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Hanna Berge

underskrift

Effekter av likt kaloriforbruk hos høy intensitet intervall og moderat intensitet kontinuerlig trening på hypotensjon etter trening hos middelaldrende inaktive kvinner.

Berge, Hanna S.

Nord Universitet

Sammendrag

Hensikten med denne oppgaven var å studere om isokalorisk høyintensiv intervalltrening (INT) kunne gi en større og lengre post exercise hypotensjon (PEH) effekt enn kontinuerlig trening med moderat intensitet (CON). Seks friske kvinner med høyt normalt blodtrykk (BT) i en alder av 56 ± 6 år gjennomførte denne studien. Totalt tre tester ble utført, en test av maksimalt oksygenopptak etterfulgt av INT og CON. Designet brukt i denne studien var et kontrollert cross-over within-participants design, hvor deltagerne selv var sin egen kontroll. Før, under og etter INT og CON ble BT målt kontinuerlig. Etter INT og CON, ble BT målt i 30minutter mens deltagerne satt i en stol. Hovedfunnene var ingen signifikant forskjell mellom INT og CON på PEH. Det var heller ingen signifikant PEH, men med justering for multiple sammenligninger var det systoliske BT 30 minutter etter trening lavere enn BT i hvile før trening (gjennomsnitt differanse: -18mmHg, 95% CI: -29.1 til 8.1, $p < 0.006$). Diastolisk BT og GAT målt 5minutter etter trening var også lavere enn BT i hvile (gjennomsnitt differanse: -5 mmHg, 95% CI: -9.4 til -0.7 $p < 0.03$) (gjennomsnitt differanse: -8 mmHg, 95% CI: -12.1 til -3.3 $p < 0.006$). Total perifer motstand (TPM) derimot var signifikant lavere 5minutter etter trening sammenlignet med målingen tatt i hvile ($p <0.05$). Funnen fra denne studien demonstrerer at fysisk aktivitet kan redusere systolisk og diastolisk BT, GAT og TPM etter høyintensiv isokalorisk INT og moderat CON trening, men at intensiteten ikke var viktig i styringen av PEH responsen.

Nøkkelord: Hypotensjon, Høyt normalt blodtrykk, Post exercise hypotensjon, Utholdenhets trening, Varighet.

Effects of isocaloric high intensity interval and moderate intensity continuous exercise on post exercise hypotension in middle aged inactive women.

Berge, Hanna S.

Nord University College

Abstract

The purpose of this thesis was to study whether isocaloric high intensity interval exercise (INT) could cause a lager and longer post exercise hypotension (PEH) effect than continuous exercise with moderate intensity (CON). Six healthy women with high normal blood pressure (BP) at the age of 56 ± 6 conducted this study. A total of three tests were completed, one test of maximal oxygen uptake followed by INT and CON. Design used in this study was a controlled cross-over within- participants design, where the participants were their own control. Before, during and after the INT and CON were the BP measured continuously. After the INT and CON, BP were measured for 30min while the participants sat in a chair. Main findings were no significant difference between INT and CON on PEH. There were also no significant PEH, but with adjustment for multiple comparisons the systolic BP 30 min post exercise was lower than the BP in rest (mean diff.: -18.6 mmHg, 95% CI: -29.1 to -8.1, $p < 0.006$). Diastolic BP and MAP measured 5min post exercise was also lower than BP in rest (Mean diff: -5 mmHg, 95% CI: -9.4 to -0.7 $p < 0.03$) (mean diff: - 8 mmHg, 95% CI: -12.1 to -3.3 $p < 0.006$). Total peripheral resistance (TPR) was significantly reduced 5minutes post exercise compared to the value in rest ($p < 0.05$). The findings from this study demonstrates that physical exercise can reduce systolic and diastolic BP, MAP and TPR after isocaloric high intensity INT and moderate intensity CON exercise, but that the intensity was not important in governing the PEH response.

Keywords: Aerobic endurance exercise, Duration, Hypotension, High normal blood pressure, Post exercise hypotension.

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Preface

First of all, I want to sincerely thank my supervisor associate professor Terje Gjøvaag who took a graduate student under his wings and made this thesis possible. Without your commitment and coffee maker I would not been able to accomplish this task. A big thank you to associate professor Boye Welde who got me interested in the topic blood pressure and for all your' guidance and help with the statistical analyzing of the data.

Finally, I would like to thank my fellow master student Marianne Olsrud for her cooperation during the exercise interventions, and of course a huge thank you to our participants for their participation.

Introduction

Hypertension is the term we associate with having high blood pressure (BP), and is viewed as the main modifiable cause of mortality and as an independent risk factor for heart and cardiovascular diseases (Mancia et al. 2007). Untreated high BP can cause very serious consequences like thickening of the vessel walls, impaired endothelial function and atherosclerosis in the arteries. In addition, enlargement (hypertrophy) of the left ventricle and/or albumin leakage from the kidneys, which in itself is associated with increased mortality and morbidity (Lewington et al. 2002; Mancia et al. 2007; Vasan et al. 2001).

The size of our arterial BP depends on several factors. First is the extensibility of our arteries. If the extensibility is small, the pressure will reach higher values when the heart pumps out blood meaning that the pulse pressure will escalate. It is normal for the BP to increase with age because the extensibility of the artery declines with age (Sand, Sjaastad and Haug, p 315-316. 2010). When the cardiac output (CO) increases without change in total peripheral resistance (TPR), also increases the blood volume in the arteries, the arterial pressure and the tension of the arterial walls. In other words, a heightened CO and constant TPR rises the systolic BP more than the diastolic (Sand, Sjaastad and Haug, p 315-316. 2010).

Another important factor that affects the arterial BP is the resistance blood encounters when flowing through the branched vascular system. The size of the TPR is first and foremost decided by the diameter of the artery. So if the TPR were to increase, the blood volume in the arteries and the arterial BP would increase too. Increased blood volume affects arterial BP because it affects the venous pressure, the end-diastolic volume, stroke volume and thus the CO. This is why adjusting the blood volume is an important factor for the long-term regulation of BP (Sand, Sjaastad and Haug, p 315-316.2010).

There are arguments that men are at greater risk for developing cardiovascular and renal diseases than women. This statement is supported by studies that have shown that BP was higher in men than in women (Khoury et al. 1992; Staessen et al.1990). Wiinber et al. (1995) researched 352 normotensive men and women in the age span of 20-79 years and concluded that the BP was 6 - 10mmHg higher in men than in women, until they reached 70-79 years of age, when the BP was equal between the genders.

However, after women reach their menopause some researchers have found that their BP can increase to levels higher than reported for men. Burl et al. (1995) confirmed that non-Hispanic white women had higher BP than men after their menopause by 70-79 years of age.

Physical inactivity alone is believed to account for 5-13% of the development of hypertension (Geleijnse, Kok and Grobbee. 2004). Based on the fact that inactivity induces hypertension one may assume that repeated bouts of physical activity may be a strategy to help reduce BP and prevent hypertension. Recommended physical activity to prevent hypertension is mainly dynamic endurance exercise like aerobic exercise, walking, swimming and cycling (Fagard and Cornelissen. 2007; Mancia et al. 2007). Additionally, studies show that moderate resistance exercise at a low load (40-50% of maximum capacity) with many repetitions can also be recommended to help control BP in both normotensive and hypertensive individuals (Bermudes et al. 2004; Melo et al. 2006; Mota et al. 2009).

The opposite of hypertension is hypotension, and is associated with having a low BP. Hypotension occurs per definition when the systolic BP is less than 90 mmHg, or if there is a decrease in systolic BP of 30 mmHg over a 2-hour period (Kane-Gill et al. 2014). In the recent years there has been more research on hypotension focusing on arterial BP regulation during exercise, rest and everyday activities. In addition, it seems to be a wider focus on acute BP reduction after a single exercise session, a phenomenon called post exercise hypotension (PEH).

The definition of PEH is a reduction in systolic and/or diastolic BP after a single bout of exercise while the values are below those observed during the pre exercise i.e. at rest (Mota et al. 2009). Post exercise hypotension is an effect from exercise that researchers have shown can help control and/or reduce BP in both hypertensive and normotensive individuals (Collier et al. 2007; Forjaz et al. 2000; MacDonald, MacDougall and Hogben 1999a). So if individuals that are normotensive or hypertensive can achieve a reduction in BP after only one exercise session, then physical activity and exercise has to be one of the most important non-pharmacological treatments we have against hypertension.

There is little knowledge and limited studies on gender differences regarding the regulation of BP. A review written by Reckelhoff (2001) suggests that components of the renin angiotensin system plays a role in the control of BP within gender differences. The causes of gender difference are, however, still vague and remains unclear. Moreover, to the best of my knowledge I was not able to find any studies comparing possible PEH differences between men and women. Nor can I find any articles suggesting separate recommendations of exercise to achieve PEH within genders. As far as I could see, there are also less research on only female participants compared to only males.

To attain the best PEH effect, the Norwegian directorate for health recommends moderate continuous aerobic exercise at 40-70% of maximal oxygen uptake ($VO2_{max}$) five to seven days a week with at least 30 minutes per session for hypertensive individuals (Björjesson, Kjeldsen and Dahlöf 2008). Researchers have also concluded that moderate continuous aerobic exercise (70-75% VO_2 max) for 30-45 minutes can reduce BP in healthy normotensive individuals (Forjaz et al. 2004; Jones et al. 2007; MacDonald, MacDougall and Hogben.1999a; MacDonald, MacDougall and Hogben.1999b).

Since moderate intensity exercise can influence a reduction in resting BP, it was reason to believe that high intensity exercise (~80-90% of VO_2 max) might have a PEH effect as well. Bonsu and Terblanche (2016) and Scott et al. (2007) confirms this by finding resting BP reductions after high intensity exercise in normotensive individuals. High intensity exercise has also been proven to effect the resting BP in hypertensive participants (Molmen-Hansen et al. 2011; Santana et al. 2013). There is limited information about high intensity endurance exercise (>85% of VO_{2max}) and whether it can evoke a PEH response. Since high intensity exercise seems to have increased its popularity recent years, it may be worth exploring this type of exercise on the PEH response.

Although PEH is well documented for moderate endurance exercise, its magnitude (normotensive = 8-10 / 3-5 mmHg) (hypertensive = 18-20 / 7-9 mmHg) and duration (2-4 hours) varies considerably (Kenney and Seals, 1993). Indicating that factors such as exercise characteristics like intensity or duration may be involved in influencing PEH. Even though the endurance exercise effect is significant and can last up till several hours there are still questions about which exercise characteristics have the biggest impact on the PEH response.

Potential mechanisms behind PEH is not completely identified yet and requires more research, but several studies suggest that reduced stroke volume and decreased TPR are important factors (Halliwill, 2001; MacDonald, 2002; Mota et al. 2009).

In the present study the BP was measured before, during exercise and 30minutes post exercise. The PEH was measured for only 30minutes because of time constraints and because studies have shown that one can expect the largest BP reduction at the beginning of the recovery period. A published pilot study concluded that similar endurance exercise as the present study, gave the largest effect on PEH in the first ten minutes post exercise and that post exercise MAP values remained unchanged from 10 to 60minutes (Gjøvaag et al. 2015). Based on this, in the present study, it was chosen to focus on the immediate BP responses in the first 30minutes post exercise.

I could not find any studies during extensive literature searches that compare the effects of two isocaloric endurance exercise sessions with different intensities upon PEH in women. To the best of my knowledge, this master thesis is the first to do this comparison in only female participants.

The purpose of this current study was to compare the effects of isocaloric high intensity interval (INT: ~85% HF maximum) and moderate continuous exercise (CON: ~75% HF maximum) upon the PEH response in middle aged physically inactive women with high normal BP. The significance of correcting for isocaloric consumption between the exercises is because if one is to examine the effect of *intensity* the calorie consumption between the exercise regimes has to be identical. In addition, I aimed to research the potential effects of stroke volume, TPR and CO on the PEH response. The main hypothesis was that exercise with high intensity (>85% HF max) would produce a greater and longer reduction in BP than moderate intensity continuous exercise.

Method

Experimental approach to the research question

Six healthy physically inactive women with high normal BP in the age span from 46 to 63 years volunteered to participate in the present study. The participants were mainly recruited from Oslo and Akershus Univeristy College. The participants conducted a total of three exercises on a cycle ergometer. At first, all participants completed a VO_2 max test in advance of the exercise interventions INT and CON. Results from the VO_2 max was used as a basis to calculate the relative HF target zones (% HFmax) during INT and CON exercise. Thereafter, the participants completed one INT exercise at high intensity (~85% HFmax) and one CON exercise at moderate intensity (~75% HFmax). All participants completed the INT exercise before the CON exercise to make sure the right amount of calories were equally consumed between the two exercises.

Blood pressure was measured before, during and after each exercise session. All exercises were conducted in the period between January-April 2016, with a minimum of 48 hours between each test. The INT and CON exercise with different intensities and resistance was the independent variable, while BP, heart frequency (HF), stroke volume, CO, TPR, blood lactate concentration and rating of perceived exertion (RPE) was the dependent variables in this study. Controlled cross-over, within participants was the design used for this study, meaning the participants were their own controls.

Participants

The participants were internally recruited through email at the Oslo and Akershus University College. The internal mail was sent out to 650 potential participants whereas 20 persons volunteered for this study. Two participants withdrew because of personal reasons, then six participants were excluded due to too low BP or because they were too well trained for the study. Out of the twelve participants that completed, six of them were women and took part in this study. The participants characteristics are shown in table 1.

Table 1. The women's characteristics prior to the study (N=6). Mean values and standard derivation (SD). Physiological responses where all measured in the resting state.

Variables	Mean	SD
Age	56.1	6.7
Height	168.5	5.3
Wight	67.3	8.5
BMI	23.8	3.1
BP $_{\text{(Sys, mmHg)}}$	127.6	9.0
BP $_{\text{(Dia, mmHg)}}$	82.1	1.1
Pulse wave velocity	6.5	1.6
HF	67	10
Lactate	1.2	0.4
RPE (0-10)	0.17	0.40

Explanation: SD = standard deviation, BMI = Body mass index, BP = Blood pressure, Sys = Systolic, Dia = Diastolic, mmHg = millimeter of mercury, HF = Heart frequency, RPE = Ratings of perceived exertion.

This study was approved 19.11.2015 by the regional ethics committee (REK south/east), and conducted in accordance with the Helsinki Declaration. Before the participants signed the written consent form, they were informed about the advantages and disadvantages associated with the study. The participants were also aware that they could at any time withdraw from the study without giving any reasons or facing any consequences.

Inclusion and exclusion criteria

Inclusion criteria for participation in the study was inactive women over 40 years with normal (120/80mmHg) to high normal (139/89mmHg) BP, as defined by Mancia et al. (2007). Another inclusion criterion was inactivity and therefore the participants could not exercise more than once or twice a week on a regular basis.

Exclusion criteria was BP lower than 120/80 mmHg, secondary hypertension, diabetes type 1 or 2, metabolic disorders, chronic obstructive pulmonary disease, differential disorders or use of medicines that could affects the outcome, pacemakers or orthopedic disorders that would make it difficult to conduct the exercises. The participants were also excluded if they were to enjoy alcohol, snus or smoke 24 hours prior to the exercises.

Measuring instruments

All tests were carried out by an experienced researcher and supervised by authorized personnel and took place in the Physiological Laboratory of the Faculty of Health sciences at the Oslo and Akershus University College.

The first day included an International physical activity questionnaire (Craig et al. 2003) (appendix 1, IPAQ), a consent declaration of each participant health condition (appendix 2), BP measurement in the resting state (Omron M10-IT. Hoofddorp, The Netherlands), a body composition test (InBody 720, InBody, Korea), a pulse wave velocity (i.e arterial stiffness Boso ABI 100 system, Jungingen Germany) and finally a VO_2 max test (Jaeger Vyntus CPX, Carefusion/BD USA). The VO_2 max test was performed on a cycle ergometer (Lode Excalibur sport, Groningen, The Netherlands) with a Polar H7 pulse transmitter (Polar Electro, Kempele Finland) interfaced with the Vyntus apparatus.

Body weight, fat mass, skeletal muscle mass and visceral fat area was measured by multi frequency bioelectrical impedance (DSM-BIA). There was very good correlation in lean body mass (ICC=0.95) and fat mass (ICC= 0.95) measurements, measured by DSM-BIA and DEXA (Ling et al. 2011). DEXA measurements are considered the gold standard in this context.

The ABI pulse wave velocity system was used to diagnose potential peripheral arterial occlusive disease and measure arterial stiffness. It also measures other parameters like individual BP readings in arms and legs, BP difference between sides of the body and pulse pressure. The Boso ABI 100 system has been compared to Doppler- Assisted ABI measurement and was found superior (Diehma et al. 2009).

Maximal oxygen test done with Jaeger Vyntus CPX is an ergo spirometry system that collects breath by breath data which can determine a patient’s metabolic response. The system measures and calculates parameters like oxygen consumption, carbon dioxide production, respiratory exchange ratio, lung ventilation, breathing frequency, tidal volume and many more. Rietjens et al. (2001) compared the Jeager system against the Douglas bag method during low and high exercise intensities. Their conclusion was that the Jeager system was a valid apparatus for determination of minute ventilation, VO_2 and carbon dioxide production during both intensities and that it can be used for accurate and quick determination of ventilatory and respiratory variables during exercise.

Second and third day involved the two exercises, INT and CON. The primary efficacy objectives these days were to measure the mean arterial pressure (MAP) and CO. The main instrument for measuring BP during the INT and CON exercises was Tango+ BP monitor, SunTech Medical, Morrissvile, North Carolina, USA.

The second primary efficacy objective CO was measured, along with stroke volume, HF and TPR beat for beat by using noninvasive signal morphology impedance cardiography (Physio Flow Enduro, Focsvillier. France). Charloux et al. (2000) compared PhysioFlow Enduro with the direct Fick method and concluded that the Physio Flow was a valid and reliable method for determining parameters like CO.

Secondary efficacy objectives for this study was energy metabolism and oxygen uptake during exercise, VO_2 max, body composition, activity measurement (IPAQ), blood lactate concentration and ratings of perceived exertion (RPE). Oxygen uptake, total calorie consumption and percentage distribution of carbohydrate and fat metabolism was measured with the ergo spirometry equipment from Jaeger Vyntus CPX. Blood lactate concentration was measured by finger prick samples in capillary blood analyzer (Arkray Lactate Pro2 LT-1730. Japan). Ratings of perceived exhaustion is described as a participant degree of effort, measured on a scale from 0 to 10 where 0 was no effort and 10 being maximum effort (appendix 3).

Exercise protocols

Every participant completed the tests in following order: VO_2 max, INT and CON. The INT exercise had to be completed before the CON exercise to make sure the same amount of calories was consumed during both sessions. This was because the INT exercise has the highest amount of calories per unit time. If not, we risked the CON exercise duration to be too short or too long risking the idea of isocaloric consumption for both exercises.

VO_2 max

A VO_2 max test is an incremental test to exhaustion. Blood lactate concentration was measured before a self-directed warm up for ten minutes on a cycle ergometer with >70 rounds per minute, at 75watt load. After the warm up the load increased with 10 watt for each ½ minute. The participants VO_2, RPE and HF was measured for every breath and averaged over 20 seconds throughout the test. The test was performed to exhaustion. Highest average over one consecutive minute was defined as VO_2 max. Ratings of perceived exhaustion was recorded pre and post exercise.

INT protocol

Testing started with a self-directed warm up for 10 minutes on a cycle ergometer with >70 rounds per minute, at 75watt load. After the warm up, additionally four minutes was used to get the participants HF in the right target zone (~85% HF max). The INT exercise was divided into four intervals and three active rest periods with a duration of four minutes each (4 x 4 exercise). The total exercise duration was 38minutes, including the warm up. Load during the intervals was ≥ 85% of HF max (80-85% $VO2_{max}$). The active rest periods between each interval was ~75% of the participant HF max (60-65% VO2max).

During the intervals the watt was controlled manually and continuously up and down based on each participant target zones calculated in advance. The reason for this, was to keep the HF close to the calculated HF zone during the whole test period. For every third minute both during intervals and active rest periods HF and RPE was recorded, while BP was only recorded during the intervals. Along with HF, RPE and BP, TPR, stroke volume and CO was also automatically measured continuously during the test. After the exercise the participant stopped pedaling and BP, HF, RPE and calories consumed was immediately recorded within 30 seconds. During the PEH period, BP, HF and RPE was first noted after five minutes, then after 10 and 30minutes. The participants lactate was measured before and after the exercise, and again after the 30minute recovery. During recovery the participant was placed on a chair in a room without bystanders.

CON protocol

Warm up was similar to the INT warm up. Load during the CON exercise was ~75% of HF max (60-65% $VO2_{max}$). The exercise duration was about 36 ± 7 min depending on the participant calorie consumption from the INT exercise. During the CON exercise watt was controlled manually and continuously up and down based on each participant HF target zone calculated in advance. The dosage of the number of minutes of activity in the CON exercise was adjusted for each participant to make sure that the participants consumed the same amount of calories during both exercises. When the same amount of calories from the INT exercise was reached in the CON exercise, the exercise was finished.

During the exercise CO, TPR, HF and calorie consumption was automatically measured continuously and BP, HF and RPE recorder every seventh minute. After the exercise, the participant stopped pedaling and BP, HF, RPE and calories consumed was immediately recorded within 30 seconds. During the PEH period, BP, HF and RPE was first recorded after five minutes, then after 10 and 30 minutes. The participants lactate was measured before and after the exercise, and again after the 30 minute PEH period. During recovery the participant was placed on a chair shielded in a room without bystanders.

Calculations

Mean arterial pressure (MAP)

Rest: 2/3 diastolic pressure + 1/3 systolic pressure.

During exercise: ½ diastolic pressure + ½ systolic pressure.

Respiratory quotient (RQ)

To approximately calculate the duration of the CON exercise to each participant the calorie consumption from their INT exercise was used and made a formula based on the respiratory quotient table shown in table 3 (Perronnet and Massicotte. 1991).

Example, participant 4.

Target: 290Kcal from INT exercise.

Target zone during CON exercise: 155HF

$155\ HF = 1{,}4\ L\ O2$

$RQ\ 0.94 - 0.95 = 5.12 Kcal/L$

$1.4 \times 5.12 = 7.168 Kcal/minute$

$$\frac{290}{7.2 Kcal/minute} = 40 minutes$$

Participants HF target zones

The various HF target zones was calculated for the INT and CON exercise based on each participant HF max from the VO_2 max test. Heart rate target zones was necessary to make sure the intensity was similar during the entire INT (~85%HFmax) and CON (~75% HFmax) exercise.

Example, participant 4.

$$HFmax = 193$$

$$INT\ target\ zone = 85\%\ of\ HFmax = 0.85 \times 193 = 164$$

$$CON\ target\ zone = 75\%\ of\ HFmax = \ 0.75 \times 193 = 145$$

Calorie calculations

Each participant calorie consumption during the exercise interventions were calculated continuously by the software in the Jaeger Vyntus CPX analyzer based on indirect calorimetry (Frayn 1983).

$$Oxygenic\ calorie\ value\ at\ the\ given\ RQ\ value\ \times oxygen\ uptake\ \times time\ of\ activity$$

Statistical analyses

Our data met the requirement of Shapiro Wilks test of normality. All results are presented as mean ± standard deviation. The paired samples *t*-test procedures were applied when only two averages were involved in the analyses, and repeated measures ANOVA was applied when there were more than two averages to be compared. A 2 (high intensity interval vs. continuous exercise) x 5 (rest before exercise, mean during exercise, 5 min, 10 min, 30 min post exercise) ANOVA with repeated measures on both variables was used to test for differences in systolic BP, diastolic BP, MAP, TPR, HF, stroke volume and CO between the two exercise studies before, during, and after the exercise period. Post hoc comparisons with Bonferroni corrections were conducted to locate differences. Where sphericity assumptions were violated, Greenhouse-Geisser adjustments of the *p*-values were reported. $P \leq 0.05$ was considered statistically significant.

IBM SPSS statistics 23.0 for Windows was the software used for the statistical analysis in this thesis (IBM SPSS inc, Chicago, IL, USA). Graphical representations were developed using Microsoft Excel 2016 for Windows (Microsoft Corporation).

Since this study only had six participants, it was necessary to reduce the number of measurements in the ANOVA to five. Hence, the measurements during exercise was reduced to one average representing the whole exercise session and we also excluded the 20 min post exercise registration. During analyzing of data showed in figure 4, 5 and 6 there was only one resting value for both exercises, the resting values are thus used for both exercises.

Results

Physiological responses, watt and RPE during exercise.

Mean $VO2_{max}$ for the women was 31.8 ± 3.5 ml/kg/min. Maximum HF and watt attained during the $VO2_{max}$ was 174 ± 12 beats/minute and 195 ± 15 watt. Corresponding values for respiratory exchange ratio and ventilation were respectively 1.19 ± 0.05 and 89.5 ± 10.5 L/min. Post $VO2_{max}$ the women had a blood lactate concentration of 10.2 ± 1.6 mmol/L and stated 10 ± 0 in RPE.

The response during the INT and CON exercises are shown in table 2. The INT exercise values are based on mean values during the four intervals, the four active rest periods are not included. During the INT exercise there was a reduction in watt from 114 ± 19 in the first interval to 98 ± 11 in the last interval, while the watt during the CON exercise went from 95 ± 10 to 76 ±11 watt (*P*= 0.01 for both exercises).

Table 2. Mean ± standard deviation on physiological response, watt and RPE *during* INT and CON exercise for the six women in this study. Blood lactate concentration is mean post exercise.

Variables	INT [a]	CON
Duration[b]	28 ± 0 *	36 ± 7
O2 uptake (ml/kg/min)	24.7 ± 2.8	20.2 ± 3.3
O2uptake (%$VO2_{max}$)	78 %	64 %
HF (beats/min)	151 ± 12 *	136 ± 12
HF (%HF_{max})	87 % *	78%
Watt	105 ± 17	86 ± 12
Watt (%$Watt_{max}$)	54 % *	44 %
Lactate (mmol/L)	2.5 ± 1.0	2.2 ± 1.0
RPE post exercise (0-10)	6 ± 2	4 ± 3

[a] = The values of the INT exercise is based on the mean of the four intervals, the active rest intervals are not included [b] = 10minute warm up is not included for the mean duration values. * = Significant difference between INT and CON exercise; * P < 0,05.

Explanation: O2 uptake = oxygen uptake, ml/kg/min = milliliters per kilo per minute, $VO2_{max}$ = Maximum oxygen uptake, HF= Heart frequency, HF $_{max}$ = Maximum heart frequency, Watt $_{max}$ = Maximum watt, mmol/L= millimoles per liter, RPE = Ratings of perceived exertion (0-10).

For systolic BP there was a significant main effect of time ($F_{4/20}$ = 40.70, P < 0.001. No main effects from the INT and CON exercise ($F_{1/5}$ = 2.66, p= 0.16) or interaction between time and exercise ($F_{4/20}$ = 0.62, p = 0.65) were found.

Figure 1 displays changes in systolic BP for the INT and CON exercise from rest until 30 min post exercise with Bonferroni adjusted post-hoc comparisons for the time effect. There was no significant PEH, however, with no adjustment for multiple comparisons, systolic BP 30 min post exercise was lower than in rest (mean dif.: -18.6 mmHg, 95% CI: -29.1 to -8.1, p = 0.006).

The systolic BP in rest, PE 5min, PE 10min and PE 30min were all significantly different from the mean systolic BP during exercise with following p < 0.05 and p < 0.01.

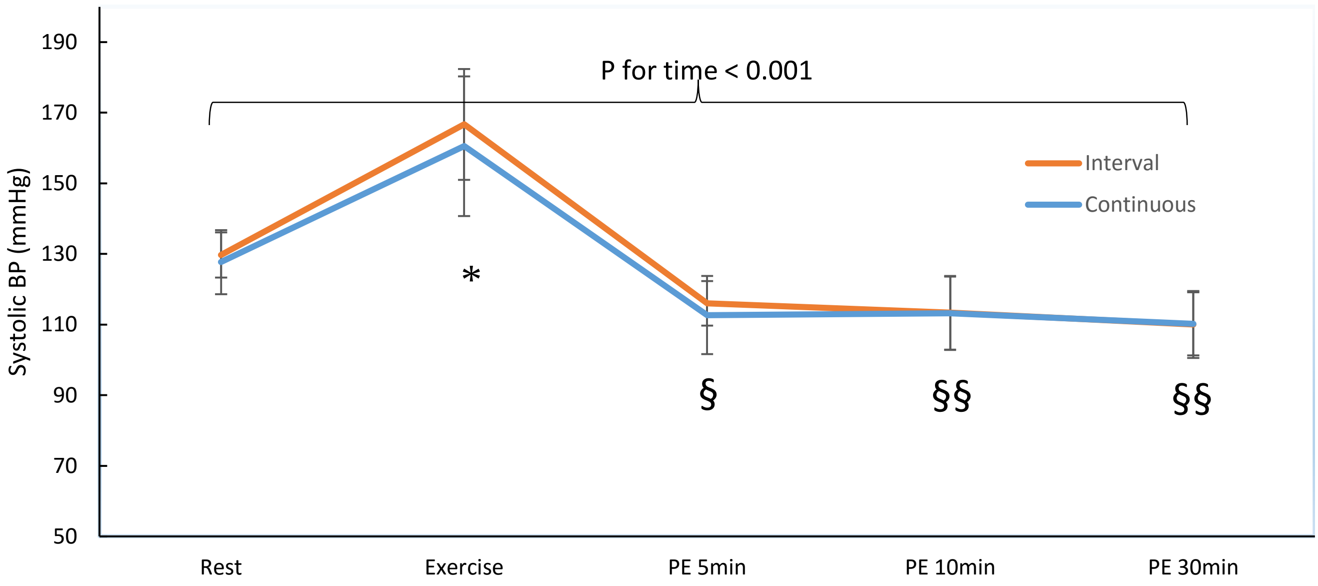

Figure 1. Systolic BP in rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

BP = Blood pressure, PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency. Max = maximum. * = significantly different from rest, * p < 0.05, § = significantly different from exercise, § p < 0.05, §§ p < 0.01.

Figure 2 displays changes in diastolic BP for the INT and CON exercise from rest until 30 min post exercise with Bonferroni adjusted post-hoc comparisons for the time effect. There were no significant changes in diastolic BP in rest, during exercise or post exercise for both INT and CON. However, with no adjustment for multiple comparisons, diastolic BP 5 min post exercise was lower than in rest (mean dif.: -5 mmHg, 95% CI: -9.4 to -7.1, p = 0.030).

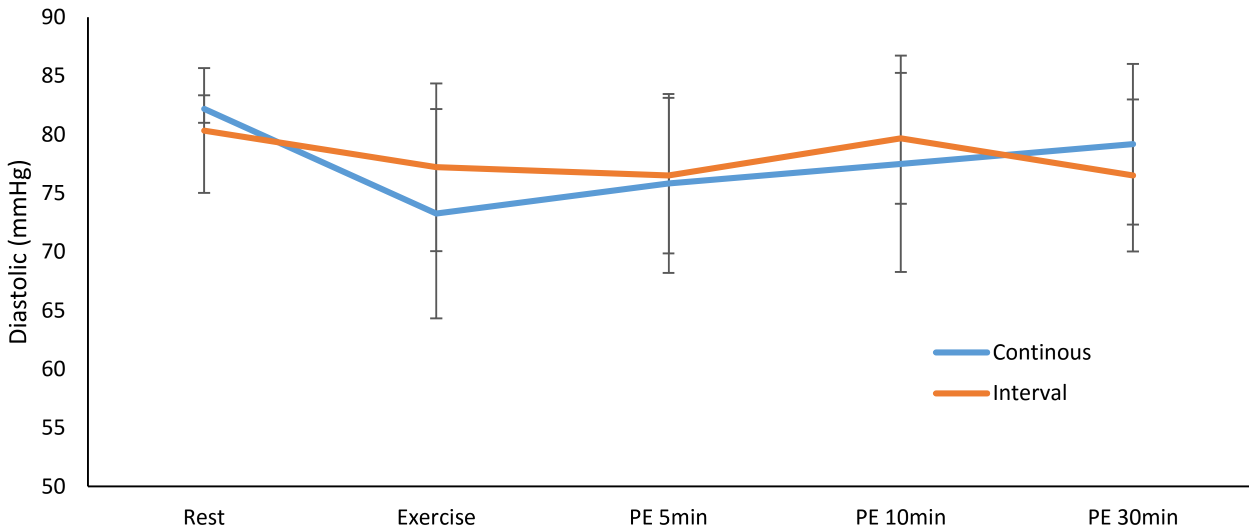

Figure 2. Diastolic BP in rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

BP = Blood pressure, PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency. Max = maximum.

Main effect of time was significant for MAP ($F_{1.9/9.6} = 28.6$, $P < 0.001$). No main effects from the INT and CON exercise ($F_{1/5} = 1.20$, $p = 0.33$) or interaction between time and exercise ($F_{1.47/7.4} = 0.62$, $p = 0.53$) were found. Figure 2 displays changes in MAP by the two exercise interventions INT and CON over time and which measurement periods that differ from each other.

Significantly differences were found in MAP at PE 5, PE 10 and PE 30min ($p < 0.01$) compared to exercise. Still no significant PEH, but with no adjustment for multiple comparisons, MAP 5 min post exercise was lower than at rest (mean dif.: - 8 mmHg, 95% CI: -12 to - 3, p = 0.006).

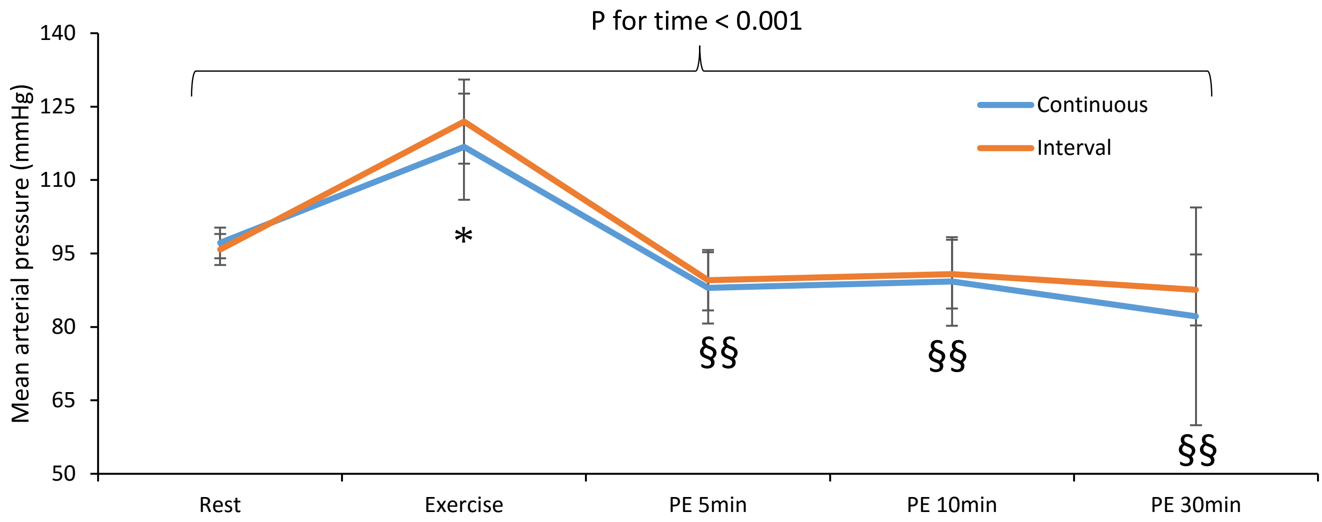

Figure 3. Mean arterial pressure from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency, Max = maximum. * = significantly different from rest, * $p < 0.05$. § = significantly different from exercise, § $p < 0.05$, §§ $p < 0.01$.

Unfortunately, no main effects in TPR from the INT and CON exercise ($F_{1/5} = 1.47$, $p = 0.28$) or interaction between time and exercise ($F_{4/20} = 1.01$, $p = 0.43$) were found. On the other hand, TPR was significant for main effect of time ($F_{4/20} = 45$, $P < 0.001$). Figure 4 presents changes in TPR by the two exercise interventions INT and CON from rest to post exercise 30min and which measurement periods that vary from each other.

Both TPR during exercise and PE 5min were significantly changed compared to the TPR measured in rest ($p < 0.01$ and $p < 0.05$). Again we see that all the measurement periods are significantly different from exercise ($p < 0.01$). Total peripheral resistance PE 30min was additionally significantly changed from both PE 5min ($p < 0.001$) and PE 10min ($p < 0.05$).

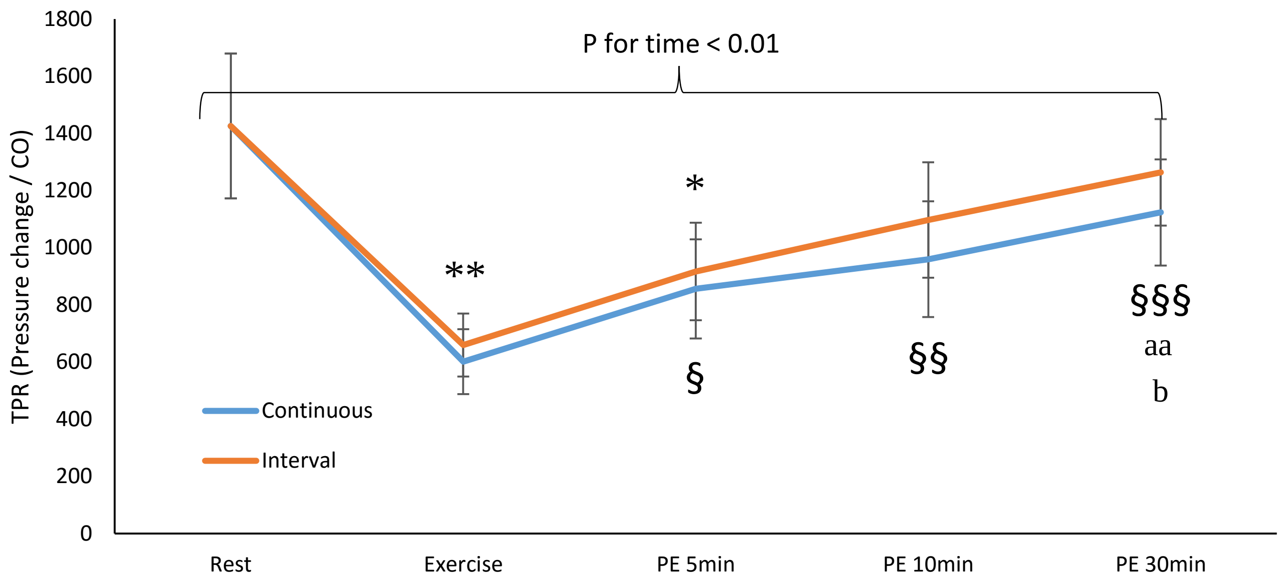

Figure 4. Total peripheral resistance from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

PE = post exercise, Min = minutes, CO = cardiac output, INT = interval, CON = continuous. HF = heart frequency, Max = maximum. * = significantly different from rest, * $p < 0.05$. ** $p < 0.01$, § = significantly different from exercise, § $p < 0.05$, §§ $p < 0.01$, §§§ $p < 0.001$. a = significantly different from PE 5min, aa $p < 0.01$. b = significant differently from PE 10min, b $p < 0.05$.

Figure 5 Presents changes in stroke volume by INT and CON from rest to post exercise 30min and which measurement periods that diverge from each other. Stroke volume was found significant for main effect of time ($F2/10 = 10.6$, $P < 0.01$). Again, no key effects from the INT and CON exercise ($F1/5 = 2.0$, $p = 0.22$) or interaction between time and exercise were found ($F4/20 = 1.0$, $p = 0.44$).

Stroke volume in PE 30min was significantly changed from both exercise and PE 5min both with $p < 0.05$. Stroke volume during exercise was also the only measurement period that differed significantly from rest ($p < 0.05$).

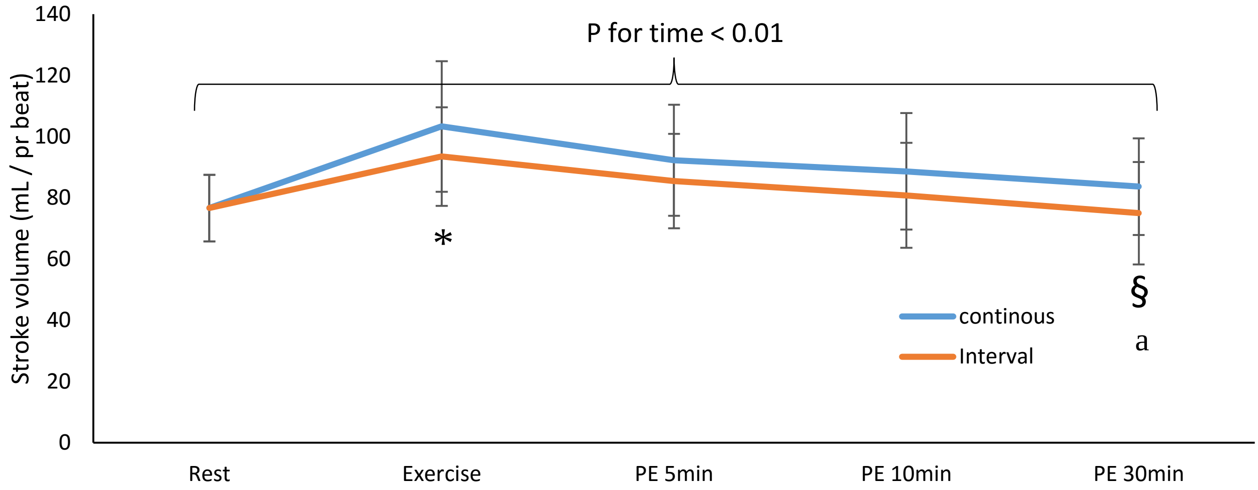

Figure 5. Stroke volume from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

mL = milliliter, PE= post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency, Max = maximum. * = significantly different from rest, * $p < 0.05$, § = significantly different from exercise, § $p < 0.05$, a = significantly different from PE 5min, a $p < 0.05$.

Significance for main effect of time were too found in CO ($F_{4/20} = 75{,}1$ $P < 0.001$). Key effects from the INT and CON exercise ($F_{1/5} = 0.23$, $p = 0.65$) and interaction between time and exercise were not significant ($F_{4/20} = 0.4$, $p = 0.8$). Figure 6 Shows changes in CO by the two exercise interventions INT and CON over time and which measurement periods that diverge from each other.

Cardiac output measured at PE 5min and PE 30min was both significantly altered compared to CO during rest ($p < 0.05$), along with exercise ($p < 0.05$). There were additionally significant changes in PE 30min matched to PE 5min ($p < 0.05$). Cardiac output was also significantly changed in rest, PE 5min, PE 10min and PE 30min compared to exercise ($p < 0.01$).

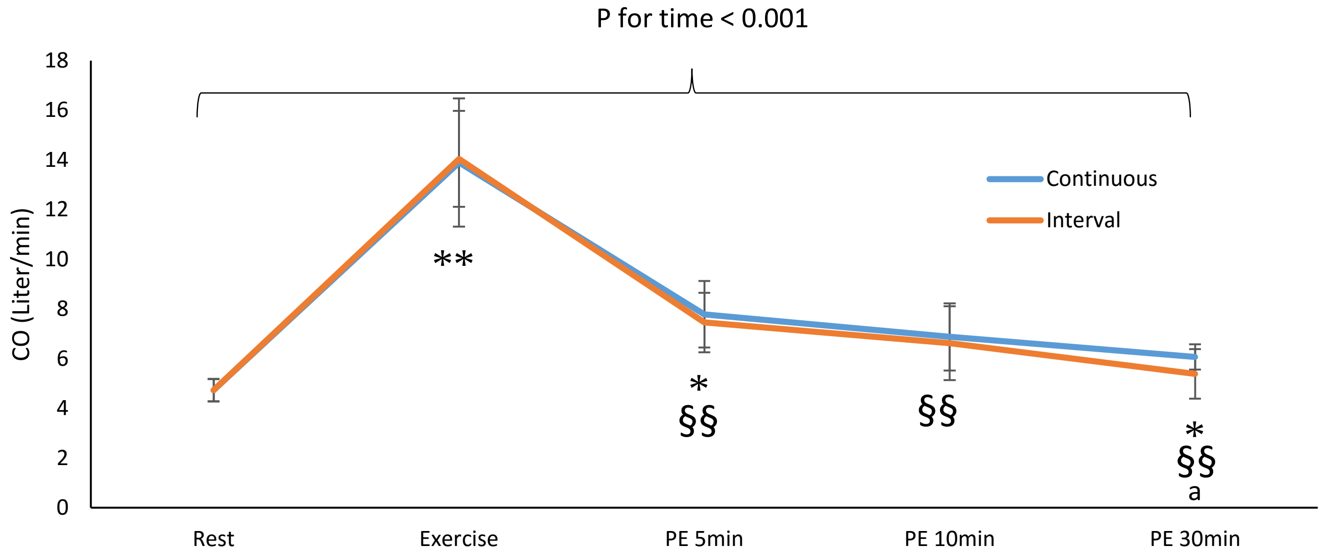

Figure 6. Cardiac output from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency, Max = maximum * = significantly different from rest, * p < 0.05. ** p < 0.01, § = significantly different from exercise, §§ p < 0.01, a = significantly different from PE 5min, a p < 0.05.

Discussion

The purpose of this thesis was to study whether isocaloric high intensity INT could cause a lager and longer PEH effect than CON exercise with moderate intensity.

Main findings were no significant difference between INT and CON on PEH. There were also no significant PEH, but with adjustment for multiple comparisons the systolic BP 30 min post exercise was lower than the BP in rest (mean diff.: -18.6 mmHg, 95% CI: -29.1 to -8.1, $p < 0.006$). Diastolic BP and MAP measured 5min post exercise was also lower than BP in rest (Mean diff: -5 mmHg, 95% CI: -9.4 to -0.7 $p < 0.03$) (mean diff: - 8 mmHg, 95% CI: -12.1 to -3.3 $p < 0.006$). Total peripheral resistance was on the other hand significantly reduced 5minutes post exercise compared to the value in rest ($p <0.05$).

Acute effects from endurance exercise

The results from Table 2 clearly indicates that the exercises INT and CON were completed with the intended exercise intensities (75% and 85% of HF max) for this present study. Mean intensity for the CON exercise were 87% of HF max and 78% of HF max for the CON exercise. The results also show a significant difference in HF between the two exercise studies, which confirms a significant intensity variation between INT and CON.

Post exercise hypotension is recognized by the BP values being lower after the exercise is finished compared to the BP values taken before the beginning of the exercise. The clear effect aerobic endurance exercise has on lowering BP in both hypertensive and normotensive individuals makes it one of the most important non-pharmacological treatments we have. Better fitness/increased physical activity with hypertension is also associated with a lower degree of mortality in both hypertensive (Evenson et al. 2004) and normotensive individuals (Sandvik et al. 1993).

Although PEH was well documented for moderate endurance exercise, its magnitude (normotensive = 8-10 / 3-5 mmHg) (hypertensive = 18-20 / 7-9 mmHg) and duration (2-4 hours) varied considerably (Kenney and Seals, 1993). This indicates that exercise characteristics like intensity and duration may be involved in influencing PEH. Even though the aerobic exercise effect is significant and could last up to several hours there is still questions about which exercise characteristics that can influence PEH and to what extent.

Blood pressure responses before and after exercise

During our statistical analyses of the data, Bonferroni adjusted pairwise posthoc- comparisons were used. This analyze method may have been too strict to use. When further analysis with no adjustment for multiple comparisons were used, the results displayed clear reductions in both systolic and diastolic BP and MAP post exercise.

Compared to the BP values taken at rest pre exercise, the systolic BP were at most reduced by 18mmHg 30minutes post exercise. The diastolic BP was reduced by 5 mmHg, 5minutes post exercise and MAP were reduced by 8 mmHg, 5minutes post exercise.

Another potential reason why a reduction in BP post exercise did not materialize from our study may have been because we only measured the BP 30minutes post exercise after the INT and CON exercise. Which was opposite to the majority who had examined PEH subsequent 60-120minutes post exercise in a review done by MacDonald et al. 2002.

The PEH was measured for only 30minutes because of time constraints and because studies have shown that one can expect the largest BP reduction at the beginning of the recovery period. A published pilot study concluded that similar endurance exercise as the present study, gave the largest effect on PEH in the first ten minutes post exercise and that post exercise MAP values remained unchanged from 10 to 60minutes (Gjovaag et al. 2015). Based on this, in the present study, it was chosen to focus on the immediate BP responses in the first 30minutes post exercise.

During the exercises the resistance (Watt) was adjusted continuously on the bike after each participant heart rate target zone which was based on the intensity of the exercises (75% and 85% HFmax). This could have contributed that the duration of the moderate CON exercise was too long. Including the warmup for 10minutes, the duration of the CON exercise ended at 46 ± 7 minutes (Table 2). Our duration of the CON exercise were therefore approximately 15minutes longer than the recommended duration of 30minutes to attain the best PEH response (Björjesson, Kjeldsen and Dahlöf 2008).

Even though there were no significant reductions in systolic BP, diastolic BP and MAP after isocaloric high intensity INT or moderate CON exercise, does not mean that the main hypothesize was entirely incorrect. A recent study done by Molmen-Hansen et al. (2012) researched on a total of 88 patients (52±8 years, 39 women) with essential hypertension. The participants were randomized to isocaloric aerobic interval exercise (85-90% of VO2max), moderate intensity continuous exercise (60% of VO2max) or a control group. Twelve weeks of exercise resulted in significantly reduced systolic and diastolic 24 hour ambulatory BP after both types of exercises. Diastolic myocardial function improved in the interval exercise only, together with a reduced TPR and increased flow mediated vasodilatation. This study points toward that the BP reduction in essential hypertensive was intensity dependent. Since Molmen-Hansen et al. (2012) exercised their subjects three times per week for 12 weeks, this could mean that that the exercise effect on 24 h ambulatory BP was not just intensity dependent, but also dependent on a high exercise volume.

Exercise intensity and duration

Exercise intensity

Pescatello et al (1991) studied six mildly hypertensive and six normotensive participants to quantify the duration of PEH at different exercise intensities. In similarity to this present study Pescatello et al (1991) found no significant reductions in systolic BP or MAP after moderate intensity CON exercise at 40% and 70% of VO_{2max} in normotensive participants. He did however find a significant PEH response in the hypertensive group, strengthening the claim that reductions in BP are greater in individuals with higher initial BP (Forjaz et al. 2000). This could mean that the initial BP to the participants in this present study were not high enough to provoke a significant and large enough PEH response.

One of the reasons why findings might have failed to materialize in the normotensive participants in Pescatello's et al (1991) study, may be due to post exercise BP were measured with an ambulatory monitor while they went about their usual activities of daily living. The participants could have been more stressed during the daily conditions which could have led to a higher BP compared to the quiet rest of a laboratory.

Pescatello et al (1991) also found that exercise at 40% and 70% of VO_{2max} provoked similar PEH responses in systolic BP, as well as low intensity exercise reduced systolic BP as effectively as moderate intensity exercise (11 versus 9 mm Hg) in hypertensive compared with non-exercise days. Looking closer to the method Pescatello et al (1991) used, one discovers that the participants abstained from food four hours prior to the exercise studies. Having fasting participants could have had an influence on the PEH response.

Jones et al. (2007) also refers to results of reduced BP with moderate intensity at 40% of VO_{2max} on seven normotensive physically active men (28 ± 6). Jones et al. (2007) additionally concluded that the reduction in BP was similar following high intensity short duration (70%

VO2max, 30minutes) and moderate intensity of longer duration (40% VO2max, 50 ± 8minutes) exercise that was matched for total work done. The researchers findings displayed that total work done is possibly an important factor in determining the degree of acute PEH for normotensive. It could also mean that BP can be lowered by a session of rather low intensity exercise given that it is prolonged in nature, making their results very unique. Low intensity exercise could be advantageous as it may reduce the risks associated with high intensity exercise in vulnerable populations like hypertensive participants.

Comparing the use of how the intensity were controlled in this present study to Jones et al. (2007), one is to see that the intensity was controlled by keeping the resistance (watt) constant during the whole exercise. This mean that important variables like VO_2, HF, CO and stroke volume would change from start to end of exercise. Hence, intensity would differ not only between exercise modalities, but also within each exercise modality. Based on this, it was difficult to ascertain the effect of intensity on the PEH response in this study.

The magnitude of the PEH response may be affected by the intensity of the exercise and it was concluded that higher intensity exercise elicits longer duration of PEH (Forjaz et al. 2004). Forjaz et al. (2004) did nonetheless have the benefits of 23 participants compared with this study six participants. In addition to Forjaz et al. (2004), the research of Angadi, Bhammar and Gaesser (2015) supports this statement after they also discovered that the duration of PEH were greater after high intensity interval exercise (90-95% HFmax) than after moderate exercise (75-80% HFmax). Both Forjaz et al (2004) and Angadi, Bhammar and Gaesser (2015) exercises studies was, however, not isocaloric.

Unlike this present study which measured post exercise BP for only 30minutes after the exercise studies, Angadi, Bhammar and Gaesser (2015) measured their participants BP for three hours post exercise. Discovering that the PEH measured during the third hour were lower than control. Which could potentially mean that the 30 minutes of BP measuring in this present study were not long enough to detect a significant PEH response. Angadi, Bhammar and Gaesser (2015) exercise studies were also performed with a constant load making it problematic to ascertain the effect of intensity on the PEH.

Unlike the limited research on high intensity exercise, there is a larger collection of studies presenting significant PEH responses after moderate intensity CON exercise (Collier et al. 2008; Forjaz et al. 2004; Jones et al. 2007; Keese et al. 2011; MacDonald, MacDougall and Hogben. 1999a; MacDonald, MacDougall and Hogben. 1999b). The common denominator for the PEH response for the articles above was exercise intensities at ~65-75% of VO_{2max}, durations of 30-45minutes and an average of 16 participants. Supporting the earlier statement from the Norwegian directorate for health recommending moderate continuous aerobic exercise at 40-70% $VO2_{max}$ for 30 minutes to attain the best PEH effect (Björjesson, Kjeldsen and Dahlöf, 2008). But, if we were to compare the articles participanst against this present study six participants could mean the difference between their results and this study lack of.

If one takes a look at all the mentioned articles above low, moderate and high intensity exercise can all be used in the control of hypertension as long as the duration is regulated in proportion to the intensity degree. Considering the differences in results between studies can be related to the relative intensity at which participants completed the exercise, meaning what seemed as a high intensity for one population could be considered as low or moderate in another.

Exercise duration

Another variable that can affect the magnitude of PEH is the exercise duration, as it is believed that longer exercise durations may affect a larger PEH response. Meaning it is possible that the duration of exercise can affect the duration of the PEH effect.

To look further into this statement MacDonald, MacDougall and Hogben (1999a) created two different studies comparing exercise duration on PEH in one article. Study 1 included normotensive participants performing bouts of cycling for 15, 30 and 45minutes at 70% VO2peak. Study 2 included hypertensive participants performing bouts of cycling for 10 and 30minutes at 70% VO2peak.

Both studies documented that the duration of exercise did not play a significant role in determining the occurrence or the magnitude of PEH. In study 1 all bouts of 15, 30 and 45minutes of cycling induced similar PEH response in normotensive men. More interestingly study 2 showed that only 10minutes of exercise were sufficient to induce PEH in hypertensive individuals. The hypertensive participants in study 2 had higher resting BP and the magnitude of their PEH was larger than the normotensive participants in study 1. This again supports the suggestion stated earlier that the PEH effect after exercise are greater in those with higher initial BP (Forjaz et al. 2000). This study also suggest that a short duration endurance exercise may act as a non-pharmacological aid to hypertension.

In contrast, there is Forjaz et al. (1998), which also considered that exercise duration could play a role in achieving PEH. Forjaz et al. (1998) concluded that a longer exercise bout leads to a higher and longer PEH was opposite to MacDonald, MacDougall and Hogben (1999a). Forjaz et al. (1998) studied 10 normotensive participants with the hypothesis that CON sub maximal exercise could lead to a greater and longer BP fall after exercise than a short exercise bout. They used two cycle exercise trials with 25 and 45minutes at 50% of VO2peak. Results were that the PEH was higher and lasted longer after the 45minute exercise. Which also is in contrast with this present study which had no significant PEH after CON exercise (36 ± 7 minutes).

Possible mechanisms behind post exercise hypotension

Several articles and reviews have looked into the potential causes and mechanisms behind PEH. Knowledge of PEH can be useful help in the work of designing strategies against hypertension. The PEH response begins with the cessation of exercise. Cardiac output reduces from high exercising values more rapidly than TPR recovers. This imbalance in the two contributing factors of arterial pressure results in a hypotension that may be maintained for hours (Halliwill, 2001). Unfortunately, the mechanisms behind these responses are not fully identified yet.

This present study conversely resulted in a significant higher CO at 5 and 30minutes post exercise compared to the values taken at rest. The TPR on the other hand were significantly reduced 5minutes post exercise compared to the values taken at rest. The significantly higher CO values could be the reason why a PEH response did not materialize in the present study. These results agree with Kenney and Seals (1993) who stated that MAP are determined by CO and TPR which means that a decrease in MAP post exercise can be determined by a reduction in either one or both variables.

Even though the mechanisms behind PEH are not yet identified several studies suggests that reduced stroke volume and reduced TPR are important factors (Mota et al 2009; MacDonald, 2002; Halliwill, 2001). This statement is supported by a meta-analysis by Cornelissen and Fagard (2005), which concluded that aerobic exercise reduces BP due to a reduction in systemic vascular resistance. The 73 randomized controlled trials reported that the BP reduction was based on a fall in systemic vascular resistance in which the sympathetic nervous system and the renin angiotensin system seemed to be involved. The meta-analysis also revealed a significant decrease in systemic vascular resistance without changes in CO. The fact that a reduction of HF is compensated by a rise in stroke volume without change in CO is well-matched with the commonly accepted effect of aerobic endurance exercise on resting hemodynamics.

Despite limited literature on how the sympathetic nervous system and the renin angiotensin system can influence PEH, we cannot exclude them as potential underlying mechanisms. The arterial baroreflex and cardiopulmonary receptor reflex regulates the sympathetic nervous system during rest. During exercise the body has an increased need for oxygen and the baroreflex system is set to operate at higher BP and HF (Ichinose et al. 2008). Then after exercise the sympathetic vasoconstrictor nerve activity loss to skeletal muscle vascular beds reduces and the baroreflex system are reset to a lower BP than the BP taken before exercise (Floras et al. 1989). The resetting of baroreflex system to higher functioning levels during exercise to low levels post exercise indicates that the sympathetic nervous system has a central role in the mechanisms behind PEH.

Considering the limited research on the renin angiotensin system one can only speculate its influence on the PEH response. There is one study by Kiyonaga et al (1985), they found significant reductions in BP after 10 and 20 weeks of aerobic exercise at 50% $VO_{2\ max}$. Compared to the non-responders (1.95 $ng.ml^{-1}.h^{-1}$), the responders displayed a significant reduction in the plasma renin angiotensin activity (0.6 $ng.ml^{-1}.h^{-1}$). In that case, since the renin angiotensin system is an important contributor in blood volume homeostasis, reduced levels of plasma renin and angiotensin could perhaps play a role in the PEH response.

While many studies have focused on the sustained reduction in regional and systematic vascular resistance after exercise, Halliwill (2001) suggests that this sustained vasodilatation are associated with two alterations in the sympathetic vascular regulation defined as a neural and vascular component. Their conclusion was that hypotension was a result from persistent decreases in vascular resistance, mediated by the autonomic nervous system and vasodilator substances.

Changes in TPR seems to be the common PEH factor among the researchers. Although there was a significant reduction in TPR 5minutes post exercise in this present study, it was not sufficient enough to cause a PEH response. Brandaö et al. (2002) also observed no changes in TPR when they explored the impact of hemodynamic left ventricular function on post exercise BP in elderly participants. Participants were 23hypertensive and 18normotensive who completed a 45 min bicycle exercise at 50% $VO_{2\ max}$. The results were a significant higher reduction in BP and CO already at 15 and 30minutes post exercise in the hypertensive. Brandaö et al. (2002) suggested that the PEH response may had been more related to a reduction in CO rather than changes in TPR.

There are yet many unanswered questions about how PEH occurs and several unknown factors and mechanisms are still unidentified. It also raises the question if the occurrence of PEH are different between genders and if there are other mechanisms involved in the response for women than for the men. This topic can therefore be an exciting research arena for future work.

Constraints

There were some constraints in this study, some are already deliberated in the discussion chapter. Other constraints were the number of participants which was unfortunately lower than intended (N=6). Although the selection was small, it supports the findings of future work in a larger scale which is also necessary in order to generalize the present findings. There was also a desire to only research on un-medicated pre hypertensive participants, but unfortunately this population was too hard to find with the time that was at hand.

The exercise protocol of this thesis was not randomized which could have helped strengthened the design. But this was due to the fact that it was necessary to perform the INT exercise first, since the energy expenditure is the highest per unit time.

Another constraint was that it was not possible to measure the participants BP longer than 30minutes post exercise due to time limitations. The INT and CON sessions were all completed during the work day and to have the participants stay up to three hours was not feasible. The use of Bonferroni adjusted pairwise posthoc- comparisons for statistical analyzes may also have been too strict on this data set.

Conclusion

Based on the present results it looks like intensity has little to say on the PEH response. There is a possibility that perhaps the chosen intensities at 75% and 85% of HF max were too mild and therefore not strenuous enough due to the continuously watt adjustments during the INT and CON exercise.

Although there were no significant reductions of systolic BP, diastolic BP and MAP there was still a clear reduction in these factors post exercise with no adjustment for multiple comparisons. The systolic BP were at most reduced by -18mmHg, 30minutes post exercise. The diastolic BP was reduced by – 5 mmHg 5minutes post exercise and MAP were also reduced by – 8 mmHg 5minutes post exercise. Although there was a significant reduction in TPR 5minutes post exercise it was not sufficient enough to cause a PEH response.

The effects duration may have on the magnitude of the PEH response is still uncertain but one should consider the duration variable to be assessed together with the intensity variable when considering the exercise interventions.

In conclusion: The findings from this study demonstrates that physical exercise can reduce systolic and diastolic BP, MAP and TPR after isocaloric high intensity INT and moderate intensity CON exercise, but that the intensity was not important in governing the PEH response.

Future work

Regarding the future, there should be more research on isocaloric exercises with different intensities and durations. It is with isocaloric adjustments one can only truly compare the effect of the exercise characteristics one are looking for. Future work should also contain more research on women and their BP responses during and after endurance exercise.

Are women more or less susceptible to changes in BP? If one is to attain a PEH response, is this PEH response higher or longer in women compared to men? Is there a difference in genders when it comes to the contribution of factors and mechanisms behind PEH? These are all questions researchers should consider.

Literature

1. Angadi SS, Bhammar DM, Gaesser GA. 2015. Postexercise hypotension after continuous, aerobic interval and sprint interval exercise. *Journal of strength and conditioning research.* 29(10):2888-2893.
2. Bermudes AM, Vassallo DV, Vasquez EC and Lima EG. 2004. Ambulatory blood pressure monitoring in normotensive individuals undergoing two single exercise sessions: resistive exercise training and aerobic exercise training. *Arq Bras Cardiol.* 82:65-71, 57-64.
3. Bonsu B and Terblanche E. 2016. The training and detraining effect of high-intensity interval training on post-exercise hypotension in young overweight/obese women. *Eur J Appl Physiol.* 116(1): 77-84.
4. Börjesson M, Kjeldsen S and Dahlöf B. 2008. ‘’Hypertension’’. Bahr, R (ed). *Aktivitetshåndboken: Fysisk aktivitet I forebygging og behandling.* Oslo. Norwegian directorate for health. 25:327-342.
5. Brandão RMUP, Alves MJNN, Braga AMFW, Teixeira OTUN, Barretto ACP, Krieger EM & Negraõ CE. 2002. Postexercise blood pressure reduction in elderly hypertensive patients. *Journal of the American College of Cardiology*. 39(4):676-682.
6. Burl VL, Whelton P, Roccella EJ, Brown C, Cutler JA, Higgins M, Horan MJ, Labarthe D. 1995. Prevalence of hypertension in the US adult population: results from the Third National Health and Nutrition Examination Survey, 1988–1991. *Hypertension*. 25: 305–313.
7. Charloux A, Lonsdorfer EW, Richard R. Lampert E, Mammosser MO, Mettauer B, Geny B, Lonsdorfer J. 2000. A new impedance cardiograph device for the non-invasive evaluation of cardiac output at rest and during exercise: comparison with the ‘’direct’’ Fick method. *Eur J appl Physiol.* 82:313-320.
8. Collier SR, Kanaley JA, Carhart Jr R, Frechette V, Tobin MM, Hall AK, Luckenbaugh AN and Fernhall B. 2008. Effect of 4 weeks of aerobic or resistance exercise training on arterial stiffness, blood flow and blood pressure in pre-and stage 1 hypertensive. Journal of human hypertension. 22: 678-686.
9. Cornelissen VA and Fagard HR. 2005. Effects of endurance training on blood pressure, blood pressure regulating mechanisms and cardiovascular risk factors. *Hypertension.* 46(4): 667-75.

10. Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE, Pratt M, Ekelund U, Yngve A, Sallis JF, and Oja P. 2003. *American College of Sports Medicine.* 0195-9131/03/3508-1381.
11. Diehma N, Dickb F, Czuprina C, Lawalld H, Baumgartnera I, Diehmd C. 2009. Oscillometric measurement of ankle-brachial index in patients with suspected peripheral vascular disease: comparison with Doppler method. *Swiss Med Wkly*. Jun 27;139 (25-26):357-363.
12. Evenson KR, Stevens J, Thomas R, Cai J. 2004. effects of cardiorespiratory fitness on mortality among hypertensive and normotensive woemen and men. *Epidemiology*. 15:565-72.
13. Fagard RH and Cornelissen VA. 2007. Effect of exercise on blood pressure control in hypertensive patients. *Eur J Cardiovasc Prev Rehabil.* 14:12-7.
14. Floras JS, Sinkey CA, Aylward PE, Seals DR, Thoren PN and Marl AL. 1989. Postexercise hypotension and sympathoinhibition in borderline hypertensive men. *Hypertension* .14:28-35.
15. Forjaz CL, Santaella DF, Rezende Lo, Barretto AC and Negrão CE. 1998. Effect of exercise duration on the magnitude and duration of post-exercise hypotension. Arq Bras Cardiol. 70(2): 99-104.
16. Forjaz CL, Tinucci T, Ortega KC, Santaella DF, Mion D and Negrao CE. 2000. Factors affecting post-exercise hypotension in normotensive and hypertensive humans. *Blood Press Monit*. 5:255-62.
17. Forjaz CLM, Cardoso CG, Rezk CC, Santaella DF and Tinucci T. 2004. Postexercise hypotension and hemodynamics: the role of exercise intensity. *J sports med phys fitness.* 44:54-62.
18. Frayn KN. 1983. Calculation of substrate oxidation rates in vivo from gaseous exchange. *The American Physiological Society.* 83: 0161-7567.
19. Geleijnse JM, Kok FJ, Grobbee DE. 2004. Impact of dietary and lifestyle factors on the prevalence of hypertension in western populations. *Eur J Public Health.* 14:235-9.
20. Gjøvaag T, Øygarden M and Wold IM. 2015. Effects of moderate and high intenisty intermittet endurance exercise on post exercise hypotension. *European Congress of Sport Science.* Abstract ID 1963.

21. Halliwill JR. 2001. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc. Sports Sci. Rew*. 29(2):65-77.
22. Ichinose M, Sala-Mercado JA O'leary DS, Hammond RL, Coutsos M, Ichinose T, Pallante M and Iellamo F. 2008. Spontaneous baroreflex control of cardiac output during dynamic exercise, muscle metaboreflex activation, and heart failure. *The American Journal of Physiology and Heart Circulation Physiology*. 294:H1310-H1316.
23. Jones H, George K, Edwards B and Atkinson G. 2007. Is the magnitude of acute post exercise hypotension mediated by exercise intensity or total work done? *Eur J Appl Physiol.* 102: 33-40.
24. Kane-Gill SL, LeBlanc JM, Dasta JF and Devabhakthuni S. 2014. A Multicenter Study of the Point Prevalence of Drug-Induced Hypotension in the ICU. *Society of Critical Care Medicine and Lippincott Williams & Wilkins*. 42(10):2197-2203.
25. Keese F, Farinatti P, Pescatello L and Monteiro Walace. 2011. A comparison of the immediate effects of resistance, aerobic and concurrent exercise on post-exercise hypotension. Journal of strength and conditioning research. 25(5): 1429-1436.
26. Kenney MJ and Seals DR. 1993. Postexercise hypotension. Key features, mechanisms and clinical significance. *Hypertension*. 22:653-664.
27. Khoury S, Yarows SA, O'Brien TK, Sowers JR. 1992. Ambulatory blood pressure monitoring in a nonacademic setting: effects of age and sex. *Am J Hypertens.* 5:616–623.
28. Kiyonaga A, Arakawa K, Tanaka H and Shindo M. 1985. Blood pressure and hormonal responses to aerobic exercise. *Hypertension.* 7:125-131.
29. Lewington S, Clarke R, Qizilbach N, Peto R, Collins R, prospective studies collaboration. 2002. Age specific relevance of usual blood pressure to vascular morality. A meta-analysis of individual data from one million adults in 61 prospective studies. 360:1903-13.
30. Ling CHY, de Craen AJM, Slagboom PE, Gunn EDA, Stokkel MPM, Westendorp RGJ, Maier AB. 2011. Accuracy of direct segmental multi-frequency bioimpedance analysis in the assessment of total body and segmental body composition in middle-aged adult population. *Clinical Nutrition 30.* 610-615.

31. MacDonald JR, MacDougall JD and Hogben CD. 1999a. The effects of exercise duration on post-exercise hypotension. *Journal of human hypertension*. 14:125-129.
32. MacDonald JR, MacDougall JD and Hogben CD. 1999b. The effects on exercise intensity on post-exercise hypotension. *Journal of human hypertension*. 13: 527-531.
33. MacDonald JR. 2002. Potential causes, mechanisms and implications of post exercise hypotension. *Journal of human hypertension*. 16:225-236.
34. Mach C, Foster C, Brice G, Mikat RP and Porcari JP. 2005. Effect of exercise duration on postexercise hypotension. *Journal of Cardiopulmonary Rehabilitation*. 25:366-369.
35. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G et al. 2007. 2007 Guidelines for the management of arterial hypertension: the task force for the management of arterial hypertension of the European society of hypertension (ESH) and of the European society of cardiology (ESC). *J hypertension.* 25:1105-87.
36. Melo CM, Alencar Filho AC, Tinucci T, Mion Jr T and Forjaz CLM. 2006. Post exercise hypotension induced by low-intensity exercise in hypertensive women receiving captopril. *Blood press monit*. 11:183-189.
37. Molmen- Hansen HE, Stolen T, Tjonna AE, Aamot IL, Ekeberg IS, Tyldum GA, Wisloff U, Ingul CB, Stoylen A. 2011. Aerobic interval training reduces blood pressure and improves myocardial function in hypertensive patients. *European Journal of Preventive Cardiology.* 19 (2): 151-160.
38. Mota MR, Pardono E, Laila CJL, Arsa G, Bottaro M, Campbell CSG and Simões HG. 2009. Effects of treadmill running and resistance exercise on lowering blood pressure during the daily work of hypertensive participants. *Journal of strength and conditioning research.* 23 (8): 2331-2338.
39. Pescatello LS, Fargo AE, Leach CN, Scherzer JR and Scherzer HH. 1991. Short-term effect of dynamic exercise on arterial blood pressure. *Circulation.* 83:1557-1561.
40. Perronnet F, Massicotte D. 1991. Table of nonprotein respiratory quotient: An update. *Can J Sport Sci.* 16:23-29.
41. Reckelhoff JF. 2001. Gender differences in the regulation of blood pressure. Hypertension. *Americcan Heart Association.* 37:1199-1208.

42. Rietjens GJ, Kuipers H, Kester AD, Keizer HA. 2001. Validation of a computerized metabolic measurement system (Oxycon-Pro) during low and high intensity exercise. *Int J Sports Med.* May;22(4):291-4.
43. Sand O, Sjaastad ØV and Haug E. 2010. Menneskets fysiologi. Gyldendal Norsk Forlag AS. 7: 315-316.
44. Sandvik L, Erikssen J, Thaulow E, Erikssen G, Mundal R and Rokdahl K. 1993. Physical fitness as a predictor of mortality in healthy middle aged men. N Engl J Med. 328:533-7.
45. Santana HA, Moreira SR, Asano RY, Sales MM, Cordova M, Campbell CS, Espindola FS, Sposito AC, Nobrega OT, Simoes HG. 2013. Exercise intensity modulates nitric oxide and blood pressure responses in hypertensive older women. *Aqing Clin Exp Res.* 25(1): 43-8.
46. Scott JM, Esch BTA, Lusina SJC, McKenzie DC, Koehle MS, Sheel AW, Warbuton DER. 2007. Post exercise hypotension and cardiovascular response to moderate orthostatic stress in endurance trained males. *Appl. Physiol. Nutr. Metab.* 33:246-253.
47. Staessen J, Fagard R, Lijnen P, Thijs L, Van Hoof R, Amery A. 1990. Reference values for ambulatory blood pressure: a meta-analysis. *J Hypertens*. 8 (suppl 6): S57–S64.
48. Vasan RS, Larson MG, Leip EP, Evans JC, O'Donnell CJ, Kannel WB, et al. 2001. Impact of high normal blood pressure on the risk of cardiovascular disease. *N Engl J Med.* 345:1291-7.
49. Wiinber N, Hoegholm A, Christensen HR, Bang LE, Mikkelsen KL, Nielsen PE, Svendsen TL, Kampmann JP, Madsen NH, Bentzon MW. 1995. 24-h Ambulatory blood pressure in 352 normal Danish participants, related to age and gender. *Am J Hypertens.* 8:978–986.

Tables

Table 1. The women's characteristics prior to the study (N=6). Mean values and standard derivation (SD). Physiological responses where all measured in the resting state.

Variables	**Mean**	**SD**
Age	56.1	6.7
Height	168.5	5.3
Wight	67.3	8.5
BMI	23.8	3.1
BP (Sys, mmHg)	127.6	9.0
BP (Dia, mmHg)	82.1	1.1
Pulse wave velocity	6.5	1.6
HF	67	10
Lactate	1.2	0.4
RPE (0-10)	0.17	0.40

Explanation: SD = standard deviation, BMI = Body mass index, BP = Blood pressure, Sys = Systolic, Dia = Diastolic, mmHg = millimeter of mercury, HF = Heart frequency, RPE = Ratings of perceived exertion.

Table 2. Mean ± standard deviation on physiological response, watt and RPE *during* INT and CON exercise for the six women in this study. Blood lactate concentration is mean post exercise.

Variables	INT [a]	CON
Duration[b]	28 ± 0 *	36 ± 7
O2 uptake (ml/kg/min)	24.7 ± 2.8	20.2 ± 3.3
O2uptake (%$VO2_{max}$)	78 %	64 %
HF (beats/min)	151 ± 12 *	136 ± 12
HF (%HF_{max})	87 % *	78%
Watt	105 ± 17	86 ± 12
Watt (%$Watt_{max}$)	54 % *	44 %
Lactate (mmol/L)	2.5 ± 1.0	2.2 ± 1.0
RPE post exercise (0-10)	6 ± 2	4 ± 3

[a] = The values of the INT exercise is based on the mean of the four intervals, the active rest intervals are not included [b] = 10minute warm up is not included for the mean duration values. * = Significant difference between INT and CON exercise; * P < 0,05.

Explanation: O2 uptake = oxygen uptake, ml/kg/min = milliliters per kilo per minute, $VO2_{max}$ = Maximum oxygen uptake, HF= Heart frequency, HF_{max} = Maximum heart frequency, $Watt_{max}$ = Maximum watt, mmol/L= millimoles per liter, RPE = Ratings of perceived exertion (0-10).

Table 3. Respiratory quotient (RQ)

" Non- protein " respiratory quotient, caloric value and percentage contribution from carbohydrate and fat to energy metabolism (Perronnet and Massicotte. 1991).

Vedlegg 3. "Non-protein" respiratorisk kvotient (RQ), kalorisk verdi og prosentvis bidrag fra karbohydrat og fett til energistoffskiftet

Respiratorisk kvotient	Kcal per liter oksygen	Prosent kalorier fra	
		Karbohydrat	Fett
0.70	4.851	0	100.0
0.71	4.858	2.3	97.7
0.72	4.870	6.0	94.0
0.73	4.881	9.6	90.4
0.74	4.893	13.2	86.8
0.75	4.904	16.8	83.2
0.76	4.916	20.4	79.6
0.77	4.927	23.9	76.1
0.78	4.939	27.4	72.6
0.79	4.951	31.0	69.0
0.80	4.962	34.5	65.5
0.81	4.974	38.0	62.0
0.82	4.985	41.4	58.6
0.83	4.997	44.9	55.1
0.84	5.008	48.3	51.7
0.85	5.020	51.7	48.3
0.86	5.032	55.1	44.9
0.87	5.043	58.5	41.5
0.88	5.055	61.9	38.1
0.89	5.066	65.3	34.7
0.90	5.078	68.6	31.4
0.91	5.089	71.9	28.1
0.92	5.101	75.3	24.7
0.93	5.112	78.6	21.4
0.94	5.124	81.8	18.2
0.95	5.136	85.1	14.9
0.96	5.147	88.4	11.6
0.97	5.159	91.6	8.4
0.98	5.170	94.8	5.2
0.99	5.182	98.0	2.0
1.00	5.189	100.0	0

Perronnet F, Massicotte D. Table of Nonprotein Respiratory Quotient: An update. Can J Sport Sci 16:23-29, 1991

Figures

Figure 1.

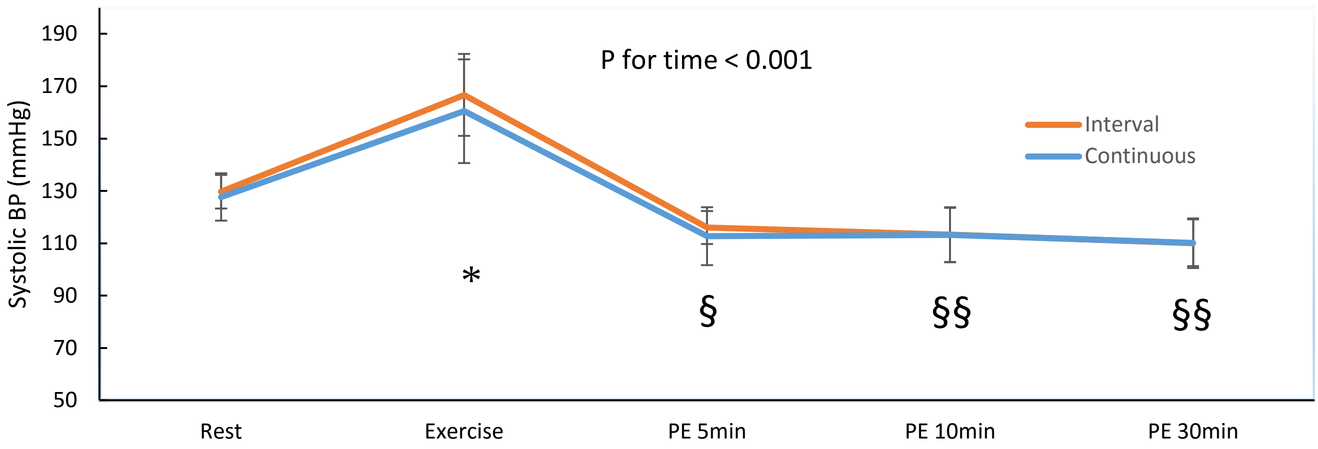

Figure 1. Systolic BP in rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

BP = Blood pressure, PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency. Max = maximum. * = significantly different from rest, * $p < 0.05$, § = significantly different from exercise, § $p < 0.05$, §§ $p < 0.01$.

Figure 2.

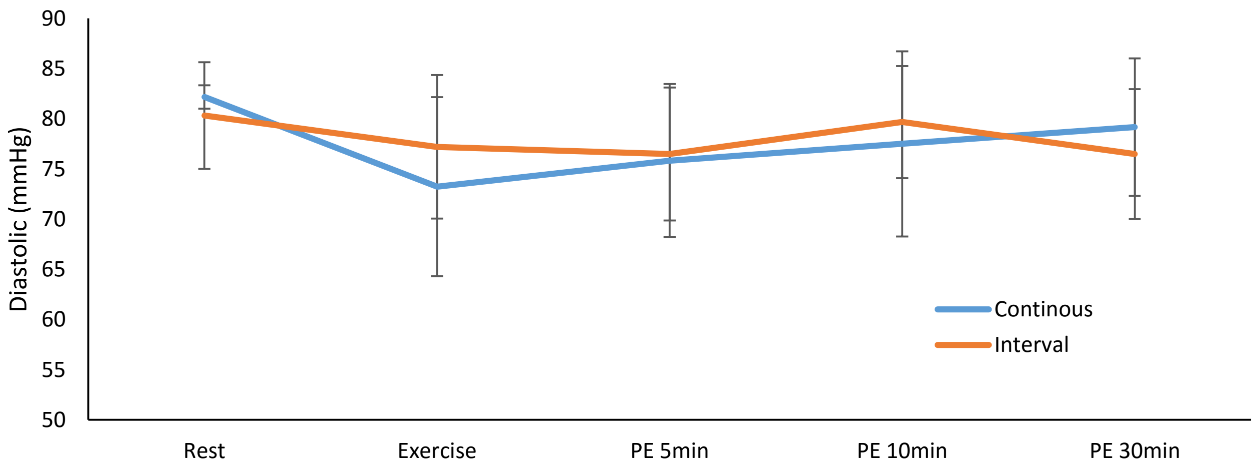

Figure 2. Diastolic BP in rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

BP = Blood pressure, PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency. Max = maximum BP = Blood pressure, PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency. Max = maximum.

Figure 3.

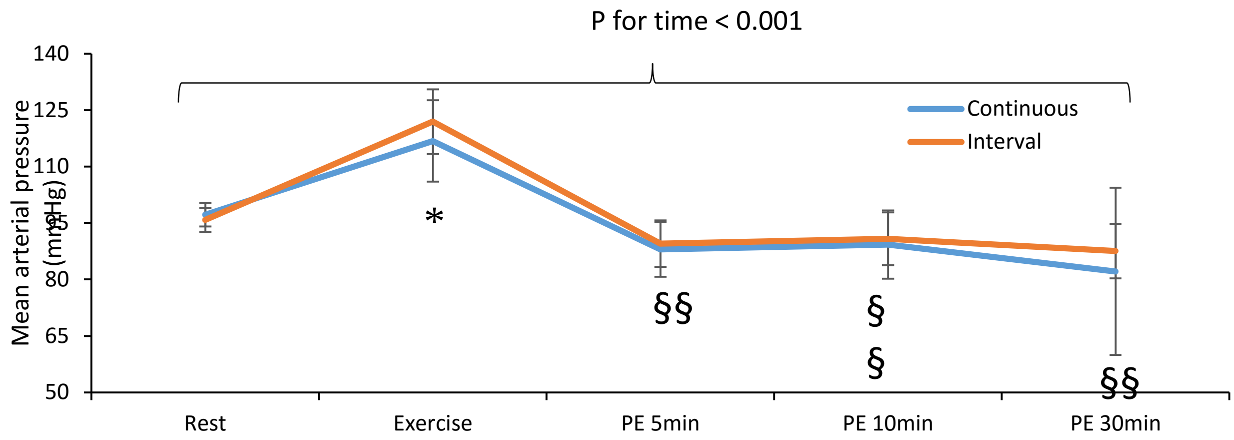

Figure 3. Mean arterial pressure from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency, Max = maximum. * = significantly different from rest, * $p < 0.05$. § = significantly different from exercise, § $p < 0.05$, §§ $p < 0.01$.

Figure 4.

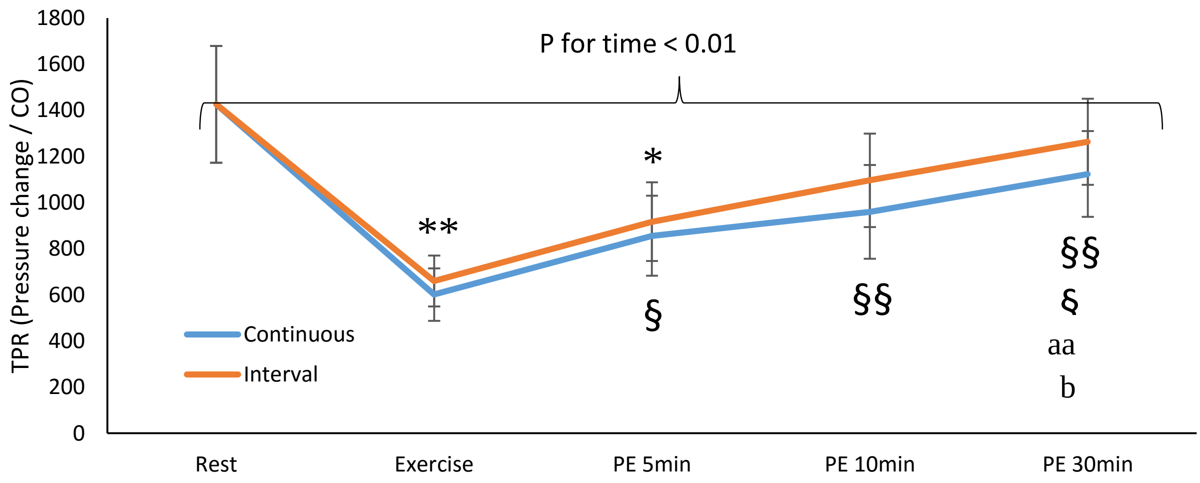

Figure 4. Total peripheral resistance from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

PE = post exercise, Min = minutes, CO = cardiac output, INT = interval, CON = continuous. HF = heart frequency, Max = maximum. * = significantly different from rest, * $p < 0.05$. ** $p < 0.01$, § = significantly different from exercise, § $p < 0.05$, §§ $p < 0.01$, §§§ $p < 0.001$. a = significantly different from PE 5min, aa $p < 0.01$. b = significant differently from PE 10min, b $p < 0.05$.

Figure 5.

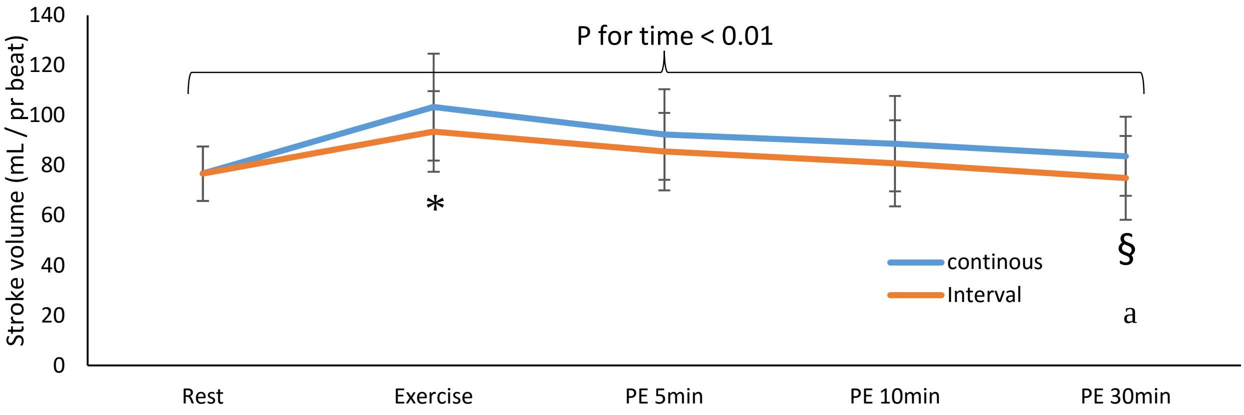

Figure 5. Stroke volume from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

mL = milliliter, PE= post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency, Max = maximum. * = significantly different from rest, * $p < 0.05$, § = significantly different from exercise, § $p < 0.05$, a = significantly different from PE 5min, a $p < 0.05$.

Figure 6.

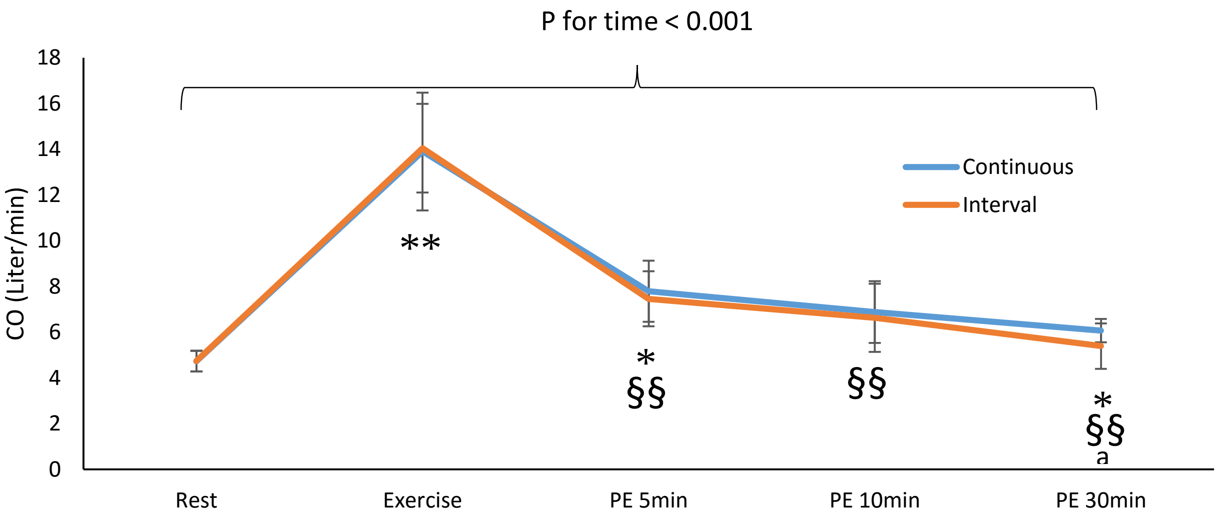

Figure 6. Cardiac output from rest, during exercise, and post exercise at 5, 10 and 30minutes for six normotensive women with high normal BP carrying out high intensity INT and CON endurance exercise (mean and standard derivation). Exercise are mean values for the entire exercise session. The active rest intervals are excluded for the high intensity INT exercise. The high intensity INT exercise was carried out at 85% HF max. The CON exercise was carried out at 75% HF max.

PE = post exercise, Min = minutes, INT = interval, CON = continuous. HF = heart frequency, Max = maximum * = significantly different from rest, * $p < 0.05$. ** $p < 0.01$, § = significantly different from exercise, §§ $p < 0.01$, a = significantly different from PE 5min, a $p < 0.05$.

Appendix

Appendix 1. Physical Activity Questionnaire (IPAQ).

Vi er interessert i informasjon om ulike former for fysisk aktivitet som folk driver med i dagliglivet. Spørsmålene gjelder tiden du har brukt på fysisk aktivitet de **siste 7 dagene**. Vennligst svar på alle spørsmålene uansett hvor fysisk aktiv du selv synes du er. Tenk på aktiviteter du gjør på jobb, som en del av hus- og hagearbeid, for å komme deg fra et sted til et annet, og aktiviteter på fritiden (rekreasjon, mosjon og sport). Tenk på all **meget anstrengende** aktivitet du har drevet med de **siste 7 dagene**. **Meget anstrengende** aktivitet er aktivitet som krever hard innsats og får deg til å puste mye mer enn vanlig. Ta bare med aktiviteter som varer minst 10 minutter i strekk.

1. Hvor mange dager i løpet av de **siste 7 dagene** har du drevet med **meget anstrengende** fysisk aktivitet som tunge løft, gravearbeid, aerobics, løp eller rask sykling?

   ____ dager

   Ingen meget anstrengende aktivitet *Gå til spørsmål 3*

2. Hvor lang tid brukte du vanligvis på **meget anstrengende** fysisk aktivitet på en av disse dagene?

   ____ timer per dag

   ____ minutter per dag

   - ☐ Vet ikke/usikker

Tenk på all **middels anstrengende** aktivitet du har drevet med de **siste 7 dagene**. **Middels anstrengende** aktivitet er aktivitet som krever moderat innsats og får deg til å puste litt mer enn vanlig. Ta bare med aktiviteter som varer minst 10 minutter i strekk.

3. Hvor mange dager i løpet av de **siste 7 dagene** har du drevet med **middels** anstrengende fysisk aktivitet som å bære lette ting, jogge eller sykle i moderat tempo? Ikke ta med gange.

   ____ dager

   Ingen middels anstrengende aktivitet *Gå til spørsmål 5*

4. Hvor lang tid brukte du vanligvis på **middels anstrengende** fysisk aktivitet på en av disse dagene?

   ____ timer per dag

   ____ minutter per dag

   - ☐ Vet ikke/usikker

Tenk på tiden du har brukt på å **gå** de **siste 7 dagene**. Dette inkluderer gange på jobb og hjemme, gange fra et sted til et annet eller gange som du gjør på tur eller som trening på fritiden.

5. Hvor mange dager i løpet av de **siste 7 dagene gikk** du i minst 10 minutter i strekk?

   ____ dager

   - ☐ Gikk ikke → *Gå til spørsmål 7*

6. Hvor lang tid brukte du vanligvis på å **gå** på en av disse dagene?

   ____ timer per dag

   ____ minutter per dag

   - ☐ Vet ikke/usikker

Det neste spørsmålet omfatter all tid du tilbrakte **sittende** på ukedagene i løpet av de **siste 7 dagene**. Inkluder tid du har brukt på å sitte på jobb, hjemme, på kurs og på fritiden. Dette kan tilsvare tiden du sitter ved et arbeidsbord, hos venner, mens du leser, eller sitter eller ligger for å se på TV.

7. Hvor lang tid brukte du på å **sitte** på en **vanlig hverdag** i løpet av de **siste 7 dagene**?

   ____ timer per dag

   ____ minutter per dag

   - ☐ Vet ikke/usikker

8. Tenk tilbake i tid. Hvor ofte drev du med fysisk aktivitet eller idrett så mye at du ble andpusten og/eller svett da du var:

   Sett ett kryss for hver aldersgruppe

	< 10 år	**10-14 år**	**15-20 år**
Aldri			
Mindre enn en gang/måned			
1-3 ganger/måned			
1 gang/uke			
2-3 ganger/uke			
4-6 ganger/uke			
Hver dag			

Appendix 2. Consent declaration.

Jeg er villig til å delta i prosjektet

Sted og dato

Deltakers signatur

Deltakers navn med trykte bokstaver

Jeg bekrefter å ha gitt informasjon om prosjektet

Sted og dato

Signatur

Rolle i prosjektet

Appendix 3. Rated Perceived Exertion scale.

1 - 10 Borg Rating of Perceived Exertion Scale	
0	Rest
1	Really Easy
2	Easy
3	Moderate
4	Sort of Hard
5	Hard
6	
7	Really Hard
8	
9	Really, Really, Hard
10	Maximal: Just like my hardest race