THE EFFECT OF HIGH-INTENSITY INTERVAL EXERCISE PROGRAM ON BLOOD LIPIDS AND HORMONES IN RECREATIONALLY ACTIVE ADULTS

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ABSTRACT

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Introduction. Type 2 diabetes and cardiovascular diseases have become more and more common and the best known prevention is physical activity. People in Finland are poorly following physical activity regulations and the most common reasons for inactivity are lack of time and lack of motivation. High-intensity interval training has become very trendy and popular as it is time efficient, and the effects to fitness and health are thought to be at least the same than after traditional aerobic training. The purpose of this study was to investigate, if there are differences in blood lipids and hormones between the two different types of HIIT protocols (HIIT running versus HIIT circuit) when the amount of work and intensity are the same but the training method is different. Currently, the existing data is limited. The objective was also to study, if these two HIIT protocols differ from traditional continuous aerobic running when considering fitness and health parameters.

Methods. In this study, there were 24 healthy, recreationally active adults as subjects. The subjects were randomly assigned to one of the tree groups: high-intensity interval running (HIIT, n=8), high-intensity interval circuit training (HICT, n=8) or steady-state running (n=8). The subjects trained three times per week for 8 weeks. The HIIT consisted of 8–10 x 1 min submaximal running intervals separated with 30 s active recovery, HICT consisted of 8–10 exercises performed maximally as circuit training (one minute per exercise) separated with a 30 s recovery between the movements and steady-state running consisted of 40–60 minutes steady state running. Blood samples were taken before and after the eight week training period and TC, HDL, LDL, triglycerides, glycerol, FFA, insulin, leptin and cortisol were analyzed. In addition, the subjects kept a 5-day food diary twice during the study period and the diaries were analyzed with NutriFlow-program.

Results. The main result was that there were no differences between the HIIT and HICT groups in any of the measured variable. Insulin decreased significantly after 8-week high-intensity interval training (both interval groups combined) by 16,7 % (from 60,0±29,8 pmol/l to 50±15,4 pmol/l). There were no other significant changes.

Discussion. Based on this study, the 8-week high-intensity interval training decreases insulin concentration in blood without affecting blood glucose concentration. It seems that high-intensity interval training facilitates insulin to function more efficiently, so less insulin is needed for glucose transportation.

Key words: blood lipids, hormones, high-intensity interval training, high-intensity circuit training, steady-state training

TIIVISTELMÄ

Malmivaara, Susanna (2015). Korkeatehoisen intervalliharjoitteluohjelman vaikutus veren lipideihin ja hormoneihin kuntoliikuntaa harrastavilla aikuisilla. Liikuntabiologian laitos, Jyväskylän yliopisto. Pro gradu -tutkielma, 76 s.

Johdanto. Tyypin 2 diabetes sekä verenkieroelimistön sairaudet ovat yleistyneet ja niiden tehokkaimpana ehkäisynä pidetään liikuntaa. Suomessa ihmiset eivät noudata liikuntasuosituksia ja syyksi inaktiivisuuteen sanotaankin olevan ajan ja motivaation puute. Korkeatehoinen intervalliharjoittelu on ollut suosittua siksi, että se säästää aikaa ja sen vaikutuksien terveyteen ja kuntoon ajatellaan olevan vähintäänkin samanlaiset kun aerobisen harjoittelun. Tämän tutkimuksen tarkoituksena oli tutkia onko kahden erityyppisen intervalliharjoittelun (juoksu ja kuntopiiri) välillä eroja veren lipideissä ja hormoneissa, kun työmäärä ja intensiteetti pysyvät samana. Tällä hetkellä tutkimuksia tästä aiheesta on hyvin rajallisesti. Tarkoituksena oli lisäksi tutkia, kuinka korkeatehoinen intervalli harjoittelu eroaa perinteisestä tasavauhtisesta harjoittelusta. **Menetelmät.** Koehenkilöinä oli 24 tervettä, kuntoliikuntaa harrastavaa aikuista. Koehenkilöt arvottiin satunnaisesti yhteen kolmesta ryhmästä: korkeatehoinen intervalliharjoittelu juosten, korkeatehoinen intervalliharjoittelu kuntopiirinä tai tasavauhtinen harjoittelu juosten. Koehenkilöt harjoittelivat kolme kertaa viikossa 8 viikon ajan. HIIT juosten sisälsi 8–10 x 1 min submaksimaalisia vetoja 30 sekunnin palautuksella, kuntopiiri sisälsi 8–10 lihaskuntoliikettä (minuutin per liike) 30 sekunnin palautuksella ja tasavauhtinen juoksu koostui 40–60 minuutin aerobisesta juoksemisesta. Verinäytteet otettiin ennen ja jälkeen harjoittelujakson ja niistä analysoitiin kokonaiskolesteroli, HDL, LDL, triglyseridit, glyseroli, vapaat rasvahapot, insuliini, leptiini sekä kortisoli. Koehenkilöt pitivät tutkimuksen aikana kahdesti ruokapäiväkirjaa, jotka analysoitiin NutriFlow -ohjelmalla. **Tulokset.** Päätulos tässä tutkimuksessa oli se, että 8 viikon harjoittelun jälkeen rasvoissa ja hormoneissa kahden erilaisen korkeatehoisen intervalliharjoittelun välillä ei ole eroja. Toinen päätulos oli se, että insuliini laski merkitsevästi 16,7 % molempien intervalliharjoittelujaksojen seurauksena (ennen 60,0±29,8 pmol/l ja jälkeen 50±15,4 pmol/l). Tutkimuksessa ei havaittu muita merkittäviä muutoksia. **Pohdinta.** Tämän tutkimuksen perusteella, 8 viikon mittainen korkeatehoinen intervalliharjoittelu laskee veren insuliinipitoisuutta vaikuttamatta veren glukoosipitoisuuteen. Näyttäisi siltä, että intervalliharjoittelu tehostaa insuliinin toimintaa ja siten vähemmän insuliinia tarvitaan glukoosin kuljettamiseen soluihin.

Avainsanat: Veren rasvat, hormonit, korkeatehoinen intervalliharjoittelu, korkeatehoinen kuntopiiriharjoittelu, tasavauhtinen harjoittelu

ABBREVIATIONS

HIIT	High-Intensity Interval Training
HICT	High-Intensity Circuit Training
LDL	Low density lipoprotein
HDL	High density lipoprotein
TC	Total cholesterol
VLDL	Very low-density lipoprotein
FFA	Free fatty acid
GLUT 4	Glucose transporter type 4
VO_{2max}	Maximal oxygen uptake
ATP	Adenosine triphosphate
CHO	Carbohydrate
PCr	Phosphocreatine
GLYC	Glycerol
GH	Growth hormone
IGF	Insulin-like growth factor

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1 INTRODUCTION

Different diseases like type 2 diabetes (T2D) and cardiovascular problems have become more and more common worldwide. Regular physical activity is the best known prevention method for these kinds of diseases. (For example Babraj et al. 2009.) In Finland, the current exercise recommendation for normal adults is to do aerobic physical activity several days in a week for a total of at least 2,5 hours of moderate intensity, or 1 hour 15 minutes of vigorous intensity. In addition, the recommendation is to increase also muscular strength and balance at least two times per week. (UKK-institute.) It seems though, that people are not following these recommendations and the most common reasons for inactivity are lack of time and lack of motivation. High-intensity interval training has been very popular in nowadays. The biggest reason for that is that it is time efficient, and the effects to fitness and health is thought to be at least the same than after traditional aerobic training. (Babraj et al. 2009; Gibala et al. 2012; Peake et al. 2014.)

High-intensity interval training consists of short but high intensity bouts interspersed by active low-intensity or passive rest periods. There are basically two different ways to do HIIT training: short but maximal bouts (for example 30 second all-out bursts) or longer (30 sec–4 min) intervals with submaximal levels (approximately 85–95 % VO_{2max}). In maximal type HIIT training, the rest periods have been about 4 minutes in studies whereas in submaximal HIIT the rest periods have been varying from 30 seconds to 3 minutes. All-out maximal HIIT is extremely hard and not suitable for all people like elderly and sick and that is why submaximal HIIT is better choice for most of the people. In a couple of studies, HIIT training has caused favorable changes in physiological parameters (blood variables, VO_{2max}, body composition) and in some cases it has been seen as more effective than traditional aerobic training. (Gibala et al. 2012.)

HIIT is mostly done by running or cycling but it can be done also as circuit training (Klika & Jordan 2013). The high-intensity circuit training (HICT) is a method that combines high-

intensity endurance training and high-intensity resistance training (Paoli et al. 2013). In high-intensity circuit training, person moves from one exercise to another quickly and the rest periods are short and that leads to short exercise sessions. (Miller et al. 2014). At this point, there is no data available about the effects of HICT on blood lipids and hormones in normal weight, healthy adults (Paoli et al. 2013). High-intensity interval training has grown its popularity because it can be done using own bodyweight so there is no need for expensive equipment, and it can be done anywhere. Also, it is time efficient. HICT is a combination of aerobic and resistance training done with high intensity and short rest periods. (Klika & Jordan 2013.) Training with own bodyweight is very common, popular at the moment and it is easy to everybody.

There are not a lot of studies that have investigated the effects of HIIT or HICT on hormones and lipids long-term, but there are several studies about acute effects of one interval training session. The hormones and lipids that we have selected in this study are important when thinking of health and fitness and those were easy and possible to measure and analyze in our laboratory.

The purpose of this study was to investigate, if there are differences between the two different types of HIIT protocols (HIIT running versus HIIT circuit) when the amount of work and intensity are the same but the training method is different. We wanted also to see, if these two HIIT protocols differ from traditional continuous aerobic running when considering fitness and health parameters. The objective was also to study how long-term interval training does affect to blood parameters (lipids and hormones) because the data of these parameters is currently lacking.

2 ENERGY METABOLISM IN HUMANS

2.1 Energy production from glucose

Glucose is a carbohydrate and it is a simple monosaccharide consisting of 6 carbon skeleton (figure 1). In the body, glucose is stored in the form of glycogen that is a polysaccharide. (Silverthorn et al. 2010, 28.) After glucose has been absorbed by the small intestine, glucose is used for cellular metabolism as energy source, stored as glycogen in the liver or muscles or converted to fat (McArdle et al. 2010, 8). Plasma glucose levels are kept in narrow limits (4–7 mmol/l) even during fasting and feeding and the homeostasis is maintained by the balance between absorption from the intestine, production by the liver and other tissues (Saltiel & Kahn 2001). Glucose is transported to skeletal muscles, adipose tissue and heart muscle with insulin and GLUT4 transporters. To some other cells like red blood cells, brain cells and liver cells, glucose is transported with concentration gradient. (McArdle et al. 2010, 101.) GLUT4 concentration has been seen to increase after endurance training and it is suggested to be a crucial phase to regulate the insulin sensitivity (Babraj et al. 2009).

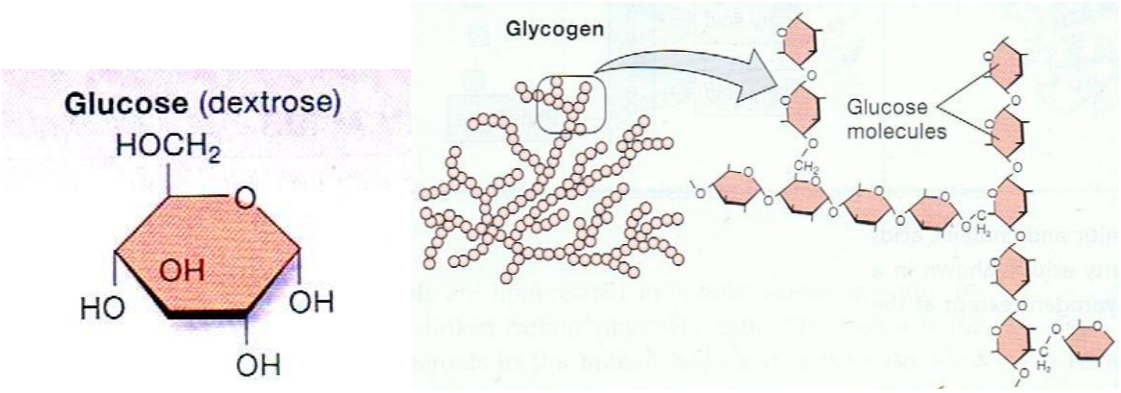

FIGURE 1. Structures of glucose and glycogen molecules (Silverthorn et al. 2010, 29).

Energy production from glucose or other molecules is a catabolic reaction cycle where the biomolecule is broken down and ATP is formed. When energy is produced aerobically from glucose, normally there are following main processes: glycolysis, citric acid cycle (also known as Krebs cycle), electron transport chain and oxidative phosphorylation. (Silverthorn et al. 2010, 107.)

Glycolysis. Glycolysis is a 10 step process that takes place in the cytosol. In glycolysis, one molecule of glucose is converted to two molecules of pyruvate. Glycolysis does not require oxygen and is therefore possible both in aerobic and anaerobic situations. (Robergs et al. 2004.) NADH is a molecule that carries high energy electrons to mitochondria where they are used for ATP formation (Silverthorn et al. 2010, 108–109).

Citric acid cycle. The pyruvate is converted to acetyl-CoA that enters the citric acid cycle, which takes place in the cell's mitochondria (McArdle et al. 2010, 149). Citric acid cycle is an 8 step process and the result of it is three NADH molecules, one $FADH_2$, one ATP molecule and two CO_2 molecules (Silverthorn et al. 2010, 110–111).

Electron transport chain and oxidative phosphorylation. The last phase in aerobic energy production is electron transport system, where the NADH and $FADH_2$ molecules, that carry high energy electrons, transfer their energy to form ATP. This process takes place in the inner mitochondrial membrane. The complexes that are part of the electron transport chain are enzymes and proteins. The high energy electrons move from complex to another and in those processes some energy is released to pump hydrogen ions from mitochondrial matrix to intermembrane space. This will cause hydrogen gradient across the membrane and as hydrogen moves back across the membrane, the potential energy that is stored in the concentration gradient is transferred to form ATP. The electron transport chain needs oxygen to work. (Silverthorn et al. 2010, 111–112.) The net energy from aerobic glucose metabolism is 36 ATP of which the majority (32 ATP) comes from the last step (Robergs 2004).

Anaerobic metabolism. If there is not enough oxygen available or the need for energy is high and immediate, the pyruvate formed in glycolysis is converted to lactate and at the same time NADH is converted into NAD^+ (Silverthorn et al. 2010, 109–110). This anaerobic system provides energy quite rapidly, but the gain is only two molecules of ATP from one glucose molecule. (Robergs et al. 2004; Silverthorn et al. 2010, 109.)

2.2 Fat metabolism

Lipid is the general term for heterogeneous group of different compounds and it includes oils, fats, waxes and related compounds. About 98 % of dietary lipids exist as neutral fat, also known as triglycerides. Lipids are categorized into three main groups: simple lipids, compound lipids and derived lipids. (Guyton & Hall 2006, 840; McArdle et al. 2010, 20–25.)

The simple lipids consist mainly of triglycerides. Fat is stored in body's fat cells, also called as adipocytes, mainly as triglycerides and the main role of triglycerides in human body is to provide energy for different metabolic functions. Another role is to form the cell membranes together with other lipids. Triglycerides consist of glycerol molecule and three fatty acids that are acylated to the glycerol (see figure 2). Fatty acids can be built up by 4 to 20 carbon atoms, although chain lengths of 16 or 18 carbon atoms are the most common ones and fatty acids can be either saturated or unsaturated. In human body, the most common triglycerides are stearic acid (18 carbons, only single bonds), oleic acid (18 carbons and one double bond) and palmitic acid (16 carbon and only single bonds). The structures of palmitic and oleic acids are shown in figure 3. The synthesis of triglyceride molecule (esterification) produces three molecules of water and vice versa, in the breakdown of triglyceride molecule (also referred as hydrolysis) three molecules of water is needed. (Guyton & Hall 2006, 840; McArdle et al. 2010, 20–25.)

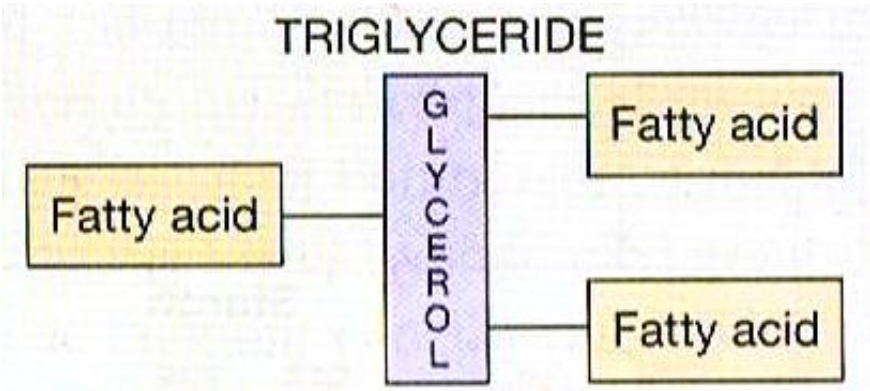

FIGURE 2. The structure of triglyceride (Silverthorn et al. 2010, 30).

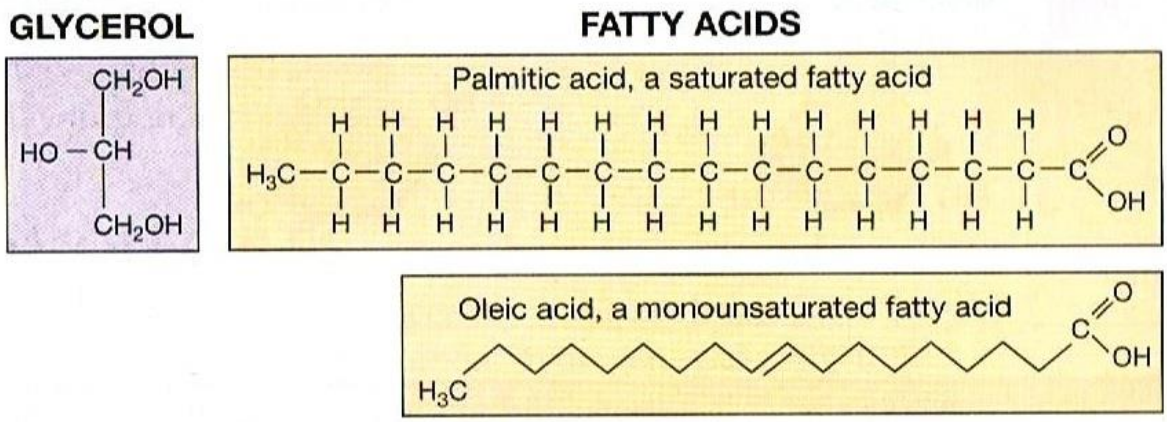

FIGURE 3. The structures of glycerol and two common triglycerides in human body, oleic acid and palmitic acid (Silverthorn et al. 2010, 30).

Saturated fatty acids contain only single bonds between carbon atoms and they exist mostly in animal products such as beef, lamb, pork, chicken, milk and cheese. Also coconut and palm oil contain saturated fatty acids. Unsaturated fatty acids contain one (monounsaturated fatty acid) or more (polyunsaturated fatty acid) double bonds between the carbon atoms in the main carbon chain. Canola oil, peanut oil and olive oil are examples of monounsaturated

fatty acids and examples of polyunsaturated fatty acids include sunflower oil, soybean and corn oil. (McArdle et al. 2010, 20–25.)

Triglyceride components that have combined with some other chemicals are called compound lipids. There are three most known compound lipids: phospholipids, glycolipids and lipoproteins. Lipoproteins are the main carrier of lipids in the blood. Lipoproteins are further divided into four subgroups according to their size, density and whether they carry cholesterol or triglycerides. These groups are chylomicrons, high-density lipoprotein (HDL), very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL). Chylomicrons transport fat-soluble vitamins (A, D, E and K) and they are metabolized in the liver and sent to adipose tissue for fat storage. HDL cholesterol contains a lot of protein (about 50 %) and little of total lipid (20%) and cholesterol (20 %) compared to other lipoproteins. VLDL cholesterol is formed in the liver from fats, carbohydrates and alcohol and VLDL is degraded in the liver to form LDL. VLDL transports triglycerides to muscles and adipose tissue. LDL carries from 60 to 80 % of the total serum cholesterol and it has the greatest affinity for cells of the arterial wall. LDL oxidation will influence to smooth cell proliferation and other unfavorable cellular changes that damages and narrows artery. (McArdle et al. 2010, 20–25.)

It is well established that low levels of HDL is strongly correlated with elevated risk of cardiovascular diseases (Camont et al. 2011). The most well-known good effect of HDL is that it removes clustered LDL cholesterol from the arterial walls and transports it to the liver. Normally, when evaluating the risk of coronary heart diseases, HDL-LDL ratio is more useful than looking at their values separately. (McArdle et al. 2010, 25.) Furthermore, HDL has other biological characteristics such as anti-oxidative, anti-inflammatory, anti-infectious and vasodilatory actions. HDL cholesterol has many sub-populations and they differ in size, density and activity. It is also important to look at the quality of the HDL rather than quantity when considering the risk of some diseases. (Camont et al. 2011.)

In humans, there are three main energy storages for fat: 1. triglycerides in the muscles, 2. circulating triglycerides and 3. adipose tissue, where fat is stored as triglycerides. In a pro-

cess called lipolysis (the breakdown of fat), triglycerides are broken down to one glycerol molecule and three free fatty acid molecules. The free fatty acids are then transported to muscles via circulation and the glycerol can be used for example as a substrate in glycolysis. The long-chain fatty acids are transported to mitochondrial matrix where the process called β-oxidation takes place. In β-oxidation, 2-carbon units are split off the chain and acetyl-Coa is formed which can be used in citric acid cycle. After this, the process is similar to carbohydrate oxidative metabolism and this is explained in chapter 5. As a result, a lot of ATP is formed. It must be noted, that fatty acid breakdown and the whole process needs lots of oxygen to work efficiently. (Guyton & Hall 2006, 842–84; McArdle et al. 2010, 155–157; Silverthorn et al. 2010, 114–155.) The energy production from fats is a slow process, because triglycerides are first converted to glycerol and free fatty acid molecules (Wilmore & Costill 2004, 121).

3 HORMONES IN ENERGY METABOLISM

In this chapter, the hormones that are related to energy metabolism are shortly introduced. There are lots of different hormones that influence energy metabolism but the most important ones are chosen here. These hormones include growth hormone, catecholamines, glucagon and testosterone. After that, cortisol, insulin and leptin are presented in more detailed because these are the hormones that were investigated in this thesis.

Hormones are chemical substances that are synthesized by specific host glands and they are secreted to bloodstream for transport throughout the whole body. Hormones are normally divided into two subgroups: steroid-derived hormones and amine or polypeptide hormones that are synthesized from amino acids. Amine and peptide hormones are soluble in blood but steroid hormones are not. Hormones act in their target cells in four ways: 1. they modify the rate of intracellular protein synthesis by stimulating nuclear DNA, 2. they change the rate of enzyme activity, 3. they alter plasma membrane transport via a second-messenger system or 4. they induce secretory activity. (McArdle et al. 2010, 401.)

Hormones bind to their specific receptors in the target cells. The receptors are located either outside the cell membrane (polypeptide hormones) or inside the cell (steroid hormones). The hormone activity is regulated by other hormones, neural stimulation and humoral changes. (McArdle et al. 2010, 402–406.)

Growth hormone, GH (also known as somatotropin), is secreted from the anterior hypophysis that is located in the brain. Growth hormone stimulates cell division, proliferation and protein synthesis in almost all cells in the body. Growth hormone also increases the mobilization and the use of fat as energy and it limits the carbohydrate breakdown. Growth hormone release is increased during physical activity, especially during short bouts. (McArdle et al. 2010, 407, 430, 438, Stokes et al. 2013.) In one study, GH increased about 7,7 micrograms per liter after intense interval exercise (Felsing et al. 1992). Physical activity also

increases the release of growth hormone isoforms that enhance growth hormone's action. During physical activity, growth hormone decreases glucose uptake by the tissues and increases fat usage as energy. It also increases protein synthesis, muscle and bone growth. Training doesn't affect GH resting values but strength training increases growth hormone release at least in men. (McArdle et al. 2010, 407, 430, 438.)

Many of the growth hormone effects are carried out by insulin-like grow factors (IGFs) also called as somatomedins. IGFs have similar effects on growth than insulin and they are synthesized in the liver. (Guyton & Hall 2006, 923–924.) IGFs attach to binding proteins that carry them in the bloodstream to target cells (McArdle et al. 2010, 410). Four types of IGFs have been found and the most important one currently is IGF-1. It is assumed that IGF-1 is responsible for the most of the GH growth processes in the body rather than direct effect of GH itself. (Guyton & Hall 2006, 923–924.)

Testosterone is produced and secreted from the testes in males and much smaller amounts in the ovaries in females. As the concentration of testosterone is much smaller in women compared to men, women have less muscle mass and strength than men. Testosterone is an anabolic hormone that stimulates muscle tissue synthesis and it also increases growth hormone release which leads to muscle protein synthesis. Testosterone also raises neurotransmitter release which leads to better force-production capabilities of skeletal muscles. Physical activity increases testosterone secretion in both sexes, after approximately 15 to 20 minutes of moderate intensity, in men more than in women. (McArdle et al. 2010, 417–419.) In one HIIT study (Wahl et al. 2014) testosterone increased significantly after one exercise session, and even more when resting periods were active compared to passive recovery. In Stokes et al. (2013) study, testosterone increased significantly after both HIIT and moderate intensity continuous running exercise.

Adrenaline and noradrenaline (also known as catecholamines) are hormones that are synthesized in the adrenal medulla, and the catecholamine release is controlled by neural impulses from the hypothalamus. Noradrenaline acts as a neurotransmitter in sympathetic nervous

system and it also intensifies lipolysis in the adipose tissue. Adrenaline increases glycogenolysis in the liver and muscles and lipolysis in adipose tissues and muscles. Physical activity increases catecholamine release, but noradrenaline secretion increases significantly after 50 % VO_{2max} when adrenaline secretion starts to rise notably after 60 % VO_{2max}. Training causes decreased catecholamine secretion at rest and during submaximal exercise loads. (McArdle et al. 2010, 414–415, 430, 432.) After intense interval training session, both adrenaline and noradrenaline have been proven to rise significantly, noradrenaline more than adrenaline (Peake et al. 2014).

Glucagon is produced in the α-cells of pancreas and basically, glucagon actions are reverse to insulin's. Glucagon enhances glycogenolysis and gluconeogenesis in the liver and also increases lipid catabolism. Glucagon secretion is controlled by glucose and insulin concentrations in the blood. Training causes smaller increase in blood glucose during exercise. (McArdle et al. 2010, 429–430.) Right after one intense interval training session, glucagon concentration doesn't change but after 2 hours after the exercise bout it has been seen to be significantly lower than in resting stage (Peake et al. 2014).

3.1 Insulin

Insulin is a peptide hormone that is secreted from islets of Lagerhans that are located in the pancreas. The islets include four types of cells and 2 of them are dominant: α-cells (20 %) that secrete glucagon and β-cells (75 %) that secrete insulin and amylin. (McArdle et al. 2010, 420.) The islets are in close connection with capillaries where the hormones are secreted. One regulation mechanism to control the production and secretion of hormones is the nervous system. In pancreas there are both sympathetic and parasympathetic nerve pathways. Insulin and glucagon regulate bloods glucose concentration and their actions are opposite and, the ratio of glucagon and insulin in the blood determines which one dominates. Insulin is an anabolic hormone and it increases glucose transport to most of the cells (not to brain), it activates glycogen-, fat- and protein synthesis (see figure 4). (Silverthorn et al. 2010, 736–741.) Insulin doesn't affect to liver's glucose uptake but it blocks glycogenol-

ysis and gluconeogenesis and increases glycogen synthesis in liver (Saltiel & Kahn 2001). In short, insulin decreases the amount of glucose in the blood and inhibits the conversion of proteins and fats to glucose. Increase in blood glucose concentration activates the release of insulin into bloodstream and when glucose concentration drops below normal values (70–110 mg/ml) more glucagon and less insulin is secreted. Glucose is very important regulator of insulin secretion but there are others regulators as well. (Reece et al. 2011, 1028–1029.) Other regulators are increased amino acids concentration in blood, other hormones (growth hormone and catecholamines for instance) and nervous system. Parasympathetic activity in β-cells increases insulin secretion and sympathetic activity decreases it. (Silverthorn et al. 2010, 738.) Insulin is an anabolic hormone that affects to protein metabolism by increasing amino acid uptake and protein synthesis in muscles and it also inhibits protein breakdown (Rooyackers & Nair 1997).

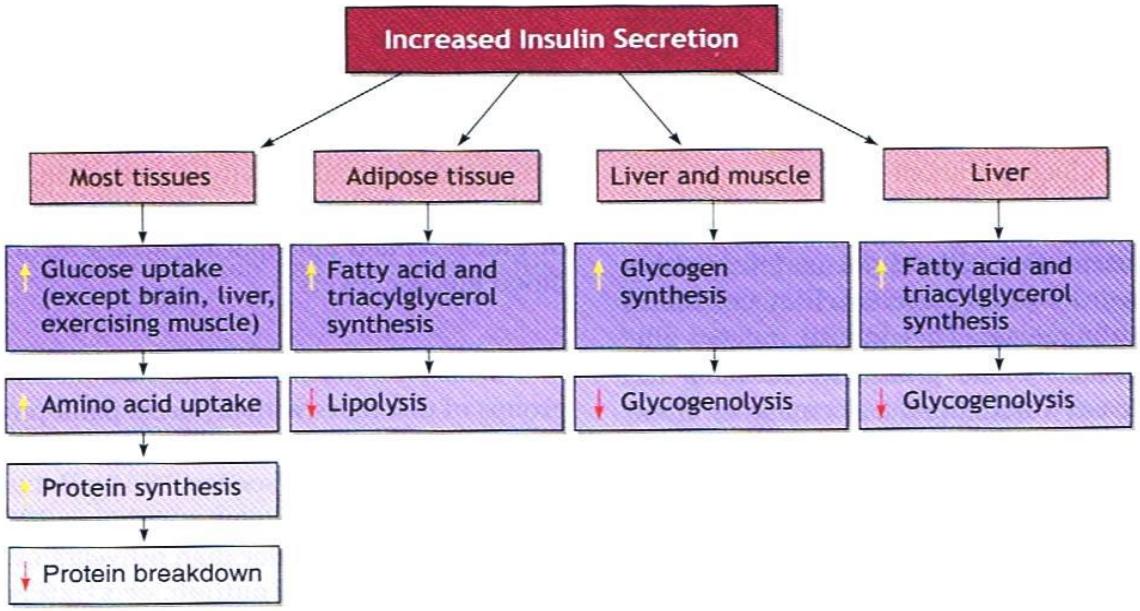

FIGURE 4. The influence of insulin to different tissues. (McArdle et al. 2010, 422)

Insulin receptor is part of tyrosine kinase receptor -family and the receptors are tetrametric proteins including two α- and two β-subunits (Saltiel & Kahn 2001.) There are two different

isoforms of insulin receptor, isoform A, that is lacking the exon 11, and isoform B that includes the exon 11. There have been found some differences in receptor activation and signaling between these two isoforms and thus the functions of these differ a little bit. The insulin receptors are found primarily in insulin sensitive tissues like liver, skeletal muscle and adipose tissue but also in other tissues like heart, brain, blood cells and so on. About 80 % of the insulin receptors in liver are type B receptors and in adipose tissue and skeletal muscles the number is about 40 %. (Belfiore et al. 2009).

Glucose is transported to cells with proteins called glucose transporters or GLUTs. In adipose tissue and skeletal muscles, the major transporter is GLUT4. If there is no insulin available, GLUT4 transporters are located in cytoplasmic vesicles inside the cells. When insulin binds to its receptor, it will activate a cascade that will cause a translocation of GLUT4 to the plasma membrane allowing glucose to enter the cell (see figure 5). In muscle contractions GLUT4 will also move to cell membrane even without insulin. The precise mechanism of GLUT4 action is still not known. (Saltiel & Kahn 2001; Silverthorn et al. 2010, 739–740; McArdle et al. 2010, 420–421.)

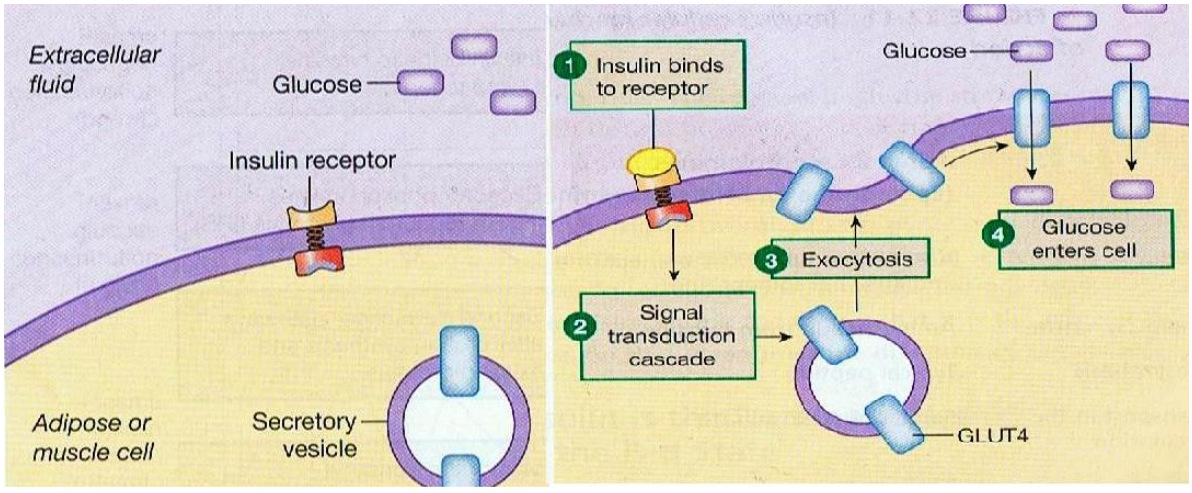

FIGURE 5. Glucose uptake to muscles and adipose tissue with GLUT4. Without insulin, glucose cannot enter to the cells (left picture) and insulin activates GLUT4 which allows glucose to enter the cell (right picture). (Silverthorn et al. 2010, 740.)

3.2 Leptin

Leptin is a hormone that is expressed in the adipose tissue in mammals and it is coded by the ob-gene. The ob-gene includes three exons with two coding regions separated by two introns. Leptin's role is to inform the body about the energy sources that are found in the adipose tissue and thus leptin affects to appetite and metabolism. In short-term fasting, leptin is down-regulated and excessive caloric intake results in up-regulation of leptin. The amounts of leptin mRNA levels are regulated by changes in body fat and changes in food intake. Leptin concentrations are much higher in people with greater percentage of body fat and leptin is down-regulated during weight loss. In other word, the more you have fat in your body, the more leptin is secreted. Inadequate leptin production is very rare in human obesity and in obese individuals it is more likely that they have some kind of leptin resistance. The expression of leptin is regulated by some hormones like insulin and glucocorticoids. (Tartaglia 1997; Houseknecht & Portocarrero 1998.)

Leptin enters the brain by a specific transport mechanism. According to Schwartz et al. (1996) and Caro et al. (1996) the difference in leptin levels is higher in blood than in brain when comparing normal weight and obese people, which means that the transport system doesn't work as efficiently in obese people than in lean individuals. This means that in obese people, their brain doesn't necessarily know what the actual leptin concentration in the blood is. This clearly indicates that this transport step is very critical and leptin resistance could be a result from inadequate leptin transport. (Houseknecht & Portocarrero 1998.)

In humans, there are several different forms of leptin receptors (OB-R). The short form (OB-R_S) is most common in humans; however in hypothalamus (and especially in those regions that have been thought to be important for body weight regulation) the long form (OB-R_L) is dominant. (Tartaglia 1997.)

In some studies where leptin is administrated directly to the brain, food intake has reduced and weight loss has occurred. This could imply that leptin affects directly to the receptors in central nervous system (CNS) and leptin administration could be a proper treatment for obesity in some cases. The way leptin is thought to affect to weight loss is due the decrease in food intake but also by increasing energy expenditure. The mechanism is complex and not fully understood but the activation of brown adipose tissue may have some role in it. (Tartaglia 1997.)

Leptin affects many organs and thus to whole body homeostasis (see figure 6). Leptin is secreted from adipocytes and locally, it affects negatively to insulin action. Leptin decreases appetite and food intake in hypothalamus. In skeletal muscles, leptin increases the amount of fatty acids oxidation and in adrenal cortex it decreases the secretion of cortisol. In pancreas, it inhibits insulin secretion and also increases fatty acid oxidation. In liver, leptin affects to insulin action, but it is still unclear how. In brown adipose tissue, leptin increases thermogenesis. (Houseknecht & Portocarrero 1998).

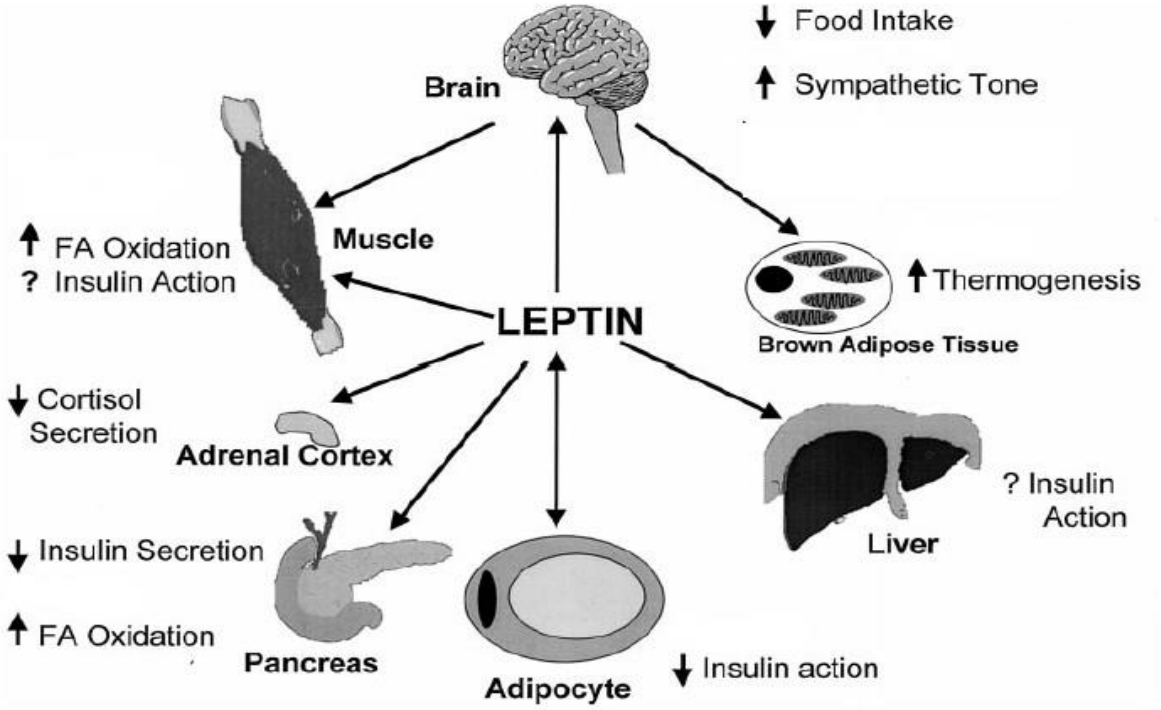

FIGURE 6. Leptin's influence on other organs. Modified from Houseknecht and Portocarrero (1998.)

3.3 Cortisol

Cortisol is a steroid hormone that is secreted from the adrenal cortex and it is synthesized from cholesterol. In humans, there are two adrenal glands and they are located above the kidneys. Each gland consists of two different sections, adrenal medulla and the adrenal cortex. The hormones epinephrine and norepinephrine are secreted from the adrenal medulla and from the adrenal cortex mainly two types of hormones are secreted: mineralocorticoids and glucocorticoids. (Guyton & Hall 2006, 944–945, 950–951.) The most secreted glucocorticoid (95 %) from the adrenal cortex is cortisol (McMahon et al. 1988). Cortisol is a stress hormone that affects body's metabolism, inflammatory responses and to appetite and food intake (Christiansen et al. 2007). The chemical structure of cortisol is shown in figure 7.

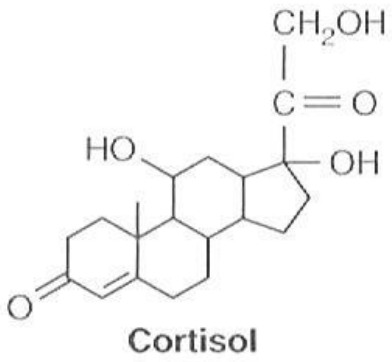

FIGURE 7. The chemical structure of cortisol (Guyton & Hall 2006, 908).

Cortisol affects to glucose, protein and fat metabolism in many ways and it has also other functions. These are listed shortly next.

Effect on glucose metabolism. The one major effect of cortisol in the body is that it increases the amount of gluconeogenesis in the liver. This means that cortisol increases glycogen storages in the liver. (Hers 1986.) The mechanisms behind this are that cortisol stimulates the amount of enzymes needed and cortisol induces transport of amino acids from other tissues, mainly from muscles. Cortisol also affects to glucose metabolism by decreasing the amount of glucose oxidation in most of the cells. Increased gluconeogenesis and decrease in glucose oxidation cause increase in blood glucose concentration and this is a stimulus to insulin secretion. Insulin cannot work as effectively as in normal conditions when cortisol levels are high in the blood because tissues become less sensitive to insulin. The reason for this is not clear but it might have something to do with elevated fatty acid levels in the blood. (Guyton & Hall 2006, 951.)

Effect on protein metabolism. Cortisol inhibits protein synthesis and stimulates protein catabolism, thus cortisol decreases protein storages all over the body, except the liver. (Rooyackers & Nair 1997.) In liver and plasma, the amount of proteins increases when cor-

tisol is present. Cortisol also inhibits amino acid transport to the muscles. (Guyton & Hall 2006, 952.)

Effect on fat metabolism. Cortisol induces fatty acid release from the adipose tissue and thus it raises the concentration of FFAs in the plasma. Cortisol also stimulates oxidation of fat in multiple cells and more fat is used for energy. (Guyton & Hall 2006, 952.)

Effect on stress and inflammation resistance. Almost all stress (physical and emotional) causes increased cortisol release and cortisol is said to be a stress hormone (Rooyackers & Nair 1997). The reason for this is not quite clear but one speculation is that increased cortisol activity mobilizes amino acids and fats and those can be used for repair, energy or substances for other compounds. Cortisol has also inflammatory abilities, it can prevent the inflammation and if the inflammation has already started, it can heal the inflammation and accelerate recovery. (Guyton & Hall 2006, 952–953.)

4 METABOLISM AND EXERCISE

4.1 Glucose metabolism and exercise

Humans have three different systems to provide energy: the ATP-PCr system, the lactic acid system and the aerobic system. In short duration high-intensity bouts, like sprints the energy is provided by immediate energy substances: ATP and PCr (phosphocreatine) molecules that are located in the muscles and these compounds can provide energy for about 20 to 30 seconds. When the exercise lasts about 60 to 180 seconds, energy is provided from glucose and glycogen anaerobically and as a byproduct, lactate will accumulate. If the exercise lasts more than couple of minutes, the aerobic energy release from glucose becomes dominant. In rest and light physical exercise, fat is used for energy but when the intensity rises, glucose becomes more important. (McArdle et al. 2010, 163–170.)

4.1.1 High-intensity interval training and glucose

In the study by Peake et al. (2014), they studied the effect of high-intensity exercise (HIIT) versus moderate intensity exercise (MOD) on glucose. They had ten well-trained men as subjects. After VO_{2max} test and familiarization exercise, participants completed HIIT and MOD exercise in randomized order with at least 7 days rest between the exercises. The HIIT exercise was 10 times 4 minute intervals at 80 % VO_{2max} and MOD exercise was done at 65 % VO_{2max}. They calculated the total work in HIIT exercise and matched it with MOD so that the work was same in both exercises but time and intensity were different. Blood samples were collected before, 5 minutes, 1 and 2 hours after the exercises. From the samples, glucose concentration was analyzed.

Plasma glucose concentration increased significantly after both HIIT and MOD trials. When they compared differences between groups, they showed that glucose concentration was significantly higher after the HIIT than MOD (see figure 8).

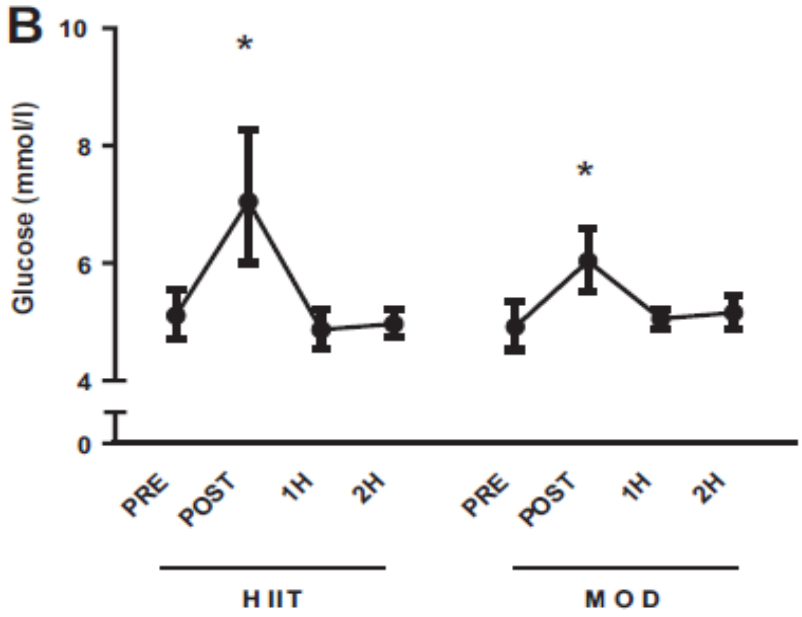

FIGURE 8. The acute effect of HIIT exercise (left) and moderate-intensity exercise (right) on glucose before, right after, 1 and 2 hours after the exercise. * = difference compared to pre value, when p<0,05. (Peake et al. 2014.)

In one other study there were 16 young men and they participated in a 2 week study where they did maximal HIIT training. The protocol was 6 training sessions in two weeks and they did four to six times 30 second sprints on a cycle. Plasma glucose and insulin were measured before and after the intervention. The researchers didn't see any changes in glucose or fasting insulin after two weeks. The authors didn't comment the reasons for this at all. (Babraj et al. 2009.)

So, high-intensity interval training has a potential to increase glucose concentration acutely after one training session but long-term effects are not clear yet.

4.2 Blood lipids and exercise

Fat is the main energy source in rest and during light physical activity in humans. Fat is also the most important source for energy during long, aerobic and low-intensity activities. The muscles use free fatty acids (FFA) and triglycerides from the circulation or muscle's internal fat storages for energy. (Van Hall et al. 2002.) In physical activities, 30–80 % of energy is from fats but there are several factors affecting the exact amount of fat used. The most common ones are nutrition, intensity, length and training background. In light or moderate intensity activities, fat is used 3 times more than in rest. When the intensity increases, the usage of fat decreases and more glycogen is needed for energy. In light activities fat is released from fat tissue and it is transported as free fatty acids via circulation to muscles. In moderate exercises, about half of the energy is from fats and the other half from carbohydrates. If the moderate exercise lasts over an hour, the usage of fats increases. Regular aerobic training will improve fat oxidation. Trained people improve fat oxidation capacity and it will spare glycogen storages and thus they can perform at a higher absolute submaximal level and fatigue will be delayed. (McArdle et al. 2010, 28–30.)

4.2.1 Aerobic training and blood lipids

Endurance training demonstrates significant increases in HDL in both men and women after a training period. There seems to be also a dose-response relationship between the: 1. Amount of exercise performed and the increase in HDL as well as, 2. Intensity of the exercise and increase in HDL. (Musa et al. 2009.)

It is well known that endurance training will improve lipid profile in the long run (For example Henderson et al. 2010; Kelley & Kelley 2006). For example, Hu et al. (2001) have studied the effects of everyday physical activity on blood cholesterols. They found out in their study that in men, increased daily activity (like biking or walking) decreased total cholesterol, LDL and triglyceride levels. In women, HDL concentration increased as a result of

increased daily activities. In other study by Panagiotakos et al. (2003) the researchers showed that the physical activity and bloods lipid concentration are inversely correlated, that is to say when you are more physically active, the lipid concentrations (cholesterol, LDL, triglycerides) are lower, except for HDL which is higher. In some studies, aerobic endurance training did increase HDL cholesterol and decrease triglyceride concentration in men. In women, aerobic training also increased HDL levels and decreased total cholesterol, LDL and triglyceride concentrations. (Kelley & Kelley 2006; Kelley et al. 2004.)

Endurance training session will not alter total cholesterol or LDL concentrations acutely. (Henderson et al. 2010, Lee et al. 1991). In Henderson et al. (2010) study triglycerides decreased significantly below resting values 3 hours after the exercise in women. Men didn't have any changes in triglycerides. Gill et al. (2003) found similar results: in women triglycerides decreased acutely after the exercise. In one study by Gordon et al. (1996), men's HDL concentration increased 24-hour after running exercise. Lee et al. (1991) found out that slightly overweight women had a significant increase in HDL immediately after an exercise but it returned to resting stage after 1,5 hours. They also saw that triglycerides were below resting values 1,5 and 23 hours after the exercise.

As a conclusion, endurance training has a potential to lower total cholesterol, LDL and triglycerides and increase HDL. The important factors that affects to the amount of change include the volume of exercise, intensity and gender.

Also, both strength and power training have been shown to affect blood's lipid profile. In one study (Tambalis et al. 2009) LDL concentration decreased significantly after strength training. Also combined endurance and strength training has been seen to increase HDL and decrease LDL levels. However, the studies about strength and combined endurance-strength training and lipids are contradictory.

4.2.2 High-Intensity interval training and blood lipids

Despite the well-known benefits of aerobic training on blood lipids, the effect of other modes of physical training on blood lipid profiles has not been so completely studied. Interval training, for example, is one of the most widely used methods of physical training in young men and women. Interval training studies using typical work:rest intervals (1:3 or 1:2) have shown little effect on blood lipid profiles, but it is yet not perfectly clear whether longer work intervals at high intensity, with prolonged periods of continuous physical activity, would have more favorable effects on blood lipids. (Musa et al. 2009.)

Musa et al. (2009) studied whether an 8-week program of HIIT training with a longer work:rest interval would significantly elevate HDL and reduce the total cholesterol (TC) and atherogenic index of untrained young adult men. The measured variables in the study were HDL, TC and TC/HDL. All these variables were obtained at baseline and after an 8-week interval training intervention using high-intensity, prolonged periods of continuous activity (1:1 work:rest ratio). They had 45 healthy men 21–36 years of age as subjects in the study and they were randomly assigned to either experimental (n = 23) or control (n = 22) group before training. Participants in the experimental group had 3 training sessions per week throughout the 8-week period.

In the training program, the experimental participants ran a distance of 3.2 km, 3 days per week for the total of 8 weeks. The control group was instructed not to undertake any vigorous exercise during the training period. Participants ran 4 sets of 800m intervals (i.e., 4x800m intervals, 1:1 work:rest ratio) at approximately 90% of their age-predicted HRmax. HR was recorded during training to ensure proper training intensity. The workload for the experimental group (energy expenditure per exercise session) was estimated. The estimated energy expenditure per training session was 423.2 kcal or approximately 1270 kcal per week. Each training session included a 10 minute warm-up and each training session was followed by an 800m mild cool-down.

TABLE 1. Pre- and post-training blood lipids and aerobic performance after 8 week interval training period compared to control group. *p<0.001; †p<0.0001. (Musa et al. 2009.)

	Test	Experimental ($n = 20$)	%Δ	Control ($n = 16$)	%Δ
HDL-C ($mmol \cdot L^{-1}$)	Pre	1.1 ± 0.3		1.1 ± 0.2	
HDL-C ($mmol \cdot L^{-1}$)	Post	1.3 ± 0.4	18.1*	1.1 ± 0.2	0.0
TC ($mmol \cdot L^{-1}$)	Pre	3.9 ± 0.7		3.9 ± 1.1	
TC ($mmol \cdot L^{-1}$)	Post	3.8 ± 0.8	−2.0	4.1 ± 1.1	5.1
TC/HDL-C	Pre	3.8 ± 1.2		3.7 ± 1.2	
TC/HDL-C	Post	3.1 ± 1.0	−18.1†	3.9 ± 1.2	5.4
2.4-km run time (min)	Pre	11.9 ± 0.8		12.3 ± 1.6	
2.4-km run time (min)	Post	10.8 ± 0.9	−9.2†	12.6 ± 1.9	2.4

In the study, they found significant increase in HDL cholesterol (p<0.001) and significant decreases were found for distance run times and TC/HDL at post-testing after the intervention period (Table 1). The HDL cholesterol increased 18.1 % after 8 week interval training program. Total cholesterol did not show any significant change at post-test. For the control group, none of the 4 variables showed any significant change at post-testing.

In that study they concluded that 8 weeks of HIIT training can cause favorable changes in HDL and the lipoprotein ratio in young adult men. They also thought that it is possible that longer continuous intervals at high intensity may be necessary to improve HDL. It was not surprising to observe that TC did not change significantly with training, given that exercise produces reciprocal changes in TC, especially with regard to HDL and LDL. In most studies, as HDL increases, LDL decreases, and this leads to either no change or a slight reduction in TC. They didn't control the diet in this study so that might have some effect on the results. (Musa et al. 2009.)

Nybo et al. 2010 studied the influence of HIIT, moderate-intensity running and strength training on plasma lipid profile and glucose tolerance. They had 36 untrained, healthy men as subjects. The subjects were divided into four groups: 1) intense interval running (HIIT); 2) a strength-training group (STR); 3) prolonged moderate intense continuous running (MOD); and 4) a control group performing no physical training (CON). The three interven-

tion groups completed 12 week training program consisting of 3 sessions per week. HIIT group ran 5 times 2 min intervals at heart rate above 95% of their HR_{max}. The prolonged running sessions consisted of 1 h of continuous running at 80% of individual HR_{max} and strength training was progressive heavy-resistance strength training. The strength training consisted of three to four sets, 6-12 repetitions of squats, leg press, isolated knee extension, hamstring curls, and calf rises with 1 minute rest periods. The total exercise time was 60 min per session. Venous blood samples were taken before and after the intervention and total cholesterol, HDL and LDL were analyzed. In addition, also glucose and insulin concentrations were measured but those results are discussed in chapter 4.3.2.

The researchers found out that total cholesterol, HDL, LDL and TC/HDL-ratio remained unchanged in the HIIT group. In the MOD group TC/HDL-ratio decreased significantly. Total cholesterol increased significantly in strength group. In the strength group, also HDL/TC ratio was lower after 12 week intervention but there were no significance and there were no change in fasting blood glucose. (Table 2).

TABLE 2. Total cholesterol, HDL and LDL, concentrations before and after 12 week interval training (HIIT), prolonged running and strength intervention. * = Significantly higher than the pre training value ($P < 0.05$). Modified from Nybo et al. 2010.

	HIIT		**Prolonged Running**		**Strength**	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
Total cholesterol (mM)	5,1±0,2	5,0±0,2	4,1±0,3	3,8±0,4	4,8±0,3	5,3±0,3*
HDL cholesterol (mM)	1,2±0,1	1,2±0,1	1,2±0,1	1,3±0,1	1,2±0,1	1,2±0,1
LDL cholesterol (mM)	3,4±0,2	3,3±0,3	2,5±0,2	2,4±0,3	3,1±0,3	3,5±0,3

The researchers concluded that the volume rather than intensity might be more important when thinking plasma lipoprotein-lipid profile in untrained people, because there were no changes in lipids in the HIIT group, and prolonged running group significantly improved TC/HDL-ratio. Therefore, intense but short interval training seems to be less effective than prolonged training when thinking of lipid profile in untrained people. The one reason for this based on these results might be that the MOD group lost fat during the intervention program. Previous studies (for example Katzmarzyk et al. 2001) have shown that loss of body fat and changes in lipoprotein-lipid profile do correlate with each other. (Nybo et al. 2010.)

Grieco et al. (2013) studied the effects of three different training intensities on total cholesterol and HDL. They had 45 healthy young individuals as subjects and they were randomly divided in to four groups: moderate intensity (MOD) 50% of heart rate reserve, vigorous intensity (VIG) 75 % HRR, maximal intensity intervals (MAX), 5 minutes at 90-100 % of HRR or control (CON). They trained for 6 weeks on a bicycle ergometer and the duration and amount of training varied because the total energy expenditure was match to MAX training.

As a result, they showed that there were no differences between groups in TC or HDL in baseline or after training. The reasons for not finding any significant changes according to the authors were that the subject number was so low and the subjects were recreationally active, healthy adults and the changes are so small that it is hard to get any statistical differences. They also had a big individual variation among subjects. Also, they didn't control the diet and that could have influence on blood lipids. (Grieco et al. 2013.)

In the study by Peake et al. (2014) the effect of high-intensity exercise versus moderate intensity exercise on free fatty acids was studied. They had ten well-trained men as subjects at the aged of 30. First, VO_{2max} was measured in cycle ergometer. After one familiarization training, the actual study begun. Participants completed HIIT and MOD exercise in randomized order so that there was at least 7 days rest between. Blood samples were collected before exercise and after that 10 to 15 minute warm-up was done. The HIIT exercise was 10

times 4 minute intervals at 80 % VO_{2max} and MOD exercise was done at 65 % VO_{2max}. They calculated the total work in HIIT exercise and matched it with MOD so that the work was same in both exercises but time and intensity were different. Blood samples were collected 5 minutes, 1 hour and 2 hours after the exercises. From the samples free fatty acids was analyzed.

The results were that serum free fatty acid concentrations increased significantly after both interventions (HIIT and MOD) but there were no difference between the groups. Serum FFA concentration was elevated still 1 and 2 hours after both exercises (see figure 9).

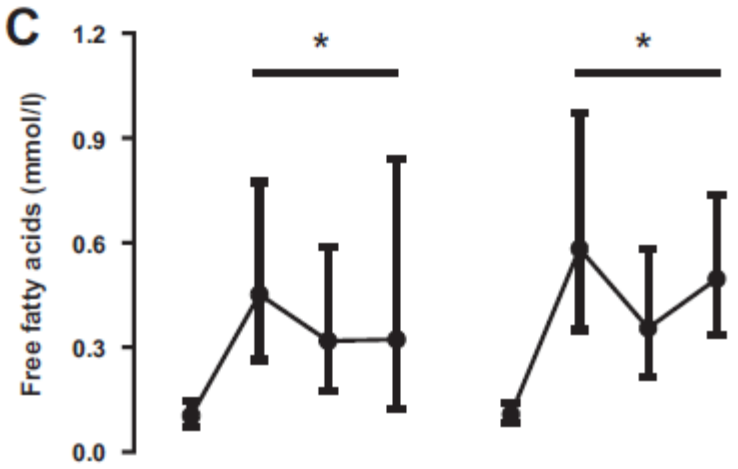

FIGURE 9. The acute effect of HIIT exercise (left) and moderate-intensity exercise (right) on FFA's before, right after, 1 and 2 hours after the exercise. * = difference compared to pre value, p<0,05. (Peake et al. 2014.)

Another study by Perry et al. (2008) investigated the long-term HIIT training on FFA's. Eight recretionally active adults trained for 6 weeks on a cycle ergometer and the protocol was 10x4 minute intervals with 2 minute rest for three times per week. There were no change in FFA's after 6 week training. (Perry et al. 2008.)

Paoli et al. (2013) studied the effect of high-intensity circuit training and endurance training on blood lipids. They had 58 healthy but slightly overweight untrained men, aged 61 as subjects. The subjects were randomly assigned to one of the three groups: Endurance (ET), Low-Intensity Circuit (LICT) or High-Intensity circuit (HICT) group. The subjects trained three times per week for 12 weeks. Blood samples were taken before and after the intervention and total cholesterol, LDL, HDL and triglycerides were analyzed.

In the study, there were no differences between the groups at the baseline. HICT group had a significant decline in TC and LDL when comparing to the other groups. HICT group had also significant increase in HDL compared to other groups. Also TG decreased significantly in HICT group. The results are presented in table 3.

TABLE 3. The effect of 12 week High-intensity circuit training (HICT), low-intensity circuit training (LICT) and endurance trainig (ET) on cholesterol and triglycerides in adult men. * =p<0.05 HICT vs LICT, #=p<0.05 LICT vs ET, ° = p<0.05 HICT vs ET. Modified from Paoli et al 2013.

	HICT		LICT		ET	
	pre	post	pre	post	pre	post
TC	5,52±0,05	5,0±0,06 *°	5,88±0,09	5,73±0,09	5,6±0,1	5,44±0,11
HDL	1,32±0,02	1,45±0,03 *°	1,3±0,03	1,33±0,04	1,28±0,04	1,27±0,05
LDL	2,98±0,1	2,51±0,08 *°	3,05±0,12	2,95±0,12 #	3,12±0,11	3,03±0,12
TG	2,66±0,05	2,26±0,02 *°	2,66±0,44	2,46±0,04	2,61±0,03	2,53±0,05

The main finding in the study was that HICT method was superior compared to LICT and ET when considering blood lipids. The researchers also concluded that that there is a dose-response manner which means that changes in lipid concentrations seems to depend on the total amount of calories (total work) burned. (Paoli et al. 2013.)

Miller et al. (2014) studied the effect of 4 week HICT intervention on blood lipids and insulin in sedentary obese men. They had eight subjects, aged 34 years. Blood samples were taken before and after the intervention and also, every week during the study. Total cholesterol, LDL, HDL, triglycerides and glucose were analyzed from the samples. The diet was not controlled but subjects were asked not to make any changes to their diets during the study.

Total cholesterol decreased significantly after 2, 3 and 4 weeks compared to baseline. Triglycerides decreased significantly after 1, 2, 3 and 4 weeks compared to baseline. There were no changes in HDL, LDL or glucose at any point. The results are shown in table 4. (Miller et al. 2014.)

TABLE 4. The results from 4 week HICT study were subjects were healthy, obese men (Miller et al. 2014).

Blood serum measurements	Baseline	Week 1—HICT	*P* value	Week 2—HICT	*P* value	Week 3—HICT	*P* value	Week 4—HICT	*P* value
Total cholesterol (mmol/L)	4.90 ± 0.60	4.75 ± 0.53	0.08	4.50 ± 0.45	**0.03**	4.26 ± 0.75	**0.03**	4.43 ± 0.63	**0.01**
Triacylglycerol (mmol/L)	2.64 ± 1.94	1.83 ± 1.15	**0.04**	1.71 ± 1.15	**0.00**	1.67 ± 1.09	**0.01**	2.05 ± 1.61	**0.04**
HDL cholesterol (mmol/L)	0.98 ± 0.21	1.04 ± 0.23	0.23	1.00 ± 0.29	0.75	0.96 ± 0.27	0.49	0.96 ± 0.28	0.64
LDL cholesterol (mmol/L)	2.72 ± 0.69	2.88 ± 0.52	0.39	2.72 ± 0.27	0.98	2.55 ± 0.63	0.52	2.53 ± 0.53	0.25

To sum up this current scientific data presented above, it is not still completely clear, wich type of high-intensity interval training program is the best when considering blood lipids and the results are a little bit contradictory. High-intensity interval training has the potential to increase HDL-concentration and decrease LDL- and TC concentrations. There is no evidence that HIIT affects to FFA concetration in long-term but there can be acute increases. High-intensity circuit training has the possibility to increase HDL concentration and decrease TC and LDL concetrations. Triglycerides have seen to decrease both acutely and long-term after high-intensity interval training but the subjects were obese men. It must

be noted, that in many interval studies, subject number has been low, diet has not been controlled and subjects have been recretionally active, healthy adults. Also, protocols have varied a lot.

4.3 Hormones and exercise

4.3.1 Aerobic training and hormones

Cortisol. Plasma cortisol levels are lower in trained individuals than in sedentary people when doing the exercise in the same absolute submaximal level (McArdle et al. 2010, 432). Secretion of cortisol increases during exercise, and cortisol facilitates the breakdown of triglycerides to glycerol and fatty acids. Cortisol also mobilizes glycogen from the liver and thus the glucose concentration in plasma increases. (Silverthorn et al. 2010, 816.)

Leptin. There are not many studies done with humans that have investigated the effects of acute exercise or endurance training on leptin. In one study (Hickey et al. 1996) there were no acute changes in leptin concentration in lean, long-distance runners after 20-mile run at 70 % VO_{2max}. The study concluded that in trained people exercise doesn't affect to leptin acutely. Perusse et al. (1997) studied the acute and chronic effects of exercise on leptin in men and women. They had 97 subjects who underwent 20 week endurance training intervention. As a result, leptin concentration decreased in men after 20 weeks of training but not in women. There was a huge individual variation in the results both after the acute and chronic measurements. They concluded that exercise doesn't have big effect on leptin in humans.

Insulin. Aerobic endurance training depresses insulin responses during exercise so that in trained individual insulin levels do not change so much compared to untrained (McArdle et al. 2010, 434). During exercise, the glucose concentration increases but insulin secretion is suppressed. Normally increased glucose concentration stimulates insulin release but during

exercise, the beta cells sympathetic stimulation is increased and that inhibits insulin secretion. As there is less insulin in the circulation during exercise, other cells than muscles cannot use glucose effectively and thus more glucose is available for muscles. During exercise the muscles do not need insulin to transport the glucose in because the muscle contractions stimulate GLUT4 and glucose intake in muscles will increase. (Silverthorn et al. 2010, 816.)

4.3.2 High-intensity interval training and hormones

There is some evidence that improved insulin action may be intensity depend and several studies have shown that intensity level above 70 % VO_{2max} has a bigger influence on insulin than lower intensities. It must be noted though that the protocols in these studies have been different and the volumes of training have been varying. (Grieco et al. 2013.)

Because there is not a clear consensus, which intensity is best when considering insulin action, Grieco et al. (2013) studied the effect of three different training intensities on insulin and glucose when protocols were isocaloric. They had 45 healthy young individuals as subjects and they were randomly divided in to four groups: moderate intensity (MOD) 50% of heart rate reserve, vigorous intensity (VIG) 75 % HRR, maximal intensity intervals (MAX), 5 minutes at 90-100 % of HRR or control (CON). They trained for 6 weeks on a bicycle ergometer and the duration and amount of training varied because the total energy expenditure was match to MAX training.

As a result they showed that there were no differences between groups in insulin or glucose in baseline or after training. The results are shown in table 5.

TABLE 5. The effect of three training intensities on blood glucose, and insulin CON=control group, MOD= moderate intensity group, VIG=vigorous intensity group, MAX= maximal intensity interval group. Modified form Grieco et al. 2013.

	CON		MOD		VIG		MAX	
	pre	post	pre	post	pre	post	pre	post
Glucose mmol/l	4,9±0,3	4,7±0,3	4,8±0,5	4,8±0,4	5,0±0,5	4,5±0,5	4,8±0,3	4,6±0,5
Insulin pmol/l	36,0±20,1	23,3±16,8	36,4±19,5	55,5±73,5	43,6±42,8	29,1±14,1	22,2±9,8	27,4±21,3

The reasons for not finding any significant changes according to the authors were that there were so small groups and the subjects were recreationally active healthy adults and the changes are so small that it is hard to get any statistical differences. Also, they didn't control the subjects' diet and that could have some influence. They also noted that the timing of blood insulin sample after training (immediately after or 48 hours after) could cause some differences between different studies. (Grieco et al. 2013.)

In one study by Kordi et al. (2013) they investigated the effect of HIIT training on blood insulin and glucose. In the study, they had 22 sedentary female students as subjects and they were divided into two groups: control or intervention. The intervention group trained 3 times per week for 6 weeks and the training was 4 to 6 maximal sprints with 30 second recovery between. Fasting blood samples were taken before and after the 6 week intervention. After the training period, insulin and glucose concentrations decreased but the difference was not statistically significant. In the control group, they found no changes. The results are listed in table 6.

TABLE 6. Changes in insulin and glucose levels after 6 weeks HIIT -training period in sedentary females. * P≥ 0.05 significance of pre-test vs. post-test. Modified from Kordi et al. 2013.

Variable	Group	Pre-test	Post-test	Significance level (P-value)
Insulin (micro unit/ml)	Control	12.17±7.73	12.27±7.04	0.472
	Experimental	11.90±5.22	9.37±3.58	0.092
Glucose (mg/dl)	Control	95.88±9.71	95.22±8.26	0.306
	Experimental	92.36±8.06	89.00±7.97	0.166

In Nybo et al (2010) study, which was sited previously, they studied the influence of HIIT and moderate-intensity running on plasma lipid profile and glucose tolerance. In the study, they had 36 untrained, healthy men as subjects. The subjects were divided into four groups: 1) intense interval running (HIIT); 2) a strength-training group; 3) prolonged moderate intense continuous running (MOD); and 4) a control group performing no physical training. The three intervention groups completed 12 week training program consisting of 3 sessions per week. HIIT group ran 5 times 2 min intervals at heart rate above 95% of their HRmax. The prolonged running sessions consisted of 1 h of continuous running at 80% of individual HRmax. Venous blood samples were taken before and after the intervention and glucose and insulin were analyzed. The results of lipid variables are discussed in chapter 4.2.2.

There were no changes in fasting insulin but fasting glucose decreased significantly in both HIIT and MOD groups. The results are shown in table 7.

TABLE 7. Blood glucose and insulin concentrations before and after 12 week interval training (HIIT) and prolonged running intervention (MOD). * Significantly lower than the pre training value ($P < 0.05$). Modified from Nybo et al. 2010.

	HIIT		**Prolonged Running**	
	Before	**After**	**Before**	**After**
Fasting glucose (mM)	5,7±0,2	5,2±0,1 *	5,6±0,7	5,1±0,4*
Fasting insulin ($\mu U \cdot mL^{-1}$)	7,1±1,1	7,8±2,2	5,0±1,7	4,1±0,9

The researchers concluded that the volume of weekly training (time) may be important when considering acute changes in insulin concentrations but it seems that moderate- and high-intensity exercises may have the same beneficial long-term effects. They also said, that based on their study, it seems that when training at high intensities, as little as 40 minutes of training per week is enough to cause similar improvements in glucose tolerance as is 150 minutes training at moderate intensity. (Nybo et al. 2010.)

Miller et al. (2014) studied the effect of 4 week HICT intervention on insulin in sedentary obese men. They had 8 subjects (34 years old) and blood samples were taken before and after the intervention and also, every week during the study. Insulin decreased 19,1 % after 4 weeks but the result was not significant (p=0,06). Results are shown in table 8. (Miller et al. 2014.)

TABLE 8. The effect of 4 week HICT on insulin. Insulin decreased 19,1% but it wasn't significant finding. (Miller et al. 2014.)

Blood serum measurements	Baseline	Week 1—HICT	*P* value	Week 2—HICT	*P* value	Week 3—HICT	*P* value	Week 4—HICT	*P* value
Insulin (pmol/L)	71.68 ± 52.37	71.44 ± 55.34	0.67	77.80 ± 51.91	0.92	66.08 ± 62.27	0.29	58.04 ± 59.43	0.06

Peake et al. (2014) studied the acute effects of high-intensity (HIIT) and moderate intensity exercise (MOD) on insulin and cortisol. They had ten well-trained men as subjects. Participants completed HIIT and MOD exercise in randomized order so that there was at least 7 days rest between. The HIIT exercise was 10 times 4 minute intervals at 80 % VO_{2max} and MOD exercise was done at 65 % VO_{2max}. They calculated the total work in HIIT exercise and matched it with MOD so that the work was same in both exercises but time and intensity were different. Blood samples were collected before, 5 minutes, 1 and 2 hours after the exercises. From the samples cortisol and insulin were analyzed. (Peake et al. 2014.)

They found out that cortisol concentration increased significantly after HIIT but not after MOD. When comparing groups, cortisol was significantly higher right after HIIT exercise. There were no change in insulin after either training session (HIIT or MOD) but cortisol level was significantly lower when comparing pre value to 1 and 2 hours post both exercises (see figure 10). (Peake et al. 2014.)

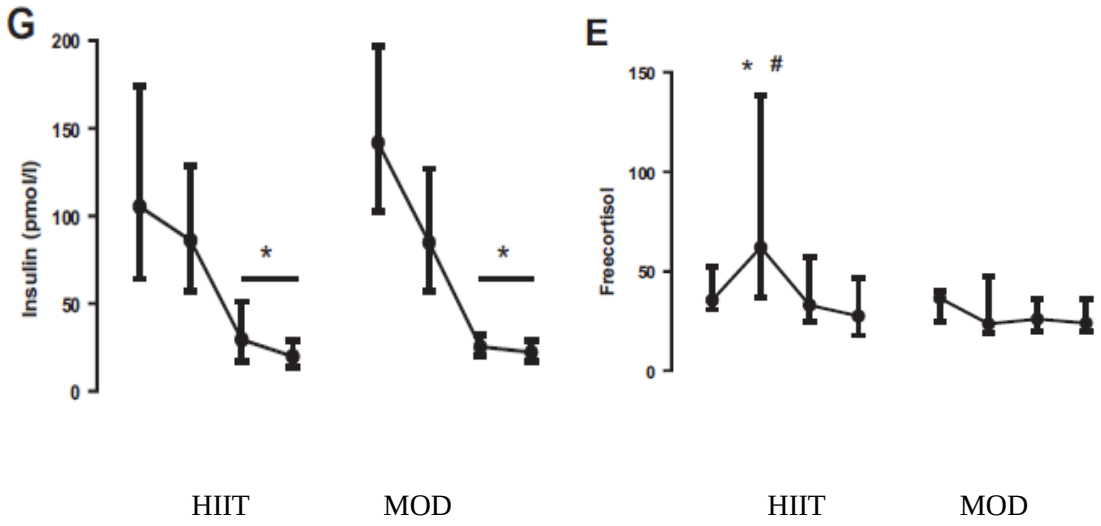

FIGURE 10. The acute effect of HIIT exercise and moderate-intensity exercise on insulin and cortisol before, right after, 1 and 2 hours after the exercise. * = difference compared to pre value, # = difference between groups when p<0,05. (Peake et al. 2014.)

Gray et al. (1993) studied the acute responses of intense, interval running exercise on cortisol (both total and free). The exercise consisted of warm-up and 1 minute intervals with 1 minute active recovery. The intensity was selected individually using the same speed and incline at which VO_{2max} was achieved. The subjects performed as many intervals as they could with that intensity. As subjects, they had eight trained male triathletes, aged 31,5 years. Total cortisol was analyzed using commercially available RIA kits and free cortisol was calculated. (Gray et al. 1993.)

They found out that total cortisol concentration was significantly higher ($p<0,001$) immediately after exercise (43 %) and 1h after the exercise (34 %). Free cortisol was also elevated significantly 1h after the exercise (82 %). There were significant decreases in total cortisol and free cortisol 6h after the exercise. These results are presented in table 9. Researchers suggested that one reason for athletes to be in bigger risk for infections during periods of intense training and competition might be because cortisol will weaken body's immune system. In this study, they examined only acute responses to interval training. (Gray et al. 1993.)

TABLE 9. Cortisol concentration in acute interval exercise. * = $p<0,05$, ** = $p<0,01$, ND = not determined. (Gray et al. 1993.)

	Sampling Time				
Rest	Postwarm-up	Posttest	1 h Post	6 h Post	24 h Post
Total cortisol ($ng \cdot ml^{-1}$)					
196.3	176.9	281.5**	263.9**	88.6**	184.3
(21.4)	(38.8)	(37.2)	(33.3)	(30.6)	(40.4)
Cortisol binding globulin ($\mu g \cdot ml^{-1}$)					
47.3	ND	ND	49.2	47.8	48.2
(3.1)			(4.2)	(3.7)	(5.6)
Free cortisol ($ng \cdot ml^{-1}$)					
12.2	ND	ND	20.2**	3.9**	11.1
(2.2)			(6.4)	(1.9)	(4.5)
% Free cortisol					
6.2	ND	ND	7.5**	4.5**	6.3
(0.7)			(1.5)	(1.1)	(1.7)

Wahl et al. (2010) studied the acute hormonal responses to a short term high-intensity training (HIIT) versus a high volume endurance training (HVT). Cortisol was measured to indicate the stress of the different exercises and they also measured acid-base balance to see if it has some influence to measured variables.

They had 11 healthy sport students (men) as subjects in the study. At first, VO_{2max} was determined. Each subject participated in three experimental trials, each separated by one week. In the two first measurements subjects did interval training (HIIT) interventions (1. HIIT bicarbonate [HIIT (B)], and 2. HIIT placebo [HIT (P)]) and the third trial was the high volume endurance training (HVT) intervention. Participants got either bicarbonate or placebo before the HIIT-exercise. The HIIT exercise consisted of four 30 seconds "all-out" bouts with 5 min passive rest. The HVT exercise consisted of a constant load exercise for 1 h at 50% VO_{2max}. Venous blood samples were collected and cortisol was analyzed using ELISA -method. Samples were taken before ingestion, before exercise, and 10, 60 and 240 min post exercise.

The study showed that the pH was much more acidic after HIIT exercise with bicarbonate than with placebo and serum cortisol was significantly elevated 10 minutes after both HIIT exercises ($p<0,05$). It was also seen that the increase in cortisol acutely after the HIIT exercise was significantly higher when pH was lower, in other words, when the body is more acidic, the incline in cortisol is much greater. This can be seen in figure 11. After HVT there were no changes in cortisol.

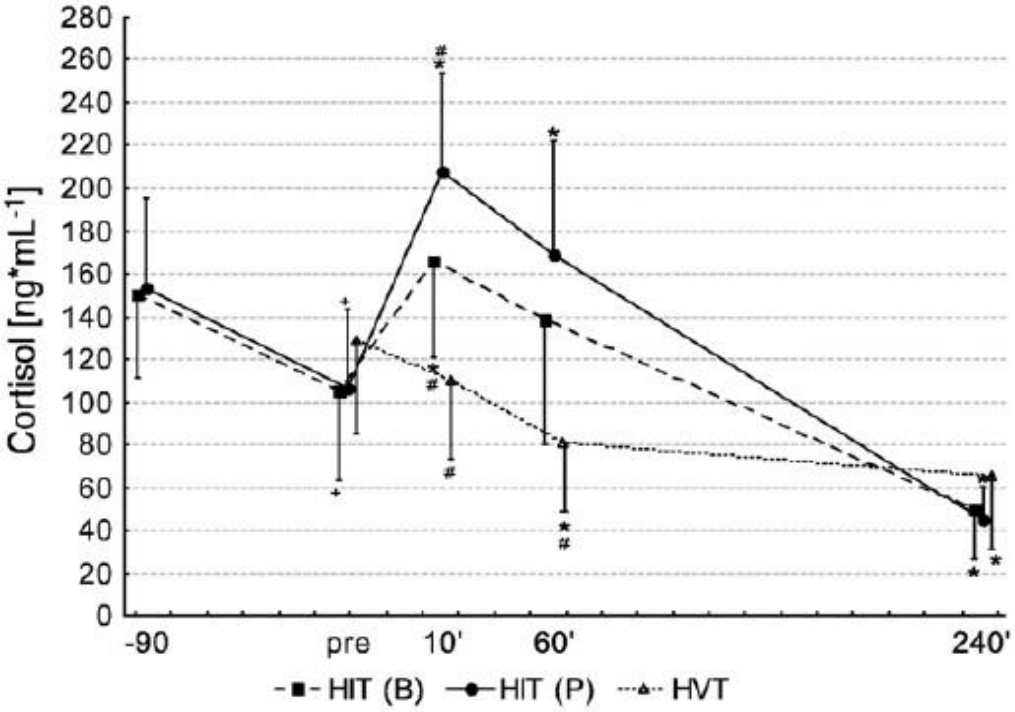

FIGURE 11. Acute changes in cortisol concentrations at different time points before and after three different exercise bouts: HIIT (P), HIIT (B) and HVT. # = p<0,05. (Wahl et al. 2010.)

As a conclusion based on the scientific studies presented above, there is no existing data about the effect of high-intensity interval training on leptin and also, the data about the effects of high-intensity interval training on cortisol in long-term is currently lacking. The acute effect of single interval training session on cortisol has been studied and it seems that cortisol concentration in blood increases immediately after training session. Insulin concentration tends to decrease after high-intensity interval training but not all the results are significant. High-intensity circuit training has not yet been studied with normal weight, healthy adults but the data with obese people shows that insulin tends to decrease after a training program. The current data is quite limited and it must be noted, that the subject number has been low and the studies has been done with healthy, active adults. Also, the changes in hormone levels are often so small that it is difficult to get significant results.

5 REASEARCH QUESTION

Currently, there is no data available about the effect of high-intensity circuit training on blood lipids and hormones and also, the existing information about high-intensity interval training on the same variables is limited. For these reasons, this study was conducted and the aim was to find an answer to the following research question.

1. Are there differences in blood lipids and hormones between the three different training protocols (HIIT running, HIIT circuit and steady-state running)?

   Hypothesis:

   Based on the previous data presented in the literature, the hypothesis is that high-intensity interval training will cause bigger changes in blood lipids and hormones than steady-state training in recreationally active, healthy adults. The hypothesis is that HIIT training will increase HDL and lower total cholesterol and LDL. Also, HIIT training will decrease insulin concentration more than steady-state running.

6 METHODS

6.1 Subjects

In this study, the objective was to recruit 20–40 recreationally active males and females at the age of 25–40 years. The recruitment was based on voluntariness. All the subjects went through a doctor examination which included resting ECG, health questionnaires and interview by a medical doctor. After this, 24 healthy subjects were included in the study. The subjects were randomized in to three groups: 1. HIIT running (n=8), 2. HIIT circuit (n=8) and 3. Steady-state running (n=8). The basic information about the subjects is listed in the table 10. For medical and personal reasons, there were 5 drop-outs in this study: 3 from HIIT running-group, one from HICT-group and one from steady-state running -group. All the subjects were informed personally about the details of the study. The study was approved by the ethical committee of Central Finland's hospital district and informed written consent was obtained from all subjects. This study was done by three master degree students and there will be three separate master's theses. The focus in this thesis is on blood lipids and hormones, Aino Kari's thesis focuses on insulin tolerance and body composition and Mari Stenman's thesis focuses on fat oxidation capacity and physical fitness.

TABLE 10. Basic information about the subjects in each group. Column "Combined HIIT" includes subjects from both interval groups. Above are all the subjects and below are just women.

ALL	**Steady-State running**		**HIIT running**		**HICT circuit training**		**Combined HIIT**	
	pre (n=7)	post (n=7)	pre (n=5)	post (n=5)	pre (n=7)	post (n=7)	pre (n=12)	(n=12)
Height (cm)	167±4	167±4	171±7	171±7	173±5	173±5	173±8	173±8
Weight (kg)	63.2±5.5	61.7±4.6	65.7±8.2	65.8±8.3	69.1±9.6	69.3±10.1	67.7±9.8	67.9±9.7
BMI	22.7±1.3	22.6±1.3	22.2±1.1	22.0±1.0	22.9±2.4	23.0±2.6	22.6±1.9	22.7±2.0
Fat percent (%)	21.8±9.3	22.0±10.5	24.1±3.0	24.6±3.2	25.2±7.5	25.3±7.8	24.7±7.3	25.0±7.8
VO_{2max} (ml/kg/min)	40.6±6.8	41.1±8.7	38.8±3.7	39.4±4.4	40.6±6.2	40.6±6.2	39.8±4.8	39.7±4.5

WOMEN ONLY	**Steady-State running**		**HIIT running**		**HICT circuit training**		**Combined HIIT**	
	pre (n=5)	post (n=5)	pre (n=4)	post (n=4)	pre (n=5)	post (n=5)	pre (n=9)	post (n=9)
Height (cm)	165±4	165±4	168±2	168±2	168±5	168±5	168±4	168±4
Weight (kg)	60.7±4.3	60.1±4.0	61.9±3.7	62.0±3.3	65.3±9.6	66.1±10.1	63.8±7.7	65.3±8.1
BMI	22.6±1.5	22.4±1.4	21.9±1.0	22.0±1.0	23.0±2.4	23.2±2.6	22.5±2.0	22.7±2.1
Fat percent (%)	26.2±5.9	26.7±7.8	25.2±2.2	25.6±2.7	29.2±7.5	29.9±7.8	27.4±6.1	28±6.4
VO_{2max} (ml/kg/min)	37.2±4.1	37.2±6.6	39.5±3.8	39.8±4.8	40.2±6.2	38.23.6	39.9±5.3	38.9±4.2

6.2 Study design

In this study there were pre- and post-exercise period measurements. The duration of the training period was eight (8) weeks including one familiarization exercise after the pre-measurements. The subjects received diet instructions in order to standardize nutritional status and they were asked to keep food diaries for five days in the beginning of intervention and five days at the end of the intervention. Every participant exercised three times a week and the exercises were:

1. HIIT running group

- 8–10 x 1 min. exercise at the intensity of 85–95 % of VO_{2max} divided by 30 seconds active recovery at the intensity of 40–60 % of $VO_{2max.}$
- The loading was progressive: Weeks 1–3 subjects did 8 intervals, weeks 4–6 they did 9 intervals and weeks 7–8 they did 10 intervals.
- Running on a treadmill.

2. HICT-group

- 8–10 x 1 min exercise followed by 30 seconds active recovery at the intensity of 50–60 % of $VO_{2max.}$
- The goal was to perform as many repetitions as possible in 1 minute
- The loading was progressive: Weeks 1–3 subjects did 8 exercises, weeks 4–6 they did 9 exercises and weeks 7–8 they did 10 exercises.
- Resistance training exercises with own body weight. The exercises were a jumping jack, push-up with a jumping jack, burbee, spider push-up, jumping lounge, rotating plank, skating jump, mountain climber, long jump from a spot and squat.

3. Steady-State running group

- 40–60 min. continuous work (65–75 % VO_{2max})
- The loading was progressive: Weeks 1–3 subjects ran 40 minutes, weeks 4–6 50 minutes and weeks 7–8 60 minutes.
- Running on a treadmill

The pre-measurements included DEXA, blood tests, oral glucose tolerance test and VO_{2max} test on a bicycle ergometer (Ergoline bike, CareFusion breath analyzer). From the VO_{2max} test, HR_{max} and VO_{2max} values were obtained for every subject and those were used to calculate the intensities individually for everyone. For the training period, HR monitors weren't available, so speed was used to measure the training intensity in the running groups. The first exercise session was done supervised and the training speeds (intensity) were determinated individually using heart rate, oxygen consumption and RPE-scale. Also, blood lactate was obtained to evaluate the intensity of the exercise. In the middle of the intervention (week 4), the training speed (intensity) was adjusted again.

6.3 Data collection and analyses

The following measurements were conducted to all subjects before the intervention and as soon as possible after the last training session.

Maximal aerobic capacity (VO_{2max}) with indirect calorimeter (cycle ergometer)
DEXA body composition
Blood tests for lipid and hormonal concentrations and oral glucose tolerance test

VO_{2max} was measured separately from the other measurements, since the other tests require overnight fasting. VO_{2max} was done with cycle ergometer and the test was started with 5 min warm-up with 50 W. The test was started with 50 W and each step lasted for 2 minutes. After the 2 minutes, the load was increased by 25 W until exhaustion. At the end of every stage, blood lactate was obtained from the fingertip, heart rate was monitored (Polar heart rate monitor) and subjects were asked the stress they were feeling using RPE-scale. During the test, respiratory gases were also measured. Respiratory gases were measured with portable indirect calorimeter during the first and last training exercise and as well during 30 minutes after the end of the exercise.

DEXA (GE Lunar Prodigy Advance) measurement was done in the morning after overnight fast as well as the blood tests. The subjects came in to the measurement in the morning after a night fast between 7.30 and 9.00 am. In the blood tests, 3,5 ml of blood was obtained to Vacuette gelheparintube (cholesterols, triglycerides, glucose, cortisol and insulin) and 3,5 ml f blood was obtained to gelserumtube (leptin, glycerol and FFAs). The blood was obtained from an arm vein. The blood samples were resting for 10 minutes and then centrifugated 15 minutes at the speed of 3500 rpm. The plasma was separated and total cholesterol, HDL, LDL, triglycerides, glucose, glycerol and FFAs were analyzed using the Konelab™ 20XT Clinical Chemistry Analyzer and insulin and cortisol were analyzed using Immulite 1000 Immunoassay System. Leptin was analyzed using ELISA-method.

After the first blood sample, subjects drank a glucose drink and blood samples were obtained again 1 hour and 2 hours after that. From those samples, glucose and insulin were analyzed same way as described above.

Food diaries were analyzed with Nutri-Flow program (Nutri-Flow Oy, Finland). Whole energy consumption, carbohydrate, protein and fat intakes were calculated using the program.

6.4 Statistical methods

All the statistical analyses were done using IBM SPSS 20 for Windows. At first, means and standard deviations were calculated. Then, it was checked that all the data were normally distributed and if not, the data were normalized using logarithmic transformation. Data analyses were done to all subjects and also for women separately because the majority of subjects were women.

After that, the differences between the three groups (HIIT, HICT and steady-state) were checked. For that, the pre-post changes between groups were compared with paired-samples T-test. There were no differences between HIIT and HICT, so it was decided to combine the two interval groups to one "Combined Interval" -group. The reason for this was that the subject number was so low in the interval groups and they trained the same amount of time, so it was thought that it would be more reasonable to combine the groups.

The next step was to determine if there are differences between pre and post measurements and, is there a difference between steady-state running group and combined interval group. Because the data was normally distributed, paired-samples t-tests were used. P-value lower than 0,05 ($P<0,05$) was considered as statistically significant result and it is marked with "*".

Food data were analyzed also with IBM SPSS 20. Means and standard deviations were calculated first. The data were normally distributed so Paired-Samples T-test was used to compare pre-post changes in each group. After that, groups were compared so that the pre-post changes between groups with Paired-Samples T-test were tested. The food data is presented in Appendix 2.

7 RESULTS

At first, the two different HIIT training groups were compared and all the variables are shown in figures 12 and 13. The main finding in this study was that there are no significant differences between HIIT running and HIIT circuit training groups in measured lipid or hormone variables.

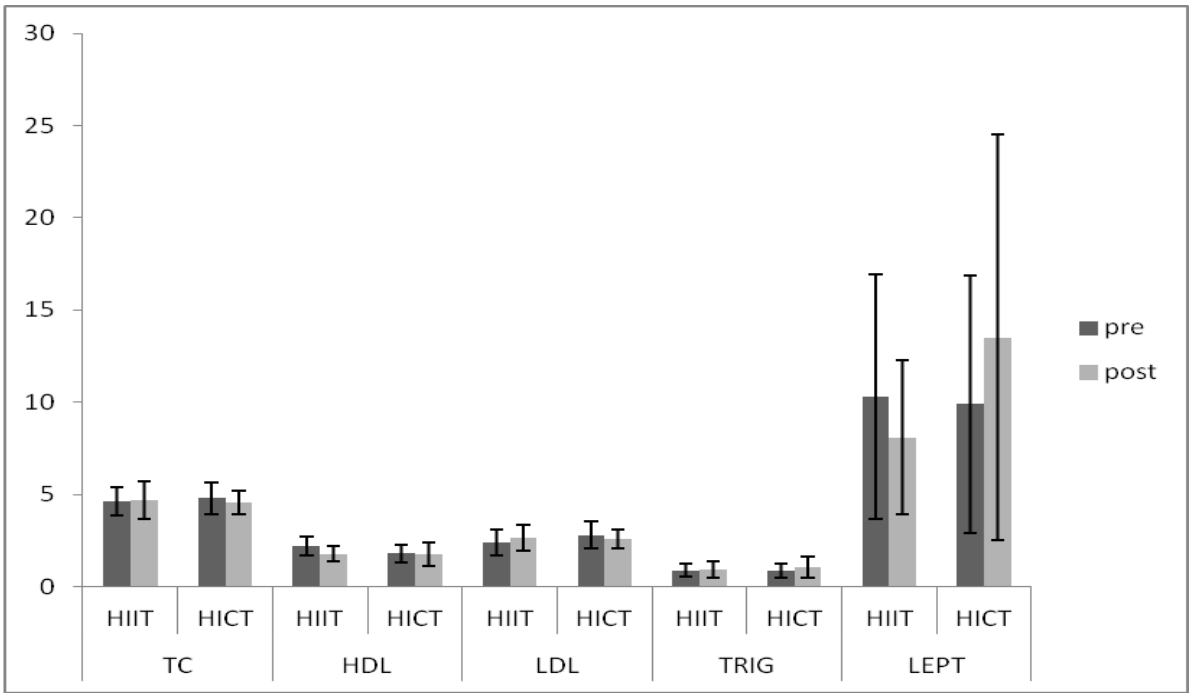

FIGURE 12. The comparison between the different HIIT groups. No differences between the two groups were observed. Measurement units are: TC, HDL, LDL, Trig (mmol/l), Lept (ng/l).

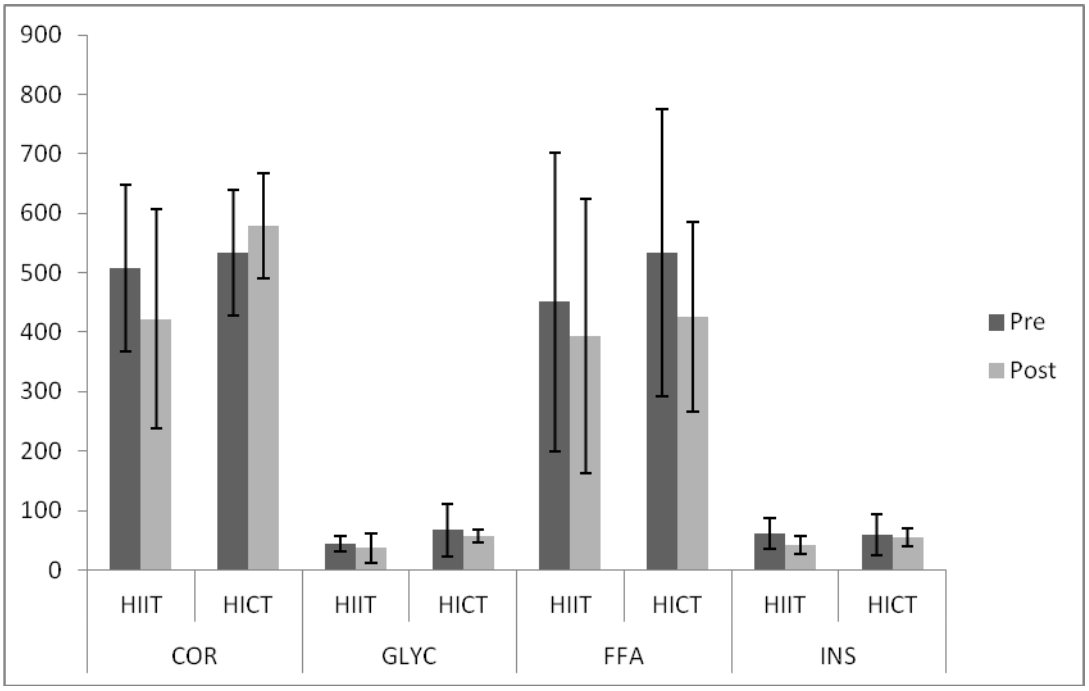

FIGURE 13. The comparison between the two different HIIT groups. No differences between the two groups were observed. Measurement units are: Cor (nmol/l), Glyc (µmol/l), FFA (µmol/l), Ins (pmol/l).

Because there were no significant differences between the two interval groups, they were combined as one bigger group called “Combined HIIT” from now on. The reason for this is that the subject number is bigger and the statistical analyses are more reasonable. In figures 14–22 are shown all the measured variables from combined HIIT and steady-state groups. A star (*) means that there is a statistically significant difference between the values.

When we compared the differences between groups, there were no significances, meaning that there were no differences. The only significant finding was, that insulin decreased 16,7 % following the HIIT intervention (shown in figure 14).

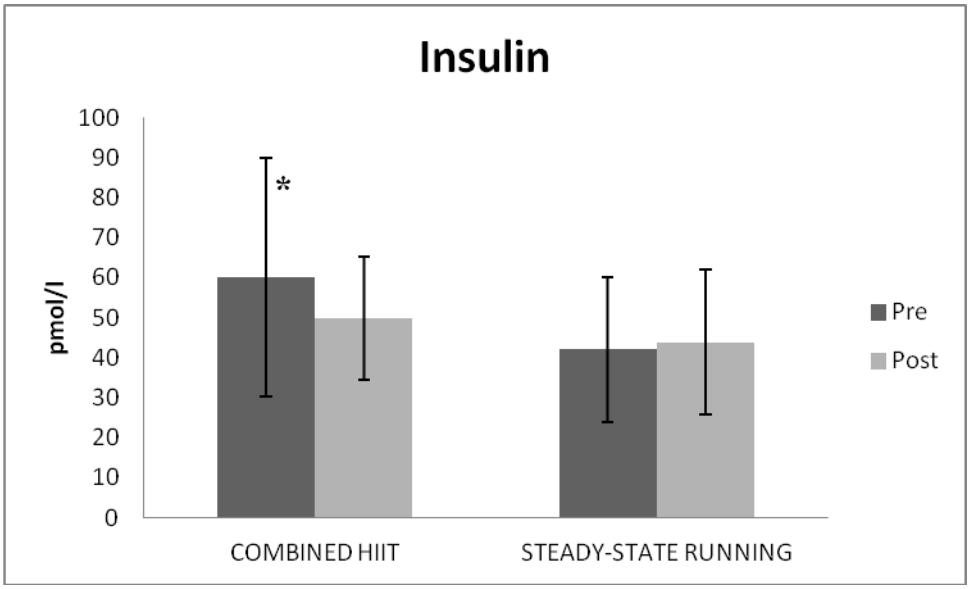

FIGURE 14. Insulin concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups. The difference between pre and post value is significant (*) when $p<0,05$.

FIGURE 15. Total cholesterol concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

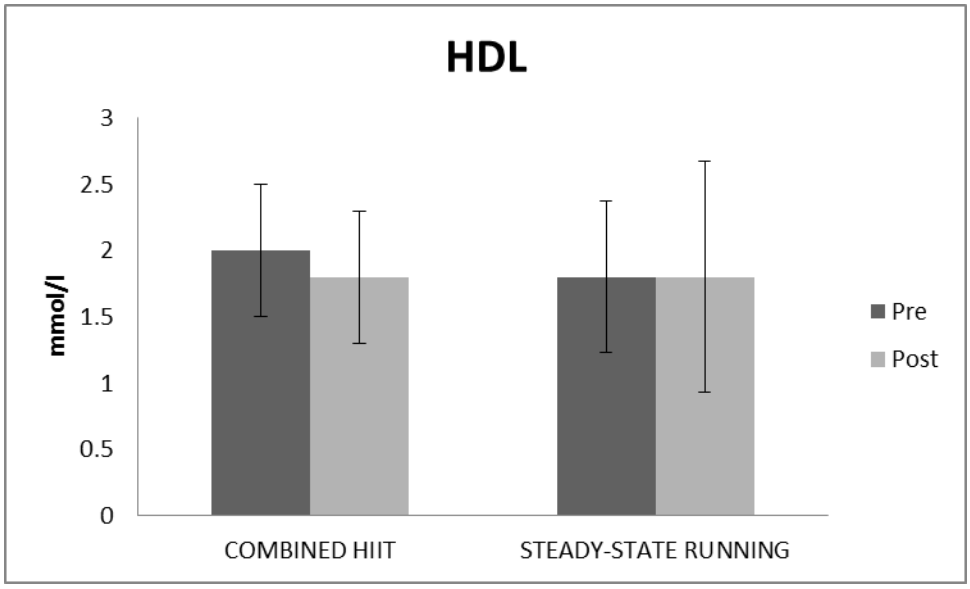

FIGURE 16. HDL-cholesterol concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

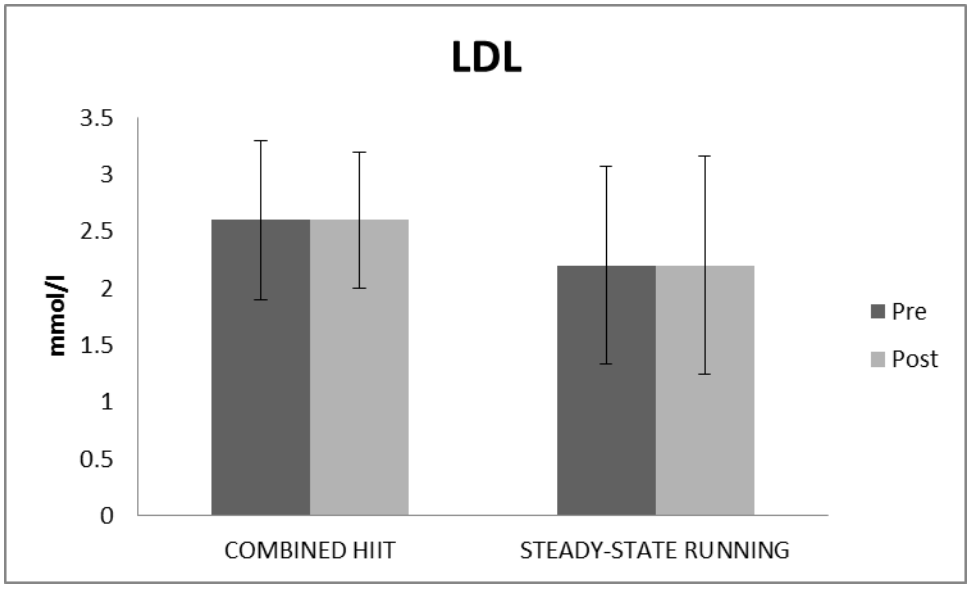

FIGURE 17. LDL-cholesterol concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

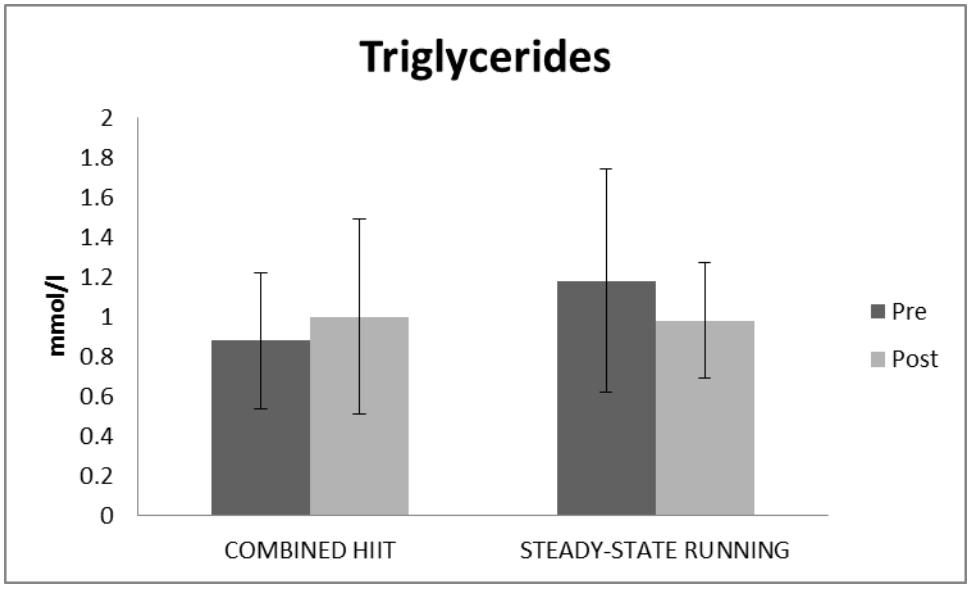

FIGURE 18. Triglycerides concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

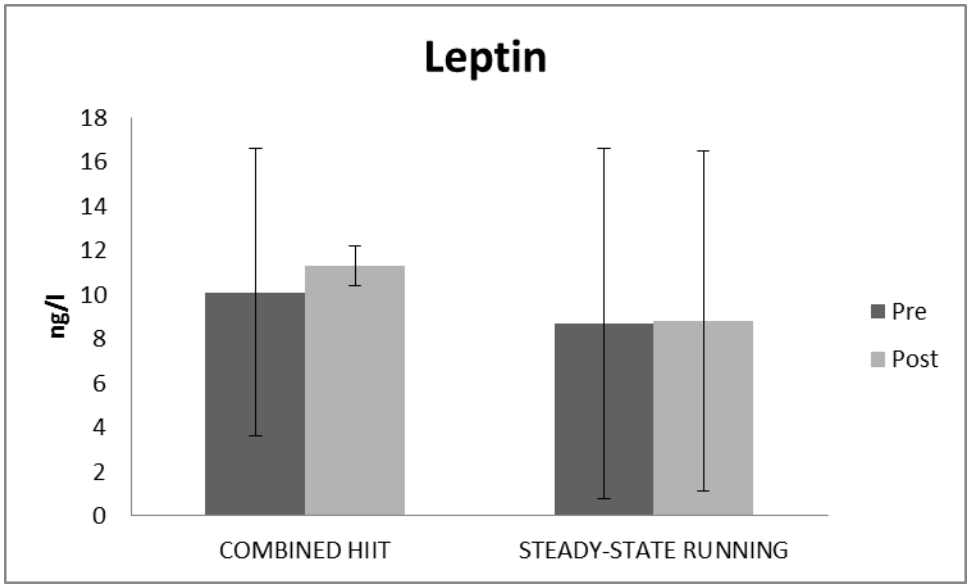

FIGURE 19. Leptin concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

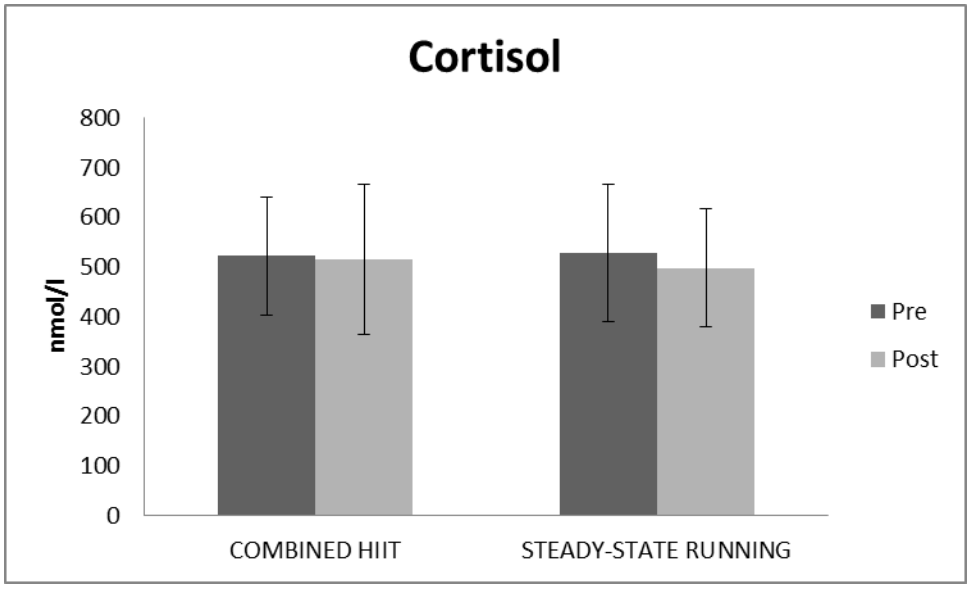

FIGURE 20. Cortisol concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

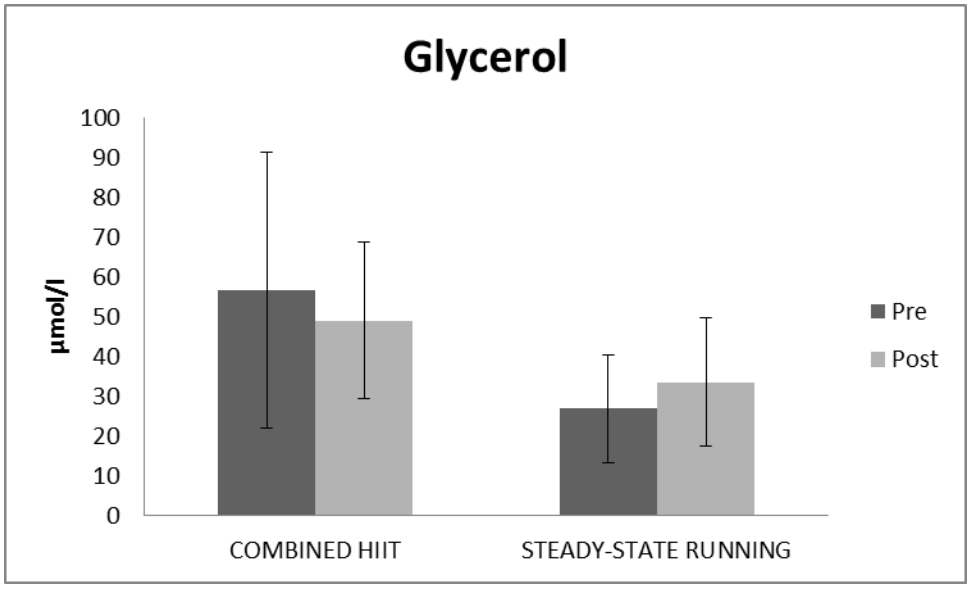

FIGURE 21. Glycerol concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

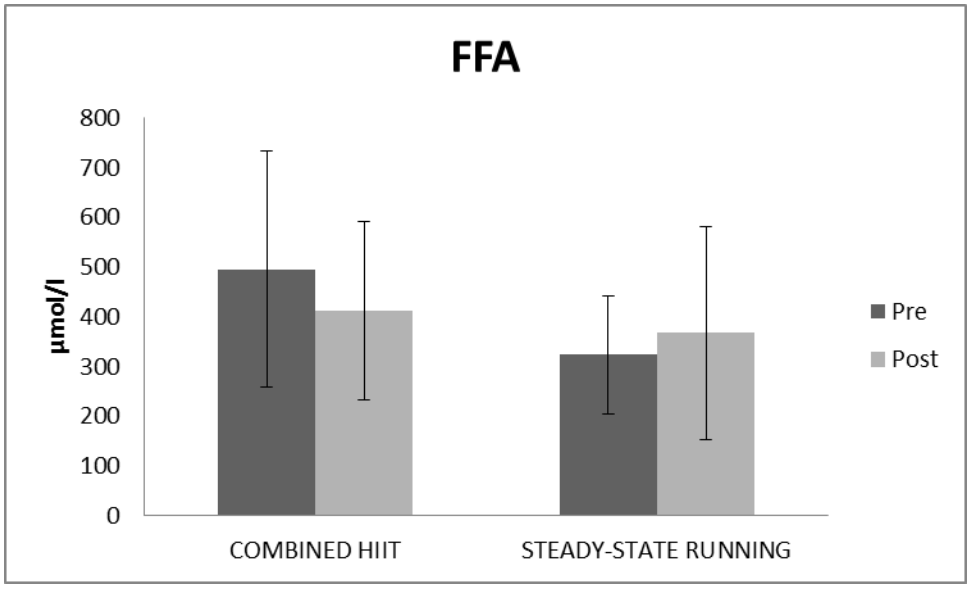

FIGURE 22. FFA concentration at rest before and after the 8 week intervention in combined HIIT and steady-state running groups.

Because the majority of the subjects were women, analyses were done also using only women's data. There were no differences between groups and the only significant finding was again in insulin which decreased significantly in the HIIT-group following the intervention. The decrease was 21,6%.

8 DISCUSSION

The main finding in this study was that the two different type of HIIT training methods (running and circuit training) didn't differ at all after 8 week intervention when considering blood lipids and hormones (cortisol, leptin, insulin). Another important finding was that insulin decreased significantly 16,7 % after 8 week training period in the combined HIIT group.

Lipids. Total cholesterol has been seen to decrease in previous studies (Musa et al. 2009; Miller et al. 2014) but we didn't get any significant results. In HIIT group there was a little decrease but the individual variation is so big that it is not a significant change. Also TC in women in HIIT group tended to decrease a little. HDL has been seen to rise in some studies (Musa et al. 2009; Paoli et al. 2013) but not in this study. Actually in HIIT group, HDL tended to lower slightly both in men and women but there were no change in steady-state running group. LDL has been seen to decrease after HIIT training (Paoli et al. 2013) but no such finding was observed in this study, LDL cholesterol didn't change almost at all. Triglycerides and HIIT have not been studied in normal weight adults. In obese people, triglycerides have decreased both acutely after one HIIT session but also after long-term intervention. (Paoli et al. 2013; Miller et al. 2014.) It must be noted that weight loss itself causes decline in triglycerides (Hobkirk et al. 2013) so we cannot say is the observed reduction in TG caused by the exercise or weight loss. In this study, there was a slight increase in HIIT group and a decrease in steady-state running group but these weren't significant because the variation was so big.

There is no previous data available about the effect of HIIT on fatty free acids long-term, but acutely FFA's has seen to increase (Perry et al. 2008; Peake et al. 2014). In this study, no significant changes were observed in FFA's but the concentrations tended to decrease in HIIT and increase in steady-state running group. Again, the individual variation is so huge

that the results are not significant. Currently, there is no data existing either on glycerol and in this study we didn't see any changes.

Hormones. The second main finding in this study was significant reduction in insulin concentration after 8 week HIIT training program. Insulin's main role in the body is to help glucose move into the cells from circulation. Glucose is transported in to the cells with GLUT4 molecules that are activated by insulin. (Silverthorn et al. 2010, 736–741.) In one HIIT study (Hood et al. 2011) GLUT4 content increased 260 % after 2 week training intervention in sedentary middle-aged women. In that study, they had 7 women as subjects and they did 6 training sessions in two weeks. The training protocol was 10 times 1 minute intervals with minute recovery periods between. In this study, GLUT4 content was not measured but it could be assumed that when insulin concentration decreased significantly, GLUT4 content and activity has risen. Less insulin is needed when GLUT4 activity and content is bigger for glucose to move into the cells at the same rate. Based on this hypothesis, one explanation for decreased insulin concentration could be that less insulin is needed to transport glucose in to the cells, the glucose would move in to the cell more effectively. In this study, no muscle biopsies were taken. It would have been important because they could have shown us the mechanisms and the changes in cell and molecule level that occurred due the training.

Glucose was also measured but the results are not presented in this study, it is discussed in Aino Kari's thesis. Glucose values are presented in table 11. Even though insulin decreased in HIIT group, there was no significance change in glucose. There was a little tendency for increase in HIIT group but not in steady-state. Insulin transports glucose in to the cells and in normal situation, if insulin concentration increases, more glucose is transported to the cells and glucose concentration in blood decreases. (McArdle et al. 2010, 420.) In our study, insulin decreased but glucose didn't change so we can conclude that due to the training, less insulin is needed to transport glucose from blood to the cells in other word, insulin functions more efficiently.

TABLE 11. Glucose concentrations in combined HIIT group and steady-state running group before and after 8 week intervention.

	Combined HIIT		**Steady-State running**	
	pre	**post**	**pre**	**post**
n	15	10	8	7
Glucose	4,95±0,40	5,04±0,36	5,04±0,44	5,01±0,41

There were no significant changes in cortisol or leptin. In this study, cortisol tended to decrease a little and in leptin, there was a slight decrease. There were no existing data available about these variables and based on this study, high-intensity interval training doesn't affect to leptin or cortisol levels in long-term. Obviously, more studies are needed to support this finding.

In previous hormone studies, there have been seen a huge inter-individual variability in hormone responses to training even if all the basic characteristics (age, gender, mass and physical fitness) are similar (Stokes et al. 2013). The variation was really big in this study also, and that affects to the results.

Nutrition. Subjects were told keep the diet as unchangeable as possible and the subjects were given the Finland's diet recommendations. To control the diet, subjects were asked to keep food diaries 3–5 days at the beginning of the study and also 3–5 days at the end of the study. The results from the diaries are presented in Appendix 2. Finnish nutrition recom-

mendations (Ravitsemusneuvottelukunta 2014) says that for an adult man, the energy need is about 2700–3150 kcal and for women 2200–2500 kcal based on the activity level. In this study, the energy consumption was way too low compared to the recommendations. Probably the diet have influenced to the results in this study because nutrition has so big influence on blood lipids. Vegetable-based nutrition induces positive results in blood lipids and animal protein-based diet induces negative results in blood lipids (Mensink & Katan 1989; Barnard et al. 2000; Ferdowsian & Barnard 2009.) meaning, that vegetable-based diet increase HDL and decrease LDL and TC and animal protein does the opposite. Diet can also affect to the hormone metabolism. Diet including plenty of protein has been associated with increased cortisol secretion and increased cortisol activity can affect negatively to insulin action. (McCarty 2005.)

Gender differences. In our study, majority of the subjects were women. There were some gender differences: HDL, cortisol and leptin were higher in women in all groups compared to whole subjects population. Because there were only three men in total, there is no sense to look men's values separately. To get more reliable results, it would have been better to include only men or only women because there are differences in lipid and hormone metabolism between sexes. In our study, the subject number was so low already so we included both sexes. Other limitation in this study was that we didn't have control group at all.

Study protocol. It must be noted, that in many interval studies, subject number have been low, diet has not been controlled and subjects have been recretionally active, healthy adults. Also, protocols have varied much so all the results are not comparable and conclusions and general concensus are hard to make.

In the middle of the study, training was interrupted by the Christmas. During Christmas, almost all the gyms were closed and majority of the subjects were out of town. Subjects were advised to do the training in indoors if possible, but if that wasn't possible, there were instructed to do the exercises outside. Because we didn't have heart rate monitors, the intensity (speed) was not possible to define so the subjects were asked to do the training based on

their own feeling (RPE-scale). This could have some influence to the results. Also, eating during Christmastime was far from normal and it could have influenced to especially to lipid variables.

The idea was that all the measurements (blood tests, DEXA, VO2max) were done also in the middle of the intervention. The halfway was during the Christmas so we had to cancel those after all. Some of the subjects were already away, our facilities were closed and the staff was already on a holiday so it became impossible. It would have been interesting to see, if the four weeks were enough to cause some changes and did the holiday time have some effect.

If there would have been more money and resources available, acute measurements after the exercise sessions would have been informative. It would have been interesting to see, what kind of changes the two different type interval training sessions have on lipids and hormones acutely. To valid the study, it would have been important that all the training sessions were done supervised, now only the first, the last and the one in the middle were done supervised, otherwise subjects were instructed to do the training by themselves. The monitoring of the intensity would have been more accurate if heart rate monitors would have been available in every training session but in this case, it was not possible.

The biggest limitation in this study was the low subject number. It was really hard to get suitable, healthy adults to commit to this training program for 8 weeks without doing any own training. When the groups were so little, even one divergent value influence to the means and deviation so much that it was hard to get significance results. The individual variation between subjects was big. In figure 23 is shown the individual variation in total cholesterol, HDL and LDL in combined HIIT group. For some people, HIIT training decreased TC for over 20 % but for some, TC increased over 10 %and same goes with LDL. For one subject, HDL decreased for 40% while other person's HDL increased about 5%. This means that all the subjects didn't response to the training same way. For the majority, interval training caused better lipid profiles but for some people, the interval training did the oppo-

site. It is important to find suitable training method for every individual so that exercise training has positive effects on fitness and health. Also, it is important that the training is varying and diverse. It is also important that the training is meaningful and enjoyable. In one study, (Bartlett et al. 2011), recreationally active men experienced high-intensity interval training more enjoyable than moderate-intensity continuous running. Continuous running was said to be boring and monotonous. That is why different type of exercises are recommended.

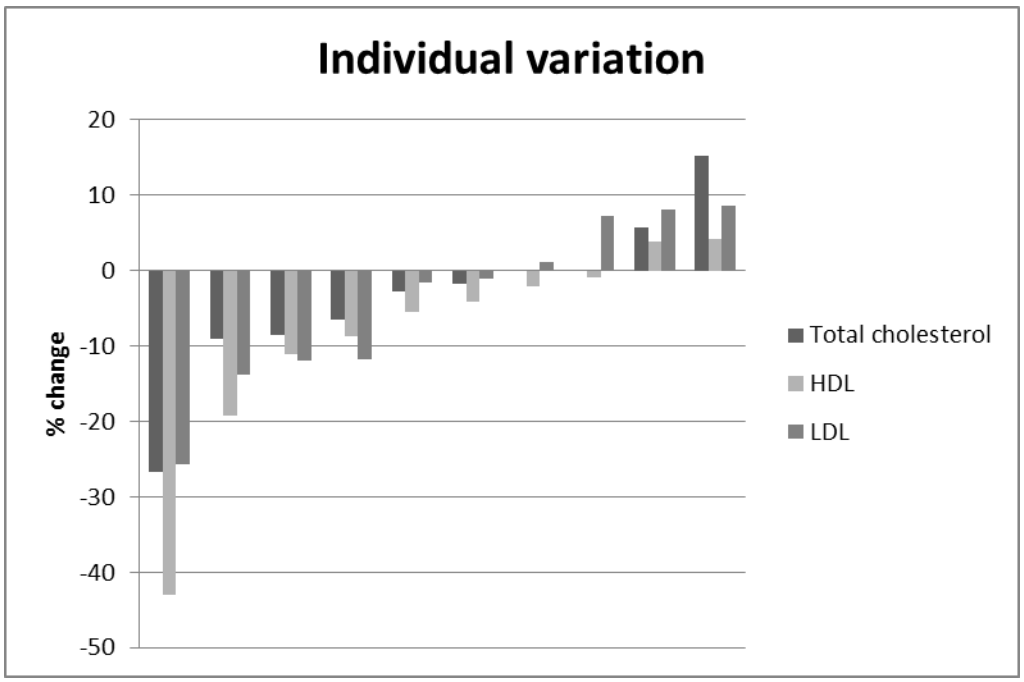

FIGURE 23. Individual variation in cholesterols in both HIIT groups (n=10).

It must be remembered the subjects in this study were recreationally active adults. These people were already in quite good shape and it is always harder to get significant improvements than with inactive population. These subjects were also healthy and it is easier to get significant changes in lipids with exercise if people are for example obese or have dyslipidemia.

In conclusion, high-intensity interval training seems to decrease blood insulin concentration without affecting blood glucose concentration in healthy, active adults. The results didn't differ between the two different type of interval training (running versus circuit training) when considering blood lipids and hormones. Therefore it seems that the volume and intensity of the training is more important than the type of physical activity (running, biking, circuit training etc.).

9 REFERENCES

Babraj J., Vollaard N., Keast C., Gruppy F., Cottrell G. & Timmons J. 2009. Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. BMC Endocrine Disorders. 9.

Barnard, N., Scialli, A., Bertron, P., Hurlock, D., Edmonds, K. & Talev, L. 2000. Effectiveness of a Low-Fat Vegetarian Diet in Altering Serum Lipids in Healthy Premenopausal Women. Am J Cardiol 85, 969–972.

Bartlett, J., Close, G., Maclare, D., Gregson, W., Drust, B. & Morton, J. 2011. High-intensity interval running is perceived to be more enjoyable than moderate-intensity continuous exercise: Implications for exercise adherence. Journal of Sports Sciences, 29, 547–553.

Belfiore, A., Frasca, F., Pandini, G., Sciacca, L. & Vigneri, L. 2009. Insulin Receptor Isoforms and Insulin Receptor/Insulin-Like Growth Factor Receptor Hybrids in Physiology and Disease. Endocrine Reviews 30, 586–623.

Camont, L., Chapman, MJ. & Kontush, A. 2011. Biological activities of HDL subpopulations and their relevance to cardiovascular disease. Trends in Molecular Medicine 17, 594–603.

Caro, J., Kolaczynski, J., Nyce, M., Ohannesian, J., Opentanova, I., Goldman, W., Lynn, R., Zhang, PL., Sinha, M. & Considine, R. 1996. Decreased cerebrospinal-fluid/serum leptin ratio in obesity: a possible mechanism for leptin resistance. The Lancet, 348, 159–161.

Christiansen, J., Djurhuus, C., Gravholt, C., Iversen, P., Christiansen, J., Schmitz, O., Weeke, J., Jørgensen,J. & Møller, N. 2007. Effects of Cortisol on Carbohydrate, Lipid, and

Protein Metabolism: Studies of Acute Cortisol Withdrawal in Adrenocortical Failure. J Clin Endocrinol Metab 92, 3553–3559.

Felsing, N., Brasel, J. & Cooper, D. Effect of low and high intensity exercise on circulating growth hormone in men. The Journal of clinical endocrinology and metabolism 75, 157–162.

Ferdowsian, H. & Barnard, N. 2009. Effects of Plan-Based Diets on Plasma Lipids. Am J Cardiol 104, 947–956.

Gibala, M., Little, J., MacDonald, M. & Hawley, J. 2012. Physiological adaptations to low-volume, high-intensity interval training in health and disease. The Journal of Physiology, 590, 1077–1084.

Gray, A., Telford, R.,Collins, M. & Weidemann, M. 1993. The response of leukocyte subsets and plasma hormones to interval exercise. Med Sci Sports Exerc 25, 1252–1258.

Grieco, C., Swain, D., Colberg, S., Dowling, E., Baskette, K., Zarrabi, L., Gandrakota, R., Kotipalli, U., Sechrist, S. & Somma, T. 2013. Effect of intensity of aerobic training on insulin sensitivity/resistance in recreationally active adults. Journal of Strength and Conditioning Research, 27, 2270–2276.

Guyton, A. & Hall, J. 2006.Textbook of Medical Physiology. Elsevier Inc, Philadelphia.

Hers, H. 1986. Effects of glucocorticoids on carbohydrate metabolism. Agents and Actions, 17, 248–254.

Hickey, M., Considine, R., Israel, R., Mahar, T., McCammon, M., Tyndall, G., Houmard, J. & Caro, J.1996. Leptin is related to body fat content in male distance runners. American Journal of Physiology - Endocrinology and Metabolism. 34, 938–940.

Hobkirk, J., King., R., Davies, I., Harman, N., Gately, P., Pemberton, P., Smith, A., Barth, J. & Carroll, S. 2013. The metabolic inter-relationships between changes in waist circumference, triglycerides, insulin sensitivity and small, dense low-density lipoprotein particles with acute weight loss in clinically obese children and adolescents. Pediatric Obesity 9, 209–221.

Hood, M., Little, J., Tarnopolsky, M., Myslik, F. & Gibala, M. 2011. Low-Volume Interval Training Improves Muscle Oxidative Capacity in Sedentary Adults. Medicine & Science in Sports & Exercise 43, 1849–1856.

Houseknecht, K. & Portocarrero, C. 1998. Leptin and its receptors: regulators of whole-body energy homeostasis. Domestic Animal Endocrinology, 15, 457–475.

Hu, G., Pekkarinen, H., Hänninen, O., Tian, H. & Guo, Z. 2001. Relation between commuting, leisure time, physical activity and serum lipids in a Chinese urban popula-tion. Ann Hum Biol 28, 412–421.

Katzmarzyk, T., Gagnon, J., Leon, A., Skinner, J., Wilmore, J., Rao, D. & Bouchard, C. 2001. Fitness, fatness and estimated coronary heart disease risk: the HERITAGE Family Study. Med Sci Sports Exerc. 33, 585–590.

Kelley, G. & Kelley, K. 2006. Aerobic exercise and lipid and lipoproteins in men: s meta-analysis of randomized controlled trials. J Mens Health Gend 3, 61–70.

Kelley, G., Kelley K. & Zung, V. 2004. Aerobic Exercise and Lipids and Lipoproteins in Women: A Meta-Analysis of Randomized Controlled Trials. Journal of Women's Health 13, 1148–1164.

Klika, B. & Jordan, C. 2013. High-intensity circuit training using body weight. ACSM's Health & Fitness Journal, 17, 8–13.

Kordi, MR., Choopani, S., Hemmatinafar, M. & Choopani, Z. 2013. The effects of the six week high intensity interval training (HIIT) on resting plasma levels of adiponectin and fat loss in sedentary young women. Journal of Jahrom University of Medical Sciences, 11, 20–27.

McArdle, W., Katch, F. & Katch, V. 2010. Exercise Physiology. Lippincot Williams & Willkins, USA.

Musa, D., Adeniran, S., Dikko, A. and Sayers, S. 2009. The effect of a high-intensity interval training program on high-density lipoprotein cholesterol in young men. J Strength Cond Res 23, 2, 587–592.

McCarty, M. 2005. Acid–base Balance May Influence Risk for Insulin Resistance Syndrome by Modulating Cortisol Output. Medical Hypotheses 64, 380–384.

McMahon, M.,Gerich, J. & Rizzat, R. 1988.Effects of Glucocorticoids on Carbohydrate Metabolism. Diabetes/metabolism Reviews, 4, 17–30.

Mensink, R. & Katan, M. 1989. Effect of a Diet Enriched with Monounsaturated or Polyunsaturated Fatty Acids on Levels of Low-Density and High-Density Lipoprotein Cholesterol in Healthy Women and Men. N Engl J Med 321, 436–441.

Miller, M., Pearcey, G., Cahill, F., McCarthy, H., Stratton, S., Noftall, J., Buckle, S., Basset, F., Sun, G. & Button, D. 2014. The Effect of a Short-Term High-Intensity Circuit Training Program on Work Capacity, Body Composition, and Blood Profiles in Sedentary Obese Men: A Pilot Study. BioMed Research International, Article ID 191797.

Nybo, L., Sundstrup, E., Jakobsen, M., Mohr, M., Hornstrup, T., Simonsen, L., Bülow, J., Randers, M., Nielsen, J., Aagaard, P. & Krustrup, P. 2010. High-intensity training versus traditional exercise interventions for promoting health. Med. Sci. Sports Exerc., 42, 1951–1958.

Panagiotakos, D., Pitsavos, C., Chrysohoou, C., Skoumas, J., Zeimbekis, A., Papaioannou, I. & Stefanadis, C. 2003. Effect of leisure time physical activity on blood lipid levels: the ATTICA study. Coron Artery Dis 14, 533–539.

Paoli, A., Pacelli, Q., Moro, T., Marcolin, G., Neri, M., Battaglia, G., Sergi, G., Bolzetta, F. & Bianco, A. 2013. Effects of high-intensity circuit training, low-intensity circuit training and endurance training on blood pressure and lipoproteins in middle-aged overweight men. Lipids in Health and Disease, 12, 131.

Peake, J., Tan, SJ., Markworth, J., Broadbent, J., Skinner, T. & Cameron-Smith, D. 2014. Metabolic and hormonal responses to isoenergetic high-intensity interval exercise and continuous moderate-intensity exercise. Am J Physiol Endocrinol Metab 307, 539–552.

Perusse, L., Collier, G., Gagnon, J., Leon A., Rao, D., Skinner, J., Wilmore, J., Nadau, A., Zimmet, P. & Bouchard, C. 1997. Acute and chronic effects of exercise on leptin levels in humans. J. Appl. Physiol. 8, 5–10.

Perry, C., Heigenhauser, G., Bonen, A. & Spriet, L. 2008. High-intensity aerobic interval training increases fat and carbohydrate metabolic capacities in human skeletal muscle. Appl. Physiol. Nutr. Metab. 33, 1112–1123.

Ravitsemisneuvottelukunta 2014 http://www.ravitsemusneuvottelukunta.fi/files/attachments/fi/vrn/ravitsemussuositukset_2014_fi_web.3.pdf 22.5.2015.

Reece, J., Urry, L., Cain, M.,Wasserman, S., Minorsky, P. & Jackson, R. 2011. Campbell Biology. Pearson Education, Inc. San Fransisco.

Robergs, R. A., Ghiasvand, F. & Parker, D. 2004. Biochemistry of exercise-induced metabolic acidosis. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 287, R502 – R516.

Rooyackers, O. & Nair, K.S. 1997. Hormonal Regulation of Human Muscle Protein Metabolism. Annual Review of Nutrition, 17, 457–485.

Saltiel, A. & Kahn, R. 2011. Insulin signalling and the regulation of glucose and lipid metabolism. Nature, 414, 799–806.

Schwartz, M., Peskind, E., Raskind, M., Boyko, E. & Porte, D. 1996. Cerebrospinal fluid leptin levels: relationship to plasma levels and to adiposity in humans. Nature Medicine, 2, 589–593.

Silverthorn, D., Johnson, B., Ober, W., Garrison, C. & Silverthorn, A. 2010. Human Physiology – an integrated approach. 5th edition, Pearson Education Inc. San Francisco.

Stokes, K., Gilbert, K., Hall, G., Andrews, R. & Thompson, D. 2013. Different responses of selected hormones to three types of exercise in young men. Eur J Appl Physiol, 113,775–783.

Sydänliitto http://www.sydanliitto.fi/kolesteroli#.VUok7iHtmko 6.5.2015.

Tambalis, K., Panagiotakos, D., Kavouras, S. & Sidossis, L. 2009. Responses of Blood Lipids to Aerobic, Resistance, and Combined Aerobic With Resistance Exercise Training: A Systematic Review of Current Evidence. Angiology 60, 614–632.

Tartaglia, L. 1997. The Leptin Receptor. J. Biol. Chem., 272, 6093–96.

UKK-institute. http://www.ukkinstituutti.fi/liikuntapiirakka. 3.2.2015.

Van Hall, G., Sacchetti, M., Rådegran, G. & Saltin, B. 2002. Human Skeletal Muscle Fatty Acid and Glycerol Metabolism During Rest, Exercise and Recovery. Journal of Physiology 543, 1047–1058.

Wahl, P., Zinner, C., Achtzehn, S., Bloch, W. & Mester, J. 2010. Effect of high- and low-intensity exercise and metabolic acidosis on levels of GH, IGF-I, IGFBP-3 and cortisol. Growth Hormone & IGF Research 20, 380–385.

Wahl, P., Mathes, S., Achtzehn, S., Bloch, W. & Mester, J. 2014. Active vs. passive recovery during high-intensity training influences hormonal response. International Journal of Sports Medicine. 35, 583–589.

Wilmore, J.H. & Costill, D.L. (2004) Physiology of sport and exercise. 3rd edition. Pearson Education, Inc. San Francisco.

10 APPENDIX 1. Cholesterol recommendations.

The cholesterol recommendations for adults in Finland (Sydänliitto):

Total cholesterol below 5,0 mmol/l

LDL-cholesterol below 3,0 mmol/l

HDL-cholesterol above 1,0 mmol/l

Triglycerides below 2,0 mmol/l.

11 APPENDIX 2. Results from the food diaries.

The data from food diaries is presented in table 12. Between groups, there was a significant difference between HIIT and steady-state running groups in CHO (p=0,007). In CHO intake, there was a significant difference between pre and post values in HICT group (p=0,021).

TABLE 12. The data from food diaries at the beginning and end of the 8 week intervention. The difference between pre and post value is significant (*) when p<0,05.

	HIIT	n=4	**HICT**	n=4	**SS**	n=6
	pre	**post**	**pre**	**post**	**pre**	**post**
Energy kcal	2138±240	1787±120	2216±380	1862±389	1931±169	1880±412
Protein g	106±22	94±17	94±15	84±19	99±44	102±34
Carbohydrate g	216±21	184±42	229±62	201±59*	199±46	203±55
Fat g	85±18	65±23	88±8	71±11	71±19	60±13

12 APPENDIX 3. Diet instructions.

DIET INSTRUCTIONS:

- Make sure that you get energy and the diet is versatile
- Make sure that you get enough protein (for example meat, fish, poultry, cottage cheese, quark)
- Make sure that you get enough good, soft fats (oils, vegetable fats) and avoid bad, hard fats
- Eat regularly, something at every 3 to 4 hours.
- Eat vegetables and fruits at least 500g per day
- 4 to 6 meals per day
- Avoid salt and white sugar and favor low-fat and multigrain products
- Drink enough (about 1 liter per day) favor water

NUTRIENTS:

FAT

- 25-35 % of energy should come from fats. The amount of saturated fat should be less than 10 %. Products that include saturated fat are for example cheese, meat, sweets and pastries.

CARBOHYDRATES

- 50-60 % of energy should come from carbohydrates. Multigrain products should be favored and white sugar avoided. Berries, fruits, vegetables contain good carbohydrates.

PROTEIN

- 10-20% of energy should come from protein. Products that contain good protein include poultry, seeds, nuts, leguminous plant and soya.

VITAMINS

- Multiple and versatile diet include enough vitamins. Fruits, berries and vegetables contain lots of vitamins.

DAILY ENERGY NEED

- The daily energy need for active man is about 2800 kcal and for active women 1900-2600 kcal.

13 APPENDIX 4. Health questioner (in Finnish).

TERVEYSKYSELY

On tärkeää, että tiedämme elintavoistasi ja aiemmista liikuntatottumuksista ennen kuin testaamme sinut. Vastaa seuraaviin kysymyksiin huolellisesti.

Nimi_____Syntymäaika_____Paino_____Pituus_____

	Ei	Kyllä
1. Onko sinulla todettu hengitys-, sydän-, tai verenkiertoelimistön sairauksia?	□	□
2. Käytätkö säännöllisesti lääkkeitä? Mitä?_____	□	□
3. Onko sinulla ollut rintakipuja tai ahdistustuntemuksia		
a. levossa?	□	□
b. rasituksessa?	□	□
Miten usein ja millaisia?_____		
4. Onko sinulla selkävaivoja tai muita tuki- ja liikuntaelinten pitkäaikaisia tai usein toistuvia vaivoja? Mitä_____	□	□
5. Oletko viimeisen kahden viikon aikana sairastanut jotakin tulehdustautia (flunssa, kuumetauti)?	□	□

6. Tupakoin □ _____savuketta/vrk, en tupakoi □, olen lopettanut □ (vuosi_____)
7. Koska olet viimeksi nauttinut alkoholia?_____Kuinka paljon?_____
8. Mikä on nykyisen työsi fyysinen rasittavuus?

-toimistotyö tai vastaava □

kevyt ruumiillinen työ □

raskas ruumiillinen työ □

9. Miten kuljet työmatkasi?_____ Työmatkan kesto _____min/päivä
10. Kuinka usein olet harrastanut liikuntaa viimeisen kolmen kuukauden aikana?

    -en lainkaan □

    -kerran viikossa □

    -2-3 krt viikkossa □

    -säännöllisesti yli 4 kertaa viikossa □

    Mitä liikuntaa olet harrastanut?

    _____
11. Arvio oma kuntotasosi asteikolla 1=heikko, 2=välttävä, 3=keskitasoinen, 4=hyvö, 5=erinomainen

    Kuntoarvio:_____
12. Verenpaine _____/_____mmHg, kolesteroli_____mmol/l

Vakuutan antamani tiedot oikeiksi, tunnen testaustavan ja osallistun siihen omalla vastuullani

Jyväskylässä _____/_____/2013

Allekirjoitus_____