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Title: Acute effects of exercise and sauna as a single intervention on arterial compliance

Year: 2020

Version: Accepted version (Final draft)

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Please cite the original version:

Lee, E., Willeit, P., Laukkanen, T., Kunutsor, S. K., Zaccardi, F., Khan, H., & Laukkanen, J. (2020). Acute effects of exercise and sauna as a single intervention on arterial compliance. European Journal of Preventive Cardiology, 27(10), 1104-1107. https://doi.org/10.1177/2047487319855454

The acute effects of exercise and sauna as a single intervention on arterial compliance

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

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Word count: 1111

According to the World Health Organization (WHO), 1 in 4 adults are not active enough on a global level,[1] and a substantial percentage of the current world population have at least one cardiovascular risk factor.[2,3] However, physical activity accumulated in bouts of at least 10 minutes in duration may mitigate these factors and improve a wide range of health-related outcomes, according to the latest physical activity advisory.[4,5] Aerobic exercise and sauna bathing have each been shown to provide health-related benefits through plausible pathways which include beneficial changes in arterial stiffness and hemodynamic indices.[6,7] Recent evidence has also shown that aerobic fitness is inversely associated with arterial stiffness,[8] and even repeated exposure to strenuous exercise does not appear to compromise vessel integrity.[9] Although the cardiovascular health benefits of aerobic exercise and sauna exposure seem to be comparable in nature, the effects of using exercise and sauna exposure in a single session has yet to be elucidated. Furthermore, using sauna bathing as an adjunct to a shortened length of aerobic physical activity has the potential to serve as a gateway toward habit change in populations who are inactive. As such, we investigated arterial stiffness and hemodynamic-related alterations associated with using aerobic exercise and sauna bathing as a single intervention, in a population with at least one cardiovascular risk factor.

Participants (n=77) were recruited from the city of Jyväskylä, Central Finland region, through the local out-of-hospital health care center. To be eligible for inclusion, participants had to be free of a prior diagnosis of cardiovascular disease (CVD) and exhibited at least one of the following cardiovascular risk factors: a history of smoking, diabetes, hyperlipidemia, hypertension, obesity, or family history of coronary heart disease. Participants were free from diagnosed and/or symptomatic CVD, musculoskeletal injury or any other physical or mental condition that will prohibit the participation in the experiment. Prior to the participation in the study, all participants were informed about the research purposes and measurement procedures, provided written informed consent, before being screened by a cardiac specialist.

The research protocol and study design were in accordance with the ethical standards approved by the institutional review board of the Central Finland Hospital District ethical committee, Jyväskylä, Finland (Dnro 5U/2016), and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

The experimental design consisted of 15 minutes of aerobic exercise on a cycle ergometer (Ergoselect 200K, Fysioline Oy, Tampere, Finland), followed immediately by 15 minutes of sauna exposure. Participants were asked to keep the cadence between 65-70 revolutions per minute (rpm) for the entire 15-minutes. Cycling cadence and its indicator lights were in full view of the participant at all times and would flash red when the cadence fell below 65 or surpassed 70 rpm. Cycling load in watts (W) was monitored and adjusted throughout the duration to ensure that the heart rate (HR) for each participant was kept at 75% of their individual maximal exercise HR; pre-calculated using the data obtained from a clinical exercise test that was conducted on a separate day. The transit time from the cessation of exercise to entering the sauna was under 120 seconds. Sauna exposure was based on a typical Finnish sauna characterized by low humidity and relatively high temperatures (73 ± 2 °C).[7] Fluid was consumed *ad libitum* throughout the entire experiment.

Blood pressure (SBP and DBP), mean arterial pressure (MAP), pulse pressure (PP), pulse wave velocity (PWV) as a measure of arterial stiffness, augmentation index (AIx), left ventricular ejection time (LVET), diastolic time (DT) and HR were taken at three different time points in the respective orders; before (PRE), immediately after (POST), and after a 30-minute recovery period (POST30). All measurements at the different time points were measured using the PulsePen device (DiaTecne s.r.l., Milan, Italy; www.pulsepen.com) composing of one tonometer and an integrated ECG unit, by the same assessor to minimize ascertainment biases and adhered closely to published guidelines.[10] Data is presented as means ± SD. Data was analyzed for within-group (time) changes with a repeated measures

analysis of variance (ANOVA). Within-group differences between POST to PRE and POST30 to PRE values were analyzed using pairwise *t*-tests, and *p*-values were corrected for Bonferroni by dividing all pairwise p-values with the number of comparisons conducted for each variable. The level for significance was set at $p \leq 0.05$. All statistical analyses were carried out with IBM SPSS Statistics v.22 software (IBM Corporation, Armonk, New York, USA).

Descriptive characteristics of the population are shown in **Table 1**. **Figure 1** shows the changes in outcome parameters from the experimental protocol of 15 minutes cycling followed by 15 minutes of sauna exposure. Compared to pre-intervention values, significant changes were observed post-intervention for PWV (9.4 vs 8.9 m/s, $p<0.001$), AIx (6.8 vs 0.5, $p=0.002$), MAP (101 vs 97 mmHg, $p<0.001$), LVET (300 vs 269 m/s, $p<0.001$), and DT (608 vs 446 m/s, $p<0.001$). Effects persisted after 30-minutes for MAP (101 vs 96 mmHg, $p<0.001$), AIx (6.8 vs 1.7, $p<0.001$), LVET (300 vs 292 m/s, $p<0.001$) and DT (608 vs 590 m/s, $p=0.016$). In addition, PP was significantly reduced compared to pre-intervention values (41 vs 37 mmHg, $p<0.001$).

The study showed that AIx decreased significantly after a combination of exercise and sauna, and although it may be associated with peripheral vascular changes such as vasodilation from the sauna bathing, this decrease in AIx was not mediated by an increase in PP as seen in sauna exposure alone, after 30 minutes of recovery.[7]

The similar and parallel changes seen in LVET and PWV were not expected, as they have been shown to share an inverse relationship.[11] The decrease seen in LVET is indicative of the shortened time taken by the left ventricle to do mechanical work, which normally leads to an increase in PWV. However, our results showed that PWV was reduced as well; which could be attributed to our study protocol. The current study protocol of aerobic exercise followed by sauna exposure also led to significantly lowered PP during the recovery period. This is

comparable to a recent study,[12] where changes to PP were retained 45 minutes after cessation of an acute bout of aerobic exercise. However, our results showed a reduction in MAP as well, after a 30-minute recovery period.

Our findings indicate that a combination of aerobic exercise and sauna led to positive alterations on MAP, PP and AIx, and these changes were retained after a 30-minute recovery period. Therefore, the benefits of combining aerobic exercise with sauna use should not be discounted, as it may be a gateway to encouraging a more optimum lifestyle. Long-term interventions involving the use of both aerobic exercise and sauna bathing should be investigated, as beneficial cardiovascular adaptations may be a possibility.

Acknowledgments

We sincerely thank Timo Harvia and the staff of Harvia Oy for sauna test facilities of the research, Poikonen Sanna and Pihlajalinna Oy for the recruitment and logistics assistance, and all the participants for dedicating their time to this study.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work supported by Tekes, the Finnish Funding Agency for Technology and Innovation, Helsinki, Finland.

Authorship

EL, TL, SKK, PW and JAL contributed to the conception and design of the work. EL, TL, SKK, FZ and JAL contributed to the acquisition, analysis or interpretation of data for the work. EL, PW, SKK, and JAL drafted the manuscript. EL, SKK, HK, PW, FZ and JAL critically

revised the manuscript. All gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

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Table 1. Participant characteristics (n=77)

Parameters	**Mean ± SD**
Age (years)	53.3 ± 9.8
Body mass (kg)	83.2 ± 14.8
Body mass index (kg/m^2)	27.8 ± 4.2
Resting SBP (mmHg)	137.1 ± 15.5
Resting DBP (mmHg)	83.3 ± 9.6
Resting HR (bpm)	68 ± 10
Maximum exercise HR (bpm)	174 ± 14
Aerobic exercise HR (bpm)[a]	131 ± 11
Sauna habits	**Number (Percentage)**
Less than once a week	15 (19.5%)
Once a week	14 (18.2%)
2 – 3 times per week	30 (38.9%)
4 or more times per week	18 (23.4%)
Cardiovascular risk classification	**Number (Percentage)**
Active Smoker/History of smoking	12 (15.8%)
Diabetes (Type 1 or 2)	3 (4%)
Dyslipidemia[b]	51 (67.1%)
Hypertension[c]	15 (19.7%)
Obesity (BMI >30 kg/m^2)	22 (28.6%)
Respiratory diseases[d]	10 (13.2%)
Family history of coronary heart disease[c]	31 (40.8%)

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; BMI, body mass index, [a] exercise heart rate HR was defined as 75% of individual maximal exercise HR, [b] based on use of cholesterol drugs or serum low-density lipoprotein (LDL) cholesterol over 3.5 mmol/L, [c] defined as SBP >140 mmHg, DBP >90 mmHg, or use of antihypertensive therapy, [d] Positive family history of CHD if father (< 55 years) or mother (< 65 years) had premature CHD,[d] respiratory diseases includes asthma and chronic obstructive pulmonary diseases

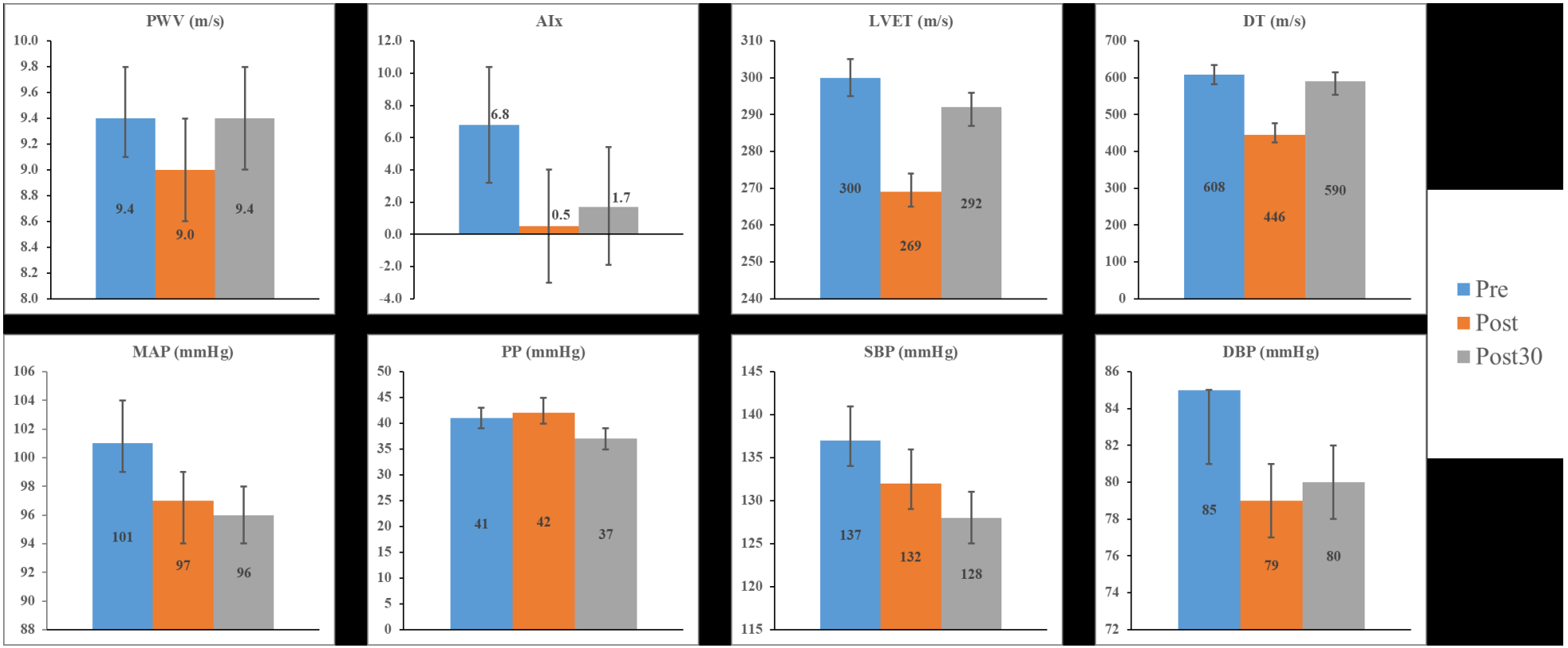

Figure 1. Changes in arterial stiffness and hemodynamic parameters. Error bars represent 95% confidence intervals (CI)