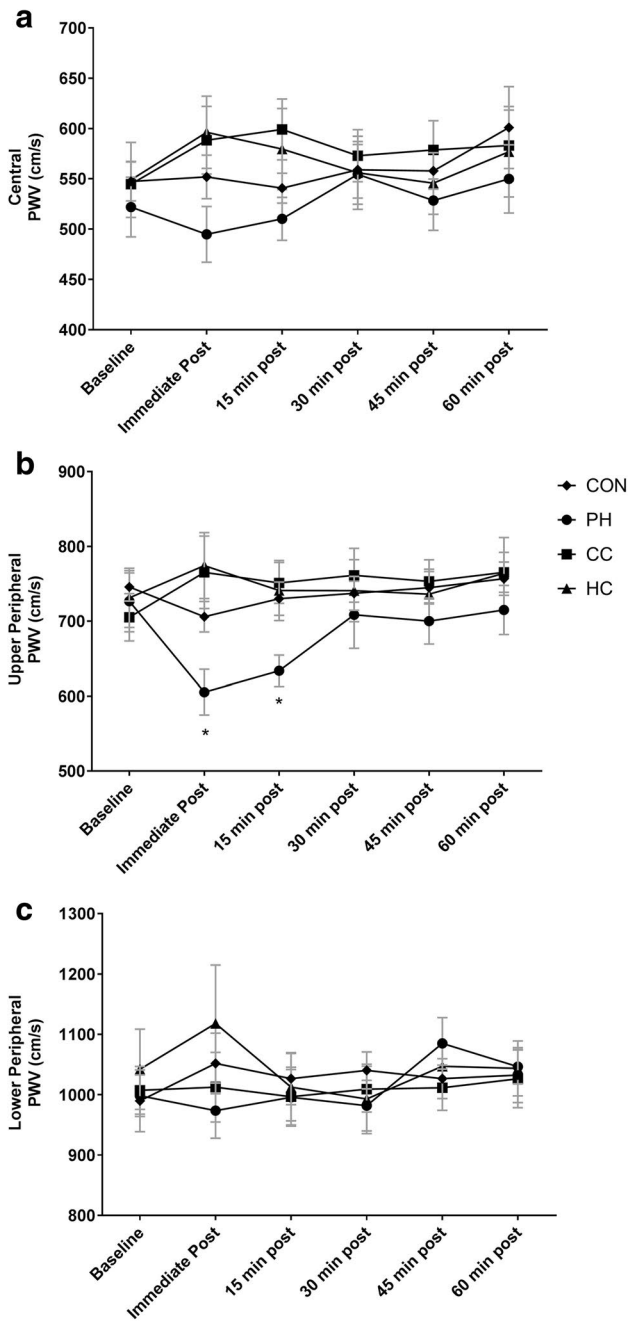

Fig. 4 Central (*panel* **a**), upper peripheral (*panel* **b**), and lower peripheral (*panel* **c**) pulse wave velocity (mean ± SEM) over the course of each trial. *Significantly different from all other trials ($p < 0.05$)

heat stress independently leads to a transient reduction in U_{PWV} (carotid to radial). Interestingly, when heat stress was coupled with exercise, there were no longer changes in any measure of PWV. These data suggest that passive heat stress has an independent effect on upper peripheral arterial stiffness that does not occur when heat stress occurs with exercise in the heat.

Similar to previous studies (Ganio et al. 2011; Kaldur et al. 2016; Moyen et al. 2016), passive heating caused reduced U_{PWV}. Previous work indicates that the response to heat stress is related to baseline stiffness, with higher stiffness being associated with larger reductions in U_{PWV} with passive heating (Ganio et al. 2011; Kaldur et al. 2016; Moyen et al. 2016). Therefore, in this study we tested individuals who had elevated arterial stiffness in resting normothermic conditions. Therefore, not surprisingly, our participants had reduced arterial stiffness with passive heat stress (Fig. 4). We provide additional insight into this response by examining participants for 60 min after the heat stress is removed and they rest in an ambient environment (~25 °C). We observed that PWV remained lower, relative to control/normothermic conditions, for 15 min post-heat stress but returned to normal after 30 min (Fig. 4). This is in parallel to changes in the mean body temperature, such that there were large decreases from immediate post to 15 min post-heat stress (Fig. 2). Although the mean body temperature decreased in the PH trial, it remained elevated relative to CON at 30 and 45 min post-heat stress, while PWV had returned to normal. This suggests that passive heat stress can reduce PWV, but a certain threshold may need to be achieved for changes to occur. Future studies will need to investigate this relationship more closely.

Surprisingly, exercise, both in cold and hot conditions, did not lead to changes in PWV. Previous studies have observed reductions in arterial stiffness with cycling exercise (Kingwell et al. 1997; Nickel et al. 2011). However, these studies presumably had participants exercising in an ambient environment of ~22–25 °C. Pilot testing confirmed that this environment leads to increases in mean body temperature. To isolate the effect of mean body temperature on PWV, we chose to have individuals cycle in a cool (15 °C) and warm environment (40 °C). This allowed us to have a condition in which the mean body temperature was no different from CON or CC (Fig. 2), thus isolating the effect of exercise on PWV. Our results indicate that acute exercise does not affect the measures of PWV. It should be noted that we only examined the acute responses to exercise, and that chronic adaptations to exercise may still improve arterial health, regardless of the environmental condition.

Combining heat stress and exercise in the HC trial did not change any measures of PWV (Fig. 4). This was somewhat surprising given that increases in body temperature with PH led to decreases in U_{PWV}. There are several possibilities that may explain this finding. Mean body temperature was greater in the PH trial versus the HC immediately post-intervention (Fig. 2). Therefore, there may be a threshold for the degree of heat stress needed to induce changes in PWV, and the HC did not reach it. The other possibility is that exercise counteracted the positive effect of heat stress, leading to no net change in PWV.

This could be a result of increased sympathetic firing during exercise, which can independently increase PWV (Swierblewska et al. 2010). Although possible, this does not explain prior findings showing reductions in PWV with acute aerobic exercise (Nickel et al. 2011; Kingwell et al. 1997).

The mechanisms for changes in PWV are not entirely clear, especially since changes were not observed in all conditions. The PH trial was the only trial where changes in PWV were observed, and this effect was localized to U_{PWV}. Heat stress can reduce retrograde shear stress, particularly in the brachial artery (i.e., upper peripheral), and is accompanied by dilation of the brachial artery (Pyke et al. 2004). Reductions in retrograde shear stress, during exercise, have been observed with prolonged cycling and are associated with improvements in endothelial function (Birk et al. 2012). This reduction in retrograde shear stress, through an improvement in endothelial function, may then transiently improve compliance leading to reductions in U_{PWV}. However, as heat and exercise are generally known to affect vascular compliance, it is interesting that we did not find any additive effects of heat-induced vascular function during exercise. In fact, exercise mitigated heat-induced alterations in PWV. This suggest that there is a mechanism occurring during exercise that may temporarily block acute reductions in PWV. This paradox of high body temperatures in both the PH and HC trials, yet differential responses in PWV, is a novel finding and warrants further investigation.

It is possible that exercise intensity is a moderating factor for observing changes in arterial stiffness. The exercise intensity used in this study (~50% $\dot{V}O_{2peak}$) was lower than that in previous studies which observed changes in PWV (~65% $\dot{V}O_{2peak}$) (Kingwell et al. 1997). Therefore, the present study, regardless of environmental temperature, may have provided a stimulus insufficient to induce vascular changes that alter PWV.

As per consensus guidelines (Townsend et al. 2015), MAP also can influence measurements of arterial stiffness. During PH, when U_{PWV} was reduced, MAP was also reduced (Figs. 3, 4). Blood pressure changes post-exercise or lack thereof may also explain why we did not see changes in PWV in the exercise trials. It is well established that higher-intensity exercise bouts result in greater acute reductions blood pressure post-exercise (Mundel et al. 2015). Therefore, the low-intensity exercise used in the present study may not have been sufficient to induce changes in PWV. However, further statistical analysis revealed that U_{PWV} was still reduced even when adjusting for MAP as a time-varying covariate. This would indicate that these changes are not MAP driven. Regardless, our study shows that low-intensity aerobic exercise (independent of heat stress) does not result in any significant changes in PWV.

In contrast to U_{PWV}, there were no significant changes in L_{PWV} throughout any of the trials. The exact mechanism by which this site-specific difference occurred is not clear. One possibility is that the arterial segments were differently affected by the increase in the central nervous system activity that is known to occur during passive heat stress (Keller et al. 2006). Specifically, passive heat stress increases muscle sympathetic nerve activity (MSNA), which could affect vasodilation/vasoconstriction, but the exact effects this has on arterial stiffness is unknown. However, there is evidence that in non-heat-stressed situations, long-duration sitting affects arteries depending on the location (e.g., popliteal artery versus brachial artery), as indexed by flow-mediated dilation (Restaino et al. 2015). Although speculative, this may explain why changes in the upper and lower PWV differed during passive heat stress. Future work should investigate these mechanisms further.

Limitations and future directions

Previous research has demonstrated that shear stress can influence acute changes and adaptations regarding arterial stiffness, and can be used to manipulate arterial adaptations to exercise (Birk et al. 2012). Future studies should attempt to manipulate shear stress during passive heat exposure to observe if changes in PWV during heat stress are due to shear stress. Furthermore, more research is necessary to fully understand how heat exposure and exercise elicit different changes in arterial compliance to recognize the advantages and disadvantages of exercise and heat-based therapies.

There were some differences in T_{sk} and T_{rec} between trials. Therefore, the mean body temperature was used as an indicator of thermal stress because it incorporates both core and skin temperature. Many thermal and cardiovascular responses are closely tied to the mean body temperature (Nadel et al. 1971). Future studies should investigate independent manipulations of skin and core temperature to examine if any of these factors independently effect changes in PWV.

Further, we cannot exclude the influence of postural changes on PWV and MAP. In PH and CON, participants were supine, but exercised upright in CC and HC. This was done purposefully to replicate the testing of previous studies using both passive heating (Moyen et al. 2016) and exercise (Kingwell et al. 1997). Further, Nickel et al. (2011) demonstrated that post-exercise reductions in arterial stiffness occur following upright exercise even when using a fully supine control trial. However, more work would be necessary to demonstrate the effect of posture on post-exercise or heat stress arterial stiffness responses.

In the HC and PH trials, participants had significant increases in T_{sk}, T_{rec}, and T_B, but the modes of heating

differed. In the HC trial, participants were exposed to a hot environment and cycled, which increased the temperature via surrounding ambient air temperature and through metabolic heat production. In the PH trial, participants were exposed to both the ambient air temperature and the 49 °C water in the water-perfusion suit. The water-perfusion suit led to a much higher skin temperature in the PH trial compared to the HC. This was purposefully done to increase the mean body temperature in a timely fashion (~50 min). However, the significantly higher skin temperature in the PH may be the underlying factor driving the reduction of U_{PWV}, thereby confounding our results. Because the heat stress was imposed in different manners, it is difficult to conclusively determine the independent or combined effects of heat stress and exercise per se. Instead, this study indicated that passive heating using a tube-lined suit transiently reduced PWV in some arterial branches, which did not occur when heat stress was imposed with exercise in the heat. Previous work indicating an effect of passive heating (Ganio et al. 2011) used a similar heating protocol. Therefore, it is possible that high skin temperatures are a confounding factor in the context of heat stress and arterial stiffness; future studies should explore this further.

Conclusions

Although exercise is very effective for improving cardiovascular health, it may not always be possible because of risk factors or mobility impairments. Previous literature suggests that heat stress may be a viable option for improving cardiovascular health in those unable or unwilling to exercise. We demonstrate passive heating, but not aerobic exercise, significantly reduced upper peripheral PWV in adults. This indicates that the mechanisms related to the effect of heat stress on arterial stiffness may be different than those observed following exercise.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest to report.

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