THE EFFECT OF PHYSICAL EXERCISE ON BODY FAT, METABOLIC HEALTH INDICATORS AND CARDIORESPIRATORY FITNESS IN OVERWEIGHT SEDENTARY WOMEN

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ABSTRACT


Introduction. Physical activity, sedentary behavior and overweight have been widely studied in health-related research. The guidelines for physical activity were updated in 2018, emphasizing the need for the reduction of sedentary behavior and thus its effects on health. It is commonly accepted that physical activity is related to weight management, cardiorespiratory fitness and healthier blood lipid profile. The purpose of this current work was to study health-effects of the eight-week exercise-intervention, following the official guidelines, within overweight sedentary females.

Methods. This study was done as a part of a larger RCT-study, in which the health effects of combined sauna-bathing and exercising were studied. The original study consisted of 49 participants, but in this study only the overweight females were included (n=37). The participants were randomly divided into a control group (no-intervention, n=13) and an experimental group (exercise-intervention, n=24). The participants age was 49 ± 8.7 yrs., BMI 31.9 ± 3.3 kg/m², height 167 ± 7 cm and body mass 89.1 ± 13 kg. The pre- and post-measurements were conducted with both groups eight weeks apart. The measurements included a body composition measurement with DXA, an indirect VO₂max-test and a fasting blood sample to determine total cholesterol, HDL, LDL, triglycerides and blood glucose. Both groups were instructed to follow their normal daily routines and eating habits. The exercise training intervention included training sessions three times a week for one hour at the time. The design of the training program followed the official guidelines for physical activity. Each training consisted of 10-minute warm-up, 20-minute strength training and 30-minute endurance training, for a total of 150 minutes of exercise in a week.

Results. Fat mass (-1.5 ± 1.5 kg), fat percent (-1.1 ± 1.1 %) and android (central adiposity) fat mass (-0.2 ± 0.2 kg) decreased and VO₂max (+4.2 ± 5.7 ml/kg/min) increased in the experimental group compared to the control group between the pre- and post-measurements (p<0.05). No changes were observed in metabolic health indicators or in fat distribution.

Conclusion. The main finding was that guideline-based exercise intervention is an effective way to decrease both total fat mass and android fat mass. In addition, an important finding was that during exercise-induced weight-loss, it is possible to maintain fat-free mass unchanged. No changes were observed in metabolic health indicators or in fat distribution, which might be due to short intervention duration or close to normal blood lipid values in the pre-measurements.

Key words: weight loss, android fat, fat distribution.
# ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td>ATP</td>
<td>adenosine triphosphate</td>
</tr>
<tr>
<td>BMI</td>
<td>body mass index</td>
</tr>
<tr>
<td>BMR</td>
<td>basic metabolic rate</td>
</tr>
<tr>
<td>DXA</td>
<td>dual-energy X-ray absorptiometry</td>
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<tr>
<td>EPOC</td>
<td>post exercise oxygen consumption</td>
</tr>
<tr>
<td>FFM</td>
<td>fat-free mass</td>
</tr>
<tr>
<td>FM</td>
<td>fat mass</td>
</tr>
<tr>
<td>HBA1</td>
<td>glycated hemoglobin</td>
</tr>
<tr>
<td>HHS</td>
<td>The U.S. Department of health and human service</td>
</tr>
<tr>
<td>HDL</td>
<td>high density lipoprotein</td>
</tr>
<tr>
<td>KCAL</td>
<td>kilocalorie</td>
</tr>
<tr>
<td>LDL</td>
<td>low density lipoprotein</td>
</tr>
<tr>
<td>MAX HR</td>
<td>maximum heart rate</td>
</tr>
<tr>
<td>MET</td>
<td>metabolic equivalent</td>
</tr>
<tr>
<td>RMR</td>
<td>resting metabolic rate</td>
</tr>
<tr>
<td>TDEE</td>
<td>total daily energy expenditure</td>
</tr>
<tr>
<td>UKK</td>
<td>UKK-Institution</td>
</tr>
<tr>
<td>VF</td>
<td>visceral fat</td>
</tr>
<tr>
<td>VO$_2$max</td>
<td>maximal oxygen consumption</td>
</tr>
<tr>
<td>WC</td>
<td>waist circumference</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
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1 INTRODUCTION

Obesity, defined as an excess of adipose tissue (Wellens et al. 1996), has been growing in numbers over the world for the past decades. Inside the European Union nearly 50 % of the adults were overweight and 17 % of them were obese (Stevens et al. 2012) and in Finland, in 2017 every fourth adult was obese (Koponen et al. 2017). The weight gain is a result of positive energy balance over time, which means the energy intake is greater than the energy expenditure (Marlatt & Ravussin, pp. 38). Sedentary lifestyle and the prolonged lack of physical activity connive to weight gain (Hu et al. 2003) and are overall primarily related to health (Lee et al. 2012). In 2018, the U.S. Department of health and human services (HHS) gave updated recommendations for weekly physical activity for health (HHS, 2018). The new guidelines highlight the importance of the reduction of sedentary behavior (HHS, 2018).

Overweight and obesity are known risk factors for metabolic abnormalities, such as dyslipidemia (Klop et al. 2013) and elevated blood sugar levels (Guyton & Hall, 2006, pp. 951). However, not all overweight and obese individuals display these conditions (Goncalves et al. 2016), which is explained by the distribution of the body fat (Ross et al. 1996). A large deposit of body fat in the abdominal section of the body in known to be an independent risk factor for metabolic abnormalities (Pouliot et al. 1992; Despres, 1993). In addition, excess amount of body fat is associated with low levels of cardiorespiratory fitness (McGavock et al. 2012).

The options for weight loss are reducing energy intake (a diet), increasing energy expenditure (exercise) or the combination of the two (a diet + exercise) (Ross et al. 2000). Exercise offers sustainability to weight loss and helps to maintain the results (Donnelly et al. 2009) Increased levels of exercise may independently lead to reductions of body fat (Schmitz et al. 2003) and improvements in metabolic abnormalities (Leon & Sanchez, 2001). The purpose of this study is to gain knowledge of exercise-induced changes in body composition, metabolic health indicators (blood lipids and blood sugar) and cardiorespiratory fitness in sedentary overweight women. Exercise-intervention was planned based on the official physical activity guidelines from HHS (HHS, 2018).
2 PHYSICAL ACTIVITY AND SEDENTARY BEHAVIOUR

Physical activity, as well as sedentary behavior, have been widely studied in health-related research. In daily life physical activity can be categorized, for example, into work-related, athletic, training, household, or other activities. “Physical activity” should not be confused with “exercise”, which differs from physical activity for being planned and organized and aiming to improve or maintain physical fitness. Physical fitness, on the other hand, is a set of qualities that are either skill- or health-related. (Carpensen et al. 1985.) Pate et al. (2008) define sedentary behavior as daily activities, in which energy expenditure does not increase considerably above the resting level. These kinds of activities include sleeping, sitting and lying down. In addition, they state that light physical activity such as slow walking, preparing dinner, and doing dishes is often grouped with sedentary behavior. (Pate et al. 2018.) Furthermore, the term inactivity is widely used and refers to a lack of physical activity, as in not meeting the recommendations (Lee et al. 2012).

2.1 Physiology of physical activity

Human energy system involves moving energy via chemical bonds. Chemical work refers to the synthetization of cellular molecules such as glycogen and triacylglycerol, while transport work refers to transfer of substances among cells. (McArdle et al. 2015, pp. 118–131.) Total daily energy expenditure (TDEE) comprises the dietary energy requirements for non-growing individuals. Three factors impacting on TDEE are thermogenic effect of feeding (10 %), thermic effect of physical activity (15-30 %) and resting metabolic rate (RMR) (60-75 %). In the waking state humans need a minimum level of energy to maintain vital functions. This energy necessity is a basal metabolic rate (BMR) and it is dependent on individuals overall body size, age, gender, health/fitness status, fat-free body mass, hormonal status and body temperature. (McArdle et al. 2015, pp 192.)

The intensity of physical activity is usually presented as the number of METs, which stands for metabolic equivalent. MET is a commonly used physiological model that embodies a simple method for stating energy cost of physical activities as multiples of resting metabolic rate
(RMR). The value that is equal to one MET (3.5 ml O₂·kg⁻¹·min⁻¹ or 1 kcal·kg⁻¹·h⁻¹) was first driven from resting O₂ consumption of one person, a 70 kg and 40-year-old man. (Byrne et al. 2005; Ainsworth et al. 2011.) Moderate-to-vigorous physical activity is all physical activity that elicits at least 3 MET (Katzmarzyk et al. 2017), as shown in figure 1. Vigorous-intensity activity requires 6.0 or more METs, when moderate-intensity is anything between 3.0 and 5.9 METs. Light-intensity activity is any waking behavior that is non-sedentary and requires less than 3 MET. (HHS, 2018.)

<table>
<thead>
<tr>
<th>Sedentary behaviour:</th>
<th>Light physical activity</th>
<th>Moderate- (3-6 METs) to vigorous (over 6 METs) physical activity, that meets the guidelines for physical activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Laying down</td>
<td></td>
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<tr>
<td>- Sitting</td>
<td></td>
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<tr>
<td>- Standing</td>
<td></td>
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<tr>
<td>1.5 MET</td>
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<tr>
<td>3 MET</td>
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<tr>
<td>6 MET</td>
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</tbody>
</table>

Figure 1. The continuum of physical activity from sedentary to vigorous physical activity (Vasankari, 2014).

2.2 Guidelines for physical activity

World Health Organization has given global recommendations on physical activity for health, with the last guidelines being published in 2010 (WHO, 2010). However, the U.S. Department of health and human Service (HHS) published updated physical activity guidelines for Americans in 2018 (HHS, 2018) and UKK-Institution updated the Finnish guidelines for physical activity in 2019 (UKK, 2019). The guidelines are pertinent for all healthy adults, who are 18-64 years old. The updated guidelines emphasize the need for the reduction of sedentary behavior and thus its negative effects on health. In addition, the obligation that physical activity has to occur in bouts of at least 10 minutes has been eliminated, pointing out that any physical activity is considered better than none. (HHS, 2018; UKK, 2019.) The new guidelines are shown in table 1.

1. Adults should move more and sit less throughout the day. Some physical activity is better than none. Adults who sit less and do any amount of moderate-to-vigorous physical activity gain some health benefits.

2. For substantial health benefits, adults should do at least 150 minutes (2 hours and 30 minutes) to 300 minutes (5 hours) a week of moderate-intensity, or 75 minutes (1 hour and 15 minutes) to 150 minutes (2 hours and 30 minutes) a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. Preferably, aerobic activity should be spread throughout the week.

3. Additional health benefits are gained by engaging in physical activity beyond the equivalent of 300 minutes (5 hours) of moderate-intensity physical activity a week.

4. Adults should also do muscle-strengthening activities of moderate or greater intensity and that involve all major muscle groups on 2 or more days a week, as these activities provide additional health benefits.

Physical activity guidelines for Americans divides levels of activity to four classes: inactive, which means there is not any moderate- to vigorous-intensity physical activity, insufficiently active, which means that there is some moderate- or vigorous-intensity physical activity but less than stated in the guidelines, active, which means the level of activity is equivalent from 150 minutes to 300 minutes of moderate-intensity physical activity per week, and highly active, which means the level of physical activity is equivalent to more than 300 minutes of moderate-intensity physical activity per week. (HHS, 2018.)

According to Sallis et al. (2016), 77% of the world population achieve the guidelines, which means 23% do not. However, variations between continents and countries were notable. In 2018, the Finnish Ministry of Education and Culture published a report evaluating Finns levels of physical activity between years 2011 and 2018 (Husu et al. 2018). The report is based on the results of FINFIT 2017 - population research, which was carried out between 2017-2018, and completed with the results of two earlier studies from Murto et al. (2017) and Koskinen et al. (2011). The report indicated that very few Finns meet the guidelines for physical activity. Only
20% of participants were physically active 150 minutes a week, in the way that physical activity occurred in sessions of at least 10 minutes. Women met the endurance guidelines (150 min/week) slightly more often than men. In addition, only 11% met the absolute guidelines, including strength training. Based on the report, strength training seems to be way more common among younger population, whereas endurance training did not differ as perceptibly.

2.3 Physical activity’s relationship with health

It is known that physical activity is essentially related to health (Kesäniemi et al. 2001). In several studies, it has been observed that physical activity reduces the risk of multiple conditions, such as cardiovascular disease (Gert et al. 1996) and metabolic syndrome (Katzmarzyk et al. 2003; Lakka & Laaksonen, 2007), as well as coronary heart disease (Morris et al. 1953; Sattelmaier et al. 2011), type 2 diabetes (Jeon et al. 2007; Aune et al. 2015), colon cancer (Wolin et. Al. 2009), breast cancer (Friedenreich, 2011; Wu et al. 2013) and all-cause mortality (Lollgen et al. 2009; Wen et al. 2011). According to Lee et al. (2012), inactivity is responsible for six percent of the burden of coronary heart disease, ten percent of breast cancer and of colon cancer, and seven percent of type 2 diabetes. Thus, according to the authors (Lee et al 2010), by eliminating inactivity, six to ten percent of these diseases could be removed and life expectancy increased. In addition, physical activity is related to weight management and to the prevention of obesity (Hu et al. 2003: Lakka & Laaksonen, 2007).

Although physical activity is strongly related to mortality and non-communicable disease, sedentary behavior is also a risk factor, independent of the amount of physical activity (Hu et al. 2003; Koster et al. 2012). Hu et al. (2003) showed in a broad prospective cohort study conducted between 1992-1998 in the USA, that each two hours per day increment in TV-watching was related with a 14% gain in risk of type 2 diabetes and a 23% gain in obesity independent of exercise levels. According to Husu et al. (2018) Finns spent in average 8.5 hours of their day sedentary, which is similar to the results from Canada, where the average sedentary time was 9.7 hours (Colley et al. 2011) and from the U.S., where the average was 8 hours (Matthews et al. 2008). It is crucial to recognize that any physical activity is beneficial, but for substantial health benefits the level of physical activity should meet the guidelines (HHS, 2018).
3 CARDIORESPIRATORY FITNESS

Cardiorespiratory fitness indicates the capacity of the cardiovascular and respiratory systems to provide oxygen during physical activity. The measure used to signify cardiorespiratory fitness is maximal oxygen consumption (VO$_{2\text{max}}$). (Blair et al. 1996.) VO$_{2\text{max}}$ is one of the components of physical fitness and along with other components, such as body composition, muscular strength and endurance and flexibility (Pollock et al. 1998), it is strongly related to health (Blair et al. 1996).

3.1 Physiology of cardiorespiratory fitness

The ventilatory system regulates the gas exchange in order to provide constant oxygen supply. Ventilation refers to the process, where oxygen is transferred from the air to the blood through the lungs. In blood, oxygen molecules join with hemoglobin, which is responsible for carrying them to cells to be used in energy production. The basal oxygen requirement of the human body is 0.2 to 0.4 L per minute, but during physical activity the requirement may increase up to 4-5 L per minute. (McArdle et al. 2015, 341–353.)

The rate of oxygen consumption and transfer is limited. It is presented as a value of VO$_{2\text{max}}$, which refers to the maximal rate at which oxygen can be transported to active muscles. (Blair et al. 1996.) The VO$_{2\text{max}}$ depends on maximal cardiac output (maximal heart rate and stroke volume) and the arteriovenous oxygen difference of the body (Mitchell & Blomqvist, 1971). VO$_{2\text{max}}$ is affected by gender (Lagestad et al. 2018), age (Radak et al. 2019), heredity (Williams et al. 2017), body mass (Lolli et al. 2017), body composition (Nogueira et al. 2016), habitual level of physical activity (Lagestad et al. 2018), exercise training (King et al. 1991) and smoking (Kim, 2018).

VO$_{2\text{max}}$ can be measured with gas analyzers, but it is rather time-consuming and expensive. This direct measurement is a standard index of cardiorespiratory fitness. However, indirect measurements are widely used, since there is no need for laboratory settings. Indirect measurements of VO$_{2\text{max}}$ are based on the widely acknowledged fact that there is a linear
relation between heart rate and the work performed, when the metabolic rate, circulation and respiration have reached a “steady-state” response to submaximal work and subsequent extrapolation to maximal heart rate. (Andersen et al. 1971.)

3.2 Cardiorespiratory fitness’ relationship with health

Higher level of cardiorespiratory fitness is related to reduced risk of mortality (Blair et al. 1996; Lee et al. 2011), metabolic syndrome (Earnest et al. 2013) and cardiovascular disease (Kodama et al. 2009). According to Pletnikoff et al. (2016), higher VO2max was related to lower risk of lung cancer in men. McGavock et al. (2012) studied cardiorespiratory fitness as a predictor of becoming overweight among children aged 6-15 years old. According to their study, decreases in fitness over time (1-2 years) and overall low cardiorespiratory fitness increased the risk of weight gain and being overweight. Children with low cardiorespiratory fitness, when compared to fit peers, had a 3.5 times greater risk of being overweight. Reductions in cardiorespiratory fitness were independently and significantly correlated with increases in BMI. (McGavock et al. 2012.) Even though low cardiorespiratory fitness is a predictor for further obesity (McGavock et al. 2012), being obese does not automatically mean having a low level of cardiorespiratory fitness (Kunutsor & Laukkanen, 2019). Furthermore, Kokkinos et al. (2019) showed in their study that high level of cardiorespiratory fitness protects from heart failure despite the BMI. In their study BMI and cardiorespiratory fitness were assessed in 20 254 male veterans and the observation was that the risk for heart failure was similar within normal weight, overweight and obese participants when adjusted for cardiorespiratory fitness.

Overall low cardiorespiratory fitness is exceedingly related to poorer health. For this reason, sedentary population and especially sedentary children should be encouraged to exercise at least to achieve recommended levels (HHS, 2018.) In the study of King et al. (1991), as well as in various other studies (Pollock et al. 1998; Huang et al. 2005; Tomoaki et al. 2014; Delextrat et al. 2016), it has been proven that endurance exercise, practiced in needed intensities, frequencies and durations, will improve cardiorespiratory fitness. However, for the sedentary population meeting the official guidelines should be a priority (HHS, 2018).
4 OVERWEIGHT AND OBESITY

‘Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health’ (WHO, 2018). ‘Obesity is defined as excess of adipose tissue’ (Wellens et al. 1996). Despite the source, obesity and overweight is described the same; an excess amount of body fat. This extra fat is a repercussion of imbalance between total energy intake and total energy expenditure. When the energy intake exceeds energy expenditure for a prolonged period of time, overweight will occur. If this balance is not corrected, it will ultimately lead to obesity. (Eknoyan, 2006.) The body does not store fat for nothing, as it is a crucial feature for human to survive during nutritional deprivation conditions, but unfortunately in today’s western society it has become a burden (Marlatt & Ravussin, 2018, pp. 38).

In history, this excess amount of body fat was seen as a sign of good health and as an advantage, but then in the first decades of 20th century, thanks for the documentaries of the insurance industry, it was defined as a disease with pathologic and pathophysiologic complications (Eknoyan, 2006). In our days, the WHO has declared it a global epidemic and a worldwide public health crisis. Overweight and obesity has grown in number over the world in the past 30 years, as shown in figure 2. In 2012, it was estimated that 35 % of all the adults over the age of 20 are overweight (BMI ≥ 25 kg/m²) and 11 % of them are obese (BMI ≥ 30 kg/m²). In the European Union, about half of all adults were at least overweight and 17 % of them obese. (Stevens et al. 2012.) In Finland, in 2017 2/3 of women and 3/4 of men were overweight and every fourth adult was obese. Furthermore, there were noticeable increments in overweight and obesity rates from 2011 to 2017. If the rates keep increasing at the same pace, in ten years every third 40-year-old would be obese. (Koponen et al. 2017.)
4.1 Classification of obesity

In 1832 Adolphe Quetelet concluded that ‘other than the spurs of growth after birth and during puberty, the weight increases as the square of the height’. This was known as Quetelet Index, later termed as Body Mass Index by Ancel Keyes in 1972. (Eknoyan, 2008.)

Body mass index (BMI) is a generally used index of weight-for-height for classifying adiposity. It defines a person’s body mass (kg) divided by the square of his/her height (m) ergo BMI = kg/m\(^2\). For adults, the WHO defines overweight as an individual with BMI ≥ 25, and obese as an individual with BMI ≥ 30. The BMI categories are shown in table 2. BMI is not a great indicator of the amount body fat, nor can it capture information on locations of fat. However, for its simplicity it is widely used in population-based studies and in determining public health-policies. (Tchernof & Despres, 2013; Saydah et al. 2014; Nuttall, 2015; WHO, 2018). BMI cannot differentiate lean mass and body fat mass, so a person can have a large amount of muscle tissue and thus a high BMI with minimal fat mass (Wellens et al. 1996).

<table>
<thead>
<tr>
<th>BMI</th>
<th>Nutritional status</th>
<th>Risk of developing health problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 18.5</td>
<td>Underweight</td>
<td>Increased</td>
</tr>
<tr>
<td>18.5 – 24.9</td>
<td>Normal weight</td>
<td>Least</td>
</tr>
<tr>
<td>25.0 – 29.9</td>
<td>Overweight (pre-obesity)</td>
<td>Increased</td>
</tr>
<tr>
<td>30.0 – 34.9</td>
<td>Obesity class 1</td>
<td>High</td>
</tr>
<tr>
<td>35.0 – 39.9</td>
<td>Obesity class 2</td>
<td>Very high</td>
</tr>
<tr>
<td>Above 40</td>
<td>Obesity class 3</td>
<td>Extremely high</td>
</tr>
</tbody>
</table>

**Percent of body fat** is a more accurate method to assess the level of obesity. It indicates the percent of body fat from the total body mass, so unlike BMI it takes into consideration the portions of fat mass and fat free mass. Laboratory methods are needed to measure body fat percentage. Therefore, BMI is more used in large population studies. (Wellens et al. 1996.) Depending on the study, BMI seems to be correlated or exceedingly correlated with the body fat percentage and it seems that the correlation is stronger at higher BMI (Steinberg et al. 2005; Meeuwsen et al. 2010; Wang et al. 2010). There are no internationally accepted guidelines for the percentage of body fat.

**Waist circumference (WC)** is widely used for the valuation of abdominal fat mass and for the rough estimate of visceral fat. The problem in using WC occurs, because not all abdominal fat is VF. WC correlates higher with total fat mass and subcutaneous fat mass than with visceral fat, and is a usable tool for the estimation of obesity. (Camhi et al. 2010.). However, WC is a better predictor for abdominal visceral fat than BMI, and should preferably be used in the evaluation of possible health risks (Janssen et al. 2002), as shown in table 3.

**TABLE 3. Guidelines for weight circumferences (The IDF Consensus, 2006).**

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Increased health risk</th>
<th>High Health risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women</td>
<td>&lt; 80 cm</td>
<td>80-87 cm</td>
<td>&gt; 88 cm</td>
</tr>
<tr>
<td>Men</td>
<td>&lt; 94 cm</td>
<td>94-101 cm</td>
<td>&gt; 102 cm</td>
</tr>
</tbody>
</table>
4.2 Physiology of obesity

A human being needs a constant source of metabolic fuel (energy), which is achieved by storing excess carbohydrates, fat and protein in the liver, adipose and muscle tissue and using them when needed. For food energy, 1-kilocalorie (kcal), indicates the quantity of heat required to elevate the temperature of one kilogram of water 33.8°F. Different food contains different volumes of absolute energy and net energy. Absolute energy values are 4.2 kcal for carbohydrates, 5.65 for proteins and 9.4 kcal for lipids, while the net energy values are 4 kcal for carbohydrates and proteins and 9 kcal for lipids. (McArdle et al. 2015, pp. 110–112.)

In the human body system energy, the metabolic fuel, is stored as triglyceride, which is an ester derived from glycerol and three fatty acids and mostly stored in fat cells founded in the adipose tissue. Adipose tissue is a specialized movable connective tissue that is greatly burdened with adipocytes fat cells, which stock triglycerides in extents as excessive as 80 to 95 % of the complete cell volume. The rest is water, protein and minerals. (Guyton & Hall, pp. 819–821.) All lipid metabolism take place in the liver, where triglycerides are derived either from the dietary triglycerides or synthesized mostly from carbohydrates, but to lesser quantities from proteins as well. When more protein is available than needed for the tissue’s use, a great share of the excess proteins is stored as fat. Regardless of which source the excess energy comes from, if it is not needed for immediate use, it will be stored as fats in the adipose tissue for later use. (Marlatt & Ravussin, 2018, pp. 40–43.) The adipose tissue can enlarge in two different ways, either by hypertrophic fat accumulation or hyperplastic fat accumulation, from which the first means an enlargement of fat cells, and the second increased number of fat cells. New fat cells are not formed until the hypertrophy of the existing cells reaches a plateau. The presence of large fat cells may trigger the formation of new ones in highly obese individuals. It seems like the hypertrophic obesity is more associated with metabolic complications than the hyperplastic obesity. (Björntorp & Sjöström, 1971.)

The fuel homeostasis, which refers to the balance between energy intake and expenditure, is sustained by the autonomic nervous system and the endocrine system, principal hormones including insulin, glucagon, epinephrine, cortisol, growth hormone, thyroxine and leptin.
Human brain is the organ regulating the process, although numerous organ systems are giving their contribution as well. It can be stated that energy homeostasis is maintained via a complicated complex of interactions between brains, which in particularly the hypothalamus, and the periphery. Regulation of food intake is composite, as is the development to overeating and obesity. In the periphery, an adipose tissue synthesizes and secretes a hormone called leptin, which exerts its action within the brain and thus plays a vital role in a reduction of food intake, by regulating several orexigenic and anorexigenic neuropeptides, which control appetite. (Jeanrenaud & Rohner-Jeanrenaud, 2001.)

Total daily caloric intake and TDEE needs to stay in balance in order to avoid weight gain. In the western society, with plenty of energy-rich food available, diet is not only a matter of individual choice (Chandon & Wansink, 2012), and the fact is that for example in Finland every fourth adult is obese (Koponen et al. 2017). In the figure 3, is shown that, how the energy intake has changed over 40 years in the United States, where one third of the adults are obese. The real concern is that the energy expenditure has not increased with the same intensity, instead it has been decreasing. (Guyenet & Schwartz, 2012.)

Achieving a weight loss by restricting caloric intake is doable (a diet), but it is almost impossible to sustain the lowered weight. Failed weight loss attempts are even related to a larger weight gain in adults. (Korkeila et al. 1999.) The body reacts to negative energy balance with a reduction of RMR (Leibel et al. 1995) and an increase of appetite (Polidori et al. 2017). The body does not want to lose weight but tries to hold on to it in multiple ways. In the study of Polidori et al. (2017), it was observed that the weight loss results in a relative increase in appetite in response to the feedback signals arising from the body weight change. Increase in appetite results in eating above baseline, which again leads to weight regain. The study of Leibel et al. (1995) points out the difficulty of sustaining weight loss by showing that with obese participant maintaining body weight at a level of ten percent or more below original weight was accompanied by a decrease in total body energy expenditure of 8±5 kcal per kilogram of fat-free mass a day.

4.3 Overweight and obesity’s relationship with health

Obesity is a known risk factor for many serious diseases and health conditions, such as cardiovascular disease (Saydah et al. 2014), type 2 diabetes mellitus (Ganz et al. 2014) and certain cancers (De Pergola & Silvestris, 2013). It is related to increased risk of all-cause mortality (HHS, 2013) and in addition, it has an enormous price tag for the society (Tremmel et al. 2017).

In 2014 Saydah and colleagues evaluated data from the National Health and Nutrition Examination Survey (NHANES) from 1999 to 2010 to observe trends in cardiovascular risk factors and obesity in the United States. Participants were categorized based on BMI, as shown in table 2: normal weight, overweight and obese. Participant with BMI ≥ 35 kg/m² were involved in the obese category but also showed separately. Results of the study concluded that in 11 years the percentage of obese adults increased from 30.1 to 34.3 % and their morbidly increased from 12.4 to 14.7 %. The study also showed the fact that adults who were morbidly obese had the highest existence of diabetes, hypertension and dyslipidemia, second highest prevalence was among obese adults and third among overweight adults. It can be concluded that increasing amount of body fat is in association with prevalence of cardiovascular risk
factors. (Saydah et al. 2014.). Like in the study of Stevens et al. (2012), as well in the study of Saydah et al. (2014), the obesity rates among adults increased over the study time.

Obesity is a recognized risk factor for type 2 diabetes (Colditz et al. 1995; Garber, 2011; Kodama et al. 2012). Ganz et al. (2014) compared BMI categories and the relative risks of type 2 diabetes diagnosis with 37 356 Americans over seven years (2004-2011). They concluded that overweight and obesity, when compared with normal BMI, was statistically significantly related with the risk of being diagnosed with type 2 diabetes among individuals without any other former evidence of type 2 diabetes. These relations are shown in figure 4, which indicates that risk for type 2 diabetes increases with increasing BMI.

![Relative risks of type 2 diabetes diagnosis by BMI categories](image)

FIGURE 4. Relative risks of type 2 diabetes diagnosis by BMI categories, modified from Ganz et al. (2014).

Obesity is associated with increased risk for different types of cancer, for example liver cancer, pancreatic cancer, breast cancer in postmenopausal women, kidney cancers and colon cancer (De Pergola & Silvestris, 2013). Data published over 16-year follow-up highlighted that obesity might explain 19 % in men and 14 % in women of all deaths from cancer in U.S., which characterize it a second leading cause after smoking (Calle et al. 2003).
4.4 Body fat and the importance of its distribution

Not only the excess amount of adipose tissue but its distribution, is associated with multiple conditions and diseases (Björntorp, 1992; Ross et al. 1996; Wildman et al. 2008). Total body adipose tissue can be classified into two quantifiable components; subcutaneous, which means the deposit originated between the dermis and the aponeurosis and fasciae of the muscles, and internal, which is all the rest than subcutaneous. The internal adipose tissue can be divided more specific to visceral component, which means the adipose tissue in chest, abdomen, and pelvis, and non-visceral component, which refers to the rest of the internal adipose. (Shen et al. 2003.)

The fact that not all obese individuals display expected metabolic abnormalities (Wildman et al. 2008) supports the statement that fat distribution of an individual does play an independent and immense role in health (Goncalves et al. 2016). The state called metabolically healthy obesity can be explained by the distribution of the excess amount of body fat (Ross et al. 1996). The amount of visceral fat (VF) is an independent factor in many conditions, such as in metabolic syndrome (Ross et al. 1996; Pouliot et al. 1992) and cardiovascular risks (Britton et al. 2013) and in addition, it is related to mortality independent of BMI (Kuk et al. 2006).

The exact meaning of the word viscera is “organs in the cavities of the body”. In a human body, there are three cavities: intra-abdominal, intrapelvic and intrathoracic. In most studies, the VF is identified as intra-abdominal fat, which is located from 5 cm below L4-L5 to the slice consistent to the liver’s superior border. (Shen et al. 2003; Tchernof & Despres, 2013.) The amount of VF can be measured with magnetic resonance imaging (MRI) and computed tomography (CT-scan). In addition, a dual x-ray absorption (DXA) can provide information of abdominal fat but does not predict VF with the same accuracy as the two others. (Samouda et al. 2012.)

DXA measures segmental body fat distribution and separates android and gynoid fat mass. GE Healthcare Lunar user’s manual, (2012) has stated the measurements as following: ‘android is measured with the lower boundary at the pelvis cut with the upper boundary above the pelvis cut by 20 % of the distance between pelvis and neck cuts. Gynoid upper boundary is below the
pelvis cut line by 1.5 times the android space and gynoid space is equal to 2 times the android space.’ Android adiposity is characterized by VF and is more common in men. Gynoid adiposity is more common in women than in men and it characterized by large quantities of subcutaneous fat. (Blouin et al. 2008.) Android-gynoid ratio is android fat percent divided by gynoid fat percent (GE Healthcare Lunar, 2012). Even though DXA does not directly measure VF, the DXA measured android fat percent predicts MRI measured VF ($R^2=0.72$) (Lee et al. 2018).

VF has been proven to be an independent factor for metabolic abnormalities, especially for insulin resistance (Despres, 1993). For metabolic syndrome, the main feature of the abnormalities is dyslipidemia. Dyslipidemia is characterized with high levels of triglycerides and low levels of HDL cholesterol. In addition, a manipulation of the lipid particle sizes is commonly seen in patients, who have large amounts of visceral fat. It is been established, that VF is a part of a multifaceted phenotype, which consisted of dysfunction in adipose tissue storage and accumulation of triglycerides in several sites, such as liver hepatocytes. (Tchernof & Despres, 2013.)

In order to have VF, there is a need for excess fat accumulation (Lanska et al. 1985). Besides of excess body fat, for example age (Lanska et al. 1985), gender (Krotkiewski et al. 1983), genetics (Bouchard et al. 1990), nutrition (Romaguera et al. 2011), ethnicity (Caroll et al. 2012) and physical inactivity (Slentz et al. 2005), all have a role in the accumulation of visceral fat. Older people and men are more likely to gain VF than young people and women (Lanska et al. 1985). Men are more likely to be shaped like an apple, in which the fat is accumulated in the abdominal area, whereas women are more likely to be shaped as a pear, in which the fat is accumulated in the gluteofemoral area (Krotkiewski et al. 1983). Both android fat and android-gynoid ratio is associated with insulin resistance and dyslipidemia (Fu et al. 2014; Samsell et al. 2014).
5 METABOLIC HEALTH INDICATORS

Metabolic health indicators consist the fasting plasma glucose and the ones interfering with dyslipidemia: plasma cholesterol (total, LDL and HDL) and plasma triglycerides. High plasma glucose is a disorder of glucose metabolism. Dyslipidemia, on the other hand, is a disorder of fat metabolism referring to the abnormal quantity and quality of lipids, as shown in figure 5. (Tarnanen et al. 2018.)

FIGURE 5. Plasma lipoprotein profile of a non-obese or healthy-obese (grey) versus the profile of a viscerally obese individual (white) (Tchernof & Despres, 2013).

Dyslipidemia is a state, where LDL concentration is over 3.0 mmol/l or HDL concentration is less than 1.0 mmol/l in men and 1.2 mmol/l in women, or the concentration of triglycerides is over 1.7 mmol/l. Dyslipidemia can be treated with medicine but only when lifestyle changes,
such as healthier nutrition and an increment of physical activity, are unable to bring up the wanted changes. (Tarnanen et al. 2018.) Obesity (Klop et al. 2013; Schwingshackl et al. 2014), and particularly visceral obesity (Hwang et al. 2016; Tian et al. 2018), is strongly related to dyslipidemia, as shown in figure 5. Other risk factors are a diet, high in saturated fats (Clarke et al. 1997), and physical inactivity (Boucher et al. 2015).

5.1 Cholesterol

Cholesterol is a waxy, fat-like substance and it is present in nutrition (exogenous cholesterol), but a larger amount is formed in body cells (endogenous cholesterol), mostly in the liver. By far most of the cholesterol is converted into cholic acid which is a necessary substance in the formation of bile salts. Rest of the cholesterol is used as a building material for cell membranes and some hormones. (Guyton & Hall, 2006, p. 826–827.)

The level of plasma cholesterol concentration may increase depending on the amount of cholesterol ingested daily. Other factors that influence the plasma cholesterol level are a diet containing a great quantity of saturated fat, lack of insulin or thyroid hormone and genetic disorders of cholesterol metabolism. The blood cholesterol concentration may be depressed by ingesting fats containing highly unsaturated fats. Contrariwise ingestion of fat containing highly saturated fat increases blood cholesterol concentration 15 to 25 percent, particularly when accompanied with overweight and obesity. (Guyton & Hall, 2006, pp. 826–827.)

Because cholesterol is a fat-like substance, it is not water-soluble. Therefore, in order it to be able to transport through blood circulation, it needs to be packed inside special lipoproteins, either inside a low-density lipoprotein (LDL), approximately 70 % of the total cholesterol, or inside a high-density lipoprotein (HDL), 20-25 % of the total cholesterol. LDL is responsible for carrying most of the cholesterol and transporting it from blood to tissues. HDL is responsible for carrying cholesterol away from the tissues, such as from arterial walls. (Guyton & Hall, 2006, pp. 826–829.)
Total cholesterol. Borderline cholesterol concentration is less than 5.1 mmol/dL and acceptable concentration less than 4.3 mmol/dL (Tarnanen et al. 2018). According to the FINRISK-research (Koponen et al. 2017) the average cholesterol concentration in Finns over 30 years old is 5.1 mmol/l in women and 5.4 mmol/l in men. Too high concentration of cholesterol causes plaques to develop on the surface of arterial walls and eventually might lead to a disease called atherosclerosis. Furthermore, the concentration of the total cholesterol is less important than the concentration of HDL and LDL, LDL being the dominant form of atherogenic hormone. (Guyton & Hall, pp. 827–828.) For this reason, HDL and LDL concentrations are used in the diagnosis of dyslipidemia and in the evaluation of the risk of the atherosclerosis, instead of total cholesterol concentration (Tarnanen et al. 2018).

HDL. High-density lipoprotein is considered to be the “good” cholesterol, because with a high ratio of HDL to LDL, the probability of developing atherosclerosis is diminished by a large margin. HDL should be over 1 mmol/dL in men and over 1.2 mmol/dL in women. (Tarnanen et al. 2018.) In the population studies the concentration of plasma HDL is contrariwise associated with the risk of atherosclerosis, but the causality of the relationship is unclear (Lewington et al. 2007). One widely accepted theory is that HDLs can absorb cholesterol crystals that have started to be deposited in arterial walls (Guyton & Hall, pp. 829). Especially visceral obesity is strongly associated with low HDL cholesterol concentration (Despres, 1993: Fox et al. 2007), but with overweight and obese patients, a decrease in body fat in general increases the concentration of HDL (Leon & Sanchez, 2001; Schwingshackl et al. 2014).

LDL. Low-density lipoprotein is responsible for carrying the larger mass of total cholesterol and transporting it from blood to tissues. LDL is known to be the “bad” cholesterol, because of its ability to attach to arterial walls. The higher the concentration, the more it is attached to the arterial walls. For that reason, LDL is a better marker to evaluate the risk for atherosclerosis, than the total cholesterol concentration. (Steinberg, 2005.) Optimal LDL concentration is less than 3 mmol/l or in some high-risk cases less than 2.5 mmol/l (Tarnanen et al. 2018). The most significant factor for high LDL concentration is a large intake of saturated fats (Clarke et al. 1997).
5.2 Triglycerides

Metabolic fuel (energy) is stored for further use as a triglyceride, which is an ester derived from glycerol and three fatty acids. All lipid metabolism take place in the liver, where triglycerides are derived either from the dietary triglycerides or synthesized mainly from carbohydrates and then stored in fat cells founded in the adipose tissue. (Guyton & Hall, pp. 819–821.) Some portion of the triglycerides circulates within a blood flow. In a fasted state the plasma triglyceride concentration is less than 2.0 mmol/l. The optimal triglyceride concentration is less than 1.7 mmol/l (Tarnanen et al. 2018).

A meta-analysis from Hokanson & Austin (1996) showed that increases in plasma triglyceride levels are related to a significant increase in the risk of incident cardiovascular disease among both women and men. The main cause for plasma triglyceride level to increase is an excess VF (Hwang et al. 2016). In the study of Hwang et al. (2016) it was observed that plasma triglyceride association with VF was extremely dominant across genders indicating that the VF has a dominant, independent impact on plasma triglyceride levels. Similar results were observed in the study of Fox et al. (2007).

5.3 Fasting glucose

Products from the ingestion of carbohydrate are almost entirely glucose, fructose and galactose, from which galactose and fructose are converted to glucose in the liver. Glucose is the most usual monosaccharide circulating in the blood. The rate of glucose transport from blood to cells is greatly increased by insulin, which causes the excess glucose to be deposited as glycogen in the muscles and the liver. (Guyton & Hall, 2006, pp. 810–811.) Insulin secretion from pancreas is dependent on a great profusion of energy-giving foods, especially carbohydrates (glucose). When the blood glucose level rises above 100 mg/100 ml, insulin is rapidly secreted to transport the glucose from blood to cells. Under normal conditions, when the insulin works as it should, in fasting state blood glucose stays constant at 80-90 mg/100 ml. A condition where insulin does not work as it should, is called diabetes mellitus. The guidelines for blood glucose are shown in table 4. Type 1 diabetes is characterized by a lack of insulin secretion, whereas type
2 diabetes the insulin sensitivity of the tissues is decreased. Out of all the cases of diabetes, type 2 presents 90 to 95 percent and is strongly affected by lifestyle choices. (Guyton & Hall, 2006, pp. 950–951.)


<table>
<thead>
<tr>
<th>Fasting blood sugar level</th>
<th>Condition</th>
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<tbody>
<tr>
<td>≤ 100 mg/dL (5.5 mmol/L)</td>
<td>Normal</td>
</tr>
<tr>
<td>100 – 125 mg/dL (5.6 – 6.9 mmol/L)</td>
<td>Prediabetes</td>
</tr>
<tr>
<td>≥ 125 mg/dL (7.0 mmol/L)</td>
<td>Diabetes (when measured on two separate test)</td>
</tr>
</tbody>
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A type 2 diabetes is characterized with a condition called insulin resistance, where plasma insulin concentration is increased but the insulin sensitivity of target tissues is diminished. Developing an insulin resistance is usually a slow process, which gets it beginning from excess weight gain and obesity. (Guyton & Hall, 2006, pp. 951.) In the study of Saaristo et al. (2008) 4500 randomly selected individual were clinically examined with oral glucose test and height, weight and waist circumference measurements. They concluded that obesity, as well as abdominal obesity were associated with a higher prevalence of abnormal glucose tolerance. In addition, central obesity was related to abnormal glucose tolerance in each of the BMI categories (normal, overweight and obese), pointing out the fact that central adiposity appears to be better predictor to type 2 diabetes than BMI, in which similar results have been observed in several studies (Kurioka et al. 2002; Wang et al. 2005; Ayubi et al. 2014). Fox et al. (2007) investigated more specifically the associations between VF and metabolic risk factors using computed tomography to assess visceral adipose tissue. They found out that VF was strongly correlated with fasting blood glucose, and the correlation was stronger than with subcutaneous adipose tissue. Even a modest weight loss, which is at least 10 % mean baseline body weight, is known to improve insulin sensitivity and enhance glycemia in the individuals with type 2 diabetes (Grams & Garvey, 2015). Davis et al. (2018) recommended lifestyle interventions, including physical activity and medical nutrition therapy, as a first-line treatment for type 2 diabetes, because of their safety and effectiveness for improving glucose control.
6 THE INFLUENCE OF PHYSICAL EXERCISE ON CARDIORESPIRATORY FITNESS, BODY FAT AND METABOLIC HEALTH INDICATORS

6.1 The effects of physical exercise on cardiorespiratory fitness

Cardiorespiratory fitness can be increased with exercising, but the response depends on the exercise duration, intensity and frequency. For example, Nielsen et al. (2019) showed in their study that a 15-week combined exercise program did not increase cardiorespiratory fitness in sedentary elderly due to the small frequency and intensity of exercise: 90 minutes once a week with moderate intensity (average heart rate 104+-12 bpm). On the contrary, King et al. (1991) showed that cardiorespiratory fitness can be improved by frequent exercise with proper duration and intensity.

In an RCT by King et al. (1991), 357 sedentary participants were randomized in a control group or one of three exercise groups: higher-intensity group-based, higher-intensity home-based and lower-intensity home-based. Two of the groups (higher-intensity group-based and higher-intensity home-based) were assigned to train three times a week for a 40 minutes per day at an intensity of 73-88 % of their peak exercise heart rate. The third exercise group (lower-intensity home-based) trained five times a week for 30 minutes at an intensity of 60-73 % of their peak exercise heart rate. The six months changes in VO\textsubscript{2}max are shown in figure 6. Compared to the control group, VO\textsubscript{2}max increased significantly in all exercise groups, but there were no significant changes observed between the exercise groups.

It is widely known, proven and accepted fact, that endurance exercising will improve cardiorespiratory fitness, when done frequently with adequate duration and intensity (Stein et al. 1990; King et al. 1991; Pollock et al. 1998; Gan et al. 2003; Huang et al. 2005; Slentz et al. 2007; Trapp et al. 2008; Delextrat et al. 2016; Kong et al. 2016; Conolly et al. 2017).
FIGURE 6. Six months’ changes in maximal oxygen consumption in women and men assigned to the control group or one of three exercise groups modified from King et al. (1991).

6.2 The effects of physical exercise on body fat and its distribution

In the interest of body fat reduction there is a requirement for a negative energy balance (Marlatt & Ravussin, 2018). This negative energy balance can be reached by consuming less energy (Strasser et al. 2014) or by increasing energy expenditure (Quist et al. 2017). Toji et al. (2012) compared the effectiveness of exercise intervention and diet on weight reduction in 62 Japanese adults, whose BMI was 24-28 kg/m². In the study they concluded that the combination of diet and exercise is the most effective way to lose weight and that diet-only is more effective than exercise-only. The decrease in BMI was significant in exercise-only-group compared to baseline but not when compared to control group. Exercise was conducted by increasing daily walked steps. (Toji et al. 2012.) The problem that occurs when the weight is lost only by the caloric restriction (diet), is that the lost weight is not only fat mass but muscle mass as well (Hoie et al. 1993; Frisch et al. 2009). Loss of the muscle mass is most likely one of the explanations for weight regain after the diet (Dulloo et al. 2012). The loss of the muscle mass can be prevented by exercise-induced weight loss programs (Quist et al. 2017).
Weight loss over an exercise intervention is highly individual. Not all people lose weight during exercise intervention (King et al. 2007), which is usually explained with increased energy intake (Blundell et al. 2003) or decreased daily physical activity during the intervention (Epstein & Wing, 1980; Goran & Poehlman, 1992). However, in multiple studies it has been observed that with most of the individuals increased levels of exercise will lead to reductions of body fat (Schmitz et al. 2003; Keating et al. 2017; Quist et al. 2017). How much weight is lost, is dependant of the exercise intensity (Quist et al. 2017), frequency and a form of exercise (Schmitz et al. 2003).

In the study of Quist et al. (2017) the effect of exercise intensity to fat loss was investigated. They compared moderate (50 % of VO$_{2\text{max}}$) and vigorous (70 % of VO$_{2\text{max}}$) intensity exercises, which both included aerobic exercises only for 5 times per week for six months. Training energy expenditure was prescribed to be 320 kcal/day for women and 420 kcal per/day for men. Body composition was measured with DXA prior and after the six months intervention. Body fat mass decreased significantly in both groups compared to control group and more in the vigorous-intensity-group compared to moderate-intensity-group. The study from Quist et al. (2017) shows that the exercise intensity matters in regard to fat loss. In addition, the fat-free mass stayed unchanged in both groups, while the weekly energy balance was negative.

A meta-analysis from Keating et al. (2017) compared the effectiveness of high-intensity interval training and moderate continuous training on fat loss. Both training methods produced a slight reduction of body fat but the reduction did not differ between the methods. The most important matter is the elevated energy expenditure both during the exercise and afterwards. Although, in the study of Quist et al. (2017), the energy expenditure during the exercise was equal in both groups (320 kcal / 420 kcal), the reduction in body fat mass was larger in the vigorous group compared to moderate. This is due because of the post exercise oxygen consumption (EPOC). A clear relationship exists between the magnitude of EPOC and both intensity and duration of exercise. (Borsheim & Bahr, 2012.)
With exercise-based weight loss interventions, it is possible to have the fat-free mass remain unchanged (Quist et al. 2017) or even increase it if resistant training is performed (Schmitz et al. 2003). The most effective way to change the body composition to healthier form (added muscle and reduced fat) is to combine aerobic exercising and resistance training (Schmitz et al. 2003).

Ross et al. (2000) compared diet-induced and exercise-induced weight loss programs on reductions in body fat, VF and subcutaneous fat. They also investigated the effect of exercise on VF without weight loss. They found out that even though the reduction of total fat was similar in the control group and in the exercise-without-weight-loss-group, the level of VF decreased significantly in the exercise-without-weight-loss-group compared to control group. Both the diet-induced-weight loss-group and the exercise-induced-weight-loss-group reduced both total fat and VF significantly more that the exercise-without-weight-loss-group or the control group. The findings from Ross et al. (2000) suggest that even exercise alone is a beneficial for reducing VF and preventing further increase in obesity. (Ross et al. 2000.) Diet- and exercise-induced fat losses were corresponding to the ones in the study of Toji et al. (2012).

In respect of VF, similar results have been reported in several other studies (Slentz et al. 2007; Johnson et al. 2009; Ross et al. 2012; Keating et al. 2015). In addition, two meta-analysis (Ismail et al. 2011; Vissers et al. 2013) found similar responses, stating that the decrease of VF tissue can be obtained with exercise-without-diet in people who are overweight and obese. In the meta-analysis of Vissers et al. (2013) the matter of a form of exercise was discussed. They stated that aerobic exercise of moderate to vigorous intensity seems to be more effective in reducing visceral fat than resistant training. Similar findings were also detailed in the meta-analysis of Ismail et al. (2011), where they assessed the efficacy of resistant training and aerobic training on visceral adiposity. The exercise-induced reduction in VF seems to be possible even with a low volume – low intensity aerobic training, while larger reductions are observed with higher intensities and volumes (Keating et al. 2015). The high-intensity interval training and the prolonged continuous exercise training will decrease abdominal VF with the same intensity, while high intensity interval training is more time efficient (Zhang et al. 2017).
6.3 The effect of physical exercise on metabolic health indicators

Total cholesterol. The effect of exercising on total cholesterol levels is somewhat unclear. Already in the 1960s the study of Golding (1961) showed that intense endurance training was able to significantly reduce total serum cholesterol levels in males, but the serum cholesterol reduced in contrast with body weight. Stein et al. (1990) did not report any significant changes in total cholesterol, nor in total body fat after 12 weeks of endurance-exercise training.

In the study of Aadahl et al. (2009), they observed that increased levels of physical activity were associated with reduced total cholesterol, independently of BMI. However, the study duration was five years and the possible changes in nutrition were not documented. Also, the result was opposite to their previous study searching for associations between physical activity levels and total cholesterol (Aadahl et al. 2007). It could be speculated that any reduction in total cholesterol levels after endurance-exercise interventions happens because of a loss of body fat. A meta-analysis from Leon & Sanchez (2001) supported this statement by showing that endurance exercise training does not provide any significant decrement in total cholesterol levels. Instead, Boyden et al. (1993) reported significant reduction in total cholesterol levels after 5 months of resistance exercise training in premenopausal women independently of changes in body composition. Similar results were reported from Fett et al. (2009) over 2 months’ circuit weight training, which could show that resistance training is effective for improving total cholesterol.

HDL. It seems that HDL cholesterol levels can be increased by adequate intensity endurance exercise training, independently of weight changes (Leon et al. 2000; Banz et al. 2003; Slentz et al. 2007; Fett et al. 2009; Tambalis et al. 2009), furthermore higher levels of physical activity are positively correlated with HDL cholesterol (Aadahl et al. 2008). Stein et al. (1990) researched the effect of three different endurance exercise intensities (65 %, 75 % and 85 % of maximal heart rate) to cholesterol levels. They showed that HDL cholesterol increased significantly in the 75 % and 85 % of maximal heart rate training groups, but not in the 65 % or control group, without any changes in percent of body fat. In the meta-analysis of Leon & Sanchez (2001), which evaluated the associations between endurance exercise training and
blood lipids, the most observed lipid change was a significant increase in HDL. Leon and colleagues (2000) studied the effects of 20-weeks endurance training on blood lipids and they observed an average 3.6% increment in HDL. However, the changes in HDL were highly wide-ranging between the participants, suggesting that exercise-induced changes in HDL are exceedingly individual. (Leon et al. 2000.)

LDL. Aadahl et al. (2009) found a correlation between a five-year change (increment) in physical activity and reduced LDL cholesterol, independently of weight changes. The finding differs from an earlier study from Aadahl et al. (2007), in which any associations between physical activity levels and LDL cholesterol were not found. In the study of Stein et al. (1990), reduction in LDL levels was reported in a group who exercised at the intensity of 75% of maximal heart rate, but not in the group, who exercised at 85% of maximal heart rate. Similar variability can be seen in a systematic review of Tambalis et al. (2009) and in two meta-analyses (Leon & Sanchez 2001; Mann et al. 2014), where all the included studies did not report changes in LDL levels in respond to exercising. It could be speculated that it either depends on the exercise intensity (Stein et al. 1990) or it is just variable between individuals. It is known that high intake of saturated fat is correlated with high LDL levels (Clarke et al. 1997).

Triglycerides. Aadahl et al. (2007) observed negative correlation between triglycerides and self-reported 24-h physical activity at 3-year follow-up. Two years later Aadahl et al. (2009) reported similar relations between physical activity and improvements in triglycerides. In a randomized controlled trial from Slentz et al. (2007), they compared three different training intensities (high-amount/vigorous-intensity, low-amount/vigorous intensity, low-amount/moderate-intensity) to changes in lipoprotein profiles, independently of weight loss. They found out that the moderate-intensity training group reduced total triglycerides twice the magnitude as the two more vigorous exercise groups and still after 15 days the lowered total triglyceride levels were sustained. Also, already in the 1970s, Gyntelberg et al. (1977) showed that exercise induces a decrease in total triglycerides without negative energy balance, in participants diagnosed with type IV hyperlipoproteinemia. Even though the energy balance of the participants stayed constant, 30 minutes of walking on a treadmill per day for four days, produced a progressive decrement in total triglycerides.
Fasting glucose. The benefit of exercise training in order to reduce blood glucose is not unequivocally convincing (Eriksson et al. 1997), but the benefits can be seen in glycated hemoglobin HBA1 (Boule et al. 2001). Glycated hemoglobin is a hemoglobin, which has joined with glucose in the blood. This occurs as a result of elevated blood glucose levels. HBA1 gives information of an individual’s average long-term levels of blood glucose. (Shervani et al. 2016.) In a meta-analysis by Boule et al. (2001), they reported significant exercise-induced changes in HBA1 values, without any changes in body mass, in 12 studies done with patients with type 2 diabetes. Magnitudes of the reductions were clinically significant. Similar results have been observed in a randomized controlled trial from Sigal et al. (2007). They showed that the combination of endurance and resistance training is more effective than either of them alone in reducing HBA1. However, both training groups reduced HBA1 more than non-training control group, but they did not state, whether the decrease in HBA1 correlated with reduced fat mass. (Sigal et al. 2007.) Both endurance (Rönnemaa et al. 1986) and resistance training (Castaneda et al. 2002; Lambers et al. 2008) has been proven to be effective to reduce HBA1 in patients with type 2 diabetes (Umpierre et al. 2011).
7 RESEARCH QUESTIONS

The purpose of the study was to investigate exercise-induced changes in body composition, metabolic health indicators and cardiorespiratory fitness. The main interest was to research the role of exercising in weight loss and the effect of exercise on fat distribution and dyslipidemia and their associations.

Question 1: Does physical exercise influence metabolic health indicators, cardiorespiratory fitness and body fat?


Question 2: Are the changes in body fat and android fat mass in association with the changes in metabolic health indicators?

Hypothesis 2: Yes. Changes in body fat and more specific in android fat, are in association with changes in metabolic health indicators. (Leon & Sanchez, 2001; Fox et al. 2007; Schwingshackl et al. 2014; Hwang et al. 2016).
**Question 3:** Does the fat distribution change as a result of exercise-intervention?

**Hypothesis 3:** Yes. The android fat mass decreases more than the gynoid fat mass, which means the android-gynoid percent ratio decreases in the experimental group (Sullivan et al. 2011; Keating et al. 2012; Vissers et al. 2013; Serra et al. 2017).
8 RESEARCH METHODS

This study was done as a part of a larger Heart, Sauna and Exercise -study from Laukkanen & Lee, which studied cardiovascular health benefits of combined exercise and sauna bathing. The study was a randomized controlled trial, which consisted of three groups: an exercise group (E), exercise + sauna group (ES) and a control group (C).

The duration of the study was 12 weeks and consisted of three measurement points: pre, post and post three weeks. This study adds together the two experimental groups E and ES and evaluates only the effect of the exercise-intervention compared to the control group (no exercise intervention). The current study included only overweight female participants. The duration was 8 weeks and the study consisted of two measurement points: pre- and post-measurements.

8.1 Participants

Participants were of both genders and had at least one conventional cardiovascular risk factor, but asymptomatic, and without previous coronary heart disease and diabetes. The study participants were recruited through mediate-to-large employers via email (appendix 1) in Central Finland, like Central Hospital, City of Jyväskylä and Energy of Jyväskylä. Anthropological data of the participants is shown in table 5.

TABLE 5. Anthropological data of the study participants.

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<th></th>
<th>Experimental n = 24</th>
<th>Control n = 13</th>
<th>All n = 37</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>49.4 ± 9.5</td>
<td>49.1 ± 7.9</td>
<td>49.3 ± 8.7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>167 ± 7</td>
<td>166 ± 7</td>
<td>1.67 ± 7</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>90.8 ± 11.4</td>
<td>87.3 ± 14.6</td>
<td>89.1 ± 13</td>
</tr>
<tr>
<td>BMI(^a) (kg/m(^2))</td>
<td>32.3 ± 3.3</td>
<td>31.5 ± 3.3</td>
<td>31.9 ± 3.3</td>
</tr>
</tbody>
</table>

\(^a\)BMI = Body mass index
The inclusion criteria of study participants consisted of a sedentary lifestyle and a conventional cardiovascular risk factor. Sedentary lifestyle was identified as a deskbound job and/or low levels of physical activity. The conventional cardiovascular risk factors were smoking, elevated cholesterol, hypertension, and family history of CHD. For the hypertension, pre-study resting blood pressure needed to be from 130-160 mmHg for systole and 75-90 mmHg for diastole and family history of CHD was positive if father (< 55 years) or mother (< 65 years) had premature CHD. Exclusion criteria included resting systolic blood pressure under 120mmHg or over 160 mmHg, BMI over 40 kg/m², any diagnosed and/or symptomatic cardiovascular disease, musculoskeletal injury or any other physical or mental condition that will prohibit the participation in the experiment.

Prior to the study an ethical statement was provided from ethical committee of Central Finland Health Care District. For personal information security, data protection officer of the University of Jyväskylä was consulted. The study was pseudonym, which means each participant was only known by their ID-code. The key code-list was held in possession of the director of the study. ECG was performed to make sure that the participants were suitable for the study and if something was found, the participant were asked to contact their doctor. Prior the study participants were asked to sign a term of agreement (appendix 2).

## 8.2 Study design

After recruiting, all interested participants were invited to information sessions, which were held twice in Liikunta building of the University of Jyväskylä. The purpose of the information session was to explain face-to-face the aim and the design of the study, as well the inclusion and exclusion criteria. In addition, participants had the chance to pose questions in regard to the study. If the participant decided he/she wishes to participate in the study, she/he booked a time for pre-screening. Final participants were selected based on the pre-screening results. After the pre-screening participants were randomly divided in three groups: exercise group (E), exercise + sauna group (ES) and control group (C), in which E and ES were combined for this study in order to form one experimental group. An overview of the study design is shown in the figure 7.
The study consisted three identical measurement days completed by both groups and an 8-week exercise intervention for the experimental group only. During the intervention experimental group exercised 150 minutes per week consisting of both endurance and strength training. Both groups were requested to maintain individual habitual physical activity and to continue with normal daily routines and eating habits throughout the intervention.

8.3 The intervention

Qualified instructors consistently supervised all prescribed training in the study. The training was designed to reflect a program aimed for the general population according to recommendations outlined by the HHS (2018). For the sedentary population the recommendation is to start with a little less than the recommended 150 minutes of endurance exercise per week (HHS, 2018). In this study the total amount of endurance exercise was 90 minutes per week and the total amount of exercise, including strength training, was 150 minutes per week.

Each training included a 10-minute warm-up, 20 minutes of strength training and 30 minutes of endurance training. Training sessions were carried out in groups of 1–5 participants and two instructors. Experimental group trained three times a week on Monday, Wednesday and Friday.
evenings (16:00 – 21:00). Both endurance and strength training were slightly progressive to ensure the improvements of physical abilities. The aim was that all the participants completes 91-100% of the training in 8 weeks, with total of 24 trainings.

The endurance training was performed with Monark bicycle ergometers. The intensity of the endurance training during the 8-week training period was controlled by participants individual heart rates and verified by the ratings of perceived exertion (RPE) scale. Participants maintained a constant pedalling frequency of 65 rpm during each training session, while the magnetic resistance of the bike ergometer was adjusted to achieve the required exercise intensity. Endurance training was progressive, as shown in table 6. In addition, 20 minutes of strength training exercise was performed by each participant in every training session.

TABLE 6. The design of the 8 weeks endurance exercising program.

<table>
<thead>
<tr>
<th>Weeks</th>
<th>Intensity (MAX HR(^a))</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2</td>
<td>60 % of MAX HR</td>
</tr>
<tr>
<td>3-4</td>
<td>65 % of MAX HR</td>
</tr>
<tr>
<td>5-6</td>
<td>70 % of MAX HR</td>
</tr>
<tr>
<td>7-8</td>
<td>75 % of MAX HR</td>
</tr>
</tbody>
</table>

\(^a\)MAX HR = Maximum heart rate

Strength training was completed with bodyweight and resistance training exercises in each participant’s individual strength level. The training program was based on Finnish exercise recommendations (UKK, 2019), and both trainings were slightly progressive and performed as a circuit training. Each circuit consisted of five movements with each movement performed for 45 seconds with a 15 second break between the movements. Completion of a circuit took five minutes followed by a one-minute break. The circuit was completed three times. The aim was to cover the whole body in one circuit. Circuits are shown in table 7. Circuit A and B were used alternately in each training for first four to five weeks. Circuit A2 and B2 were used the last three to four weeks. In resistance exercising the load increased and in bodyweight movements harder variations were introduced as the performance improved.
TABLE 7. Strength training program.

<table>
<thead>
<tr>
<th>Circuit</th>
<th>Legs</th>
<th>Back</th>
<th>Core</th>
<th>Arms</th>
<th>Extra</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circuit A</td>
<td>Squat</td>
<td>lat-pull-down</td>
<td>Dead bug</td>
<td>Horisontal Push-up</td>
<td>Balance exercise</td>
</tr>
<tr>
<td>Circuit B</td>
<td>Lunges</td>
<td>Horisonal row</td>
<td>Horse stance</td>
<td>Vertical push-up</td>
<td>Balance exercise</td>
</tr>
<tr>
<td>Circuit A2</td>
<td>Squat</td>
<td>lat-pull-down</td>
<td>Dead bug</td>
<td>Horisontal Push-up</td>
<td>One leg RDL</td>
</tr>
<tr>
<td>Circuit B2</td>
<td>Walking lunges</td>
<td>Horisonal row</td>
<td>Plank with variations</td>
<td>Vertical push-up</td>
<td>Hip thurst</td>
</tr>
</tbody>
</table>

8.4 Measurements

In the beginning of the study all interested participants were pre-screened and asked to fill up a basic health questionnaire to insure their suitability for the study. The measurements consisted of body composition measurement, blood sampling and cardiorespiratory fitness test. There were two measurement points in the study: before the intervention (pre-measurement) and after the intervention (post-measurement).

Body composition measurement and blood sampling were completed in the morning between 06.00 and 09.00 in fasting conditions and the fitness test was completed in the evening between 16.00 and 19.00. All measurements took place in the Exercise and health laboratory of the University of Jyväskylä. Participants were instructed to abstain food, drinks, alcohol and nicotine for 12 hours and to refrain from heavy physical activity for 48 hours before morning measurements. Acute meals or exercise can affect body composition assessments and blood variables (Guyton & Hall, 2006, pp. 950–951; Schubert et al. 2018; Abe et al. 2019). Breakfast was served after morning measurements. In addition, participants were also instructed to avoid physical activity 48 hours before the cardiorespiratory fitness test.
8.4.1 Pre-screening

Pre-screening took place in the Exercise and health laboratory of the University of Jyväskylä in August 2019 for all those who had shown interest towards the study. Prior to the pre-screening, each one of them was instructed to fill up a basic background questionnaire (appendix 3) to evaluate their risk-factors for cardiovascular disease, sedentary behavior and physical activity.

Pre-screening took 10 minutes per person and consisted of resting electrocardiography (ECG), waist circumference, weight and height measurements. All the measurements were taken by a professional technician. BMI was calculated with weight and height. Out of 61 participants attended in the pre-screening, 49 met the inclusion criteria and were selected for the study. Out of these 49 participants, 37 were selected for this study, which consisted of only the overweight women (BMI>25). Excluded participants (12) were either men or normal weight women.

8.4.2 Body composition

Body composition was estimated with Dual Energy X-Ray Absorption (DXA). DXA is a three-component model which measures fat mass (FM), fat free mass (FFM) and bone mass density (Mazess et al. 1990). The DXA measurement technique is based on the differential attenuation of the tissues of transmitted photons at two energy levels (Mazess et al. 1990). As photon absorptiometry requires a photon source and detector, DXA devices are composed mainly of a detector and a generator emitting x-rays of two energies (photon source) (Genton et al. 2002). DXA is different than a regular x-ray inspection, as it uses two energy spectrums to distinguish between high and low channel x-rays, as well as two detectors – one on top of each other. (Ren et al. 2018). One issue that needs to be mentioned when using DXA is the radiation dosage. Even though it is small, it might limit the usage in certain population, like children and pregnant women. The radiation dosage is less than the dosage exposed during a commercial flight (Valcovic, 2019). The DXA used in the study was Lunar Prodigy Advanced, shown in figure 8, and the software for running the measurement was enCORE™ software GE Healthcare encore version 14.10.
Before the first scan in the morning, DXA was calibrated via standard calibration block (GE Lunar Healthcare, 2016), after which the following participant’s information was input in the program: name, ID, birth date, height and weight. The program calculated the thickness of the body and choice between three programs, each with different time consumptions. The body was classified either thin (<16 cm), standard (16-25 cm) or thick (>25 cm). A single measurement lasted less than 15 minutes. For the scan participants were asked to dress up slightly (e.g. shorts and t-shirt) and to remove shoes, socks, jewelry and all metal objects.

Participants were instructed to relax and lie supine on the scanning bed with hands by their sides and palms placed in a neutral position. During all body scans, participants were asked to remain still, and a block with straps was placed against the feet and the straps were used to standardize the position. (Nickerson et al. 2019: Schubert et al. 2018). Small pea bags were
situated in armpits and between a palm and a thigh on both sides. It was confirmed that the entire body was inside the scanning area. If the participant was wider than the scanning area, a technique called MirrorImage was used, where the right side of the body and head and the spinal cord were inside the scanning area. Scanning happens only for a half of the body and the estimation of the total body composition is done from the half body scan. (Rothney et al. 2009; Tataranni & Ravussin, 1995).

8.4.3 Cardiorespiratory fitness test

Cardiorespiratory fitness was assessed with VO$_2$max, which refers to the maximum amount of oxygen that can be utilized during exercise. VO$_2$max can be measured indirectly, which means that the value is based on the linear relation between the heart rate and the work performed, when the metabolic rate, circulation and respiration have reached a “steady-state” response to submaximal work and subsequent extrapolation to maximal heart rate. (Andersen et al. 1971).

In this study, the indirect measurement used, was a 3-ladder bicycle ergometer test. In this test a participant cycles 3-4 increasing submaximal workloads, with a tempo of 60 repetitions per minute and the duration of four minutes per each load. The maximum heart rate needs to be measured or estimated, so the prediction of maximal workload is possible. (Keskinen et al. 2010, pp. 86–88.) In this study, the maximum heart rate (MAX HR) was based on the age and calculated with the following formula by Jones & Campbell (1975): MAX HR [210 - (0.65 x age)]. The Monark Bicycle ergometers, as shown in figure 9, were used in a test.
FIGURE 9. Monark bicycles.

Before the test VO$_{2\text{max}}$ was estimated based on the participant’s gender, age, weight, height and the level of physical activity by using a NEX-formula: $56,363 + (1,921 \times \text{Activity}) - (0.381 \times \text{Age}) - (0.754 \times \text{BMI}) + ((10,987 \times \text{Gender} \ (\text{man}=1, \ \text{woman}=0))$. The estimated VO$_{2\text{max}}$, was used to calculate the present values of VO$_{2\text{max}}$ for each workload: 1. 38 % of VO$_{2\text{max}}$ (warm-up), 2. 52 % of VO$_{2\text{max}}$, 3. 65 % of VO$_{2\text{max}}$, 4. 78 % of VO$_{2\text{max}}$, and the values were entered to a formula $VO_2 = \frac{12.35 \times P}{BM} + 3.5 \Rightarrow P = \frac{(VO_2 - 3.5) \times BM}{12.35}$ to get the correct watts for each workload. (Keskinen et al. 2010, pp. 86–88.)

Prior to the start, the course of the test was explained, and the bike was adjusted for the participant. At first, the participant biked a 4-minute warm-up, which was followed by the actual test including 3-4 workloads. If the heart rate was not close to the one intended, the following workload was corrected and if the intended heart rate was not reached in the third workload, one additional workload was added. In the end of each four-minute workload, the average heart rate was saved from the last 15 seconds, the RPE was asked by using a printed PRE-scale and the watts were increased based on the calculated values. RPE-table used was Borg’s original 15 state table. After the test was completed, a regression equation was formed
from heart rates and workloads to get an estimated workload for maximal heart rate. From the resulting value, \( VO_2 \text{max} \) was calculated with the following formula: 
\[
VO_2 \text{max} = (12.35 \times \frac{P\text{max}}{BM}) + 3.5,
\]
where \( P \) is W/HR and \( BM \) stands for body mass (Andersen et al. 1971; Keskinen et al. 2010, pp 86–66).

### 8.4.4 Metabolic health indicators

The following blood lipids (total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides) and blood glucose were measured from blood samples. The blood samples were done as a part of laboratory measurements in a fasted state, between 06.00 and 09.00, by a qualified and experienced technician from the university. Venous blood samples were collected from the antecubital vein into Vacuette SST 6 ml tubes using sterile needles. The sample was centrifuged for 10 minutes at 2000 rpm after which serum was removed and stored at 80 °C until chemical analyses.

Serum samples of glucose, lipids and lipoproteins were analysed with Colorimetric assay, Konelab 20 analyzer (Thermo, Vantaa, Finland). Sensitivities (mmol/l) and coefficients of variation (with-in-assay CV % average) were 0.10 mmol/l and 0.80 % for glucose, 0.10 mmol/l and 1.00 % for total cholesterol, 0.04 mmol/l and 0.80 % for HDL-cholesterol, 0.04 and 1.10 % for LDL cholesterol, 0.02 mmol/l and 1.30 % for triglycerides.

### 8.5 Statistical analysis

All statistical analysis was done with SPSS -statistical analysis program (PASW Statistics for Windows 22.0.) and Microsoft Excel (2016) was used for the calculations and for chart creation.

Shapiro-Wilk test was used for the exploration of normal distribution with the p-value set at 0.05. In addition, both kurtosis and skewness were examined. Either parametric or non-parametric test was used, based on the distribution of the values, as presented below.
For the calculation of correlation, Pearson correlation was used for normally distributed values and Spearman’s rho correlation for the values that were not normally distributed. The correlation was significant if the p value was under 0.05 (p<0.05*), 0.01 (p<0.01**), and 0.001 (p<0.001***).

There were no statistically significant differences between the control and experimental group in the pre-measurement values. Under these circumstances, it was possible to calculate changes within groups between the pre- and post-measurements and then compare the changes between the groups. Independent samples t-test was used for normally distributed values (Levene’s test for equality of variances), whereas Mann-Whitney U-test was used for the values that were not normally distributed. The difference was significant if the p value was under 0.05 (p<0.05*), and for stronger signification under 0.01 (p<0.01**), and 0.001 (p<0.001***).

The significance of change within groups between the pre- and post-measurement was tested with repeated measures ANOVA when the values were normally distributed and with Wilcoxon signed rank test when the values were not normally distributed.
9 RESULTS

9.1 Body composition

The results from body composition measurements are shown in table 8. Changes were found in body composition measures between the control and experimental groups in the pre- and post-measurements in fat mass (p<0.05), android fat mass (p<0.05) and fat percent (p<0.01). Fat-free-mass changed in the experimental group, but the change was not significant (p>0.05).

TABLE 8. Mean (±SD) body composition results of the studied variables. Significant changes between the pre- and post-measurements are marked with *. (*p<0.05, **p<0.01, ***p<0.001).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Experimental</th>
<th>Pre</th>
<th>Post</th>
<th>Change</th>
<th>Control</th>
<th>Pre</th>
<th>Post</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>32.3 ±3.3</td>
<td>31.9 ±3.3</td>
<td>-0.4 ±0.7</td>
<td>31.5 ± 3.3</td>
<td>31.4 ±3.4</td>
<td>-0.1 ±0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>90.8 ±11.4</td>
<td>89.7 ±11.3</td>
<td>-1.1 ±2.0</td>
<td>87.3 ±14.6</td>
<td>87 ±14.5</td>
<td>-0.3 ±0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>46.6 ±7.1</td>
<td>47.0 ±7.1</td>
<td>0.4 ±1.2</td>
<td>44.4 ±5.3</td>
<td>44.2 ±5.1</td>
<td>-0.2 ±1.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>41.3 ±7.4</td>
<td>39.8 ±7.3</td>
<td>-1.5 ±1.5***</td>
<td>39.9 ±10.3</td>
<td>39.9 ±10.2</td>
<td>0.0 ±1.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat (%)</td>
<td>46.9 ±5.0</td>
<td>45.8 ±5.1</td>
<td>-1.1 ±1.1***</td>
<td>46.8 ±5.1</td>
<td>46.9 ±4.6</td>
<td>0.1 ±1.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Android mass (kg)</td>
<td>4.1 ±1.0</td>
<td>4.0 ±1.0</td>
<td>-0.2 ±0.2**</td>
<td>4.0 ±1.0</td>
<td>4.0 ±1.1</td>
<td>0.0 ±0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A/G-ratio (%)</td>
<td>1.1 ±0.1</td>
<td>1.1 ±0.1</td>
<td>0.00 ±0.03</td>
<td>1.1 ±0.1</td>
<td>1.1 ±0.1</td>
<td>-0.01 ±0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Android fat (%)</td>
<td>54.6 ±4.1</td>
<td>53.3 ±4.3</td>
<td>-1.3 ±1.2*</td>
<td>55.0 ±4.6</td>
<td>54.6 ±4.6</td>
<td>-0.4 ±1.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gynoid fat (%)</td>
<td>50.8 ±6.8</td>
<td>49.7 ±7.1</td>
<td>-1.0 ±1.4*</td>
<td>50.1 ±5.1</td>
<td>50.1 ±4.8</td>
<td>0.1 ±1.6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* A/G-ratio = android-gynoid ratio
9.1.1 Total fat mass

The change from the pre- to post-measurements in total fat mass was highly significant ($p<0.01$) between the experimental group (from 41.3 ±7.4 to 39.8 ±7.3 kg) compared to the control group (from 39.9 ±10.3 to 39.9 ±10.2 kg). In the experimental group the mean decrease in total fat mass was 1.5 ±1.5 kg ($p<0.001$) and in the control group, there was no change, as shown in figure 10.

![Figure 10](image-url)  
**FIGURE 10.** The total fat mass in the pre- and post-measurements within both groups.
9.1.2 Fat percent

The change from the pre- to post-measurements in fat percent was significant (p<0.05) between the experimental group (from 46.9 ±5.0 to 45.8 %) compared to the control group (from 46.8 ±5.1 to 46.9 ±4.6 %). In the experimental group, the mean decrease in fat percent was 1.1 ±1.1 % (p<0.001) and in the control group, there was no change, as shown in figure 11.

FIGURE 11. The fat percent in the pre- and post-measurements within both groups.
9.1.3 Android fat mass

The change from the pre- to post-measurements in android fat mass was significant ($p<0.05$) between the experimental group (from 4.1 ±1.0 to 4.0 ±1.0 kg) compared to the control group (from 4.0 ±1.0 to 4.0 ±1.1 kg). In the experimental group, the mean decrease in android fat mass was 0.17 ±0.2 kg ($p<0.01$) and in the control group, there was no change, as shown in figure 12.

FIGURE 12. The android fat mass in the pre- and post-measurements within both groups.
9.2 Estimated maximal oxygen uptake

The change from the pre- to post-measurements in estimated VO$_2$max was highly significant (p<0.001) between the experimental group (from 25.6 ±5.6 to 29.8 ±4.7 ml/kg/min) compared to the control group (from 30.3 ±4.8 to 25.6 ±4.7 ml/kg/min). In the experimental group the mean increase in estimated VO$_2$max was 4.2 ±5.7 ml/kg/min (p<0.01) and in the control group the mean decrease was -4.8± 4.3 ml/kg/min (p<0.01), as shown in figure 13.

![Figure 13](image)

FIGURE 13. The estimated VO$_2$max (ml/kg/min) in the pre- and post-measurements within both groups.

9.3 Metabolic health indicators

No changes were observed in metabolic health indicators. The values are presented in table 9.
TABLE 9. Mean (±SD) metabolic health indicator results of the studied variables and the changes between the pre- and post-measurements in both groups p<0.05*, p<0.01**, p<0.001***.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Experimental</th>
<th>Control.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Change</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>5.2 ±0.8</td>
<td>5.1 ±0.7</td>
<td>-0.2 ±0.6</td>
</tr>
<tr>
<td>LDL (mmol/l)</td>
<td>3.3 ±0.6</td>
<td>3.1 ±0.6</td>
<td>-0.2 ±0.5</td>
</tr>
<tr>
<td>HDL (mmol/l)</td>
<td>1.6 ±0.4</td>
<td>1.4 ±0.4</td>
<td>-0.2 ±0.1</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>1.2 ±0.5</td>
<td>1.3 ±0.7</td>
<td>0.05 ±0.4</td>
</tr>
<tr>
<td>Blood glucose (mmol/l)</td>
<td>5.7 ±0.6</td>
<td>5.7 ±0.5</td>
<td>0.02 ±0.37</td>
</tr>
</tbody>
</table>

9.4 Correlations between the variables

The changes in estimated VO$_2$max had a significant negative correlation with the changes in total body fat mass ($r$=-0.474, p<0.01), as shown in figure 14.

FIGURE 14. Correlation between absolute changes in total fat mass and maximal oxygen uptake (VO$_2$max).
The changes in android fat mass had a significant positive correlation with the changes in total fat mass ($r=0.669$, $p<0.001$), as shown in figure 15.

FIGURE 15. Correlation between changes in total fat mass and in android fat mass.
10 DISCUSSION

Main findings. Fat mass reduced significantly in the experimental group between the pre- and post-measurements, but no changes were observed in fat distribution, which was estimated via android-gynoid ratio. In addition, fat-free mass was maintained, and cardiorespiratory fitness increased in the experimental group. In control group, no changes were observed in body composition, but cardiorespiratory fitness decreased. No changes were observed in metabolic health indicators nor in associations between them and android fat mass in either of the groups between the pre- and post-measurements.

Body composition. In order to lose weight, there is a demand for negative energy balance (Marlatt & Ravussin, 2018, pp. 41–44). In this study it was observed that negative energy balance is possible to achieve with guidelines-based exercise-intervention and the amount of body fat is possible to decrease without caloric restriction. These findings answer the research question one and support the hypothesis one.

Similar findings have been found in various previous studies (Garrow & Summerbell, 1995). For example, in the study of Quist et al. (2017) three months intervention, with bicycle exercising only, produced an average of 3.3 kg fat loss, while in this study the average fat loss was 1.5 kg in eight weeks. Since the decrease in body fat is a consequence of negative energy balance, it means that there has been an average of -11550 kcal in 8 weeks total, which would mean - 206 kcal per daily balance. The calculations (1.5 kg / 0.45 kg x 3500 kcal / 56 d) assume that 3500-kcal deficit will result in one pound of fat loss (approximately 0.45 kg) (Wishnofsky, 1958). Donelly et al. (2009) stated in their review that moderate intensity PA of 150 to 250 min per week with and energy equivalent of 1200 to 2000 kcal per week may result in modest way lost, which is supported by the findings in this study, as the weekly amount of physical activity was 150 min per week and the average weekly energy deficit 1442 kcal.

Based on the calculation of Wishnofsky (1958), the widely used and accepted weight-loss strategy is to produce a 3500-kcal deficit per week, which results in - 0.5 kg per week and 24 kilograms in a year (Thomas et al. 2013). In this study the weight-loss pace was - 0.188 kg per
week, which is less than half of the pace used in usual strategies. The 3500-kcal deficit strategy has been proven to be an invalid method for longer periods of time (Thomas et al. 2011; Thomas et al. 2013), because the body reacts to negative energy balance with a reduction of RMR (Leibel et al. 1995). According to Thomas et al. (2011) the self-limiting nature of weight loss is best captured by thermodynamically based models, not by the 3500-kcal rule.

In this study, exercising produced a moderate daily caloric deficit, which resulted in small weight loss over the course of 8 weeks within the experimental group. Chaston & Dixon, (2013) concluded that visceral adipose tissue as well can be lost preferentially with modest weight loss, as seen in this study as a reduction of android fat mass. In addition, modest weight loss is considered to be more sustainable than large weight loss (Korkeila et al. 1999). According to this study, the official guidelines for physical activity will help overweight women to reduce crucial abdominal fat mass, as well as total fat mass, without any caloric restrictions.

Android fat mass decreased significantly in the experimental group compared to the control group and the decrease in android fat mass was correlated with the decrease in total fat mass, which answers research question one and supports its hypothesis. Similar findings have been seen in the study of Ross et al. (2000), where the exercise-induced-weight-loss-group reduced both total and visceral fat more than the control group or exercise-without-weight-loss-group. However, they also showed that the exercise-without-weight-loss-group reduced visceral fat more than the control group. In this study, there was not a significant change in android-gynoid percent fat ratio between the pre- and post-measurements in neither of the groups, which means the fat distribution did not change as a result of exercise intervention. This answers the research question three, but does not support its hypothesis. This result was opposite to that of Serra et al. (2017), who showed that android-gynoid ratio decreased with weight-loss + exercise group, but not with weight-loss-only group in sedentary overweight women. This difference could be explained by the duration of the intervention and the total amount of weight lost. In the study of Serra et al. (2017) the duration of the study was 6 months and the total decrease in weight 8 %. Comparing to this study, where the both were less, as the duration of the study was only 8 weeks and the total decrease in weight only 2 %. Longer intervention could have resulted in a larger weight loss.
With exercise-based weight loss interventions it is possible to maintain the fat-free mass (Quist et al. 2017) or even increase it, when resistance training is performed (Schmitz et al. 2003). Dulloo et al. (2012) stated that the loss of muscle mass is most likely one explanation for weight regain occurring after a diet. In this study fat-free mass was maintained in the experimental group, while the fat mass was decreased. These results correspond with ones from Schmitz et al. (2003) and a parallel statement can be made that combined aerobic exercising and resistant training is an effective way to improve body composition.

The problem, which remains unsolved, is how to motivate sedentary overweight people to exercise frequently and in needed intensities. It is known that very rare Finnish adults achieve the physical activity guidelines (Husu et al. 2018) and the Finnish adolescents’ sedentary behavior has increased, and physical activity has decreased in past decades (Santtila et al. 2006). In this study it would have been interesting to observe what happens after the study: whether the participants keep up with exercising or stop right away or over time. It would have been interesting to see if the changes in total fat mass and android fat mass were sustained or lost in a longer period.

Cardiorespiratory fitness. It is a widely accepted fact that endurance exercise training improves cardiorespiratory fitness in sedentary population (HHS, 2018). The same finding was made in this study, as both the absolute and the relative VO$_2$max values significantly increased in the experimental group compared to the control group between the pre- and post-measurements. This finding answers the research question one and supports its hypothesis.

Furthermore, this result supports the findings in the meta-analysis from Valkeinen et al. (2010). The meta-analysis investigated effect of exercise training on oxygen uptake in CHD. It indicated that exercise-groups who performed endurance exercise trainings on an average 3.1 ±0.4 times per week for an average duration of 14.2 ±13.5 weeks, resulted in significant increments in VO$_2$max (2.6 ±1.6 ml/kg/min), compared to non-exercising control groups, which is similar to the VO$_2$max change (4.2 ±5.7 ml/kg/min) observed in the experimental group in this study. The meta-analysis also showed that longer interventions (over 6 months) appeared to be more effective than short ones. In the current study, the intervention was only 8 weeks, so
almost a half shorter than the average study in the meta-analysis. It can be speculated that greater increments could have been seen with longer intervention period. The changes in absolute VO$_2$max values implicate that the change in relative VO$_2$max was not just a consequence of a change in body weight.

What is also worth noting, is that the VO$_2$max decreased in control group -4.8 (±4.3) ml/kg/min, which can be a consequence of decreased habitual physical activity. The pre-measurement was done in the end of August, while the post-measurement was completed in late October and early November. In addition, the VO$_2$max is an estimated value and the errors are possible.

*Metabolic health indicators.* The role of exercising in the treatment of dyslipidemia is not trivial, as the results of different studies broadly vary from each other (Leon & Sanchez, 2001). In this study, however, no changes were observed in the metabolic health indicators between the pre- and post-measurements in either of the groups. These findings suggest that exercise itself is not a powerful method to improve dyslipidemia or high blood glucose in 8 weeks. This answers the research question one, but does not support the hypothesis one.

The mean HDL, triglycerides and blood glucose of the participants were in normal limits to begin with, whereas total cholesterol and LDL cholesterol were only slightly higher than recommended. This might be one explanation why there were no changes in these values. According to the FINRISK-research (Koponen et al. 2017) the average total cholesterol concentration in Finns over the age of 30 years is 5.1 mmol/l in women, which is slightly less than the average total cholesterol value (5.5 ± 0.8 mmol/L) measured in the pre-measurements. However, the mean value in experimental group was 5.2 ± 0.8 mmol/L in the pre-measurements.

In addition, the intervention time might not have been a long enough, at least according to Tambalis et al. (2009), who stated that an exercise intervention under 12 weeks is not long enough to observe changes in blood lipids. In addition, the guidelines for physical activity (HHS, 2018) underline the importance of the reduction in sedentary behaviour, in addition to exercise training. This was not the case in this study, since the sedentary behaviour was not
reduced. Longer intervention, with the possible reductions in sedentary behavior and participants diagnosed with dyslipidemia might have produced a different outcome. Based on the meta-analysis from Leon & Sanchez (2001), the most possible observation, among the metabolic health indicators, would have been a slight increase of HDL. However, it has to be pointed out that according to Leon et al. (2000), exercise-induced changes in HDL are highly variable between individuals. As there were no changes observed in metabolic health indicators, there were no associations shown between them and android fat or total fat mass between the pre- and post-measurements, which answers the research question two, but does not support the hypothesis two.

**Limitations** DXA does not measure VF, which refers to the fat around the organs. DXA only measures the amount of fat mass in the abdominal section of the body: android fat mass. The use of MRI or CT could have provided more specific information of visceral fat. However, based on the study of Lee et al. (2018) DXA-measured android fat percent is a predictor (Adjusted R-square = 0.73) for MRI-measured visceral fat. In addition, the use of direct VO2max measures could have provided more accurate information of cardiorespiratory fitness. However, the submaximal exercise test is considered highly reliable as an estimate of VO2max when used for women (Hartung et al. 1995). MRI, CT and direct VO2max measurements are more expensive and time-consuming, and thus mostly used in clinical settings.

Nutrition follow-up would have provided information of daily caloric intake and saturated fat intake, whereas physical activity follow-up would have provided information of off-study physical activity and the TDEE. The information of total daily caloric intake and TDEE could have offered more tools for understanding the changes observed in total fat mass and the android fat mass. The number of participants was small (n=37). Especially when the groups were analyzed individually (n=13 and n=24). A larger number of participants would have made the results more reliable. Furthermore, as the study was conducted only on females, assumptions cannot be made regarding both genders. For example, abdominal obesity and android fat is more common in males than in females (Krotkiewski et al. 1983), which could have affected the results of fat distribution. The duration of the study might play a role as a limiting factor for metabolic health indicators and fat distribution. It might be possible that changes in metabolic health indicators cannot be observed in a such a short period, as 8 weeks (Tambalis et al. 2009).
As well, possible observations in the changes of fat distribution might have demanded a larger reduce in total fat mass, thus a longer intervention time (Serra et al. 2017).

**Strengths.** The group of participants was homogenous, as they were all women, overweight, sedentary and had at least one risk factor for cardiovascular diseases. The exercise intervention was highly controlled and supervised and the attendance to practices was high among all experimental group participants (91.8 %). All the supervisors were students of the faculty of Sport Science of the University of Jyväskylä. There were no dropouts during the study, which could propose that the exercising safety and participant motivation were on a high level. The study was systematically organized and hardly any measurements were re-organized. The study was an RCT, which remains the “gold standard” for evaluating the effects of intervention. This study is readily translated outside its original experimental setting, for replication, comparison, and adaptation elsewhere.

**Scientific conclusion.** Based on this study, a statement can be made that exercise-induced weight loss is possible to achieve in 8 weeks, as well as a decrease in android fat mass. Furthermore, this weight-loss can be achieved without a decrease in fat-free mass. It can also be concluded that a moderate amount of progressive exercise training for three times a week is a valid way to improve cardiorespiratory fitness. However, an 8-week exercise-intervention is not an effective way to improve fat distribution or metabolic health indicators, despite the decrease in android fat mass.

**Practical conclusions.** When taken into consideration that in Finland 2/3 of women and 3/4 of men are overweight (Koponen et al. 2017), practical and sustainable methods for weight loss are needed. The findings from this study suggest that exercise-induced weight loss, without any changes in dietary or caloric intake, is possible and moderate, even when the amount of exercise is smaller than the recommended amount in the official guidelines (HHS, 2018). Furthermore, the study shows that even a small amount of resistant training helps to maintain fat-free mass, even during a negative energy balance. This study provides information and knowledge to fitness instructors, who work with people struggling with weight management, but do not necessarily have nutritional education. Even though exercise training did improve body
composition and cardiorespiratory fitness, it has to be kept in mind that there is also a need for reductions in sedentary behaviour (HHS, 2018), as it is independently associated with mortality (Koster et al. 2012) and non-communicable diseases (Hu et al. 2003).

Based on this study and previous literature, exercise is the best way to increase TDEE, cardiorespiratory fitness and muscle mass, enabling weight-loss and weight management. This exercise-induced weight loss was moderate and easily accessible, as the trainings could be modified as daily routines with minimal effort. 30 minutes of bicycling could be carried out as commute to work and 20 minutes strength training done at home.

_Suggestions for the future studies._ Since this study did not provide changes in metabolic health indicators or in fat distribution, further study is needed. A longer study and participants with abnormal blood lipid levels could provide more valid information. As well as a study with different exercise intervention, for example one that follows the official exercise guidelines, but where exercise intensity is more vigorous or the weekly amount increases up to 300 minutes. In addition, nutrition research could be implemented as part of the study, as it would make the research of metabolic health indicators and body composition more accurate. An intervention that is conducted independently, for example at-home work-outs, could provide a more practical point of view.
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Sydän, sauna ja liikunta


Mitattuksista saatte arvokasta tietoa omasta kehonkoostumuksesta, suorituskyvyystä, sekä terveydentilasta ja näistä kaikista teidän on mahdollista keskustella tutkimuksessa mukana ollevien asiainmuutajoiden ja lääkärin kanssa. Harjoitusohjelma suunnitellaan henkilökohtaisesti juuri teille ja jokaisesta harjoituskertaa valvoo liikuntatieteiden opiskelija, joka tarvittaessa auttaa ja vastaa kysymyksiin. Kaikki mitattukset, liikuntaharjoitukset ja saunominen tapahtuvat Vivecalla (Rautolahjankatu 8).

Olette sopiva tutkimukseen, mikäli olette noin 30-60-vuotias ei ollenkaan tai vähän liikuntaa harrastava henkilö ja teillä on vähintään yksi sydän- ja verisuonitautien riskitekijä (korkea verenpaine tai kolesteroli, tupakointi, keskivartalolihavuus, suvussa esiintyvä varhainen sepelvaltimoattai tai painoindexi yli 30 kg/m²), ilman aikaisempaa sepelvaltimoattaita tai diabetetta. Teillä ei tulisi olla tulehduskellista sairautta, hengitys- ja verenkiertoelimiten sairautta, tai sairautta, jotka haittaavat tutkimukseen liittyviä liikuntasuorituksia. Teidän ei tulisi myöskään saunoa useammin kuin kerran viikossa.

Mikäli olette mielestänne sopiva tutkimukseen, otkaaakaa yhteyttä tutkimukseen yhteyshenkilöön Jari Kohunarka, sähköpostite (iiankolu@student.jyu.fi), niin kerromme lisätietoja. Kertokaa sähköpostissasi nimi, pituus, paino ja ikä, sekä kuvaalaa nykyistä liikunta-aktiivisuutanne, saunomistotumuksianne ja teillä löytyvä riskitekijöitä.

Tutkimusryhmän puolesta

Professori, kardiologi Jari Laukkanen, Keski-Suomen sairaanhoitopiiri
APPENDIX 2.

SUOSTUMUS TUTKIMUKSEEN

Sydän, sauna ja liikunta

_Jyväskylän yliopiston liikunta- ja terveyslaboratorio, Jyväskylän yliopisto, Jyväskylä_

Minua______________________ (tutkittavan nimi) on pyydetty osallistumaan yllämainittuun tieteelliseen tutkimukseen, jonka tarkoituksena on mitata lämpöaltisteen (saunominen), lihaskuntoharjoittelun ja kestävyysharjoittelun vaikutuksia kehossa.

Olen lukenut ja ymmärtänyt saamani kirjallisen tutkimustiedotteen. Tiedotteesta olen saanut riittävän selvityksen tutkimuksesta ja sen yhteydessä suoritettavasta henkilötietojen keräämisestä, käsittelystä ja luovuttamisesta. Tiedotteen sisällö on kerrottu minulle myös suullisesti, minulla on ollut mahdollisuus esittää kysymyksiä ja olen saanut riittävän vastauksen kaikkiin tutkimusta koskeviin kysymyksiin.

Tiedot antoi   ____________________________________        ____ / ____ / 2019.

Minulla on ollut riittävästi aikaa harkita osallistumisistani tutkimukseen. Olen saanut riittävät tiedot oikeuksistani, tutkimuksen tarkoituksesta ja sen toteutuksesta sekä tutkimuksen hyödyistä ja riskeistä. Minua ei ole painostettu eikä houkuteltu osallistumaan tutkimukseen.

Tiedän, että tietojani käsitellään luottamuksellisesti eikä niitä luovuteta sivullisille.

Ymmärrän, että osallistumiseni tutkimukseen on vapaaehtoista. Olen selvillä siitä, että voin peruuttaa tämän suostumukseni koska tahansa syytä ilmoittamatta eikä peruutukseni vaikuta kohteluuni tai hoitoonni millään tavalla.

Olen tietoinen siitä, että mikäli keskeytän tutkimuksen tai peruutan suostumuksen, minusta keskeyttämiseen ja suostumuksen peruuttamiseen mennessä kerättyjä tietoja ja näyttöitä voidaan käyttää osana tutkimusaineistoa voimassa olevan lainsäädännön sallimissa rajoissa.

Suostun siihen, että tutkimuksessa käsitellään erityisiin henkilötietoryhmiin kuuluvia tietoja (terveyttä koskevat tiedot)

- Kyllä

Suostun siihen, että tutkimuksen päätettyä aineistoa arkistoistaan tunnisteellisena 5 vuoden ajan

- Kyllä
- Ei

Suostun myöhempään yhteydenottoon jatkotutkimuksia varten seuraavan viiden vuoden sisällä

- Kyllä
- Ei

1
Allekirjoituksellani vahvistan osallistumisen tähän tutkimukseen ja suostun vapaaehtoisesti tutkimushenkilöksi.

Tutkittavan nimi

Tutkittavan syntymäaika

Tutkittavan osoite

Paikka ja aika

Allekirjoitus

Suostumus vastaanotettu

Tutkimuksen vastuullisen johtajan nimi

Paikka ja aika

Allekirjoitus (Suostumuksen vastaanottaja)

Alkuperäinen allekirjoitettu tutkittavan suostumus sekä kopio tutkimustiedotteesta jäävät tutkijan arkistoon. Tutkimustiedote ja kopio allekirjoitetusta suostumuksesta annetaan tutkittavalle.
APPENDIX 3.

Sydän, sauna ja liikunta -tutkimus 2019

TAUSTATIETOKYSELY

Tutkimus ID (syntymäpäivä, syntymäkuukausi, etunimen 1. kirjain ja sukunimen 1. kirjain): ___ ___ ___ ___

Nimi ___________________________________________ Syntymäaika _____. _____.____________

Onko Teillä todettu jokin seuraavista sairauksista?

Diabetes, tyyppi I ___
Diabetes, tyyppi II ___
Kilpirauhasen sairaus ___ mikä? _____________________________
Kohonnut verenpaine tai verenpainetauti ___
Anemia ___
Rintakipua ___
Sepelvaltimotauti ___
Muu sydän- ja verisuonisairaus ___ mikä? _____________________________
Migreeni ___
Nivelreuma tai muu reumatologinen sairaus ___
Tuki- ja liikuntaelinsairaus ___ mikä? _____________________________
Ihotuma tai muu ihosairaus ___ mikä? _____________________________
Hengityselinsairaus ___ mikä? _____________________________
Huimaus ___
Masennus ___

Jos Teillä on jokin edellä mainituista sairauksista/oireista, milloin sairaus todettiin ja mikä on sairauden nykytila ja lääkitys? ___________________________________________________________________

Onko Teillä muita sairauksia tai oireita? ____________________________________________________________

Onko Teillä muita sairauksia tai oireita? ____________________________________________________________

Onko suvussanne sepelvaltimotautia nuorella iällä (miehillä alle 55- vuotiaana, naisilla alle 65- vuotiaana)? ___ kyllä ___ ei ___ en osaa sanoa

Onko suvussanne muita sydän- ja verisuonitauteja? ___ kyllä ___ ei ___ en osaa sanoa

Pituutteen (cm) _____ ja painonne (kg) ______

Harrastatteko kestävyystyypistä liikuntasäikeen (esim. kävelyä, juoksua, pyöräilyä, hiihtoa, uintia)? ___ kyllä ___ en

Jos harrastatte kestävyysliikuntaa, kuinka usein ja millaista liikuntaa harrastatte?
Kuinka kauan yksi liikuntasuoritus kestää (min.)?________________________________________________

Harrastatteko lihaskuntoa kehittävää liikuntaa viikoittain (esim. kuntosali)       __kyllä     __en

Jos harrastatte lihaskuntoa kehittävää liikuntaa, kuinka usein ja millaista liikuntaa harrastatte?
_______________________________________________________________________________________

Kuinka kauan yksi liikuntasuoritus kestää (min.)?  ______________________________________________

Harrastatteko arkiliikuntaa viikoittain (esim. työmatkaliikuntaa, puutarhanhoitoa tai muuta fyysisesti rasittavaa
askaretta)?       __kyllä     __en

Jos harrastatte arkiliikuntaa, kuinka usein ja millaista arkiliikuntaa harrastatte?
_______________________________________________________________________________________

Kuinka kauan yksi liikuntasuoritus kestää (min.)?  ______________________________________________

Tupakoitteko?       __kyllä     __en

Saunotteko?       __ en sauno      ___ harvenmin kuin kerran viikossa       ___ kerran viikossa
     ___ 2-3 kertaa viikossa       ___ 4-7 kertaa viikossa

Jos saunotte, kuinka kauan yksi saunomiskerta jäähyttelyineen kestää (min.)?
_______________________________________________________________________________________

Jos saunotte, missä lämpötilassa?
_______________________________________________________________________________________

Mikä on oma arvio terveydentilastanne?
_______________________________________________________________________________________
_______________________________________________________________________________________
_______________________________________________________________________________________
AIKATAULUTUS


Maanantaina, keskiviikkona ja perjantaina kello

___ 16:00 – 17:30
___ 16:30 – 18:00
___ 17:00 – 18:30
___ 17:30 – 19:00
___ 18:00 – 19:30
___ 18:30 – 20:00
___ 19:00 – 20:30
___ 19:30 – 21:00
___ 20:00 – 21:30

☐ Kaikki ajat sopivat minulle

Lisätietoja

_______________________________________________________________________________________
_______________________________________________________________________________________
_______________________________________________________________________________________

Kiitos vastauksistanne.