Sauna Exposure Leads to Improved Arterial Compliance: Findings from a Non-Randomized Experimental Study

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This study was supported by the Tekes, the Finnish Funding Agency for Technology and Innovation, Helsinki, Finland

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ABSTRACT

Background: Heat therapy has been suggested to improve cardiovascular function. However, the effects of hot sauna exposure on arterial compliance and the dynamics of blood flow and pressure has not been well documented. Thus, we investigated the short-term effects of sauna bathing on arterial stiffness (AS) and hemodynamics.

Design: Experimental non-randomized study

Methods: There were 102 asymptomatic participants (mean, 51.9 years) who had at least one cardiovascular risk factor. Participants were exposed to a single sauna session (duration: 30 minutes; temperature: 73°C; humidity: 10-20%). Pulse wave velocity (PWV), augmentation index (AIx), heart rate (HR), blood pressure (BP), mean arterial pressure (MAP), pulse pressure (PP), augmented pressure (AP) and left ventricular ejection time (LVET) were assessed before, immediately after, and 30 minutes after a single sauna session.

Results: Sauna bathing led to reductions in PWV, BP, MAP and LVET. Mean PWV value before sauna was 9.8 m/s and decreased to 8.6 m/s immediately after sauna ($p<0.001$ for difference), and was 9.0 m/s after the 30-minute recovery period ($p<0.001$ for ANOVA). SBP was 137 mmHg before sauna, decreasing to 130 mmHg after sauna ($p<0.001$), which remained sustained during the 30-minute recovery phase ($p<0.001$ for ANOVA). After a single sauna session, diastolic blood pressure (DBP) decreased from 82 to 75 mmHg, MAP from 99.4 to 93.6 mmHg and LVET from 307 to 278 ms^{-1} ($p<0.001$ for all differences). Pulse pressure was 42.7 mmHg before the sauna, 44.9 mmHg immediately after sauna, and reduced to 39.3 mmHg at 30-minutes recovery ($p<0.001$ for ANOVA). Heart rate increased from 65 to 81 bpm^{-1} post-sauna ($p<0.001$); there were no significant changes for AP and pulse pressure amplification (PPa).

Conclusion: This study shows that PWV, SBP, DBP, MAP, LVET and DT decreased immediately after a 30-minute sauna session. Decreases in SBP and LVET were sustained during the 30-minute recovery phase.

Key words: Arterial stiffness, pulse wave velocity, sauna bathing, heat therapy, experimental study

INTRODUCTION

Heat therapy has many benefits for the human physiology. The heat stimulates the sensory receptors in the skin, decreasing transmissions of pain signals to the brain to relieve discomfort, and increases the flow of oxygen and nutrients to the muscles, helping to heal damaged tissue, through thermoregulatory mechanisms and pathways.[1,2] Previous studies have also shown an association between controlled heat stress and augmented cardiovascular function.[3-5]

In line with these findings, it has also been suggested that heat therapy may improve microvascular function.[6] There are different ways to apply heat therapy, such as using dry heat or warm water immersion.[1] However, the use of dry heat via hot sauna exposure has been gaining popularity, especially after it has been shown to alleviate acute and chronic conditions such as asthma, headaches, hypertension, incidence of colds and other related broncho-constructive disorders.[7-9] Indeed, our prospective population-study has also demonstrated that there is a strong association between sauna exposure and a lowered risk for CVD outcomes[10]. In addition, it has been suggested that sauna therapy may improve vascular compliance, which has been previously documented in subjects with coronary heart disease (CHD) risk factors,[11] although the effects of sauna bathing on arterial compliance has not been reported.

Arterial stiffness (AS) has been recognized as a risk factor for atherosclerotic CVDs and mortality.[12,13-15] Although resting blood pressure (BP) levels is considered a close surrogate measure to AS, studies have shown that AS is a more accurate indicator of vessel function.[16] Consistently, some studies have shown that AS is considered to be a stronger risk factor for cardiovascular mortality than brachial BP alone[16,17] and, therefore, increased AS is independently associated with adverse cardiovascular events.[18] However, to our best knowledge, no experimental studies on the acute effects of sauna bathing on AS and hemodynamic parameters have been conducted.

Pulse pressure (PP) and mean arterial pressure (MAP) have been well documented as markers of cardiovascular risk in different clinical settings[19,20] Pulse pressure is a pulsatile

component of the BP curve as opposed to MAP, defined as the difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP), and has been demonstrated to predict cardiovascular mortality in the general population.[21,22]

The current gold standard for assessing AS is through the measurement of pulse wave velocity (PWV). However, a change in PWV could be supplemented via alterations in vascular pressure and/or left ventricular ejection time (LVET). This reflects a natural change in arterial wall properties without hemodynamic shifts, as the pulsatile nature of blood flow in large arteries are mainly regulated by arterial distensibility, ventricular function and arterial pressure. Better understanding of these underlying mechanisms may help in explaining the acute physiological responses of the vascular system in maintaining homeostasis against heat exposure.

Therefore, using a non-randomized experimental study, we aimed to investigate the acute hemodynamic and vascular responses among asymptomatic participants with cardiovascular risk factors and their recovery profiles after 30 minutes of sauna bathing. This study will further clarify if sauna bathing leads to significant alterations in vascular and hemodynamic function, including changes in PWV, MAP, PP and LVET among this population.

METHODS

Participants

A total of 102 (n) participants were recruited from the city of Jyväskylä, Central Finland region, through the local out-of-hospital health care center. The study group consisted of asymptomatic participants (no cardiovascular symptoms) with at least one cardiovascular risk factor, such as a history of smoking, dyslipidemia, hypertension, obesity, diabetes, or family history of CHD. All participants with acute or diagnosed CVD were excluded. Prior to the participation in the study, all participants were informed about the research purposes and measurement procedures, and were screened by a cardiac specialist. The research protocol and study design were approved by the institutional review board of the Central Finland Hospital District ethical committee, Jyväskylä, Finland (Dnro 5U/2016). All study participants provided written informed consent prior to the inclusion in the study. The study was performed as stated by the declaration of Helsinki.

Clinical Examination

A clinical evaluation with baseline data collection was conducted on a separate day prior to the experiment. All baseline and sauna measurements were conducted during June and November 2016. During the screening visit, medical history, physical examination, blood lipid levels and resting ECG were assessed. A maximal exercise test was conducted on a separate day to determine their level of fitness. Resting blood pressure was estimated as the mean of two measurements obtained while the participant was in supine with a standardized measurement protocol. Body mass index (BMI) was calculated by dividing weight in kilograms by the square of height in meters.

Sauna Exposure

Sauna exposure in this experiment was based on a traditional Finnish sauna, which is characterized by air with a relative humidity of 10-20% and high temperature.[10] In our study, relative humidity of the air was 15-20% and the temperature over the entire course of the experiment was 73 ± 2 °C. The sauna temperature in each room was recorded continuously as 10 second averages using a 2-channel internal temperature sensor designed by Harvia Oy, Finland. Sauna exposure was based on typical Finnish sauna bathing sessions; the total duration was 30 minutes, and it was interspersed with a short, two-minute shower at 15-minute intervals. Participants wore their own swim suits during the sauna session and there were separate sauna rooms for women and men; the sauna rooms were similar in the terms of space, humidity, temperature and air conditioning.

Before the sauna experiments, all participants received written and verbal instructions informing them to avoid meal, caffeine and smoking within 3 hours of the measurement, and that speaking and sleeping during the measurement was prohibited. All measurements were taken in a quiet room with a stable temperature (21 °C) on the right side of the body in the supine position. Participants were supervised by a physician and were allowed to leave the sauna at any time they felt uncomfortable. All participants underwent the recommended sauna protocol successfully. They were instructed to rest in a designated waiting lounge at room temperature (temperature 21 °C) for the whole recovery period after the immediate post sauna measurement. Water intake was ad libitum. Study participants were allowed to drink 500 ml still water during sauna and recovery period, according to guidance of the local ethical committee

Outcome definitions

Assessment of Arterial Stiffness Parameters

The measurement of AS during this experiment followed closely established guidelines.[23] Measurements of AS are performed by using The PulsePen device (DiaTecne s.r.l., Milan, Italy; www.pulsepen.com) which is composed of one tonometer and an integrated

electrocardiogram (ECG) unit. The PulsePen is made of a pressure probe the size and shape of a ball point pen with a built-in acquisition device that serves to non-invasively detect the pressure waveform by means of applanation tonometry. The unit is connected to the computer by means of an optical fiber that ensures the electromagnetic isolation for the patient undergoing the test. Data analysis is performed by specially designed software at a sample rate of 500 Hz.[24] The device software does not validate measurements if the difference between BP or heart rate values taken at the time of carotid and peripheral artery recordings was >10%. All measurements before and after the sauna were taken by a single trained operator of the tonometer. The same transit distances measured during baseline clinical evaluation were used throughout the experiment for consistency and reliability.

PWV was measured by recording carotid and peripheral (femoral) waveforms in rapid succession at a sample rate of 1 kHz, and defined as the transit distance between the measuring sites divided by the time delay between the distal pulse and proximal pulse wave, using the ECG trace as reference. Transit distances were assessed by body surface measurements using a tape measure from the suprasternal notch to each pulse recording site (carotid and femoral). Direct carotid to femoral measurement was adjusted to 80% (common carotid artery – common femoral artery x 0.8) for the calculation of PWV as recommended by current guidelines.[25] Transit time was defined as the difference between the delay of the distal pulse wave to the R wave belonging to the ECG qRs complex and the delay of the proximal pulse wave to the R wave belonging to the ECG qRs complex. The pulse wave delay was determined by calculating the time elapsed from the peak of the R wave and the "foot" of the pressure pulse wave.

The pressure values recorded by tonometry were calibrated to the BP values obtained at the brachial artery; where they were assigned to the appropriate pixels and the values for MAP and all other pressure-related parameters were re-established. The values deduced by the software apply the established concept that the MAP remains unchanged in the tract from the aorta to the peripheral arteries. MAP was calculated by the software as DBP + 1/3(SBP - DBP).[24] LVET was determined as the difference between heart period and diastolic time. Assessments of various AS parameters are shown in **Figure 1**.

Left ventricular ejection time (LVET), diastolic time (DT), and augmentation index (AIx) were obtained from the carotid pressure waveform analysis. The point corresponding to the end of LVET and the beginning of DT is identified by the dicrotic notch in the carotid pulse waveform. This point is automatically estimated by the PulsePen software.[26] Augmentation index is a parameter which provides an indication of the contribution of reflected waves to the total PP and was defined as the difference between the second and first systolic peak on arterial pulse waveform and was expressed as a percentage of central pulse pressure ((AIx) = AP/PP x 100).[27]

Assessment of Blood Pressure

Supine brachial systolic and diastolic blood pressures (SBP and DBP respectively) were obtained using Microlife BP A200 (Microlife Corp., Taipei, Taiwan) for better sensitivity and accuracy[28].Two sequential readings were measured and the mean values were used. Participants rested in the supine position for 10 minutes before PWV was measured at baseline. However, due to the nature of the study, AS was measured immediately and 30 minutes after the sauna exposure without having laid supine for 10 minutes.

Statistical Analyses

Data are presented as means ± standard deviations (SDs) and frequencies as appropriate. Normally distributed data were analyzed for within-group (time) changes with a repeated measure analysis of variance (ANOVA). Normality was checked using the Shapiro-Wilk test as well as through observing the Q-Q-plots. Non-normally distributed data was log-transformed to achieve normality and thereafter analyzed. The level for significance was set at $p \leq 0.05$. Within-group differences between before vs. immediately after, before vs. 30 minutes recovery and immediately after vs. 30 minutes recovery from sauna were analyzed using pairwise t-tests, and p-values were corrected for Bonferroni by multiplying all pairwise p-values with the number of comparisons conducted for each variable.

All statistical analyses were carried out with Stata version 14.1 (Stata Corp, College Station, Texas) and IBM SPSS Statistics v.22 software (IBM Corporation, Armonk, New York, USA).

RESULTS

Characteristics of participants

In this study population, there were 56 male and 46 female participants. The characteristics of the participants are shown in **Table 1**. The mean (SD) age and BMI of participants was 51.9 (SD 9.2) and 27.9 (kg/m^2) (4.7) respectively and the most common underlying clinical conditions were dyslipidaemia (63%) and hypertension (14%). Body weight of the participants did not change statistically significantly during the sauna session (pre-sauna: 82.7 (16.0) kg vs post-sauna: 83.0 (15.7) kg).

Changes in arterial stiffness

Table 2 shows changes in measures of AS after 30 minutes of sauna exposure. Mean PWV value before sauna was 9.8 m/s, decreased significantly to 8.6 m/s immediately after sauna, and was 9.0 m/s after a 30 minute recovery period ($p<0.001$ for ANOVA). Values of AIx and LVET decreased statistically significantly immediately after sauna; for LVET, the decrease was still significant after 30 minutes recovery. However, AIX recovered to its initial levels after 30 minutes of recovery (**Table 2**). Diastolic time decreased significantly due to sauna exposure from 635.1(±115.1) to 494.6 (±113.0) m/s, and increased back to the pre-sauna level after 30 minutes recovery.

Changes in arterial pressure, augmented pressure, pulse pressure and pulse pressure amplification

Changes in measures of central hemodynamics are shown in **Table 3**. MAP decreased significantly from 99.4 (± 15.0) mmHg before sauna to 93.6 (± 10.3) mmHg immediately after

sauna, but MAP recovered at 30 minutes after sauna to 95.9 (± 14.2). However, changes from immediately after sauna to 30 minutes recovery in MAP were not statistically significant. Pulse pressure decreased significantly from after the sauna exposure to the recovery period, being 44.9 (± 9.8) mmHg immediately after sauna, and reducing to 39.3 (± 8.3) mmHg at 30-minutes recovery (**Table 3**). There were no statistically significant changes for AP and PPa at all time points during the sauna bathing.

Changes in brachial blood pressure and heart rate

Sauna bathing had significant effects on BP (**Table 4**). SBP was 137 mmHg before sauna, which decreased significantly to 130 mmHg immediately after sauna ($p<0.001$ for difference), and remained at 130 mmHg after the 30 minutes recovery ($p<0.001$ for pre- and post 30-minutes sauna difference). The corresponding values for DBP were 82 mmHg, 75 mmHg, and 81 mmHg, respectively **(Table 4).** Heart rate increased from 65 bpm to 80 bpm as a result of the 30 minutes' sauna exposure but returned to 66 bpm after the 30 minutes' recovery period **(Table 4).**

DISCUSSION

This study showed that heat exposure of sauna has acute effects on AS among participants who have at least one cardiovascular risk factor. The main finding from this study was that sauna bathing has several important effects on vascular and hemodynamic function. We found that sauna bathing leads to lower levels of PWV, AIx and diastolic time, with short-term decreases in both MAP and DBP, which were observed immediately after sauna. On the other hand, levels of SBP and LVET were sustained until the end of 30-minute recovery phase. The indices of AS including PWV, AIx, MAP and LVET changed in the same direction during the sauna bathing. Heart rate and PP yielded a short-term response in the opposite direction; showing an increase immediately after the sauna exposure before returning to resting levels or even below the baseline level, while no notable changes were found for AP and PPa.

PWV was modulated positively after 30 minutes' heat exposure while the reduction in SBP levels remained significant when comparing baseline values to 30 minutes' recovery data. It has been previously shown that PWV is more closely related to left ventricular systolic function than to heart period.[26] The association between PWV and LVET can be attributed to myocardial function, where the two share an inverse relationship. The decrease in LVET in parallel with PWV was therefore an interesting finding.

As postulated by Salvi and coworkers,[26] when there is a decrease in systolic ejection time, the time taken by the left ventricle to do mechanical work is shortened, thus resulting in an increase in BP and velocity of travelling waves, which in turn leads to an increase in PWV. However, the opposite was found as a result of sauna exposure. Nonetheless, the authors concluded that PWV may be a determinant of LVET; this is supported by the results from our study in that the changes in PWV and LVET were similar immediately after sauna and at 30 minutes recovery period.

These findings are further supported by the observation that changes in cardiac afterload did not affect the duration of left ventricular ejection.[29] Secondly, LVET could be the time interval of the cardiac cycle responsible for the relationship between PWV and HR.[30] In this study, LVET decreased from 307 to 278 ms^{-1} after sauna exposure and recovered towards initial

levels after a 30 minute period, which may at least partly indicate a higher HR after sauna. Indeed, results from this experiment showed an acute increase in HR after the sauna exposure. This is consistent with the changes in DT due to sauna bathing, where DT was decreased immediately after sauna and returned to initial levels during the recovery.

The increase in HR seen in our study did not have a significant contribution to PPa, although it has been postulated that changes in HR and LVET would mediate changes in AP and AIx, and thus modifying PPa level.[31] Our results showed that acute sauna exposure increased HR and decreased LVET, which in turn resulted in a significant decrease in AIx, even though AP showed no change. This may be indicative of the contribution of HR to wave reflection and AIx.[32] Augmentation index is commonly accepted as a measure of enhancement of central aortic pressure by a reflected pulse wave and is considered to measure a different aspect of AS.[27] This study showed that AIx decreased significantly after sauna, which may be associated with peripheral vascular changes (vasodilation) after sauna bathing, although it is likely that the increase in PP mediated the decrease in AIx.

Interestingly, the results from our study showed a transient increase in PP with no significant concomitant decrease in PPa. High PP has been shown to be an independent predictor of mortality in patients with heart failure[33] and has been postulated to be a significant predictor of CHD risk.[34,35] In addition, a reduction in PPa has been proposed as a potential mechanical biomarker of global arterial function and cardiovascular risk.[31] The increase in PP and HR through sauna exposure also did not coincide with an increase in PPa as suggested by other studies[32]. However, in this study, the decreases in PP were found after a 30 minutes recovery period, suggesting beneficial cardiovascular effects of a 30 minute sauna exposure.

In our current study setting, sauna bathing was able to acutely reduce BP and MAP. Decreases in BP and MAP, is a clinically important finding from this study showing the effect of sauna on BP parameters. Because hot sauna bathing can produce acute vasodilation which leads to a significant drop in BP,[36] longer term sauna bathing could potentially lead to a reduction in systemic BP, which is supported by our recent findings on regular sauna bathing and a lowered risk of hypertension.[37] In patients with slightly elevated BP, a single sauna session produces positive lowering effects on systemic BP.[38] These findings are comparable

with exercise training and blood pressure-related studies[39,40] investigating the acute effects of BP changes. Decreases in systemic BP with a concomitant increase in HR seen in the current study is likely due to sympathetic and parasympathetic regulations of the heart. Furthermore, passive heating has been shown to reduce PWV.[41] In addition to increased body temperature and its positive effects, sauna bathing may have decreased plasma volume which is further related to changes in hemodynamic parameters such as decreased AS and systemic BP. Therefore, it is possible that heat therapy such as sauna may reduce AS via body dehydration.[42] Therefore, in assessment of PWV during sauna sessions, body fluid balance hydration status should be taken into account which includes ensuring adequate hydration status.

Strengths

Our study was based on a large number of participants, which provided adequate pre-defined power to assess changes in hemodynamic and vascular responses after sauna bathing. The assessment of cardiovascular parameters including BP and AS was performed using standard measurement protocols[24,43]. The measurement of the PWV is a simple and rapid way to assess the compliance of great arteries. An expert operator may assess PWV data in 5-8 minutes (including the insertion of patient's data in the computer). This allowed us to collect data that represented the respective time points more accurately. Arterial tonometry with simultaneous ECG was obtained with the use of a commercially available tonometer that has been well validated previously.[25,44,24] The measurement of the PWV is a simple, non-invasive and rapid way to assess the compliance of great arteries. An expert operator may assess PWV data quickly including the insertion of patient's data in the computer and measurement protocol is convenient for the subjects. In addition, although the assessment of AS has been documented to have reasonable levels of reproducibility, with an intra-observer coefficient of variation (CV) of 4.8% and interobserver CV of 7.3%, our utilization of a single trained operator throughout the whole study adds more reliability to the results. Sauna bathing is a safe activity in populations with cardiovascular risk factors and it has been shown that acute exposure to

Finnish sauna and cold-water immersion causes haemodynamic alterations in chronic heart failure patients without serious adverse events

Limitations

The sauna intervention was short term and we employed a before- and after-design without the use of a control group as this study was designed to explore the effects of a single sauna session. Based on data from the non-randomized study focusing on changes in AS, many vascular system alterations appear to be functional and transient due to a single sauna session. However, structural changes such as alterations in the elastin-collagen fibers and re-organization of the extracellular matrix of the arterial wall cannot be discounted, and may still occur with long-term regular sauna exposure. This is an area that warrants further research as the sauna remains as one of the more accessible, convenient, and safe modes of heat exposure that can provide possible cardiovascular benefits. Further studies utilizing a similar intervention method and longer-term randomized controlled trials are needed in order to gain a better perspective of the effects of sauna (heat stress) on the cardiovascular system and its overall health effects. Although the duration and method used for the sauna exposure mimicked a typical Finnish sauna, we did not measure PWV over the entire duration of recovery to track its time course profile in this population. As a result, we do not have complete information of when PWV and other related AS indices recovered to the baseline level. The diversity of the participants included in the study may have influenced the results, in that the youngest participant was 32 years of age, while the oldest was 75 years. In addition, the body composition of the participants also varied greatly within genders, ranging from 23.8 to 31.0 kg/m^2 for women, and between 25.1 to 30.6 kg/m^2 for men. However, the size of the study population was large enough to show significant hemodynamic and vascular effects of sauna in population with risk factors for CVD, and therefore, the results may be generalized to a similar patient population. Finally, though the target temperature for sauna exposure in our experiment as set by the sauna meter was 84 °C, the mean temperature recorded by the internal temperature sensor was 73 ± 2 °C, which may seem lower for a typical Finnish sauna session. However, this is usually the case for typical sauna sessions; when sauna

temperatures are set between 80-90 °C, the true temperature within the sauna room is usually lower. This was evident because we employed a reliable 2-channel internal temperature sensor which recorded the room temperature continuously as 10 second averages.

Conclusions

This novel study indicated that sauna bathing leads to improvements in cardiovascular function, including improved arterial compliance and decreased systemic BP. The study shows that 30 minutes of heat exposure in the sauna leads to positive changes in PWV and other arterial-related indexes. Further research is needed to show whether sauna bathing combined with physical exercise may produce similar or pronounced effect on AS parameters.

Authorship

EL, TL, SK, HK, PW, FZ and JL contributed to the conception and design of the work. EL, TL, SK, FZ, and JL contributed to the acquisition, analysis, or interpretation of data for the work. EL, TL, SK, FZ and JL drafted the manuscript. EL, TL, SK, HK, PW, FZ and JL critically revised the manuscript. All gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

Funding

This study was supported by the Tekes, the Finnish Funding Agency for Technology and Innovation, Helsinki, Finland.

Conflict of Interest: none declared

Figure legends

Figure 1. Arterial stiffness, hemodynamics and vascular parameters. (Figure is modified from Salvi et al) [26]

References

1. Shibasaki M, Umemoto Y, Kinoshita T, et al. The role of cardiac sympathetic innervation and skin thermoreceptors on cardiac responses during heat stress. *Am J Physiol Heart Circ Physiol.* 2015;308(11):H1336-1342.

2. Gagnon D, Schlader ZJ, Crandall CG. Sympathetic activity during passive heat stress in healthy aged humans. *J Physiol.* 2015;593(9):2225-2235.

3. Crandall CG, González-Alonso J. Cardiovascular function in the heat-stressed human. *Acta Physiol (Oxf).* 2010;199(4):407-423.

4. Brunt VE, Howard MJ, Francisco MA, Ely BR, Minson CT. Passive heat therapy improves endothelial function, arterial stiffness and blood pressure in sedentary humans. *J Physiol.* 2016;594(18):5329-5342.

5. Radtke T, Poerschke D, Wilhelm M, et al. Acute effects of Finnish sauna and cold-water immersion on haemodynamic variables and autonomic nervous system activity in patients with heart failure. *Eur J Prev Cardiol.* 2016;23(6):593-601.

6. Brunt VE, Eymann TM, Francisco MA, Howard MJ, Minson CT. Passive heat therapy improves cutaneous microvascular function in sedentary humans via improved nitric oxide-dependent dilation. *J Appl Physiol (1985).* 2016;121(3):716-723.

7. Ernst E, Pecho E, Wirz P, Saradeth T. Regular sauna bathing and the incidence of common colds. *Ann Med.* 1990;22(4):225-227.

8. Preisler B, Falkenbach A, Klüber B, Hofmann D. [The effect of the Finnish dry sauna on bronchial asthma in childhood]. *Pneumologie.* 1990;44(10):1185-1187.

9. Kanji G, Weatherall M, Peter R, Purdie G, Page R. Efficacy of regular sauna bathing for chronic tension-type headache: a randomized controlled study. *J Altern Complement Med.* 2015;21(2):103-109.

10. Laukkanen T, Khan H, Zaccardi F, Laukkanen JA. Association between sauna bathing and fatal cardiovascular and all-cause mortality events. *JAMA internal medicine.* 2015;175(4):542-548.

11. Imamura M, Biro S, Kihara T, et al. Repeated thermal therapy improves impaired vascular endothelial function in patients with coronary risk factors. *J Am Coll Cardiol.* 2001;38(4):1083-1088.

12. Mitchell GF. Arterial Stiffness and Wave Reflection: Biomarkers of Cardiovascular Risk. *Artery Res.* 2009;3(2):56-64.

13. Sakuragi S, Abhayaratna WP. Arterial stiffness: methods of measurement, physiologic determinants and prediction of cardiovascular outcomes. *Int J Cardiol.* 2010;138(2):112-118.

14. Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: a systematic review and meta-analysis. *J Am Coll Cardiol.* 2010;55(13):1318-1327.

15. Ben-Shlomo Y, Spears M, Boustred C, et al. Aortic pulse wave velocity improves cardiovascular event prediction: an individual participant meta-analysis of prospective observational data from 17,635 subjects. *J Am Coll Cardiol.* 2014;63(7):636-646.

16. Franklin SS. Beyond blood pressure: Arterial stiffness as a new biomarker of cardiovascular disease. *J Am Soc Hypertens.* 2008;2(3):140-151.

17. Vlachopoulos C, Alexopoulos N, Stefanadis C. Aortic stiffness: prime time for integration into clinical practice? *Hellenic J Cardiol.* 2010;51(5):385-390.

18. van Sloten TT, Schram MT, van den Hurk K, et al. Local stiffness of the carotid and femoral artery is associated with incident cardiovascular events and all-cause mortality: the Hoorn study. *J Am Coll Cardiol.* 2014;63(17):1739-1747.

19. Paultre F, Mosca L. Association of blood pressure indices and stroke mortality in isolated systolic hypertension. *Stroke.* 2005;36(6):1288-1290.

20. Safar ME, Boudier HS. Vascular development, pulse pressure, and the mechanisms of hypertension. *Hypertension.* 2005;46(1):205-209.

21. Benetos A, Gautier S, Labat C, et al. Mortality and cardiovascular events are best predicted by low central/peripheral pulse pressure amplification but not by high blood pressure levels in elderly nursing home subjects: the PARTAGE (Predictive Values of Blood Pressure and Arterial Stiffness in Institutionalized Very Aged Population) study. *J Am Coll Cardiol.* 2012;60(16):1503-1511.

22. Roman MJ, Devereux RB, Kizer JR, et al. Central pressure more strongly relates to vascular disease and outcome than does brachial pressure: the Strong Heart Study. *Hypertension.* 2007;50(1):197-203.

23. Tomlinson LA. Methods for assessing arterial stiffness: technical considerations. *Curr Opin Nephrol Hypertens.* 2012;21(6):655-660.

24. Salvi P, Lio G, Labat C, Ricci E, Pannier B, Benetos A. Validation of a new non-invasive portable tonometer for determining arterial pressure wave and pulse wave velocity: the PulsePen device. *J Hypertens.* 2004;22(12):2285-2293.

25. Van Bortel LM, Laurent S, Boutouyrie P, et al. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity. *J Hypertens.* 2012;30(3):445-448.

26. Salvi P, Palombo C, Salvi GM, Labat C, Parati G, Benetos A. Left ventricular ejection time, not heart rate, is an independent correlate of aortic pulse wave velocity. *J Appl Physiol (1985).* 2013;115(11):1610-1617.

27. Munir S, Guilcher A, Kamalesh T, et al. Peripheral augmentation index defines the relationship between central and peripheral pulse pressure. *Hypertension.* 2008;51(1):112-118.

28. Wiesel J, Arbesfeld B, Schechter D. Comparison of the Microlife blood pressure monitor with the Omron blood pressure monitor for detecting atrial fibrillation. *Am J Cardiol.* 2014;114(7):1046-1048.

29. Hamada M, Hiwada K, Kokubu T. Clinical significance of systolic time intervals in hypertensive patients. *Eur Heart J.* 1990;11 Suppl I:105-113.

30. Lantelme P, Mestre C, Lievre M, Gressard A, Milon H. Heart rate: an important confounder of pulse wave velocity assessment. *Hypertension.* 2002;39(6):1083-1087.

31. Townsend RR. Novel Uses of Office-Based Measures of Arterial Compliance. *Methodist Debakey Cardiovasc J.* 2015;11(4):219-222.

32. Wilkinson IB, Prasad K, Hall IR, et al. Increased central pulse pressure and augmentation index in subjects with hypercholesterolemia. *J Am Coll Cardiol.* 2002;39(6):1005-1011.

33. Schillaci G, Di Luzio S, Coluccini M, et al. A low pulse pressure is an independent predictor of mortality in heart failure: data from a large nationwide cardiology database (IN-CHF Registry). *Ital Heart J.* 2004;5(12):892-898.

34. Mitchell GF, Hwang SJ, Vasan RS, et al. Arterial stiffness and cardiovascular events: the Framingham Heart Study. *Circulation.* 2010;121(4):505-511.

35. Glasser SP, Halberg DL, Sands C, Gamboa CM, Muntner P, Safford M. Is pulse pressure an independent risk factor for incident acute coronary heart disease events? The REGARDS study. *Am J Hypertens.* 2014;27(4):555-563.

36. Hannuksela ML, Ellahham S. Benefits and risks of sauna bathing. *Am J Med.* 2001;110(2):118-126.

37. Zaccardi F, Laukkanen T, Willeit P, Kunutsor SK, Kauhanen J, Laukkanen JA. Sauna Bathing and Incident Hypertension: A Prospective Cohort Study. *Am J Hypertens.* 2017.

38. Ohori T, Nozawa T, Ihori H, et al. Effect of repeated sauna treatment on exercise tolerance and endothelial function in patients with chronic heart failure. *Am J Cardiol.* 2012;109(1):100-104.

39. Pescatello LS, Guidry MA, Blanchard BE, et al. Exercise intensity alters postexercise hypotension. *J Hypertens.* 2004;22(10):1881-1888.

40. Rezk CC, Marrache RC, Tinucci T, Mion D, Forjaz CL. Post-resistance exercise hypotension, hemodynamics, and heart rate variability: influence of exercise intensity. *Eur J Appl Physiol.* 2006;98(1):105-112.

41. Caldwell AR, Robinson FB, Tucker MA, et al. Effect of passive heat stress and exercise in the heat on arterial stiffness. *Eur J Appl Physiol.* 2017;117(8):1679-1687.

42. Caldwell AR, Tucker MA, Burchfield J, et al. Hydration status influences the measurement of arterial stiffness. *Clin Physiol Funct Imaging.* 2017.

43. Kunutsor SK, Laukkanen JA. Circulating active serum calcium reduces the risk of hypertension. *Eur J Prev Cardiol.* 2017;24(3):239-243.

44. Joly L, Perret-Guillaume C, Kearney-Schwartz A, et al. Pulse wave velocity assessment by external noninvasive devices and phase-contrast magnetic resonance imaging in the obese. *Hypertension.* 2009;54(2):421-426.

Table 1. Baseline characteristics	
Parameters (n = 100)	**Mean ± SD**
Age (years)	51.9 ± 9.2
Body weight (kg)	82.7 ± 16.0
Body mass index (kg/m^2)	27.9 ± 4.7
Systolic blood pressure (mmHg)	136.5 ± 16.2
Diastolic blood pressure (mmHg)	82.1 ± 9.6
Resting HR (bpm)	65.2 ± 10.4
Total cholesterol ($Chol_{tot}$; mmol/L)	5.4 ± 1.0
Low density lipoprotein (LDL; mmol/L)	3.0 ± 0.8
High density lipoprotein (HDL; mmol/L)	1.4 ± 0.4
Triglycerides (mmol/L)	2.0 ± 1.7

Table 2. Changes in arterial stiffness related outcomes

Parameters	Pre Mean (SD)	Post Mean (SD)	Post 30 min Mean (SD)	p-value (ANOVA)	Pairwise p-value for pre-post difference	Pairwise p-value for pre-post 30 min difference	Pairwise p-value for post-post 30 min difference
PWV (m/s)	9.8 ± 2.4	8.6 ± 1.6	9.0 ± 1.7	<0.001	<0.001	*N.S*	<0.001
AIx	9.8 ± 16.0	4.1 ± 15.8	7.8 ± 15.7	<0.001	<0.001	*N.S*	*N.S*
LVET (m/s)	307.4 ± 26.4	277.9 ± 37.8	299.0 ± 34.1	<0.001	<0.001	<0.001	<0.001
DT (m/s)	635.1 ± 115.1	494.6 ± 113.0	634.3 ± 121.2	<0.001	<0.001	*N.S*	<0.001

Note: PWV, pulse wave velocity; AIx, augmentation index; LVET, left ventricular ejection time; DT, diastolic time; N.S., non-significant

Table 3. Central hemodynamic variables

Parameters	Pre Mean (SD)	Post Mean (SD)	Post 30 min Mean (SD)	p-value for ANOVA	Pairwise p-value for pre-post difference	Pairwise p-value for pre-post 30 min difference	Pairwise p-value for post-post 30 min difference
MAP (mmHg)	99.4 ± 15.0	93.6 ± 10.3	95.9 ± 14.2	<0.001	<0.001	*N.S*	*N.S*
PP (mmHg)	42.7 ± 9.2	44.9 ± 9.8	39.3 ± 8.3	<0.001	*N.S*	<0.001	<0.001
AP (mmHg)	1.5 ± 0.9	1.6 ± 0.8	1.4 ± 0.9	*N.S*	*N.S*	*N.S*	*N.S*
PPa, (%)	28.8 ± 10.1	25.9 ± 13.4	27.5 ± 10.8	*N.S*	*N.S*	*N.S*	*N.S*

Note: MAP, mean arterial pressure; PP, pulse pressure; AP, augmented pressure; PPa, pulse pressure amplification; N.S., non-significant

Table 4. Heart rate and brachial blood pressure response

Parameters	Pre Mean (SD)	Post Mean (SD)	Post 30 min Mean (SD)	p-value for ANOVA	Pairwise p-value for pre-post difference	Pairwise p-value for pre-post 30 min difference	Pairwise p-value for post-post 30 min difference
SBP (mmHg)	136.5 ± 16.2	130.3 ± 14.4	129.8 ± 13.8	<0.001	<0.001	<0.001	*N.S*
DBP (mmHg)	82.1 ± 9.6	75.1 ± 9.3	80.6 ± 9.2	<0.001	<0.001	*N.S*	<0.001
HR (bpm)	65.2 ± 10.4	80.7 ± 15.1	65.9 ± 10.7	<0.001	<0.001	*N.S*	<0.001

Note: SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; N.S., non-significant