A Comparison of the Vascular Response to Acute Sauna Heating in Young and Middle-Aged Adults

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A Comparison of the Vascular Response to Acute Sauna Heating
in Young and Middle-Aged Adults

Olivia Kathryn Leach

A thesis submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of
Master of Science

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ABSTRACT

A Comparison of the Vascular Response to Acute Sauna Heating in Young and Middle-Aged Adults

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Master of Science

BACKGROUND: Age-related declines in endothelial function have been well documented with larger declines observed in middle-aged. Passive heat exposure has been shown to be a promising method to improve vascular endothelial health, with sauna specifically being linked to reduced risk of cardiovascular disease. Increases in blood flow and shear rates associated with heat exposure are often considered to have a major influence on the observed improved endothelial function following heat exposure. The magnitude of these changes in response to sauna have not yet been defined. Therefore, the purpose of this study is to quantify and compare the vascular response to an acute bout of sauna heating in young and middle-aged individuals.

METHODS: 10 young (24.9 ± 4.2 years, 6 males and 4 females) and 8 middle-aged adults (55.6 ± 3.9 years 4 males and 4 females) underwent 40 min of sauna exposure at 80 ºC. Esophageal and intramuscular temperatures were recorded throughout the duration of the experiment. Brachial and superficial femoral artery blood flow, artery diameter, and shear rates were recorded at baseline and following heat exposure. Brachial artery flow-mediated dilation (FMD) was measured at baseline and following 90 min of recovery.

RESULTS: Core and muscle temperatures significantly increased by 1.5 ± 0.53 and 1.95 ± 0.70 ºC, respectively (P < 0.05) and the magnitude of increase did not differ between young and middle-aged participants (P0.867 and 0.488, respectively). Shear rate increased by 170–200% (P < 0.001), while blood flow increased by 180–390% (P < 0.001) in the superficial femoral and brachial artery, respectively, in both groups. Importantly, the changes in shear and flow did not significantly differ between young and middle-aged subjects for either artery (P = 0.190–0.899.) Systolic blood pressure (SBP) was significantly reduced from 135.25 ± 17.50 to 122.38 ± 19.7 mmHg (P = 0.017) only in middle-aged participants and a decrease in diastolic blood pressure was observed from 81.6 ± 13.0 mmHg at baseline to 69.8 ± 8.4 mmHg (P < .001). Heat-induced dilation was strongly correlated to baseline endothelial function in the young (R = 0.86, P = 0.006), but not the old (R = 0.22, P = 0.631).

CONCLUSIONS: These results indicate that young and middle-aged adults have similar shear-rate and blood flow responses to acute sauna heating, which significantly reduces blood pressure in middle-aged, but not young individuals. Future heat therapy studies may elicit meaningful cardiovascular benefits from lower magnitudes of chronic passive heat stress.

Keywords: heat stress, vascular function, sauna, blood flow, shear rate
ACKNOWLEDGEMENTS

I feel lucky to have begun my research experience as early as I did. I am grateful to have had a space to develop my own interests and questions. I am especially appreciative to the previous graduate students who gave me the opportunity to work on their theses.

I was fortunate to have had the oversight of several fantastic scientists. To my committee, Dr. Gifford, Dr. Mack, and Dr. Hyldahl, thank you for providing the guidance and support throughout the process. Dr. Gifford, thank you for taking me on at a time that was particularly chaotic in the lab and for your investment in my education. Dr. Mack, thank you for entertaining my incessant questions and equipment malfunctions. Thanks for showing me that a future in research was possible and providing the connections to help me get there.

Having been involved in previous graduate students’ projects, I understand the value of a good research team. Thank you to my research team who gave up many Friday nights for data collection, especially Kate Strong. Thanks for being my right hand throughout the entire process, I could not have done it without you. Nani, Mo, Jared, and Landon – I owe you for my sanity, and probably a little bit of yours. Jessica Joy, you are the friend in academia I didn’t know I needed but couldn’t live without.

To my family and siblings, thanks for pretending to think what I do is interesting. Mom, thanks for being the chief listener of all my complaints and the primary editor of all my writing.
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Introduction

Age-related declines in vascular health are not linear (1, 2). The effects of age on the vascular system are evident in childhood and begin to decline at a markedly faster rate in midlife (2). Celermejer (1) reports this age threshold at which the rate of vascular function decline increases to be around the fourth decade in men and the fifth decade in women, with the rate of decline being matched by the age of 65. This age group encompasses what the National Institute of Health considers middle age (50–64 years) (3). The trajectory of cardiovascular health in older adults has also been associated with the level of subclinical atherosclerosis in middle age, independent of cardiovascular health status in younger years (4). This timeline suggests middle age is an influential time to adjust the trajectory of vascular health later in life.

Heat therapy is a mechanism for improving vascular function (5–10). This is based on epidemiological data showing a strong correlation between frequent sauna usage and a reduced risk of cardiovascular disease and all-cause mortality, particularly in the Finnish population (11, 12). For example, Laukkanen (12) reports that Finnish men who spent at least 19 min in a sauna (average temperature = 80 °C), 4 times per week had approximately 50% less incidence of sudden cardiac death and fatal coronary artery disease than those who used saunas less frequently or for a shorter duration. Thus, sauna bathing at a temperature of 80 °C for 19+ minutes per session appears to provide a stimulus sufficient to elicit major cardiovascular benefits.

Despite the known benefits on cardiovascular health associated with frequent sauna usage, few studies have looked at the physiological responses to this passive heat therapy. Passive heating interventions using hot water immersion (5–7, 13, 14) suggest that an increase in vascular shear stress in response to heating is a primary stimulus for improved vascular function.
Improvements in vascular function have been reported following several passive heating modalities (5, 6, 9, 13, 15, 16). However, these sauna alternative heating modalities lack the years of prospective data and recorded cardiovascular benefit associated with passive heating with sauna. Unfortunately, the magnitude of tissue temperature change and increased shear stress associated with sauna protocols have not been reported despite the epidemiological data which supports sauna. Rather, a variety of passive heat stress protocols for the sauna are based on simple core temperature thresholds without regard to the required vascular shear stress required for the adaptations associated with heat therapy.

Studies using sauna as passive heat therapy focus heavily on older populations (17–21). Other heat therapy studies report participants to be the middle-aged population, however their reported mean participant age of 65+ categorizes these participants as older adults. Given the pivotal time point of middle age and the influence it has on the trajectory of vascular health, the effect of passive heating on this population is of particular interest. Sauna, as the method of heating, is additionally intriguing because of the known relationship between chronic usage and overall cardiovascular health. Thus, the purpose of this study is to quantify the vascular response to an acute bout of sauna heating in young and middle-aged adults in order to better inform the development of future heat intervention studies.

Methods

Subjects

Physical characteristics of the subjects are reported in Table 1. All participants were thoroughly acquainted with all aspects of the experiments before providing written informed consent. Experimental protocols were approved by the institutional review board at Brigham Young University. Ten young adults (6 males, 4 females, 18–35 years) and eight middle-aged
adults (4 males, 4 females, 50–64 years) participated in this study. All participants were screened for cardiometabolic health prior to participation through a health history questionnaire. Young female participants were studied during the early follicular phase and middle-aged females were postmenopausal and not currently taking hormone replacement therapy.

**Experimental Approach**

All measurements were recorded in the supine posture at an ambient temperature of 23°C. Prior to an experimental visit, body composition was measured using dual-energy x-ray absorptiometry (Lunar iDXA, GE Healthcare, Chicago, IL, USA) and physical activity (steps per day) was measured through a wrist-worn accelerometer for 7 days (GT9X, ActiLife v6.13.4, ActiGraph, Pensacola, FL, USA). Subjects reported to the laboratory having fasted for 4 hr and refraining from caffeine, alcohol, and exercise for 24 hr prior to participation in the study. Upon arrival, dry, seminude weight was recorded. Esophageal and intramuscular temperature probes were placed, and the subject was placed in the supine posture. After 30 min of rest, peripheral hemodynamics, HR, blood pressure, and flow mediated dilation (FMD) vascular function were assessed. The participant then entered the 80 °C dry sauna and rested in the semirecumbent position for two 20-min bouts separated by a 10-min recovery interval at room temperature. Peripheral hemodynamic measurements, HR and blood pressure were taken during the 10-min recovery interval at the conclusion of the second 20-min exposure, and then every 30 min for the 90-min recovery period. Vascular function was assessed at the end of the 90-min recovery period.

**Esophageal and Muscle Temperature**

Esophageal temperature was used as an estimate of body core temperature. A T-type thermocouple encased in a plastic sheath (~2 mm diameter) was introduced into the esophagus
by way of a nostril at a length of one fourth the subject’s standing height. Esophageal
temperature measures were not recorded for two middle-aged participants due to the participants’
inability to properly place the thermocouple. A sterile T-type thermocouple was inserted
perpendicular to the vastus lateralis muscle at a depth of approximately 3.5 cm using an 18-
gauge sterile Cathlon I.V. catheter (Smiths Medical, Minneapolis, MN) to monitor intramuscular
temperatures throughout the intervention. Prior to insertion, local anesthesia (1% lidocaine with
epinephrine) was used to numb the area. Core and muscle temperature was recorded in 30-s
intervals using PicoLog thermocouple data logger (PicoLog Technologies, Cambridgeshire, UK;
model USB TC-08). Two muscle temperature measurements (1 young, 1 middle-aged) were not
included in data analysis due to thermocouple malfunctioning following insertion in the muscle.

**Heart Rate and Blood Pressure**

Blood pressure and heart rate were measured using an automated sphygmomanometer
from the right arm (Tango M2 Stress Test Monitor, SunTech Medical, Raleigh, NC). Blood
velocity and diameter of the brachial and superficial femoral arteries were measured using
doppler ultrasonography (12-MHz linear array transducer, LOGIQ™ e Ultrasound GE
HealthCare, Milwaukee, WI, USA). Shear rate was calculated as the quotient of 4 × blood
velocity and vessel diameter. Vascular resistance was calculated by dividing mean arterial
pressure by blood flow and expressed as mmHg⁻¹·min·ml⁻¹. Superficial femoral artery data from
two young and one middle-aged participants were excluded from analysis due to poor image
quality.

**Vascular Function**

Macrovascular dilator function was assessed using endothelial-dependent flow-mediated
dilation (FMD) of the brachial artery, in accordance with recent guidelines (22). A 9 cm blood
pressure cuff (D. E. Hokanson, Bellevue, WA) was placed on the upper forearm distal to the elbow. Before cuff inflation, brachial arterial diameter and blood velocity were recorded during a 1-min baseline period. Arterial inflow to the forearm was occluded by rapidly inflating the cuff to 250 mmHg for 5 min (E20 Rapid Cuff Inflator; D. E. Hokanson, Bellevue, WA). Diameter and blood velocity measures were recorded 20 s before rapid cuff deflation and for 2 min following deflation. Brachial artery diameter was analyzed frame-by-frame by automated edge detection software (Quipu srl., Pisa, Italy) and averaged into 1-s bins corresponding with 1-s average velocities.

**Statistical Analysis**

Baseline differences between young and middle-aged subjects were compared using independent sample t-tests. To describe the effect of age on the vascular and temperature responses to heat therapy, a mixed-model repeated measures ANOVA (presauuna, immediately postsauna, 30-, 60- and 90-min postsauna) was performed. Select planned comparisons were analyzed using t-tests with Bonferroni correction. The relationship between endothelial function, age and the observed responses to heat were examined with least squares linear regression. Alpha was set to 0.05. Statistical analysis was performed using SPSS version 28 (IBM, New York, USA).

**Results**

**Temperature**

Core temperature revealed a main effect of time (P < 0.001), reaching peak increase of ~1.5 °C in the sauna that remained elevated throughout the duration of the experiment (P = 0.010). No effect of age (P = 0.867) and no interaction (0.387) were observed, indicating that young and middle-aged participants responded similarly. There was a main effect of time (P <
0.001) on muscle temperature with no effect of age (P = 0.488) or time-by-age-group interaction (P = 0.952). There was a peak increase in muscle temperature of 1.95 ± 0.70 °C during the sauna exposure. Muscle temperature remained elevated for 30 min postexposure (P < 0.001) (Figure 1B).

**Heart Rate and Blood Pressure**

Systolic blood pressure (SBP) exhibited a main effect of time (P = 0.019), and a time-by-age-group interaction (P < 0.001). Young participants showed no change in SBP from baseline (119.4 ± 6.4 mmHg) after sauna exposure (123.1 ± 7.7 mmHg; P = 0.067). SBP in the middle-aged participants decreased from 135.1 ± 17.2 mmHg at baseline to 122.4 ± 19.7 mmHg (P = 0.005) immediately postsauna (Figure 2B). There was a main effect of time (P < .001) and age (P = 0.039) in diastolic blood pressure (Figure 2B). There was no change in DBP in young participants and a significant decrease from 81.6 ± 13.0 mmHg at baseline to 69.8 ± 8.4 mmHg (P < .001) in middle-aged participants immediately after sauna exposure. HR revealed a main effect of time (P < 0.001) with no age group interaction (P = 0.884) showing a similar increase in HR in both groups. HR in young participants increased from 57 ± 12 bpm to 75 ± 17 bpm, and middle-aged participants increased from 62 ± 9 bpm to 77 ± 14 bpm (< 0.001). HR remained elevated after 30-min recovery from the sauna exposure (P = 0.011).

**Peripheral Hemodynamics**

Brachial artery flow reveals a main effect of time (P < 0.001) with no effect of age (P = 0.899) or time-by-age-group interaction (P = 0.206). Brachial artery flow increased from baseline following sauna exposure similarly in both the young and middle-aged participants and remained elevated through 30 min of recovery (P < 0.001; Figure 3A). There was a main effect of time (P < 0.001) with no effect of age (0.488) or time-by-age-group interaction (P = 0.952) for
brachial shear rate. Shear rate in the brachial artery increased from 173.43 ± 77.78 sec\(^{-1}\) to 515.13 ± 235.30 sec\(^{-1}\) and 152.58 ± 46.84 sec\(^{-1}\) to 459.69 ± 108.07 sec\(^{-1}\) in the young and middle aged, respectively (Figure 3B). Shear rate increases remained elevated 60 min into recovery (P = 0.007). There was a main effect of time (P < 0.001) with no effect of age (P = 0.783) or time-by-age-group interaction (P = 0.185) for brachial artery diameter, indicating similar dilation in both groups from baseline to immediately after sauna (Figure 3C).

Additionally, we observed no significant difference in heat-induced brachial artery dilation between young and middle-aged groups in the brachial artery (P = 0.067). There was a main effect of time (P < 0.001) and no effect of age (P = 0.204) or time-by-age-group interaction (P = 0.562) for resistance in the brachial artery. Resistance in the brachial artery at rest was 0.78 ± 0.28 mmHg·min·ml\(^{-1}\) for the young and 1.03 ± 0.31 mmHg·min·ml\(^{-1}\) in the middle age. Resistance significantly decreased after sauna exposure in both age groups to 0.18 ± 0.05 mmHg·min·ml\(^{-1}\) in the young and 0.16 ± 0.08 mmHg·min·ml\(^{-1}\) in the middle-aged and remained lower through 30 min of recovery (P < 0.001; Figure 2C).

Baseline flow in superficial femoral artery was 207.78 ± 81.14 ml·min\(^{-1}\) in the young and 208.68 ± 30.4 ml·min\(^{-1}\) in the middle-aged participants. As reported in Figure 3D, there was a main effect of time (P < 0.001) and no effect of age (P = 0.620) or time-by-age-group interaction (P = 0.781). Superficial femoral artery flow increased to 613.71 ± 229.84 ml·min\(^{-1}\) in the young and 546.17 ± 176.81 ml·min\(^{-1}\) in middle-aged participants and remained elevated after 30-min recovery (P = 0.009). There was a main effect of time (P < 0.001) and no effect of age (P = 0.190) or time-by-age-group interaction (P = 0.145) on shear rates in the superficial femoral artery (P = 0.145) indicating similar increases in superficial femoral artery shear rates in both groups. Baseline shear rates were 80.63 ± 44.30 sec\(^{-1}\) in the young and 61.99 ± 22.08 sec\(^{-1}\) in the
middle-aged group. Shear rates increased to 227.60 ± 123.37 sec\(^{-1}\) and 154.44 sec\(^{-1}\) after sauna in the young and middle-aged groups, respectively, and remained elevated above baseline 30 min into recovery (P = 0.015). Baseline resistance was 0.49 ± 0.21 mmHg-min-ml\(^{-1}\) in the young and 0.48 ± 0.06 mmHg-min-ml\(^{-1}\) in the middle-aged group. A main effect of time (P < 0.001) was observed with no effect of age (0.472) or time-by-age-group interaction (P = 0.688). Resistance in the superficial femoral artery decreased to 0.16 ± 0.08 mmHg-min-ml\(^{-1}\) in the young and 0.18 ± 0.07 mmHg-min-ml\(^{-1}\) in the middle-aged following sauna.

**Vascular Function**

There was a main effect of time (P = 0.014) and no effect of age (P = 0.914) on FMD percent dilation, indicating that FMD percent dilation increased following heat exposure and that the change did not differ between groups (post-FMD 7.13 ± 3.28 young and 7.31 ± 3.60 middle-aged). Interestingly, post hoc analysis indicated that the change in FMD percent dilation tended to increase in each group (young: P = 0.060, middle-aged: P = 0.110), but did not independently reach the threshold for statistical significance. No main effect of time (P = 0.624), age (P = 0.654) or interaction (P = 0.570) were observed for total shear during reactive hyperemia. When normalized by total shear rate, FMD percent dilation tended to increase over time (P = 0.067), while no effect of age (P = 0.881) or interaction (P = 0.854) were observed. Brachial and superficial femoral artery heat-induced dilations were strongly correlated to baseline endothelial function in the young (R = 0.793, R = 0.798). Interestingly, this relationship was not evident in the middle-aged group (R = 0.223, R = 0.377; Figure 4).

**Discussion**

The purpose of this study was to quantify the vascular response to an acute bout of sauna heating in young and middle-aged adults. The study protocol was largely influenced by
prominent epidemiological data (11, 12) showing reduced cardiovascular risk in chronic sauna usage at 80–100 °C and for a duration of 19 min or more. We found that the response to a single sauna session was almost identical between young individuals and middle-aged individuals.

Hemodynamic Responses to Acute Sauna Heating in Young and Middle-Aged

The thermoregulatory response to passive heat stress results in an increase in blood flow to muscle and skin following rises in core temperature (23). This redistribution of blood flow increases the frictional force in the vasculature exerted by the blood, or shear stress, which stimulates nitric oxide (NO) production from the endothelium (24). Shear stress is a known stimulus for nitric oxide mediated dilation and is also considered a primary stimulus for vascular adaptation in response to heat (7, 13). The temperature and shear rate response to sauna, as well as additional heating modalities, is not well quantified.

As illustrated in Figure 1, core and muscle temperature increases were identical between groups. With identical temperature responses, we then quantified the shear response between young and middle-aged individuals. Shear has been shown to be a primary stimulus for adaptation in response to heat. As expected, 40 min of sauna at 80 °C elicited a significant increase in shear rate in both the young and middle-aged participants (Figure 3E & 3F). We observed significant increases in shear rate and blood flow in the brachial and superficial femoral arteries after 20 min of sauna, followed by even greater increases following 40 min of exposure. Shear rate also remained elevated through 30 min of recovery in the brachial artery.

Earlier studies have examined the shear rates following acute sauna exposures of shorter durations and lower temperatures. However, these studies only report changes in shear rates following 30 min of exposure. Consequently, much smaller increases were reported, due to the delay in measurement following exposure (17, 21). We found peak shear following sauna
increased approximately 200%, which remained elevated through 30 min of recovery. These changes in shear rates closely follow those reported in studies using lower limb and whole-body hot water immersion (6, 15, 25). Our data and existing data show a significant shear stimulus is present during the heat exposure and through 30 min of recovery.

Some have proposed using heat therapy to improve vascular health in aging. We found no differences between the young and middle-aged participants in the magnitude of shear rate increase in both the brachial and superficial femoral arteries following sauna, indicating that the middle-aged group received the same shear stimulus as the young. This contrasts with the changes in shear rates reported in limb-specific passive heating. Romero et al., (6) showed a blunted increase in shear in older adults when compared to healthy young adults. While Romero observed significant elevations in shear rate, the response was smaller than that observed in the young adults. These discrepancies between Romero et al. and the present study could be explained by a younger age population (middle age vs older adults) and no baseline differences in endothelial function, i.e., FMD in the present study.

The effect of age on the vascular system has also been shown to be limb-specific (26). Blood flow and shear rate measurements were recorded in both the brachial and superficial femoral artery, allowing for an understanding of potential differences in response between upper and lower extremities. We observed no differences in the magnitude of shear increase between the brachial and superficial femoral artery between young and middle-aged subjects. These data suggest whole-body heat therapy would likely have systemic benefits.

**Arterial Blood Pressure Responses to Acute Heat**

A reduction in blood pressure can reduce the risk of cardiovascular disease and all-cause mortality, irrespective of starting blood pressure (27). Additionally, high SBP is strongly
correlated with endothelial dysfunction, with the gradient of diminished FMD detectible at nonhypertensive levels (28). Heat therapy has been proposed as a potential intervention for the prevention and treatment of hypertension. For example, Zaccardi et al. (29) reported a 46% reduction in incident hypertension over 24 years in men who used sauna four or more times a week. Reductions in BP have also been reported following acute heat exposures (30, 31). For example, Lee et al. (30) showed SBP reductions in men with existing cardiovascular risk factors following 30 min of sauna that remained lower for 30 min of a recovery period.

As illustrated in Figures 3A and 3B, we saw no change in SBP or DBP in young participants following exposure, likely due to lower baseline values (Table 1). However, in agreement with previous heat therapy interventions, we observed an ~10% reduction in SBP and ~15% reduction in DBP in the middle-aged participants following sauna exposure (30, 31). This transient period of reduced BP persisted up to 30 min of the recovery period, providing approximately 60 min of reduced blood pressure. Daily exposures to sauna may offer daily reductions in blood pressure. However, it is unknown if acclimatization alters the postexposure hypotensive response.

The pressure reduction associated with acute heat exposure is likely the result of a decreased total peripheral resistance (32). Total peripheral resistance was not measured in the present study, however, a decrease in resistance in the brachial and superficial femoral arteries (Figures 2C & 2D) supports a possible systemic decrease in resistance. This transient reduction in BP and resistance following an acute bout of sauna could contribute to the reduction in cardiovascular risk associated with repeated heat therapy exposure (29, 31).
**Vascular Function Following Acute Heat**

The ability of the vasculature to dilate is altered with aging and is associated with increased cardiovascular risk (1, 33). Improved vascular function has been reported in healthy and diseased populations in acute and chronic heat therapy interventions (6, 9, 16, 34). Whether middle-aged individuals show the same dilatory response to heat therapy is unknown. Our study population showed no baseline differences, suggesting similar functional capacity in both healthy young and healthy middle-aged adults. When all subjects were grouped together, FMD increased after sauna treatment (Pre: 5.34 ± 2.885; Post: 7.21 ± 3.32; P = 0.014); however, when compared between age groups, the effect is no longer significant, likely due to small sample size.

Detecting changes in vascular function following exposure to heat is highly influenced by the duration of the recovery period. Gravel et al. (17) showed no change in FMD response in older adults following a single sauna exposure. However, post measures were taken 30 min after exposure, likely confounding results with postheat hyperemia. We tested vascular function in the brachial artery following 90 min of recovery. This recovery period was sufficient time for blood flow, shear rates, BP, and muscle temperatures to return to baseline. Core temperature, however, was still above baseline after 90 min of recovery. As a result, we feel confident that the observed increases in FMD accurately represent the acute effect of heat on the vasculature and not merely the effect of a sustained hyperemic response in muscle blood flow following heat exposure.

One interesting outcome was the relationship between heat-induced dilation of the brachial artery and baseline FMD percent dilation in the young group that was not evident in the middle-aged (Figure 4). Differences in dilatory mechanisms could influence this discrepancy between the age groups. While this study is not designed to determine any mechanistic differences in vasodilator function, there is literature to support the upregulation of other
compensatory pathways in aging, in conjunction with decreases in NO mediated dilation. Specifically, Serviente et al. (35) showed an upregulation in K+ channel-dependent vasodilator function in healthy older adults. This upregulation is thought to represent compensatory adaptation that allows for maintenance of endothelium-dependent vasodilation in the presence of reduced NO bioavailability. We found no difference between groups in baseline FMD, which has been shown to be highly NO dependent (36). Thus, it is possible that upregulation of compensatory dilator mechanisms occurs before reductions in NO bioavailability are detected by FMD. With age-related declines in NO-mediated vascular function seeing a dramatic increase around the fourth and fifth decades in men and women, respectively, the effects of heat on this specific age population shows strong preventative value.

**Implications for Future Protocols**

The heating protocol we used was based upon epidemiological data showing clear cardiovascular benefits to sauna bathing at 80 °C for at least ~20 minutes. To maximize the effect, we used a protocol of 2 × 20-min exposures at 80 °C separated by 10 min of recovery in the supine position at 23 °C. Significant increases in core temperature were observed within the first 20-min exposure of about 1 °C for both age groups (Figure 1A). As expected, core temperature continued to rise during the second exposure, however, at a lesser magnitude than the first exposure (about 0.5 °C) with peak core temperature remaining less than 38 °C. The change in core temperature is in agreement with prior heat interventions with the change in core temperature being ~1.5 °C. Our recorded peak core temperature, however, is lower than what has previously been reported by hot water immersion (38.5–39 °C) (14, 37). This discrepancy could likely be explained by a difference in core temperature measurement. We reported a slightly lower baseline core temperature using esophageal temperature (36.29 ± 0.42 °C young and 36.11
± 0.29 °C middle-aged) as an estimate of body core temperature. Additionally, the hydrostatic gradient present in water immersion studies result in hemodynamic shifts, even in normothermic conditions, that could account for higher baseline values and, consequently, higher peak core temperature values following whole-body hot water immersion (38).

The peak core temperature recorded in the present study supports those of limb-specific passive heat therapy (6) that show vascular-specific adaptations can still be obtained at a lower core temperature threshold. Despite modest but significant increases in core temperature, trends for increase in vascular function were observed. This suggests that repeated passive heat therapy could still be effective with smaller increases in core temperature, like the ~1.0 °C increase observed after 20 min of sauna, supported by Laukkanen’s epidemiological data (11, 12). Thus, high core temperatures reportedly needed for heat acclimation (e.g., 38.5 °C) are not needed to achieve vascular adaptations. The likelihood of lower required core temperature thresholds may make passive heat therapy more feasible and accessible.

Conclusions

In conclusion, the vascular response to acute sauna heating in middle-aged individuals mirrors the response in young individuals. There is a significant increase in shear rate and blood flow in the brachial and superficial femoral arteries following acute sauna exposure that remain elevated through 30 min of recovery in the brachial artery. Significant SBP reductions are observed only in middle-aged individuals, likely a result of higher baseline values. Additionally, lower absolute body core temperatures reveal the possibility of vascular adaptations in response to lower heat stress. Future heat therapy studies may illicit meaningful cardiovascular benefits from lower magnitudes of chronic passive heat stress.
References


36. **Kooijman M, Thijsen DHJ, de Groot PCE, Bleeker MWP, van Kuppevelt HJM, Green DJ, Rongen GA, Smits P, Hopman MTE.** Flow-mediated dilatation in the


Table 1. Subject Characteristics

<table>
<thead>
<tr>
<th>Subject Characteristics</th>
<th>Young</th>
<th>Middle-Aged</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>24.9 ± 4.18*</td>
<td>55.75 ± 3.91*</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72.55 ± 10.54</td>
<td>79.71 ± 14.32</td>
</tr>
<tr>
<td>Body Fat %</td>
<td>27.2 ± 10.1</td>
<td>32.13 ± 6.88</td>
</tr>
<tr>
<td>Activity, steps per day</td>
<td>12109.63 ± 2676.48</td>
<td>12203.88 ± 4948.15</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Baseline Measurements</th>
<th>Young</th>
<th>Middle-Aged</th>
</tr>
</thead>
<tbody>
<tr>
<td>T Core, °C</td>
<td>36.29 ± 0.42 (n = 10)</td>
<td>36.11 ± 0.29 (n = 6)</td>
</tr>
<tr>
<td>T muscle, °C</td>
<td>35.48 ± 0.62 (n = 9)</td>
<td>35.33 ± 0.47 (n = 7)</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>119.4 ± 6.381*</td>
<td>135.13 ± 17.192 *</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>71.2 ± 9.24</td>
<td>81.62 ± 12.96</td>
</tr>
<tr>
<td>Brachial Artery Flow, ml·min⁻¹</td>
<td>123.2 ± 37.28</td>
<td>104.47 ± 35.06</td>
</tr>
<tr>
<td>Superficial Femoral Artery Flow, ml·min⁻¹</td>
<td>207.78 ± 81.14 (n = 8)</td>
<td>208.68 ± 30.4 (n = 7)</td>
</tr>
<tr>
<td>Baseline Flow Mediated Dilation, %</td>
<td>5.57 ± 2.48</td>
<td>5.09 ± 3.44</td>
</tr>
</tbody>
</table>

Values are means ± SD. Young n = 10 and middle-aged n = 8 unless otherwise noted. *indicates significant difference between groups.
Figure 1. Esophageal and Muscle Temperature Changes
Increase in esophageal temperature (A) for (n = 10 young, n = 6 middle-aged). Intramuscular temperature (B) (n = 9 young, n = 7 middle-aged). Data recorded at baseline, during 40-min sauna exposure (2 × 20-min sauna separated by 10-min recovery at 23 °C) and 90 min of recovery at 23° C. Data presented as mean with individual data points. T = different than baseline, all groups
Figure 2. Blood Pressure Response to Heat
Change in mean SBP (mmHg) (A) and DBP (mmHg) (B). Change in brachial artery resistance (C) and superficial femoral artery resistance (D). Data recorded at baseline, during 40-min sauna exposure (2 × 20-min sauna separated by 10-min recovery at 23 °C) and 90 min of recovery at 23 °C. Data recorded at baseline, during 40-min sauna exposure (2 × 20-min sauna separated by 10-min recovery at 23 °C) and 90 min of recovery at 23 °C.

T = different than baseline, all groups, t = lower than baseline, middle-aged, a = different from other age group.
Figure 3. Peripheral Hemodynamic Changes in Brachial and Superficial Femoral Artery
Change in brachial artery blood flow (ml·min⁻¹) (A), brachial artery diameter (cm) (n = 10 young, n = 8 middle age) (B), and brachial artery shear rate (s⁻¹) (C). Change in superficial femoral artery blood flow (ml·min⁻¹) (D), superficial artery diameter (cm) (E) (n = 8 young, n = 7 middle-aged), and superficial femoral artery shear rate (s⁻¹) (F). Data recorded at baseline, during 40-min sauna exposure (2 × 20-min sauna separated by 10-min recovery at 23 °C) and 90 min of recovery at 23 °C. Data presented as with individual data points.
T = different than baseline, all groups
Figure 4. Brachial Artery Heat-Induced Dilation and Vascular Function
Relationship between brachial artery heat-induced dilation (%) and baseline brachial artery FMD percent dilation (D), $R^2 = 0.63$ and $P = 0.006$ in the young and $R^2 = 0.05$ and $P = 0.631$ in middle-aged participants.