

**EFFECTS OF TRAINING BACKGROUND TO ACUTE  
PSYCHOLOGICAL STRESS BIOMARKERS**

Aki Ikäheimo

Liikuntafysiologian Pro gradu -tutkielma

Kevät 2016

Liikuntabiologian laitos

Jyväskylän yliopisto

Ohjaaja:

Minna Tanskanen

## TIIVISTELMÄ

Ikäheimo, Aki 2015. Harjoitustaustan vaikutus fysiologisiin muuttujiin henkisen kuormituksen aikana. Liikuntabiologian laitos, Jyväskylän yliopisto. Fysiologian Pro gradu -tutkielma 79 s.

Stressi on laajalti tutkittu aihe ja kroonisen stressin aiheuttamat terveysvaikutukset ovat merkittäviä maailmassa. Stressi käsitteenä voidaan ajatella tarkoittavan tilannetta, jolloin homeostaasia järkytetään. Stressi voidaan jakaa henkiseen ja fyysiseen sekä pitkäaikaiseen ja lyhytkestoiseen. Ihmisen kokiessa stressiä, niin henkistä tai fyysistä, tapahtuu hermoston toiminnan johdosta erilaisia fysiologisia muutoksia ihmiskehossa. Reaktio stressiin johtaa esimerkiksi sykkeen kiihtymiseen, verenpaineen kohoamiseen sekä erilaisten hormonien erittymiseen, kuten: adrenaliini ja kortisoli. Tässä tutkimuksessa on tarkoituksena tutkia miten erilaisen harjoitustaustan omaavien henkilöiden fysiologiset muutokset kevyen henkisen kuormituksen aikana eroavat toisistaan.

Tutkimuksessa on mukana kolme koeryhmää, jotka kaikki suorittavat saman testin ja jokaiselta mitataan samat muuttujat: Vähän liikkuva ryhmä (N = 16), kestävyysurheilijat (N = 8) ja voimaurheilijat (N = 8). Koehenkilöille aiheutetaan kevyt henkinen kuormitus testin avulla, mikä koostuu satunnaisesti päässä lasku ja stroopin väri-sana-tehtävistä. Koehenkilöiden vastauslomakkeessa on myös epäjatkuvuus tulosten kirjausten osiossa, joka aiheuttaa henkilöille sekaantumisen, jonka tarkoitus on aiheuttaa henkistä kuormitusta. Tutkimus on jaettu kolmeen eri osaan: perustilanne, testi ja palautuminen. Näiden kolmen eri osa-alueen tuloksia vertaillaan keskenään sekä ryhmien välisiä eroja.

Ryhmät erosivat toisistaan kehonkoostumuksiensa osalta, joka oli odotettavissa. Kestävyysurheilijoilla oli alhaisempi rasvaprosentti ja voimaurheilijoilla oli enemmän lihasmassaa. Kortisolin osalta löydettiin ero perustasossa vähän liikkuvien ja urheilijoiden osalta ( $p < 0,05$ ); urheilijoilla oli korkeampi kortisolin taso. Urheilijaryhmät myös reagoivat

eri tavalla kortisolin osalta mitattuna; kestävyysurheilijoilla oli kasvava trendi kortisolin osalta ja voimaurheilijoilla laskeva trendi, tosin ilman tilastollista merkittävää eroa. Ihon sähkönjohtavuus erosi suuresti ryhmien välillä ja vähän liikkuvien ihon sähkönjohtavuus oli korkeammalla tasolla läpi testin verrattuna urheilijoihin. Systolinen verenpaine laskee vähän liikkuvien kohdalla testin ajan ja voimaurheilijoilla systolinen verenpaine on korkeampi palautumisvaiheessa kuin perustilanteessa.

Testihenkilöt raportoivat kokeneensa henkistä stressiä tutkimuksen aikana ja mitatut biomarkerit tukivat tätä. Tutkimuksessa löydettiin eroja erilaisten harjoitustaustojen omaavien henkilöiden osalta. Tämän johdosta voidaan todeta, että erilainen harjoittelutausta vaikuttaa siihen miten keho reagoi henkiseen stressiin.

Avainsanat: stressi, kestävyysharjoittelu, voimaharjoittelu, fysiologiset muuttujat, henkinen kuormitus

## **ABSTRACT**

Ikäheimo, Aki (2016). Effects of Training Background to Acute Psychological Stress Biomarkers. Department of Biology of Sport, University of Jyväskylä, Master's thesis, 79 pp.

Stress has been widely studied and negative effects of stress are considerable in the modern world. Stress can be understood as a situation in which the current state, homeostasis, is being put under the pressure to change. Stress can be divided into physical and psychological stress also stress can be acute or chronic. As human encounters a stressor, whether it is physical or psychological, the nervous system reacts and leads into changes in the biomarkers. For example heart rate is elevated, blood pressure rises and different kind of hormones are secreted like epinephrine and cortisol. In this study an experiment is made where people with different training background go through an acute psychological stress and from all the same biomarkers are measured. Participants are divided to different groups based on their training background.

In this study there are three groups. Sedentary (N=16), endurance athletes (N=8) and strength athletes (N=8). An acute psychological stress is inflicted with a randomized test consisting of mental arithmetic and Stroop's color-word test. In the answer sheet of the test there is also a discontinuity so that person will lost the flow and encounter another surprising situation. These results are then compared against the other phases of the test and between the different groups.

Body composition is different between the groups which was expected. Cortisol hormone was on a higher level in the baseline for the athletes compared to sedentary group. Also athletes reacted differently from cortisol point of view; endurance athletes presented an ascendant trend and strength athletes descendent trend although no statistically significant difference was found. Skin's conductivity was on a higher level, for the sedentary group compared to the athletes ( $p < 0.05$ ), during the whole test. Systolic blood pressure decreases

during the test for the sedentary group and for the strength athletes the recovery level is higher than the baseline for systolic blood pressure.

Test subjects reported to have experienced psychological stress during the study and this can be seen in the values of the biomarkers. We can then argue that the psychological stress was inflicted to the subjects. We did also see that people with different training background had different kind of reactions to the psychological stress for related biomarkers. We can then conclude that people with different training backgrounds react differently to psychological stress.

Key words: stress, endurance training, strength training, biomarkers, psychological stress

## **ABBREVIATIONS**

ATP	Adenosine triphosphate
BP	Blood Pressure
CNS	Central Nervous System
CO	Cardiac Output
CSA	Cross-Stressor Adaptation
ECG	Electrocardiography
EDA	Electrodermal Activity
EPI	Epinephrine
HPA	Hypothalamus-Pituitary-Adrenal cortex axis
HR	Heart Rate
HRV	Heart Rate Variability
NE	Norepinephrine
PNS	Parasympathetic Nervous System
PSS	Perceived Stress Scale
SIVAQ	Single-item Question on Leisure-time Vigorous Physical Activity
SNS	Sympathetic Nervous System
SA	Sympathoadrenal
SV	Stroke Volume
TPR	Total Peripheral Resistance

# CONTENT

1 INTRODUCTION .....	4
2 HUMAN PHYSIOLOGY .....	6
2.1 Nervous System .....	6
2.1.1 Sympathetic Nervous System (SNS) .....	8
2.1.2 Parasympathetic Nervous System (PNS) .....	8
2.2 Endocrine System.....	8
3 STRESS.....	10
3.1 Stressors.....	12
3.2 Adaptation.....	13
3.3 Acute Stress .....	14
3.4 Chronic Stress .....	14
3.5 Physical Stress.....	16
3.6 Psychological Stress .....	16
3.7 Physiology of Stress .....	17
3.7.1 Hormones .....	18
3.7.2 Nervous system .....	19
3.8 Measure Methods of Stress.....	21
3.8.1 Autonomic Nervous System .....	21
3.8.2 Endocrine system.....	24
3.8.3 Other Methods.....	24
3.9 Diseases Related to Chronic Stress .....	25

4	PHYSICAL ACTIVITY AND STRESS.....	27
4.1	Resistance Training.....	27
4.2	Aerobic Exercise.....	28
4.3	Effects of Stress to Exercise.....	29
4.4	Cross-stressor Adaptation Hypothesis.....	30
5	RESISTANCE TRAINING.....	32
5.1	Hormonal Adaptation.....	34
5.2	Nervous System Adaptation.....	35
5.3	Other adaptations.....	35
6	AIM OF THE STUDY.....	37
7	METHODS.....	39
7.1	Participants.....	39
7.2	Measurements.....	40
7.3	Processing of the data.....	44
7.4	Statistical Analyses.....	47
8	RESULTS.....	49
8.1	Anthropometry and Stress Questionnaire Results.....	49
8.2	Cortisol.....	50
8.2.1	Relative Change Between the Groups.....	51
8.2.2	Absolute Change.....	52
8.3	Electrodermal Activity.....	52
8.3.1	Relative change.....	53
8.3.2	Absolute Change.....	54
8.4	Heart Rate.....	54



8.4.1	Relative change .....	55
8.4.2	Absolute Change .....	55
8.5	Heart Rate Variation.....	56
8.5.1	Relative change .....	57
8.5.2	Absolute Change .....	57
8.6	Blood Pressure .....	57
8.6.1	Systolic Blood Pressure .....	57
8.6.2	Diastolic Blood Pressure .....	59
8.7	Breathing Frequency .....	59
8.7.1	Relative change .....	60
8.7.2	Absolute Change .....	60
8.8	Summary of Statistically Significant Differences in Biomarkers .....	61
9	DISCUSSION.....	62
9.1	Differences Between Endurance and Strength Athletes.....	63
9.2	Different Reactions for Endurance and Strength Athletes Compared to Non-athletes	64
9.3	Conclusion .....	65
	REFERENCES .....	67

# 1 INTRODUCTION

Object stays at rest or continues with the same speed and direction unless there is some unbalanced force. This is the First Law of Motion by Isaac Newton. There is something similar in human body; human body wants to balance all the forces and this is called homeostasis. One example of homeostasis in human body would be how the body controls the pH of the blood. Blood has different kind of buffers that react when blood starts to turn acid or basic (McArdle et al. 2014, 298-300). Adaptation to different kind of stressors, like the aforementioned change in pH, is the key of life.

It is safe to say that humans have been dealing with stress since the dawn of time and will continue to do so. Simplification of stress reaction in humans can be described as the fight or flight reaction to a stressful situation. In this kind of reaction the body prepares to react to the situation at hand. Stressors have changed from the days when humans prepared to outrun a lion to the modern world of work related stress. Stress can be thought to be a disturbance in the homeostasis (Tsatsoulis & Fountoulakis 2006). Endocrine system and nervous systems react to acute stress to prepare the body. Hormone secreting axes sympathoadrenal (SA) and Hypothalamus-Pituitary-Adrenal cortex axis (HPA) activate resulting in secretion of epinephrine (EPI) and Norepinephrine (NE) from SA and cortisol from HPA. Sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) also activate (Huang et al. 2013). Acute stress can have positive results but chronic stress can suppress the immunology system and lead to many kinds of diseases (Esch et al. 2002). Role of stress has been greatly studied because of the positive and negative effects to human body.

Like any exercise, resistance exercise is considered to be a stressor for the body. It presents an unbalanced force to the body and body then adapts to it by developing. In the case of resistance training it can lead to more powerful and bigger muscles (McArdle et al. 2014, 531). Resistance training can lead to positive results for example in cardiovascular function and sensitivity to glucose and insulin (Kell et al. 2001).

Scope of this study is to examine the effect resistance training has to acute mental stress biomarkers. It is thought that stress coping can be developed by stressing the body in another ways e.g. resistance training can develop person's ability to cope with mental stress. This hypothesis is called Cross-Stressor Adaptation (CSA) (Klaperski et al. 2013). Most of the reviews in the area of positive exercise effects to stress management and reactivity are about aerobic exercise. This study aims to look from the resistance training point of view the positive effects the exercise can have on person's ability to cope with stress by looking at the markers related to stress. It is important to think about different methods to measure how body reacts to different kinds of stress situations therefore different methods for measuring the stress effects are reviewed and also different kind sources for stress. Test will be designed so that it is possible to come up with new knowledge regarding how resistance training affects acute psychology stress related biomarkers. This new information can be used to understand the mechanics of stress coping that can be enhanced with resistance training.

## **2 HUMAN PHYSIOLOGY**

We are going to cover physiology around the essential areas for this study. We are going to look at the important parts of human physiology which are needed for understanding what happens for human body related to stress and physical activity: nervous system and endocrine system. Adaptive functions, which happen in the human body, are related to hormonal control that is of neural and endocrine source (Nienstedt et al. 2009 538). When looking at building of muscle mass (hypertrophy) the six important factors are: physical activity, nutritional status, genetics, environmental factors, nervous system activation and endocrine influences (McArdle et al. 2014 529). From these two sources we can identify that the important subjects to adaption in humans are nervous system and endocrine system. These two are the important ones to understand. In the later chapter we will notice that these two are also important when looking at stress. Level of details in these topics will not be highly detailed. We will be later looking at on a more detailed level when looking function of these related to stress and physical activity.

### **2.1 Nervous System**

Human nervous system can be divided into two parts: central nervous system (CNS) and peripheral nervous system. CNS is constructed out of brain and spinal cord (McArdle et al. 2014 384). Brain is the important organ of the CNS. Brain is the one that collects the information and then process the information received. (Nienstedt et al. 2009 516). Peripheral nervous system is made out of nerves, which are the channels for information transportation in the nervous system. (McArdle et al. 2014 384). We are going to focus on to the anatomy of the nervous system because we presume that the outcome of the nervous system in this thesis is important.

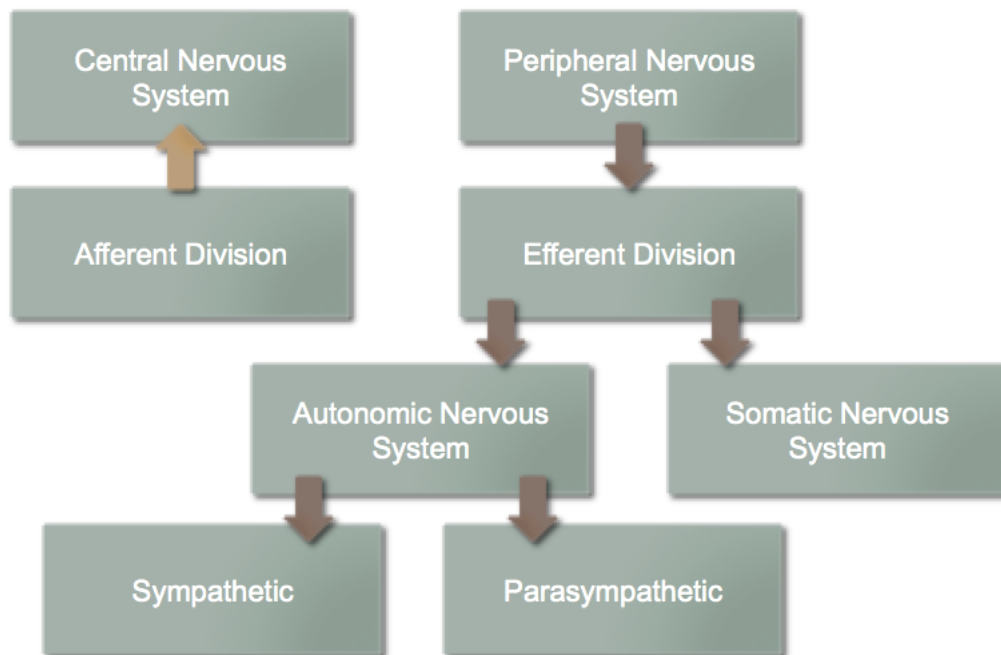


FIGURE 1. It is demonstrated here how the essential parts of human nervous systems are constructed and related to each other. CNS gets information from the sensory system called afferent division. CNS is built from brain and spinal cord. Peripheral nervous system is the pathway to control actions of the human body. Peripheral nervous system is made of cranial nerves III-XII and spinal nerves. Through the efferent division, peripheral nervous system controls the voluntary movement through somatic nervous system like movement of skeletal muscles. Through the autonomic nervous system the peripheral nervous system commands the involuntary actions and it is divided to sympathetic and parasympathetic divisions. Adapted from McArdle et al. 2014 385.

We need to take a closer look to sympathetic and parasympathetic divisions as they will be later in this thesis identified to be important factors in stress related situations. General distinction between sympathetic and parasympathetic divisions is that sympathetic is using the energy resources of human body and parasympathetic is storing energy (Nienstedt et al. 2009 516). Of course not all the situations are like this for example in sexual arousal both divisions are activated at once (Porges 1995).

### **2.1.1 Sympathetic Nervous System (SNS)**

SNS is activated in the basic fight or flight situation as the body prepares to a crisis and energy supply to the critical places needs to be secured and blood flow to the correct areas. For example the digestion system isn't critical in the acute crisis so the blood flow to there is reduced by SNS and at the same time the blood flow to the active skeletal muscles is increased. (Nienstedt et al. 2009 541-542). Examples of SNS activation are: dilated pupils, accelerated heart rate (HR) and secretion of EPI and NE (McArdle et al. 2014 332).

### **2.1.2 Parasympathetic Nervous System (PNS)**

PNS is important for recovery as it is considered to be anabolic and restoring body's energy stores. As SNS and PNS are usually reciprocally activated the activation of SNS decreases the activity level of PNS (Porges 1995; Vrijkotte et al. 2000). Heart rate variability (HRV) is linked to PNS activity (Task Force 1996). Heart rate variability is the variation between heart beats and is often used to measure stress reactions as a reduction in PNS function (Clays et al 2011). Sleeping has been studied in relation to PNS activity, stress reaction and recovery (Hynynen et al. 2011b; Pichot et al. 2002). Examples of PNS activation are: constricted pupils, slower HR and release of insulin and digestive enzymes (McArdle et al. 2014 332).

## **2.2 Endocrine System**

Endocrine system is a collection of organs in humans, which secrete hormones. These hormones then transport inside of human body and have different results like alter cell membrane permeability, stimulate fat synthesis and activate enzyme systems. Hormones have target organs, which have receptors that recognize the hormones and cause an action in the organ itself. In human there are ten primary hormones secreting organs: hypothalamus, pineal gland, pituitary gland, thyroid gland, parathyroid gland, thymus gland, adrenal gland,

pancreas, ovary (females) and testis (males). In contrast there are exocrine glands; for example sweat glands. (McArdle et al. 2014 408). There are other hormones secreting organs but they are not usually counted as part of endocrine system if they have other more typical function for example stomach (Nienstedt et al. 2009 368).

Interesting findings are that muscle has been identified to be an endocrine organ. This field of study is fairly new so lots of discoveries are still being made. Myokines is the general term for proteins and cytokines secreted by skeletal muscles. (Schnyder & Handschin 2015; McArdle et al. 439) and chronic training can lead to increase of these myokines (Kanzleiter et al. 2014). Myokines have been reported to have an impact on: inhibition of osteoblasts, inhibition on mammary cancer cell growth, increased GLP1 secretion, suppression on colon tumorigenesis, increased lipolysis, increased glucose uptake, increased energy expenditure, hypertrophy, increased thermogenesis and  $\beta$ -oxidation. (Schnyder & Handschin 2015).

### 3 STRESS

For understanding the concept of stress, it is a good idea to look through the evolution of the term itself in scientific research. We are not going to look into the psychology area of stress as it is out of the scope of this study. It is thought that the pioneer of the stress related studies is Walter Cannon. Cannon in 1932 published his theories in which the stress is a survival reaction. This is activated by pressure from the environment to the person, which means the urge to fight or flight the situation at hand. Where Cannon made the conceptual framework for stress, Hans Selye then later developed endocrine reaction to stress. (Toppinen-Tanner & Ahola 2012 22). Selye (1950) made the general adaptation syndrome theory in which he concluded three stages to stress: alarm phase in which the resources are made available for the body, resistance phase in which the stressor is being fought and exhaustion which occurs if the resistance isn't successful at removing the stressor's effect. Selye wrote that reaction to stress was an automated process. This has been later been discussed and considered to be misleading in situation where the stressor itself is smaller and the way the person evaluates the level of stress is important. McEwen (1998) conceptualized the allostatic load. In the figure 2 is the concept by McEwen of how the human body perceives stress and then the development of allostatic load. Allostasis means that homeostasis is changed and adaptation to it has happened. It can be also a case in which the adaptation doesn't happen and one can't recover from the stressor; in this kind of situation the allostatic load is developed. There are lot of issues behind which contribute to how one can handle the stress, these are individual differences in genes and experience. These factors lead to how one perceives the stress, so how great is the impact and then how the physiologic response happens. In this study we will be interested about the individual differences, which affect the physiologic responses.



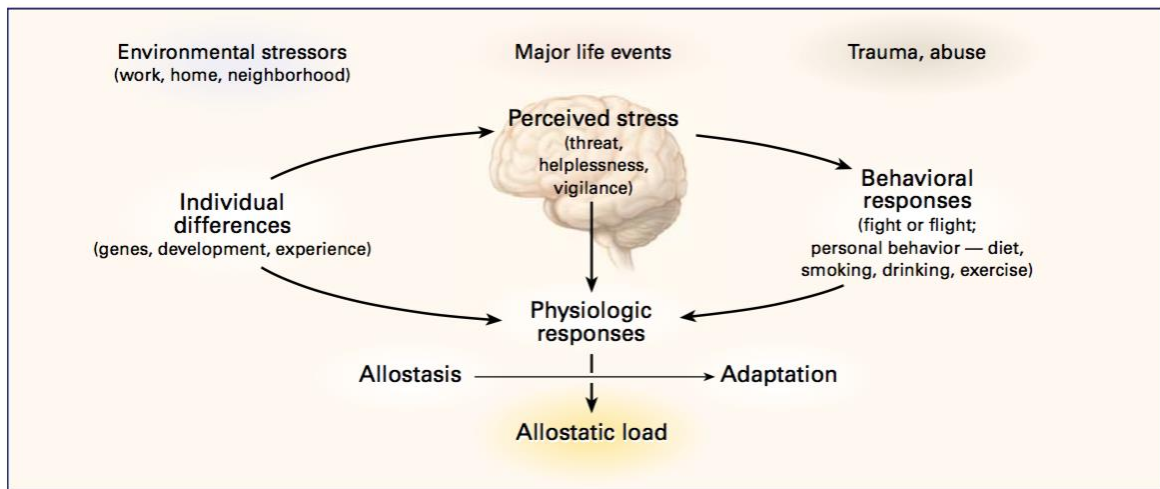


FIGURE 2. The Stress Response and Development of Allostatic Load (McEwen 1998).

Another way to look at stress is the systems science point of view in which the stress is seen as state different than the optimal attractor location (Oken et al. 2015). In figure 3. The concept of aforementioned is explained. This visualizes how stress tolerance can work for individual persons. Interesting is understand what causes the depth of the basin.

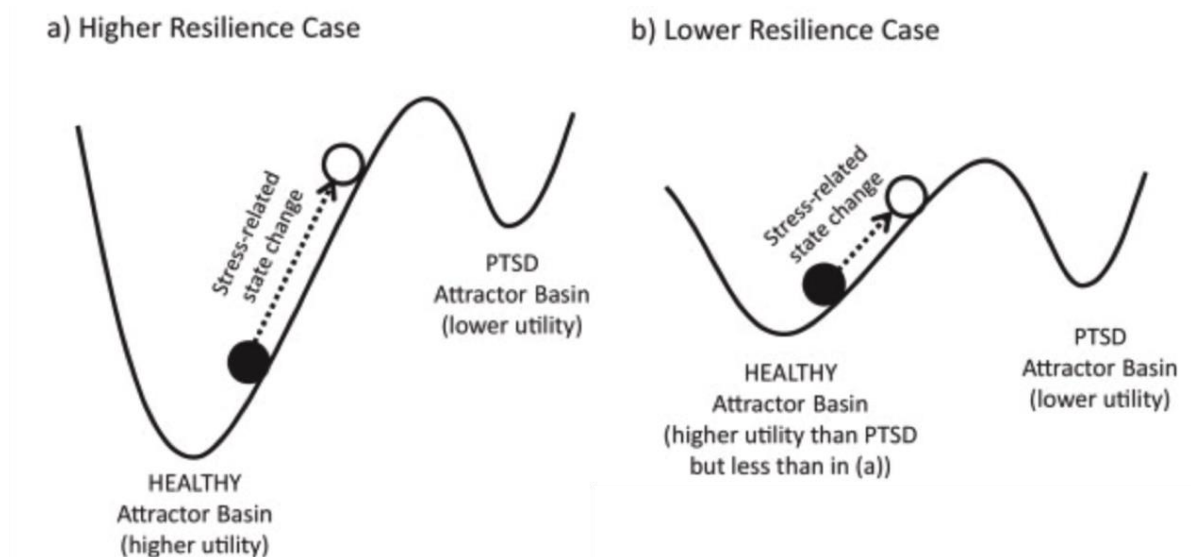


FIGURE 3. An example of physiological situation of a human to explain terminology; attractor basin, utility and resilience. Higher utility is down. Stressor can move the situation towards a lower attractor basin utility and the resilience determines how easily the shift can be made. (Oken et al. 2015)

Traditionally stress has been defined based on the stressors, impact or methods for recovery from the negative effects (Kettunen 2015). Impacts can be measured as physiological changes in human body like increased HR (Toppinen-Tanner & Ahola 2012 12). From theoretical point of view, we will be covering stress from the point of view of physiologic responses related to the source, duration, physiology, measuring and diseases. From systems science viewpoint the resilience is something that we are interested as we speculate that resistance training would be the source for resilience.

### **3.1 Stressors**

Human body can encounter many kinds of stressors. In definition stressor is the source of stress. Stressor can be physical like heat or psychological like giving a public speech. (Chun-Jung et al. 2013; Tsatsoulis & Fountoulakis 2006). In a stressful situation the human body recognizes that the situation is something that can't be handled with normal actions. Resources at hand are mobilized so that they can be used in the situation and body prepares to fight or flight the situation (Toppinen-Tanner & Ahola 2012 12).

Although in general the stress reactivity to physical or psychological stress looks to be similar in acute situations from physiology point of view e.g. elevated HR and blood pressure (BP) also activation of SA (Forcier et al. 2006; Chun-Jung et al. 2013; McArdle et al. 2014 323). Physical and psychological stressors can be shown differently in the body, differences can be found for example in salivary cortisol levels, HR and HRV (Hynynen 2011a). This should be taken into consideration then when preparing the measuring methods for this study.

### 3.2 Adaptation

One of the most common adaptation theories related to stress is the training adaptation theory. In the figure 4 is the common model for training adaptation for endurance sports. After a training, which can be considered a stressor, the level is higher after the recovery than it was prior to training. Body is adapting to the stressor. This is an example of a physical stress adaptation.

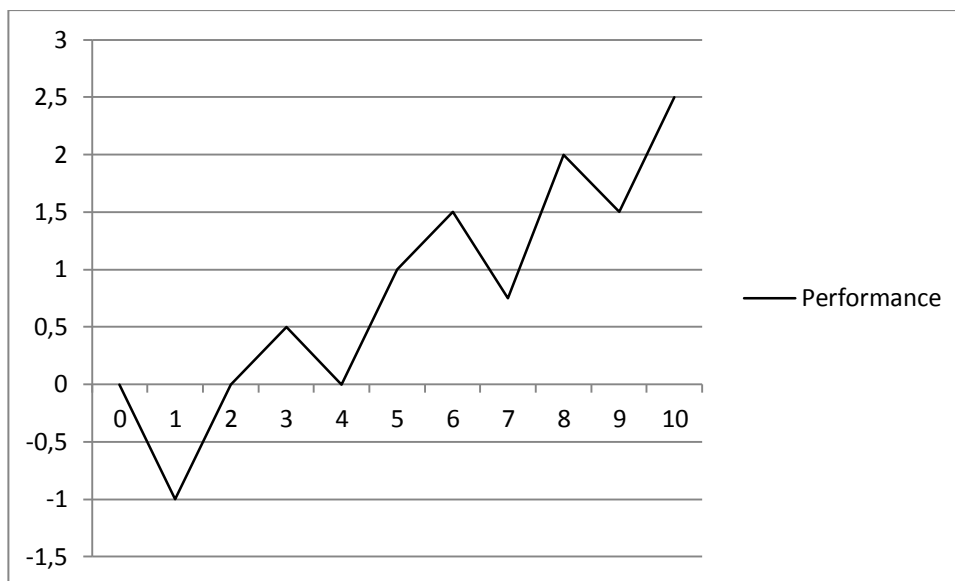


FIGURE 4. Training adaptation model. Development progress steadily as the body is stressed and then after the recovery the level of performance is higher than prior to the training. Modified from Kreider et al. (1998 10)

Like other adaptations in human body there can be also identified outline responders in stress adaptation. In a study public speaking was used as a mental stressor and cortisol levels were measured after the speech. There was a group whose cortisol level did not decrease after making more speeches and a group whose cortisol levels decreased after giving speeches, which can be thought to be a sign of adaptations. (Kirschbaum et al. 1995).

This would be a mental stress adaptation example. We can see from this that it doesn't have to be a physical stressor, which can cause adaptation.

### **3.3 Acute Stress**

Acute stress is a situation when the stressor happens only for a short period of time. Widely accepted theory of this is the fight or flight response in which the cardiovascular function is elevated and EPI, NE and cortisol are secreted, HR and BP are increased (Janset et al. 1995; Huang et al. 2013). Body is then prepared to either to fight the situation at hand or flight from the scheme and avoiding the stressor. In the modern days the stressor for acute stress can be more psychological when compared to the era when humans were in danger of being eaten by wild animals. These days humans can be more afraid about being eaten alive by their amount of work.

In resistance training happens an acute stress reaction and adaptation to it. During the resistance training BP and HR are elevated and cortisol is secreted as reaction to stressor. After the training the protein synthesis is started so that adaptation can happen (McArdle et al. 318-322; Damas et al. 2015; Hakkinen & Pakarinen 1991). In psychological stress situation the response to acute situation is similar to acute physical response: SAM response, release of NE and EPI, elevation of BP and HR (Huang et al. 2013). As discussed earlier the acute stress can lead to adaptation or in the situation that the recovery isn't achieved the acute stress can lead in the situation of allostatic load which can be then turned to chronic stress.

### **3.4 Chronic Stress**

Chronic stress has been studied a lot in the work related stress environment. This is a good source to look for changes in the body when psychological stress has become chronic. Perhaps the reason for this is the lack of chronic physical stress. One source for chronic

physical stress would be then overtrained athletes. As a field of study the overtraining isn't that clear as the outcomes are mixed for example the HR can be lowered or increased (Kreider et al. 1998 3). Aforementioned is the reason why we won't be looking at overtraining in this thesis.

In work people are exposed to stress daily. Allostatic load is a concept in which person's recovery isn't enough and stress piles up and causes severe negative outcomes. (Stults-Kolehmainen & Sinha 2014; Ritvanen et al 2005). It is important to understand that this doesn't only happen in work life and mentally.

When psychological stress comes chronic the body is in an exhausted situation. One example of this is burnout in work life. Person feels that he or she is losing his or her energy and the workload comes unbearable. As the term chronic implies the changes don't happen quickly and also there really isn't a clear point in which the burnout can be said to happen. (Toppinen-Tanner & Ahola 2012 120-126.). When a person in chronic stress situation then experiences an acute stressor the outcome can be that the body overreacts to it (Pike et al. 1997) and also the recovery of the body is hindered (Stults-Kolehmainen et al. 2014).

Physical stress can also become chronic and an example of this is overtraining in athletes. When this happen the performance of the athlete is decreased and there are abnormalities in the HPA and SA (McArdle et al. 491). SNS and PNS also indicate several issues in overtraining situation. Disturbance in SNS can be seen as: poor sleep quality, resting HR is increased and also BP. PNS side also can be seen for example in: depression, apathia and surprisingly lower resting HR and BP. (Kreider et al. 1998 21.). Overtraining can be therefore quite tricky to measure with only physiological measurements as SNS and PNS can show conflicting results.

### **3.5 Physical Stress**

Most common way of looking at physical stress effects to humans is looking at athletes. Athletes are exposed constantly to physical stress because of their physical training to their own sport. One must realize that it just isn't about training adaptations. Humans who live in high altitudes have less oxygen in the air and therefore their body adapts to this by producing more red blood cells and also when encountering heat human's blood circulation is altered and sweating occurs so that human body can adapt to the stressor (Nienstedt et al. 2009 586 & 426-427). Human body's adaptation to heat is called acclimatization and it occurs in just few weeks (McArdle et al. 2014 633).

### **3.6 Psychological Stress**

Lazarus (1993) proposed that psychological stress could be defined by the so-called stress emotions: anger, anxiety, disgust, envy, guilt, jealousy, sadness and shame. Tsatsoulis & Fountoulakis (2006) also add professional and social sources for psychological stress. From these psychological stress sources we can say that psychological stress is caused by the internal factors. Of course the root cause is usually external like keeping a public speech but the changes in human body happen because of the internal emotions in context with the external situation.

Physical stress can be quite easily understood when compared to the more complex psychological stress. Consider a situation where one is exposed to heat for 30min and then a situation where a student needs to take a very hard test in few weeks. Physical stress situation here is well defined period of time where as the psychological stress isn't so clear; one can forget that the test is next week or one can have it in mind all the time and can't sleep.

What makes psychological stress also interesting is that the body reacts similar way than in physical stress: making the body alert and mobilizing energy but the energy isn't needed in same way as in physical stress situation as the energy expenditure isn't so much higher so the released energy can be stored as visceral fat. (Tsatsoulis & Fountoulakis 2006; Rosmond 2002). Obesity can then lead to mental diseases (Stunkard et al. 2003) and/or hypertension, heart diseases and diabetes (Doll et al. 2000).

In this study we are going to look the physiological biomarkers related to psychological stress. Physiological biomarkers to psychological stress are for example elevated EPI, NE and cortisol levels in humans (Huang et al. 2013). In later parts of this study we are going to look into more details with different kinds of methods to measure the physiological biomarkers of stress.

### **3.7 Physiology of Stress**

Scope of this study is to study how resistance training affects the biomarkers of psychology stress. Therefore it is needed to take a look at the physiological side of stress. Although there are different responses to psychological and physical stress (Hynynen 2011a), in this chapter we don't differentiate those. Understanding the differences is important in interpreting the results of the study. We will be looking at the stress hormones and then the function of automatic nervous system as these are considered to be the main players in stress functions (Tsatsoulis & Fountoulakis 2006; Klaperski et al. 2013; Oken et al. 2015). Again we need to make some compromises here in this study. We are leaving out the brain as an individual player from this study and brain plays a highly important role in the field of stress (Oken et al. 2015). This study focuses on the outcome of ANS and endocrine system in stress related situations.

### 3.7.1 Hormones

It has been widely studied that in stress reaction the HPA activates (Tsatsoulis & Fountoulakis 2006; Bartholomew et al. 2008; Pruessner et al. 1999). Natural source of hormones for interest of this study would be then the ones found in HPA activation. From the figure 5 we can identify three interesting hormones: cortisol, EPI and NE. These three hormones are the common targets of research in stress-reaction (Lundberg 2005; Armstrong & VanHeest 2002). Cortisol is considered to be the most common stress hormone (Björntorp 2001; Lundberg 2005). Cortisol affects the tissues showed in the table 1.

TABLE 1. Effect of cortisol to different tissues in human body Modified from McArdle et al. 2014 425.

<b>Many Tissues</b>	<b>Adipose tissue</b>
Inhibits glucose uptake	Stimulates adipose tissue synthesis
Inhibits amino acid uptake	
<b>Muscle tissue</b>	<b>Liver</b>
Stimulates protein breakdown	Stimulates gluconeogenesis
Inhibits protein synthesis	

Activation of SAM in stress reaction leads to secretion of EPI and NE which leads to changes in the heart beat as indicated in the figure 5. Prolactin is also sometimes used to indicate the level of perceived stress (Forcier et al. 2006) but the studies haven't been very consistent as there have been reported completely reciprocal results when it comes to prolactin levels in stress studies (Gerra 2001).

In stress situation the level of cortisol is increased as discussed earlier. Cortisol then starts the breakdown mode in human body causing to: breakdown of proteins to amino acids, triacylglycerol breakdown to glycerol and fatty acids. It then also suppresses the function of immune system and causes negative calcium balance. Cortisol acts as an insulin antagonist.



(McArdle et al. 2014 424; Nienstedt et al. 2009 403-405). From the outcome of cortisol it could be said that the body seems to prepare to an acute usage of energy. Also EPI and NE are causing the body to release energy from the storages to use; EPI and NE cause the liver to release more glucose to the blood stream. NE also enhances the blood circulation to the activated muscles and stimulates the release of energy from fat also and inhibits insulin function. (Nienstedt et al. 2009 407; Vuori et al. 2013 137).

### **3.7.2 Nervous system**

ANS is the system, which controls the homeostasis of human body (Porges 1995). Therefore in stressful situation the ANS is one of the components, which reacts to the situation and activates typically the HPA (McEwen 1998). In the figure 5 is represented the stress responses in human's focusing on SAM and HPA.

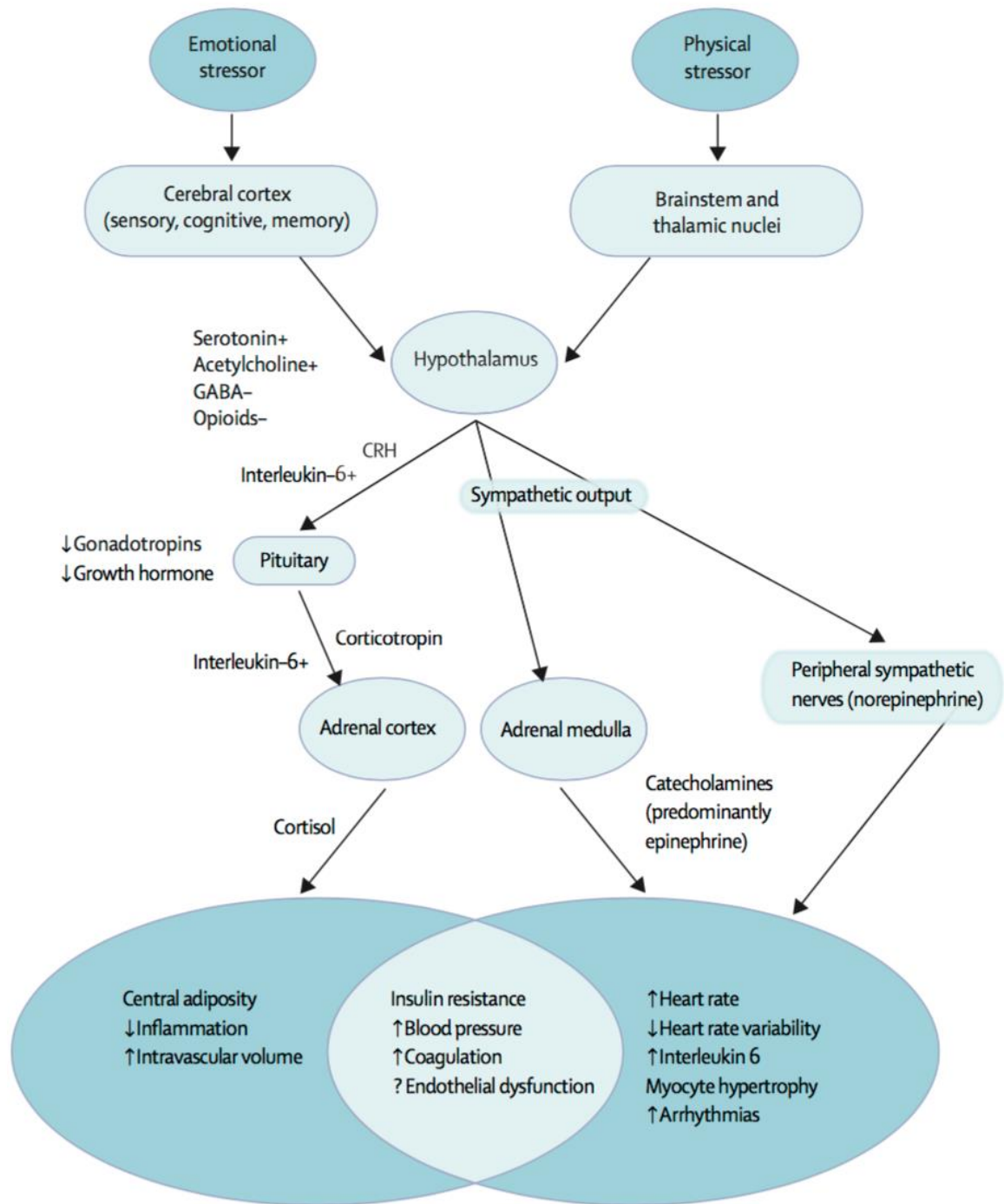


FIGURE 5. Following the stress effects of HPA, on the left, and SAM in humans. Overlapping responses are at the bottom in the middle (Brotman et al. 2007).

### **3.8 Measure Methods of Stress**

Stress has an effect on SNS, PNS, SA and HPA as discussed earlier. Therefore these are the main areas in this study when measuring the stress. When trying to understand a phenomenon it is important to measure it. Although the measurement itself isn't enough as interpretation of the data is also needed. Idea of looking through the different kind of measurement methods is to try to find measurement methods for the study at hand. Measure methods should be easy to use and feasible in a study on this level.

Like discussed earlier, allostatic load is the cost of allostasis which means that the body tries to adapt but can't do that and the stressful situation just continues. There has been a 10 point measurement for allostatic load: overnight 12h urinary cortisol, EPI, NE, both diastolic and systolic BP, waist to hip ratio, total cholesterol level compared to high density lipoproteins, high density lipoprotein cholesterol, glycosylated hemoglobin and DHEA-S (Seeman et al. 2001). From these markers we can see few ideas for the study for which markers to measure. There isn't though a clear agreed method for measuring the allostatic load from biomarkers. Allostatic load measurements can also be adjusted using different correlations like childhood poverty and work exhaustion measurement (Oken et al. 2015).

There are also a lot of different psychological methods for measuring the stress. In this thesis we are interested about physiological measurement of stress. Psychological tests, which could be used in this study, could be questionnaires to find out the level of current stress for the individuals in the study so that it can be considered in the interpretation of the results.

#### **3.8.1 Autonomic Nervous System**

Autonomic nervous system consists of SNS and PNS. SNS and PNS are responsible for the changes in HR, which is considered to be a good source to measure the activity of these

systems (Task Force 1996). SNS raises the heart rate and this is visible when measuring the HRV as higher SNS activity suppresses PNS and therefore the HRV is reduced. HRV is explained in the figure 7. BP is also controlled by autonomic nervous system. (Porges 1995). Both SNS and PNS affect the HR so heart rate is one of the good indicators of ANS functions. SNS increases the HR and PNS then decreases the HR (McArdle et al. 2014 332).

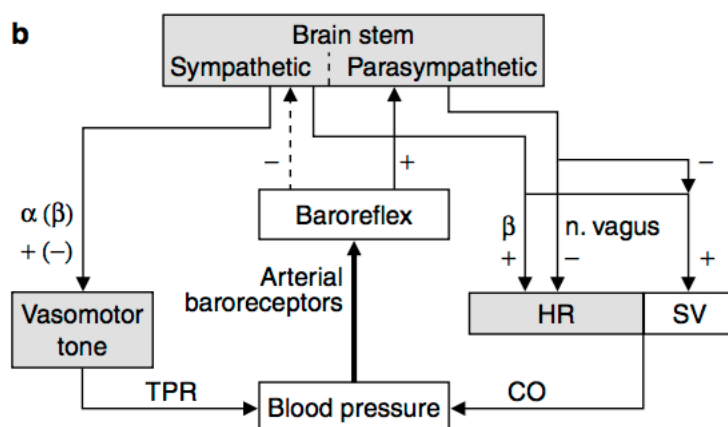


FIGURE 6. Simplification of how sympathetic and parasympathetic nervous systems influence to heart rate (HR), stroke volume (SV), cardiac output (CO) and total peripheral resistance (TPR) (Aubert et al. 2003).

In the figure 6 is shown how the SNS and PNS work together in influencing the HR, stroke volume (SV) and BP. It can be seen from the figure that the concept is little more complex than just SNS exciting the HR and PNS inhibiting the HR. Nervous vagus is the most important nerve for controlling the heart rate via PNS (Porges 1995). Parasympathetic neurons influence the sinus discharge in the heart. Parasympathetic neurons secrete acetylcholine. (McArdle et al. 2014 330).

HRV is the variability in the intervals between heartbeats. This variation is considered to be because of vagal tone and therefore PNS is considered to be active. Elevated HR is regulated by the SNS. (Task Force 1996). These days HRV is quite feasible source of data

so it has been used in studies (Hynynen et al. 2011b, Aubert et al. 2003, Henelius et al. 2014)

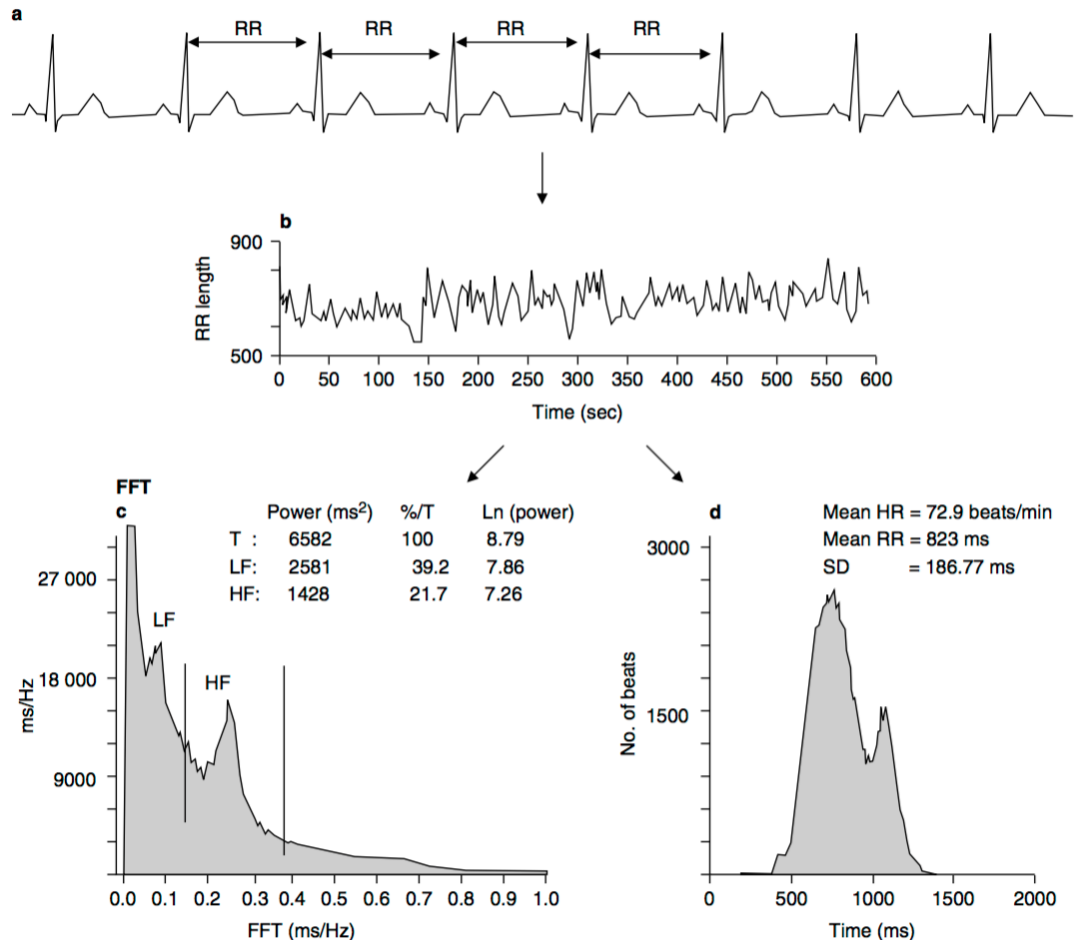


FIGURE 7. HRV measurement starts with measuring of electrocardiography (ECG), example of this is the part a. From ECG are the intervals between Rs calculated and put to tachogram, part b, and finally the tachogram is analyzed based on the frequency or time domain, which are parts c and d in, respectively. (Aubert et al. 2003)

Activation of SNS also increases the electrodermal activity (EDA) and measurement of EDA can be used as a method to measure the activation of SNS in stress related situations (Taylor et al. 2015). Other possibilities measuring the SNS would be the pupil dilation

(McArdle et al. 2014 332) and breathing frequency (Toppinen-Tanner & Ahola 2012 23). From different options it is needed to choose the practical solutions to be used in this study.

### **3.8.2 Endocrine system**

SA and HPA are both hormones regulating systems so the hormone levels of EPI, NE and cortisol are of interests when measuring stress (Chun-Jung et al. 2013; Kraama 2013). Usually hormones are measured from blood but it can be sometimes not feasible. Persons can even react to the upcoming blood testing so that the blood test itself can cause stress reaction in humans. It is possible to measure cortisol from saliva and also NE can be indirectly measured from saliva as  $\alpha$ -amylase is linked to changes of NE levels and chromogranin-A to NE and EPI. (Noto et al. 2005)

### **3.8.3 Other Methods**

As humans are highly complicated systems there are other factors also that could be taken into consideration when looking at how stress can be measured from physiological point of view. Some of these methods are related to previously mentioned endocrine system and ANS but can be looked at their own area of interest. Sleep has been studied greatly in association with stress (Maina et al. 2009; Toppinen-Tanner & Ahola 2012 84; Henelius et al. 2014). Also it has been studied that the brain itself can go through concrete structural changes (Gianaros et al. 2007; Vyas et al. 2002; Liston et al. 2006). Immune system has also been noted to be affected by stress (Clow & Hucklebridge 2001; Perna et al. 1997; Pike et al. 1997). Chronic stress has an effect to cellular and humorous measures linked to immune system (Segerstrom & Miller 2004; Gleeson 2006 140). As stress is widely studied subject there are of course other less frequently used methods to measure the level of stress including: lactic acid, myocardial oxygen consumption, cardiac index, preejection period, left ventricular ejection time, R-wave amplitude, pulse transit time, pulse volume, respiratory sinus arrhythmias, vascular conductance, finger temperature, frontalis or trapezius muscle tension, forearm blood flow and stroke volume, cardiac output (CO) and

total peripheral resistance (TPR). (Forcier et al. 2006). As these are not very often used the question arises if these are feasible as there isn't a lot of scientific data to back these up.

### 3.9 Diseases Related to Chronic Stress

One way of looking at the topic of this study is to look from another point of view. When looking into what kind of diseases stress can cause, we can perhaps learn something new from the factors affecting stress. Acute stress can actually enhance the immune system (Esch et al. 2002). This is understandable because acute stress reaction is meant to prepare the body for the situation at hand. Chronic stress can then cause lot of diseases as the unnatural situation of stress continues and body isn't meant to be in constant stress mode. In the figure 8 is explained the how stress can raise the BP.

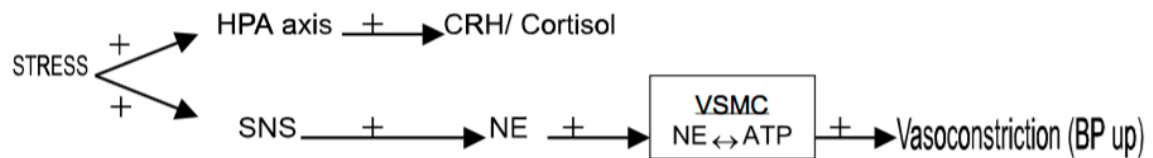


FIGURE 8. Stress activates the SNS that leads to secretion of NE. NE then interacts in vascular smooth muscle cell (VSMC) with Adenosine triphosphate (ATP) and causes vasoconstriction. Vasoconstriction then leads to an elevated BP. (Esch et al. 2002).

Stress can be the cause of many mental disorders like depression and anxiety (WHO 2015; Twenge 2000). Physical activity can help to overcome these disorders (Tsatsoulis & Fountoulakis 2006; Ströhle 2009). Chronic stress increases the level of cortisol in the blood (Bartholomew et al. 2008). Elevated cortisol levels can then cause increased glucose production in the liver, inhibition of glycogen synthase in the skeletal muscle, increased lipid accumulation in adipose tissue and decreased insulin secretion in pancreas. Decreased insulin levels can then lead to Type 2 Diabetes. (Rosmond 2002). Also increased lipid accumulation in adipose tissue can then lead to obesity and obesity is linked to many

diseases like metabolic syndrome and an increased risk to stroke, coronary heart disease and type 2 diabetes (McArdle et al. 2014 433).

It seems that many of the diseases linked to stress are kind of diseases that physical activity has a positive effect. This could be used to argue that physical activity makes the body adapt to stress. What should be kept in mind is that does the stress actually cause inactivity that then leads to stress related diseases so it is actually stress-related inactivity that is the cause of these diseases and also the obesity that becomes from inactive way of life which can also be linked to unhealthy way of living like overeating, smoking and too much usage of alcohol. (Toppinen-Tanner & Ahola 2012 132-133). Causality is an important thing to be able to prove when making stress disease related studies.



## **4 PHYSICAL ACTIVITY AND STRESS**

There are lots of studies, which conclude that physical activity has a positive effect towards stress (Spalding et al. 2004; Crews & Landers 1987; Aldana et al.1996). It has been also shown that there are differences between resistance and aerobic exercise when looking from stress measurement point of view (Bretland & Thorsteinsson 2015). We are going to cover different kind of physical activities, resistance and aerobic, in this chapter and also how does stress affect exercising in general. We will conclude this chapter with looking at cross-stressor adaptation hypothesis in which stress coping abilities could be enhanced through a different kind of stressor; stressor would be in this case physical activity.

### **4.1 Resistance Training**

It is hard to find which are clearly made out of resistance trainings effects to psychological stress from the physiological biomarkers point of view as most of the studies are made about general physical activity or then aerobic exercise like jogging. One way to look at the benefits of resistance training is in general to see that resistance training as effective as aerobic exercise when looking at mortality (Cooper et al. 2010). This can be thought to say that in general there is something in resistance training that benefits the human body regarding survival and stress is pretty much always part of human life. One big physical stressor for human body could be thought to be disease or a surgical operation. It has been studied that muscle mass can decrease the mortality in surgical operation (Weisj et al. 2014;) or survival of cancer (Baracos & Kazemi-Bajestani 2013). These are concrete examples about situation in which the physical stress is being tolerated better regarding the muscle mass.

One interesting new studied concept is that muscle mass affects to kynurenine metabolism and therefore can prevent stress related depression (Agudelo et al. 2014). We could then argue that hypertrophy can be linked directly to a better coping with stress.

Indirectly resistance training could be thought to increase stress coping with social support (Stephens 2000; Dodge et al. 2012). Social support can therefore be seen as a resource to be utilized to handle stress and resistance training can be seen as a social activity also.

## **4.2 Aerobic Exercise**

As the aerobic exercise isn't the main field of study in this thesis, we are not going to look very deeply to the changes that the aerobic exercise has on the body. However it is important to know the basic adaptations the body has to the aerobic exercise so the changes which resistance training is having can be distinguished from other forms of exercise. We consider this kind of training to be typically endurance for example long distance running.

Adaptations that occur in the body after regular endurance training are: neuromuscular, metabolic, cardiovascular, respiratory and endocrine system (Jones & Carter 2000). Neuromuscular benefits are linked to the control of muscles usage during activity, metabolic ability can be developed by the increase of the amount of the mitochondria in the muscles, cardiovascular benefits are improved blood circulation and strengthening of the heart muscle, respiratory improvements are from the adaptations of the ventilation muscles and endocrine system is more efficient when less endocrine involving is needed for the performance on the same level as before the adaptation (McArdle et al. 2014 464-475; Jones & Carter 2000).

On the neural side the endurance training causes restructuring of dendrites, enhance in protein synthesis, an increase in protein's axonal transport, better neuromuscular movement and adaptations in electrophysiological features (Gardiner 2006; Gardiner et al. 2006). These are similar kind of adaptations than in resistance training (Enoka 2008 368).

One study made by Norris et al. (1990) studied an aerobic and anaerobic exercise group's effect on stress markers. It was concluded that especially aerobic exercise group resulted in better responses to psychological stress. In that study measurement weren't made so much on the physiological side, as only HR and BP were recorded. A study made by Tsutsumi et al. (1997) showed that resistance training had improvements in general mood and anxiety. Also general improvement in stress related to questionnaires results have been linked with increase of strength (Hicks et al. 2003).

### **4.3 Effects of Stress to Exercise**

It could be also worthwhile of considering looking from the other side the problem at hand. Instead of thinking how physical activity can influence the ability to cope with stress, it could be beneficial to understand how stress can influence the ability to exercise. According to several studies stress reduces people's physical activity level and can lead to negative behavior like unhealthy eating, drinking more alcohol and smoking (Stults-Kolehmainen & Sinha 2014). It is not only negative behavior that can come from stressful life. Study also suggests that the actual strength results from the training can be reduced when experiencing a stressful life (Bartholomew et al. 2008). Also it can influence the quality of recovery from training leading to need of longer recovery periods (Stults-Kolehmainen et al. 2014).

Important would be to understand why stress hinders the gains of strength training and why the recovery from training is deteriorated because these two elements are the main factors in strength training: exercise and recovery. In the studies the reasons for longer recovery period and impaired strength gains are explained through the effect of stress to the body. Chronic stress can lead to unhealthy behavior like discussed earlier, which of course can make the recovery from exercise worse, but it can also be that the immunology system is not working properly in the situation of chronic stress, which then leads to lower quality of body's recovery from training (Stults-Kolehmainen et al. 2014). Strength gains can be lower because of several hormonal changes, which the chronic stress can initiate: increase in EPI and cortisol (Bartholomew et al. 2008). Our attempt to learn from this looking of the

problem from the other side doesn't really present us anything new regarding this study. As the factors, causing negative impacts on strength training, speculated by the authors of the articles, are only just reversed from stress' negative changes in the homeostasis.

#### **4.4 Cross-stressor Adaptation Hypothesis**

It has been studied that exercise has positive effect on people's ability to cope with stress (Schnohr 2005; Nguyen-Michel et al 2006). It is known therefore that physically active people's ability to perceive stress is better. Big question is the mechanism how the physical activity then increases the experience of stress and the ability to cope with stress.

Theory of CSA is based on the exercise induced biological changes on the HPA and SNS, which then leads lower response in other kinds of stressors than exercise. Key here is that the stress reactivity is smaller and recovery period is faster. (Klaperski et al. 2013). Comparing of exercised person to sedentary counterparts using stress related metrics can help us to understand what kind of adaptations can happen in stress reactivity (Sothmann 1996). Interesting fact is discussed earlier which stated that resistance training can actually increase the sensitivity to hormones.

One engaging finding is that HPA hormones are on a higher level on the exercised group, when compared to sedentary group after exercise. Looking at HPA hormones on the same absolute level of exercise, the hormone levels are lower on the exercised group than the sedentary group but when the level is relative then the hormone levels are the same or even higher on the exercised group. (Sothmann 1996.). This is visualized in the table 2. In this study it would be one intriguing result to see also when comparing the resistance trained individuals to endurance trained.

TABLE 2. HPA hormones compared with two different groups.

	<b>Exercised</b>	<b>Sedentary</b>
<b>Recovery</b>	Higher	Lower
<b>Absolute</b>	Lower	Higher
<b>Relative</b>	Higher or same	Same or lower

Aforementioned HPA hormone level finding suggests that the body itself doesn't secrete less HPA hormones when in stress on a trained person but suggests that the tolerance of the hormones is better. An interesting concept here to look at is overtraining. In overtraining situation the cortisol levels are also elevated but then again the response to this blunted (Kreider et al. 1998 7, 159). Perhaps the key here is not the amount of the hormones but the sensitivity to them. The reaction to hormones tells how well the body is adapted to stressful situations.

One interesting concept from resistance training is cross-education. In cross-education it has been shown that if person trains only one limb the other limb will also become stronger even without physical activity (Hortobágyi 2005; Lee & Carroll 2007). This could be also thought to be a sign of concept in which body can become stronger on one part when another part is trained.

It has been studied that posture itself affects the level of cortisol secreted as posture and muscle tone are part of the preparatory set for a response to a stressor (Payne & Crane-Godreau 2015). It could be used to argue that resistance training which leads to a better posture could therefore influence the cortisol response. Anxiety is closely related to stress as a mental health issue. It has been studied to show that resistance training has anxiolytic effects. (Strickland & Smith 2014). There is therefore a clear link with resistance training and mental issues.

## 5 RESISTANCE TRAINING

One way to define resistance training is that it is repeated voluntary muscle contractions made against a resistance bigger than normally encountered in the daily activities (Lee & Carroll 2007). Resistance training is considered in this study to mean using resistance to produce muscle contraction, which leads to the adaptation of the body, and increasing strength levels so that the maximum produced force increases. In this chapter we are looking the key adaptations that happen in human body after resistance training. We will look first the general adaptations towards resistance training, which are listed in the table 3. Then we will take a closer look at hormonal and nervous system level.

TABLE 3. Physiologic adaptations to resistance training. Adapted from McArdle et al. (2014) 531, originally Fleck & Kraemer 1988.

<u>System/Variable</u>	<u>Response</u>	<u>System/Variable</u>	<u>Response</u>
<b>Muscle fibers</b>		<b>Intramuscular fuel stores</b>	
Number	Equivocal	Adenosine triphosphate	Increase
Size	Increase	Phosphocreatine	Increase
Type	Unknown	Glycogen	Increase
Strength	Increase	Triglycerides	Not known
<b>Mitochondria</b>		<b>Aerobic capacity</b>	
Volume	Decrease	Circuit resistance training	Increase
Density	Decrease	Standard resistance training	No change
<b>Twitch contraction time</b>	Decrease	<b>Connective tissue</b>	
<b>Enzymes</b>		Ligament strength	Increase
Creatine phosphokinase	Increase	Tendon strength	Increase
Myokinase	Increase	Collagen content of muscle	No change
<b>Enzymes of glycolysis</b>		<b>Body composition</b>	
Phosphofructokinase	Increase	Percent body fat	Decrease
Lactate dehydrogenase	No change	Lean body mass	Increase
<b>Aerobic metabolism enzymes</b>		<b>Bone</b>	
Carbohydrate	Increase	Mineral content and density	Increase
Triglyceride	Not known	Cross-sectional area	Increase
<b>Basal metabolism</b>	Increase		

In the beginning of resistance training the first adaptations happen on the neural side and after the neural adaptation the strength increase comes from hypertrophy (McArdle et al. 2015 529; Lieber & Fridén 2000; Lee & Carroll 2007). Adaptations happen on the neural side and this will be interesting to see if it has an effect on the neural side of the stress reactions. In the figure 9 is illustrated a general response of strength training.

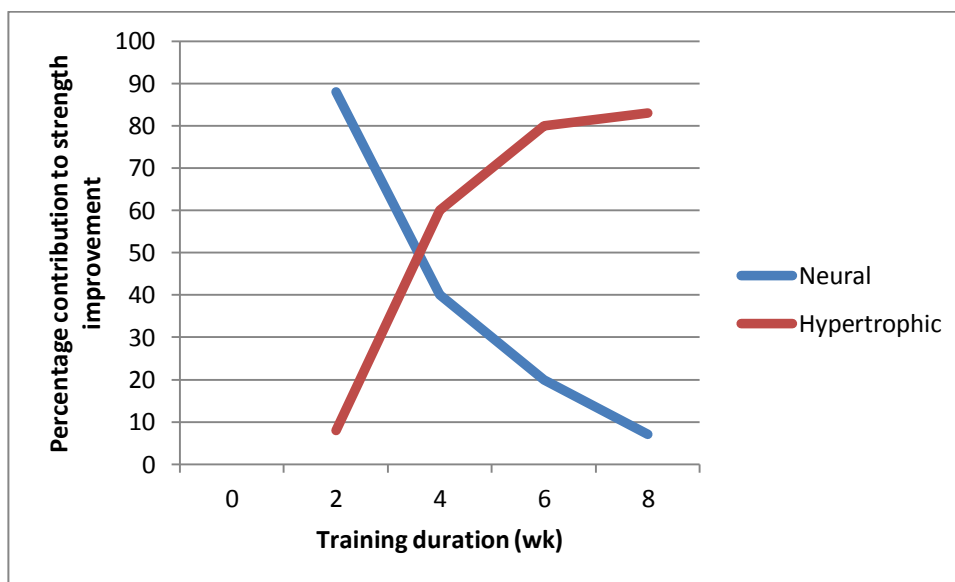


FIGURE 9. Typical figure for strength increase factors during strength training; first it is about neural adaptation and then hypertrophy. Adapted from McArdle et al. (2014 530)

Resistance training can lead to many beneficial outcomes relating one's health. Resistance training has been linked to reduced heart rate, increased stroke volume of the heart during exercise and rest, reduced blood pressure, positive lipids profile, glucose and insulin sensitivity and insulin response. It is hard to say if the benefits are solely from resistance exercise or is the higher lean body mass the key issue or the better nutrition intake compared to the control group in the studies. (Kell et al. 2001). Understanding the underlying changes that lead to these positive changes in the body might help us to understand better how resistance training is making the body better.

## 5.1 Hormonal Adaptation

On the hormone levels strength training has been linked to increased fast serum of cortisol levels (Campbell et al. 1994; Hakkinen & Pakarinen 1991), ratio of testosterone/estradiol (Kalman et al. 2007) and testosterone (McArdle et al. 2014 447). However there are studies in which the systematic changes haven't been recorded for total and free testosterone (Kraemer & Ratamess 2005; Hakkinen et al. 2000), GH (Kraemer & Ratamess 2005; Wideman et al. 2002), DHEA, DHEAS, cortisol, or SHBG (Hakkinen et al. 2000). Perhaps it isn't important to look what has changed on the datum level so much as on the secretion and other interactions on hormone levels. There are other changes in endocrinal level than just the level of hormones: clearance rate of hepatic and extrahepatic hormones, hormone secretion rates and receptor-site activation changes due to neurohumoral control (McArdle et al. 2014 447).

The two essential hormones in relation to resistance training adaptation are GH and testosterone. In an acute response to resistance training the level of testosterone is increased and cortisol decreased. When training regularly the sensitivity to certain hormones can become greater and this could lead to better effect of the hormones as also the hormones seem to degenerate slower in the trained individuals (McArdle et al. 447-449). This could be an interesting fact when thinking about the reaction created by stress hormones as the sensitivity could be altered by the physical activity.

Although we have ruled brain to be out of the scope of this study. An interesting point has to be made regarding brain-derived neurotrophic factor which can be also called BDNF. BDNF has been linked to be an important part of brain plasticity and work as an antidepressant kind of medicine. (McArdle et al. 2014 448; Cotman et al. 2007; Duman et al. 2007). It is a clear affect that physical activity has on a mental side and stress and depression are linked to each other like stress and structural changes in brain as discussed earlier.



## 5.2 Nervous System Adaptation

One could argue that neural adaptation plays a bigger role in strength adaptation as it is possible to gain more strength without hypertrophy but not possible without neural adaptation (Enoka 2008 365). Therefore it is important to understand nervous system adaptations, as it has to happen in strength training.

Discussed adaptations that happen in nervous system are:

- Increased output from supraspinal centers
- Reduced coactivation
- Increased activation of synergist muscles and agonist
- Better coupling within spinal interneurons
- Descending drive changes which decrease the bilateral deficit
- Increased motor unit synchronization
- Increased muscle activation
- Greater excitability of motor neurons

(Semmler & Enoka 2000 3-20)

Most likely the biggest strength increase in neural adaptation comes from the coordination of the muscles involved in the strength-needed action (Carson 2006; Carroll et al. 2001). Strength then that is of neural adaptation seems to be related to the function of motor neurons and then to a better control of the involved muscles.

## 5.3 Other adaptations

Strength training done with a high enough force can make the BP to rise up to 250/120 mmHg, which can cause thickening of the wall of the ventricles. This is different kind of adaptations than in endurance training, in which the volume of the ventricle increases where

as the volume of the ventricle stays the same in the phenomenon related to strength training wall thickness building. (Vuori et al. 2013 42) Resistance training can also enhance the insulin sensitivity, lipid metabolism and improve BP levels (Vuori et al. 2013 445-446).

## 6 AIM OF THE STUDY

This study is about finding measurable physiological differences between sedentary and physical active groups when it comes to psychological stress and especially trying to find differences between groups with different exercise backgrounds, which are strength and endurance. Stress coping will be defined as how the person reacts to a small psychological stress and how the person recovers from it. All groups will go through the same kind of test. We will record HR, HRV, BP, EDA and salivary cortisol levels. There would other interesting hormones regarding stress reactions but the feasibility of those methods isn't practical on a study of this level. Stress studies with the aspect to exercise have mostly been about how endurance training background or general activity has an effect to physiological markers. In this study there is a strength training background group, whose results will be compared to sedentary and endurance background groups. We want to learn new things from the physiological side of mental stress related to exercise background.

For this study we have two research questions:

1. Are we able to produce a mental stress, which can be seen in physiological markers?
2. Are there statistical differences in physiological markers between the different groups?

For research question 1 we shall introduce a hypothesis that the cortisol levels would be elevated after the mental stress phase (Gerra 2001), HR is elevated (Noto et al. 2005), EDA and BP are elevated (Salmon 2001).

For research question 2 the hypothesis is that we expect to see lower SBP for endurance group compared to strength and sedentary group and also the HR should be lower for endurance group during the test (Spalding et al. 2004). We expect to see lower changes in EDA for endurance group when compared to sedentary group (Salmon 2001), physically

active groups should have attenuated reactivity in BP compared to sedentary group (Forcier et al. 2006), cortisol response for the physically active should be lower compared to sedentary group (Huang et al. 2013).

## 7 METHODS

### 7.1 Participants

Voluntary thirty persons (10 women and 20 men) participated to this study. Participants were recruited through ads. Inclusion criterias for endurance athlete was that capability of running a marathon close or less than in three hours, and for strength athletes able to achieve over 500kg in total at powerlifting. Allocation to the groups was based on interviews with the participants. Participants' ages were from 20 years to 72 years. Mean was 42 years and the histogram of the ages can be seen in the figure 10.

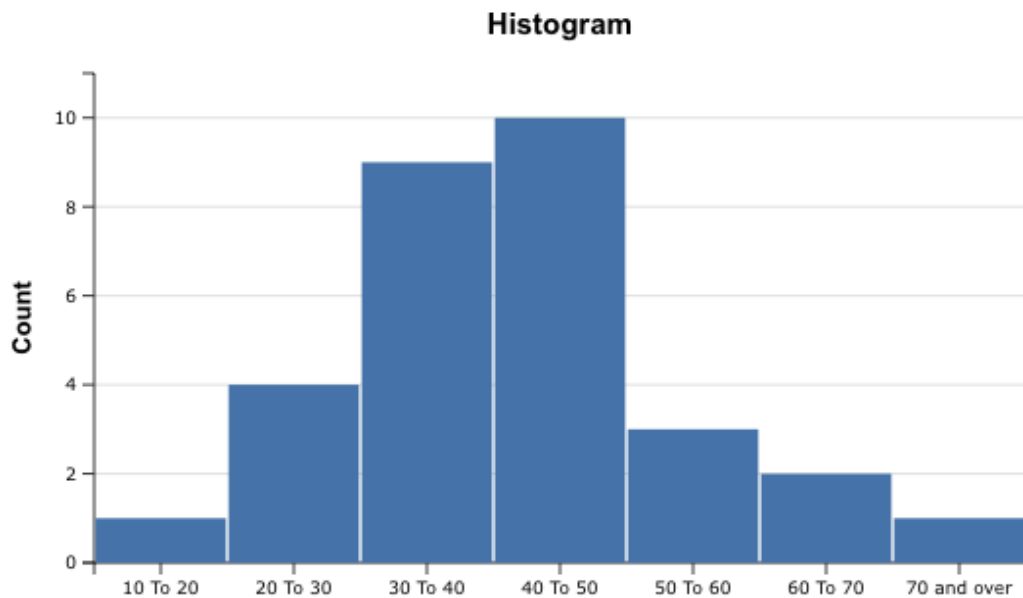


FIGURE 10. Participants age in a histogram.

Participants were divided in to three different groups:

1. Non-athlete – n = 14
2. Endurance athlete – n = 8
3. Strength athlete – n = 8

## 7.2 Measurements

Experiment consisted of three parts:

1. Baseline measurement
2. Mental stress loading
3. Recovery measurement

All participants had their height measured and their body composition using bio impedance measurement (Inbody720, Biospace Co. Ltd, Seoul, Korea).

Following measurements were then measured continuously: EDA, ECG, breathing frequency. BP and cortisol were then measured after every phase.

Participant wore sensors (EL507, BIOPAC Systems Inc., Camino Goleta, California) to measure EDA from the fingertips, ECG with sensors (BlueSensor M, Ambu, Malaysia), breathing frequency. For these measurements wireless data acquisition unit MP150 (BIOPAC Systems Inc., Camino Goleta, California) was used. Data from MP150 was collected by AcqKnowledge software (BIOPAC Systems Inc., Camino Goleta, California). Omron M3 (Omron Healthcare, Lake Forest, IL, USA) upper arm blood pressure monitor was used to measure BP both systolic and diastolic were recorded, Omron M3 has been found to be valid for achieving valid results (Topouchian et. al 2011). Cortisol was measured using cotton saliva collection tubes (Sarstedt AG&Co., Nümbrecht, Germany), which were in participants mouth for a minute that were then analyzed with an analyzer

(Dynex Technologies Co., Chantilly, USA) at the laboratory of University of Jyväskylä with the reagent (ELISA RE 52611, (IBL International GMBH, Hamburg, Germany).

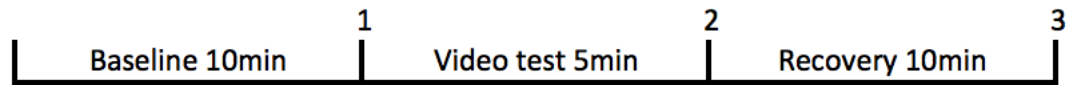


FIGURE 11. Experiment's timeline with measurement points

Figure 11 Explains how the measurements were done. Because BP and cortisol weren't measured continuously there had to be points at which these measurements were taken. These points are after each phase; marked in the figure 11 with 1,2 and 3.

There are three measurement points so that we could identify the influence of the test. BP can be higher because of anticipation and Cortisol is higher in the morning so measuring at three points should so the effect of the experiment.

All the participants filled SIVAQ - Single-Item Question on Leisure-Time Vigorous Physical Activity (Fogelholm et al. 2006) so that their current level of activity was recorded. Questionnaire asks person to estimate their weekly level of activity from the past three months and it gives then score from 1 to 6 based on the subject's level of activity.

Cohen's Perceived Stress Scale 10 was used to record the subject's current level of stress. Finnish translation was used with the permission from Jukka Putaala, MD, PhD, MSc. Questionnaire asks then perceived stress related questions to which person answers based on the feeling she/he has from the past month. Score for this questionnaire is from 0 to 40 points.

Participants were then also asked after the video test to give a number from 1 to 10 on how stressful they felt the test situation. It was instructed so that 1 was a level in which they would almost fall asleep and 10 would be the biggest stress they have ever felt.

Mental stress was inflicted by using a combination of mental arithmetic and Stroop color-word test. These two methods are easy to use and often used in scientific studies (Forcier et al. 2006). Combination was used so that persons with good arithmetic skills wouldn't be favored and vice versa with the Stroop color-word test.

Test was made with Microsoft Powerpoint and then converted to video for a smooth showing. Person was allowed to study the first slide as long as she/he wanted. Then person started the test by pressing a button and test went then on automatically. Person wrote down to a separate answer sheet the correct answer of each slide.

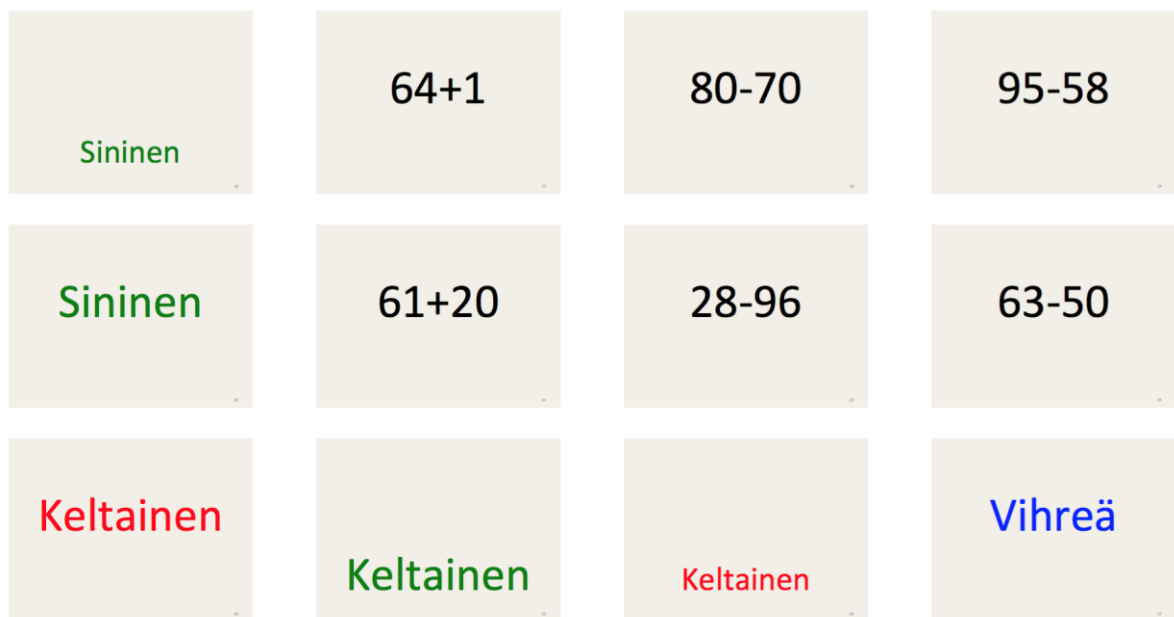


FIGURE 12. Example of slides from video test.

In the figure 12 there are example slides which were used in the video test. All the participants were Finnish so Finnish language was used. In Stroop word-color test



participant had to write down the color of the word and not color written. In mental arithmetic slides the subject wrote down the answer to it. All the slides have a number in the lower right corner.

Slides started with 3s display time, after 33 slides it changed to 2s and last 33 slides were with 2s display time. Total length for the test was 5min and there were 100 slides. Order and structure of the slides were randomized using Random.org web site, which has a randomizer. Randomized things were:

- Arithmetic or Stroop
- Word and color for Stroop
- Numbers and addition or subtraction for arithmetic
- Location and size for both

To break down the flow of the subject the answer sheet had an error on it.

1	100	21	82	31	KELT	41		81	112	101
2	SIN	22		32	57	42		82	SIN	102
3	36	23	13	33	62	43		83	52	103
4	SIN	24	PUN	34	123	44		84	PUN	104
5	93	25	VIH	35	KELT	45		85	PUN	105
6	58	26	PUN	36	90	46		86	PUN	106
7	132	27	SIN	37	32	47		87	KELT	107
8	SIN	28	22	38		48		88	KELT	108
9	KELT	29	VIH	39		49		89	VIH	109
10	123	30	VIH	40	SIN	50		90		110
11	54	31	PUN	41	VIH	51		91		111
12	VIH	32	30	42		52		92	KELT	112
13	SIN	33	VIH	43	KELT	53		93		113
14	SIN	34	PUN	44	KELT	54		94		114
15	VIH	35	84	45		55		95		115
16	VIH	36	PUN	46		56		96	KELT	116
17	65	37	106	47		57		97	PUN	117
18	10	38	KELT	48		58		98	PUN	118
19	33	39	6	49		59		99		119
20	VIH	40	VIH	50		60		100		120

FIGURE 13. One answer sheet to demonstrate the intentional error on it

In the figure 13 it can be seen that third column starts from number 31 when it should be 41 and then numbers from 70 to 80 are missing all together. This was used to add the stressful situation of the test, as subject would realize at some point during the test that she/he wasn't on the track anymore.

### 7.3 Processing of the data

Analysis for the data acquired from MP150 was done using two different methods. EDA and breathing frequency were analyzed using Matlab (version 8.5.0 Mathworks). HR was

analysed using Kubios HRV Software (version 2.2, UEF), which also then calculated the HRV.

During the experiment the different phases of the experiment, as shown in the figure 14, were marked down using stopwatch. Analyses were then made using a time slot within the phase so that there were clear marginal so that the beginning and ending wouldn't affect the results. Analyzed periods were; 5min for rest and recovery and 3min for the video test.

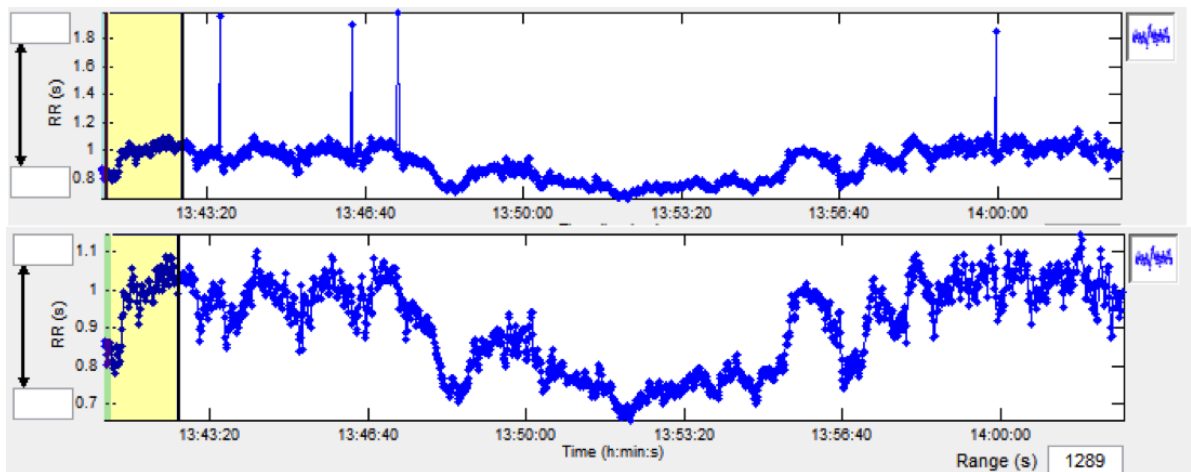


FIGURE 14. Kubios heart rate analysis without medium filter on the top and after medium filter below.

Medium filter was used in Kubios Software was used to remove the sudden spikes in the data. In the figure 14 one can see the effect of the medium filter.

EDA was filtered using a 20,000 point moving average. This was selected based on the visual look of the data. Example of this is shown in figure 15.

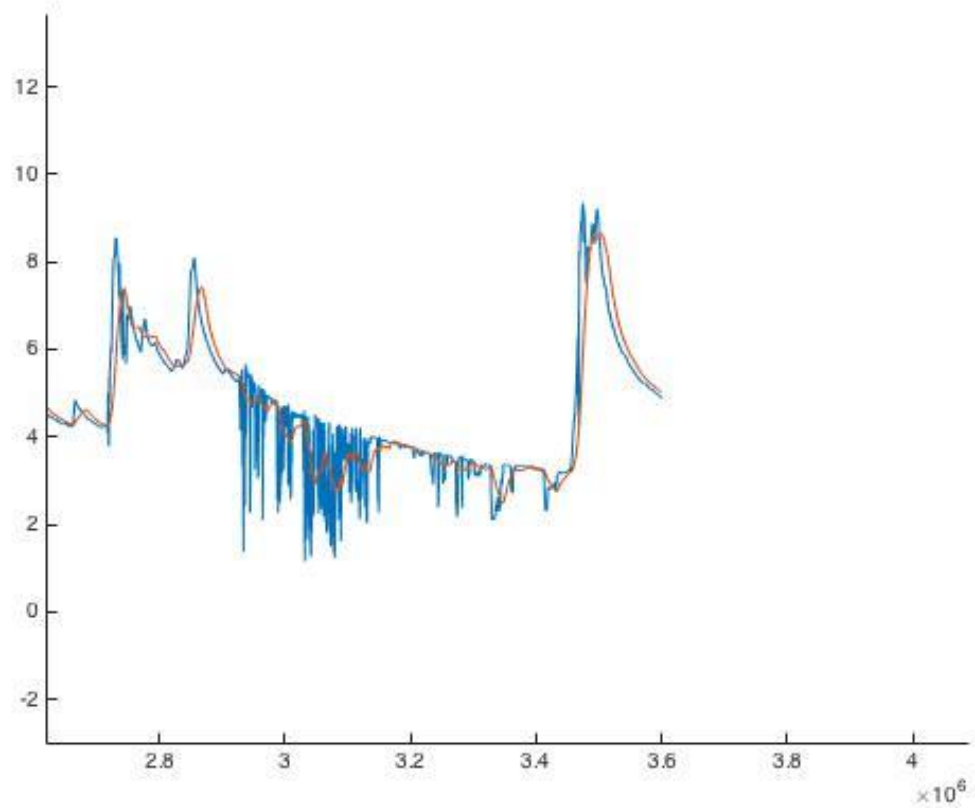


FIGURE 15. The difference of EDA data without and with 20,000 point moving average. Blue is raw data and brown is with 20,000 point moving average.

Figure 16 shows how the analysis of breathing data was done using the peak recognition function on Matlab.

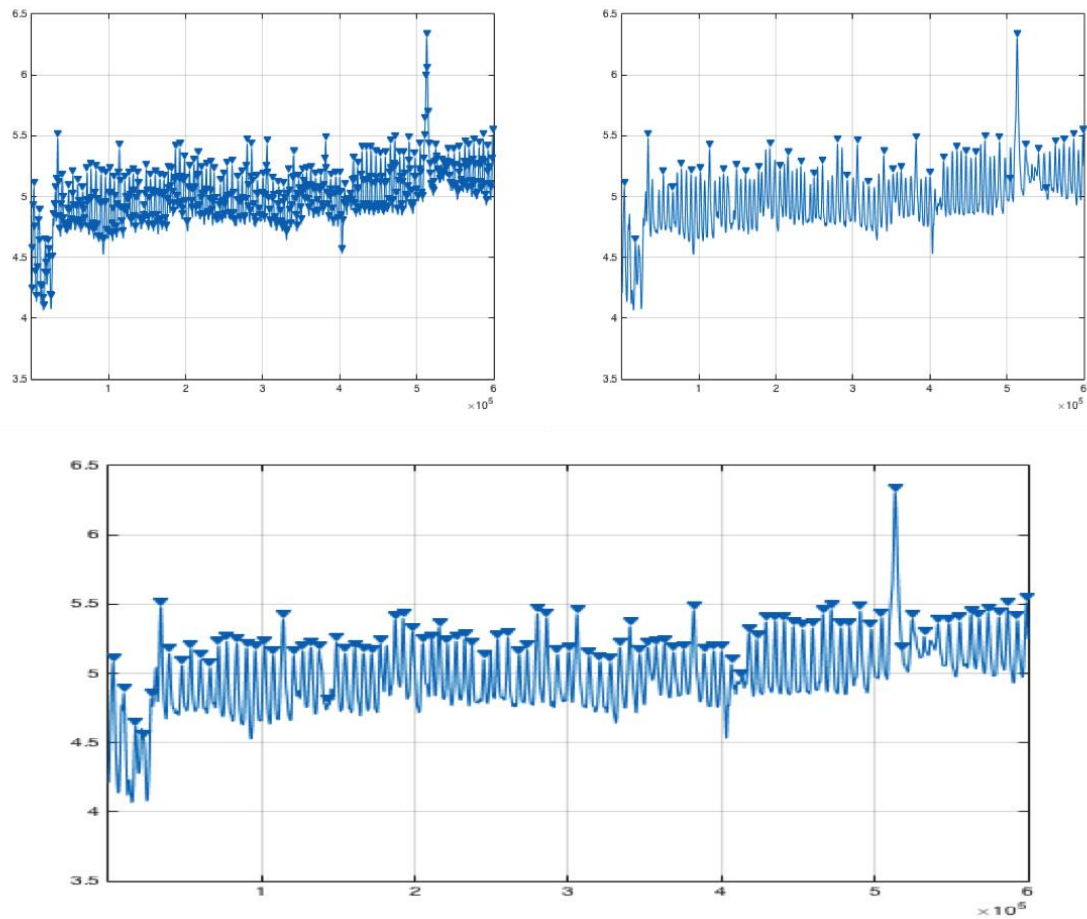


FIGURE 16. The method used to analyze breathing peaks. Top left picture shows an example in which the detail level was too high, the small triangles indicate peaks, top right shows an example in which the detail level was too low and many of the peaks isn't marked with a triangle, bottom picture shows an example in which the level of detail is adequate.

## 7.4 Statistical Analyses

Data were analyzed using SPSS Statistics software (version 22, IBM, USA) and the results are expressed as mean  $\pm$  standard deviation. The effect of mental stress, group and their interactions were calculated using general linear model with repeated measures and with One-way ANOVA. Statistical significance was set at  $p \leq 0.05$ . Test of normality has been

made for all data. If results didn't pass the test of normality then the data was analyzed using Kruskal-Wallis test as a nonparametric test.

## **8 RESULTS**

We shall first look some of the results as a whole then see how the different groups compare again each others. Most important thing is to find differences between the groups that are statistically significant ( $p < 0.05$ ). This is the reason why not all non-statistically significant results aren't shown because there is simply so much data. Relevant results for this study will be shown.

At the end of this chapter I shall conclude a summary of the statistically significant differences so that the conclusions can be based on those and it makes it easier to see the most important results.

Groups will be in the results:

1. Sedentary
2. Endurance Athletes
3. Strength Athletes

### **8.1 Anthropometry and Stress Questionnaire Results**

In this chapter we will look the general results, which give us descriptive information about the subjects.

TABLE 4. Weight, Body Mass Index, Fat Percentage, Muscle Mass and Single-Item Question on Leisure-Time Vigorous Physical Activity International Physical Activity Questionnaire results listed.

Variable	Mean of the Group and Standard Deviation			Difference between groups. - = $p \geq 0.05$ , * = $p \leq 0.05$ , *** = $p \leq 0.001$		
	Group 1	Group 2	Group 3	1->2	1->3	2->3
Weight	69.46±11.69	68.04±4.297	93.60±8.936	-	*	*
BMI	23.76±3.12	21.44±1.161	29.35±5.694	-	*	***
Fat %	26.4286±7.443	9.675±2.042	21.188±4.058	***	*	***
Muscle Mass	28.236±5.739	34.7±1.875	42.1±11.53	-	***	*
SIVAQ	4.36±1.393	5.75±0.463	6±0	*	*	-

There were no differences between the groups. However weight was higher for strength athletes when compared to sedentary and endurance athletes ( $p < 0.05$  for both comparisons) (table 4). BMI acted similarly although the difference between endurance and strength athletes was higher ( $p < 0.001$ ) (table 4). Fat percentage had difference between all the groups: strength and endurance athletes ( $p < 0.001$ ), sedentary and strength athletes ( $p < 0.001$ ) and between sedentary and endurance athletes ( $p < 0.005$ ) (table 4). Strength athletes had a higher muscle mass when compared to sedentary group ( $p < 0.001$ ) and endurance athletes ( $p < 0.05$ ) (table 4).

Nonparametric test showed statistically significant difference for sedentary group towards endurance athletes ( $p < 0.05$ ) and strength athletes ( $p < 0.05$ ) (table 4).

## 8.2 Cortisol

There were no differences between cortisol values for different phases of the experiment. Differences were found between groups in different phases. In the baseline situation the cortisol levels had difference between sedentary and endurance athletes ( $p < 0.05$ ) (figure



17). In the test phase there were differences between sedentary and endurance athletes ( $p < 0.05$ ) and sedentary and strength athletes ( $p < 0.05$ ) (figure 17). Sedentary and endurance athletes had also difference in the recovery phase ( $p < 0.05$ ) and also the sedentary and strength athletes had a difference ( $p < 0.05$ ) (figure 17).

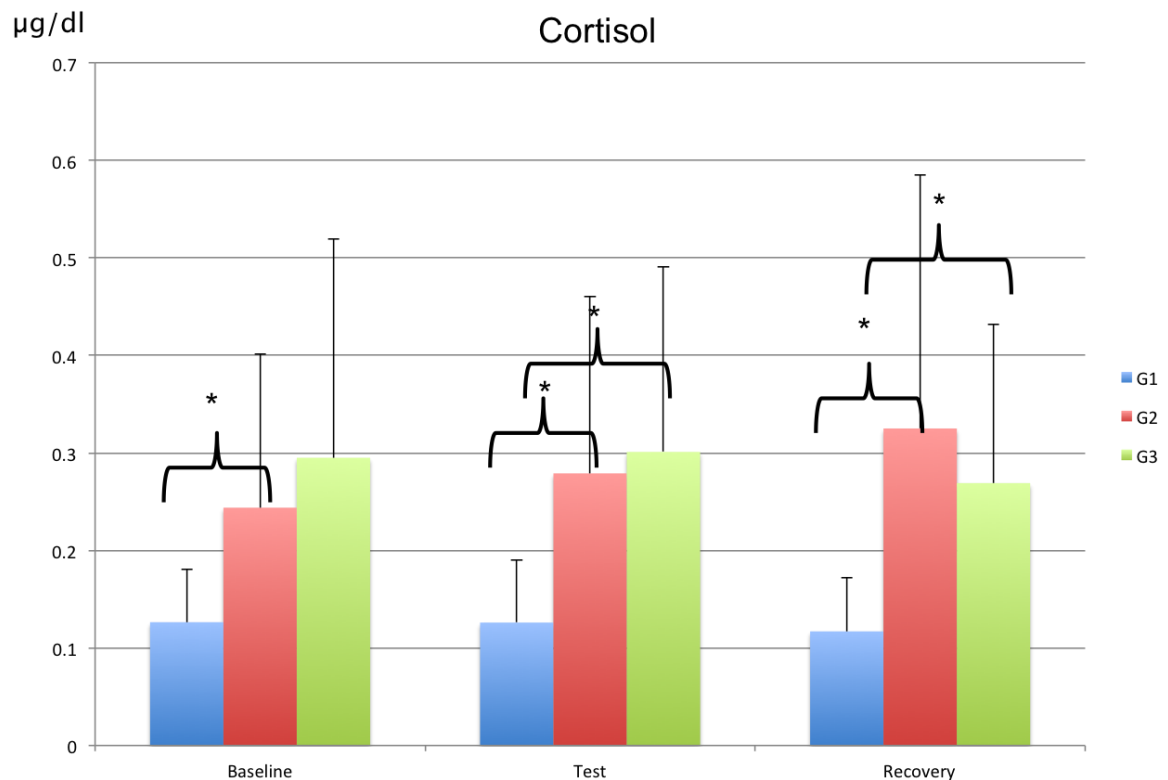


FIGURE 17. Cortisol measured for all the different three Groups (G). \* =  $p < 0.05$  when compared to another group.

### 8.2.1 Relative Change Between the Groups

There is a difference between sedentary and endurance athletes between baseline and recovery relative change in cortisol ( $p < 0.05$ ) (table 5).

### **8.2.2 Absolute Change**

In absolute change of cortisol between phases test and recovery, there is a difference between sedentary and endurance athletes ( $p < 0.05$ ) and between endurance and strength athletes ( $p < 0.05$ ). Then there is a difference in between phases baseline and recovery for sedentary and endurance athletes ( $p < 0.05$ ) and for endurance and strength athletes ( $p < 0.05$ ) (table 5).

### **8.3 Electrodermal Activity**

There is a difference for all three groups: sedentary ( $p < 0.001$ ), endurance ( $p < 0.05$ ) and strength athletes ( $p < 0.05$ ), when comparing the measurements between test and baseline (figure 18). Between the groups there were differences at baseline for sedentary between both athlete groups ( $p < 0.05$ ) (figure 18). In the test phase sedentary group had difference compared to both athletes: endurance ( $p < 0.05$ ) and strength ( $p < 0.001$ ) (figure 18). In the recovery phase the only difference was between sedentary and strength athletes ( $p < 0.05$ ) (figure 18).

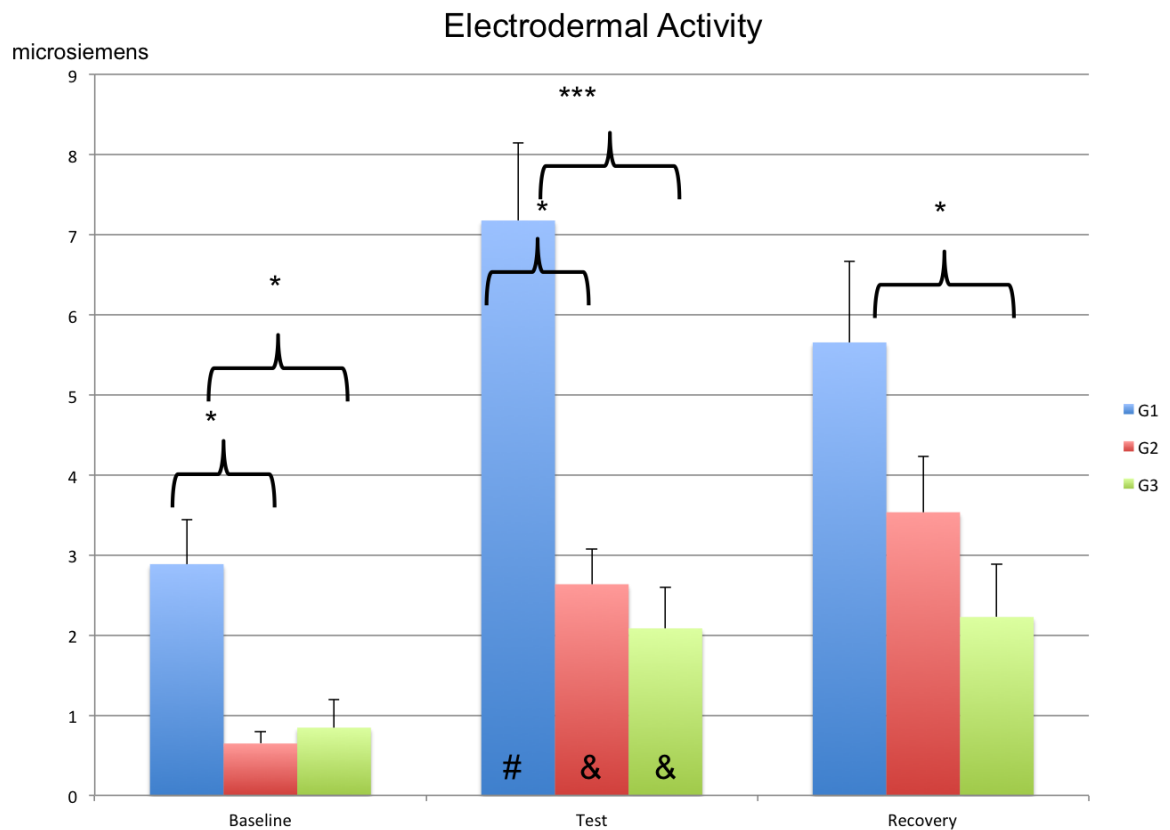


FIGURE 18. Electrodermal Activity measured for all the different three Groups (G). # =  $p < 0.001$  compared to previous phase's measurement, & =  $p < 0.05$  compared to previous phase's measurement, \* =  $p < 0.05$  when compared to another group and \*\*\* =  $p < 0.001$  when compared to another group.

### 8.3.1 Relative change

For relative change there is a difference ( $p < 0.05$ ) between baseline and recovery for sedentary and endurance athletes (table 5).

### **8.3.2 Absolute Change**

In absolute change there are differences between changes in baseline test for groups sedentary and endurance athletes ( $p < 0.05$ ) and sedentary and strength athletes ( $p < 0.05$ ) (table 5). Between test and recovery there is a difference between sedentary and endurance athletes ( $p < 0.05$ ) (table 5).

## **8.4 Heart Rate**

Sedentary group had a difference ( $p < 0.001$ ) compared to previous phase of the study. Both athlete groups also had a difference ( $p < 0.05$ ) between the steps (figure 19). In the baseline measurements all the groups had differences between each other: sedentary and endurance athletes ( $p < 0.05$ ), sedentary and strength athletes ( $p < 0.001$ ) and between the athletes ( $p < 0.05$ ) (figure 19). In the test phase strength athletes have a difference with sedentary ( $p < 0.05$ ) and endurance athletes ( $p < 0.05$ ) (figure 19). In the recovery phase the differences between groups are similar as in baseline phase (figure 19).

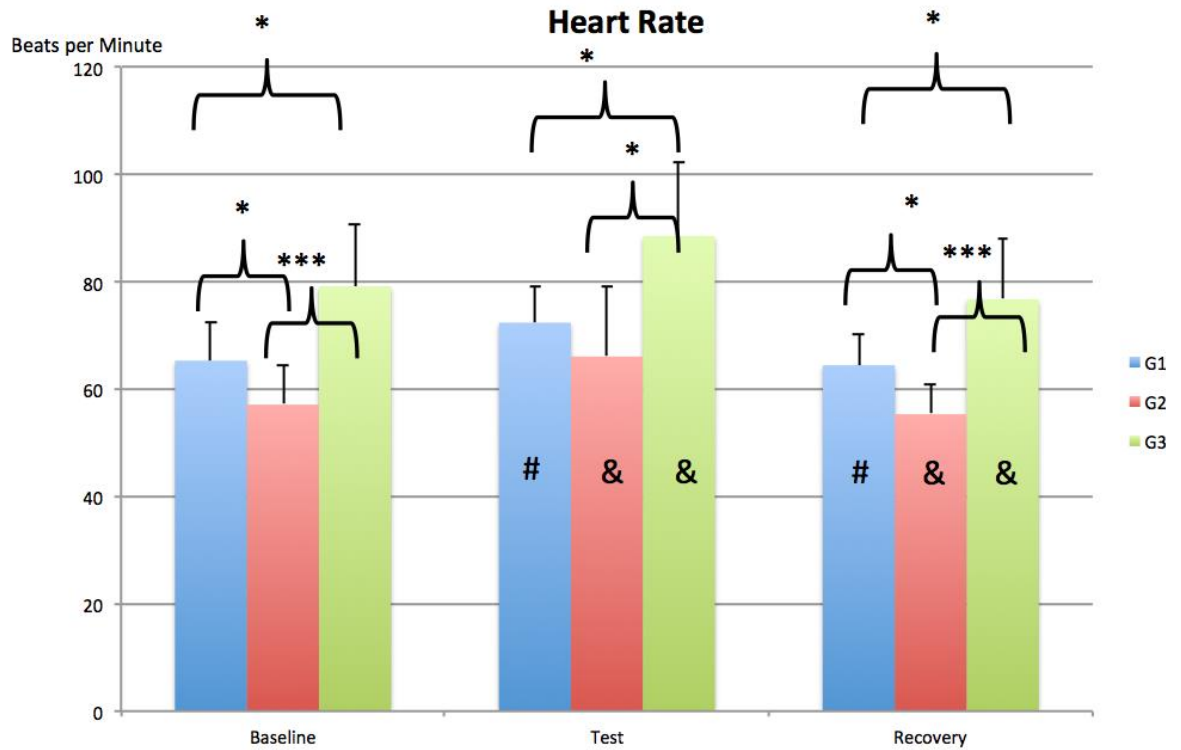


FIGURE 19. Heart rates measured for all the different three Groups (G). # =  $p < 0.001$  compared to previous phase's measurement, & =  $p < 0.05$  compared to previous phase's measurement, \* =  $p < 0.05$  when compared to another group and \*\*\* =  $p < 0.001$  when compared to another group.

#### 8.4.1 Relative change

No differences were found.

#### 8.4.2 Absolute Change

No differences were found.

## 8.5 Heart Rate Variation

Sedentary group had the only difference when compared to previous phases: they were between recovery and baseline ( $p < 0.05$ ) and between recovery and test ( $p < 0.05$ ) (figure 20).

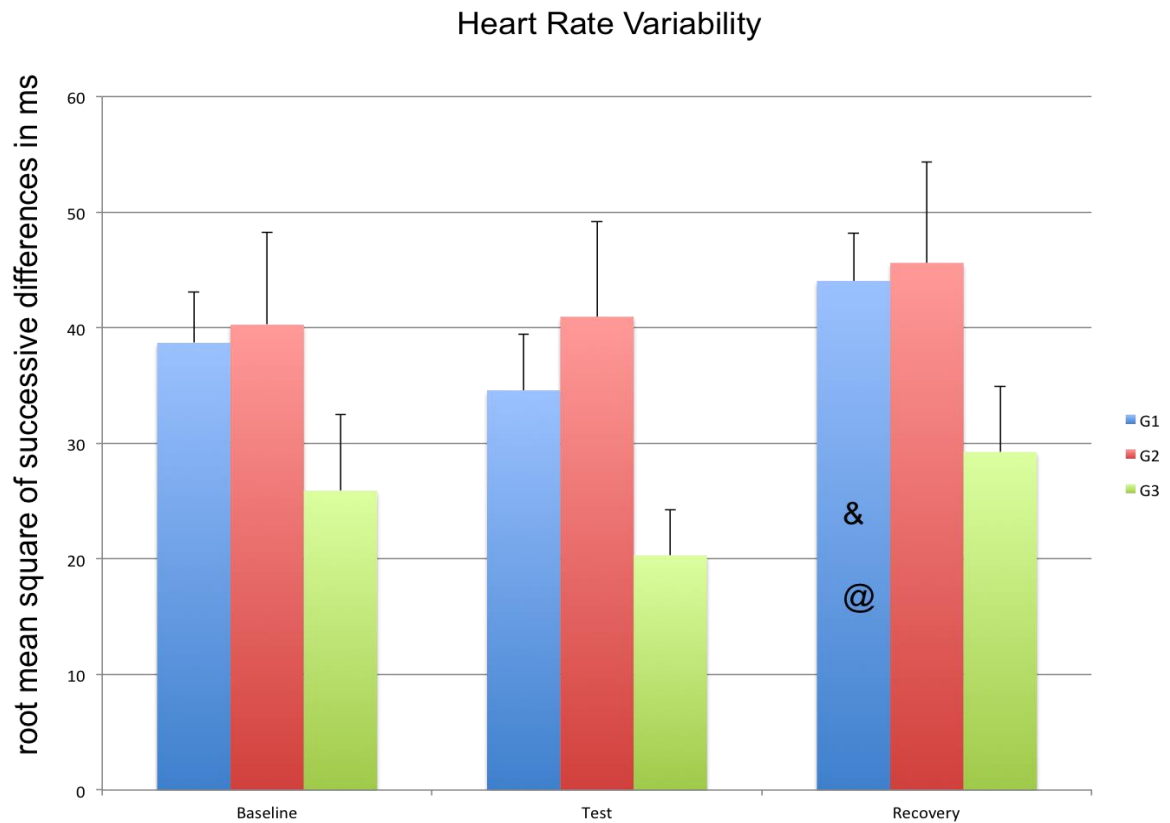


FIGURE 20. Heart Rate Variability measured for all the different three Groups (G)., & =  $p < 0.05$  compared to previous phase's measurement, @ =  $p < 0.05$  when compared to baseline measurement

### **8.5.1 Relative change**

No differences were found.

### **8.5.2 Absolute Change**

No differences were found.

## **8.6 Blood Pressure**

Both systolic and diastolic blood pressure was measured so we shall look them individually in this chapter

### **8.6.1 Systolic Blood Pressure**

Differences between phases were found for endurance athletes between test and baseline ( $p < 0.05$ ) and between recovery and test phase ( $p < 0.05$ ) (figure 21). Sedentary group had a difference ( $p < 0.05$ ) between recovery and baseline measurement (figure 21).

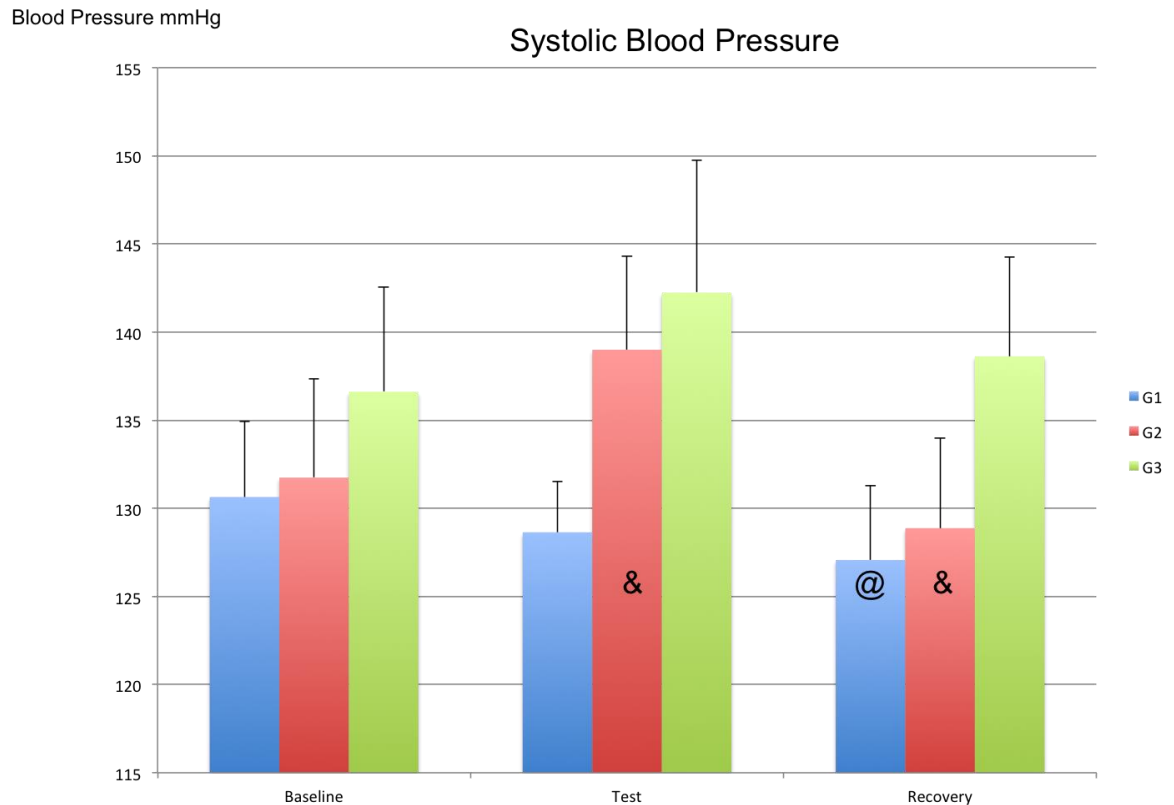


FIGURE 21. Systolic Blood Pressure measured on all the different three Groups (G). & =  $p < 0.05$  compared to previous phase's measurement, @ =  $p < 0.05$  when compared to baseline measurement

### 8.6.1.1 Relative change

The change between baseline and test had a difference ( $p < 0.05$ ) between sedentary and endurance athletes (table 5). In the change between baseline and recovery there was a difference ( $p < 0.05$ ) between sedentary and strength athletes (table 5).

### 8.6.1.2 Absolute Change

Between sedentary and endurance athletes there was a difference ( $p < 0.05$ ) in the change between baseline and test and between test and recovery (table 5). The change between baseline and recovery had a difference ( $p < 0.05$ ) between sedentary and strength athletes (table 5).



## **8.6.2 Diastolic Blood Pressure**

No differences were found.

### **8.6.2.1 Relative change**

Between sedentary and strength athletes there is a difference ( $p < 0.05$ ) in the change between baseline and test (table 5).

### **8.6.2.2 Absolute Change**

Between sedentary and strength athletes there is a difference ( $p < 0.05$ ) in the change between baseline and recovery (table 5).

## **8.7 Breathing Frequency**

All the groups have a difference ( $p < 0.05$ ) towards the previous phase and in the recovery phase there is a difference ( $p < 0.05$ ) between sedentary and endurance athletes (figure 22).

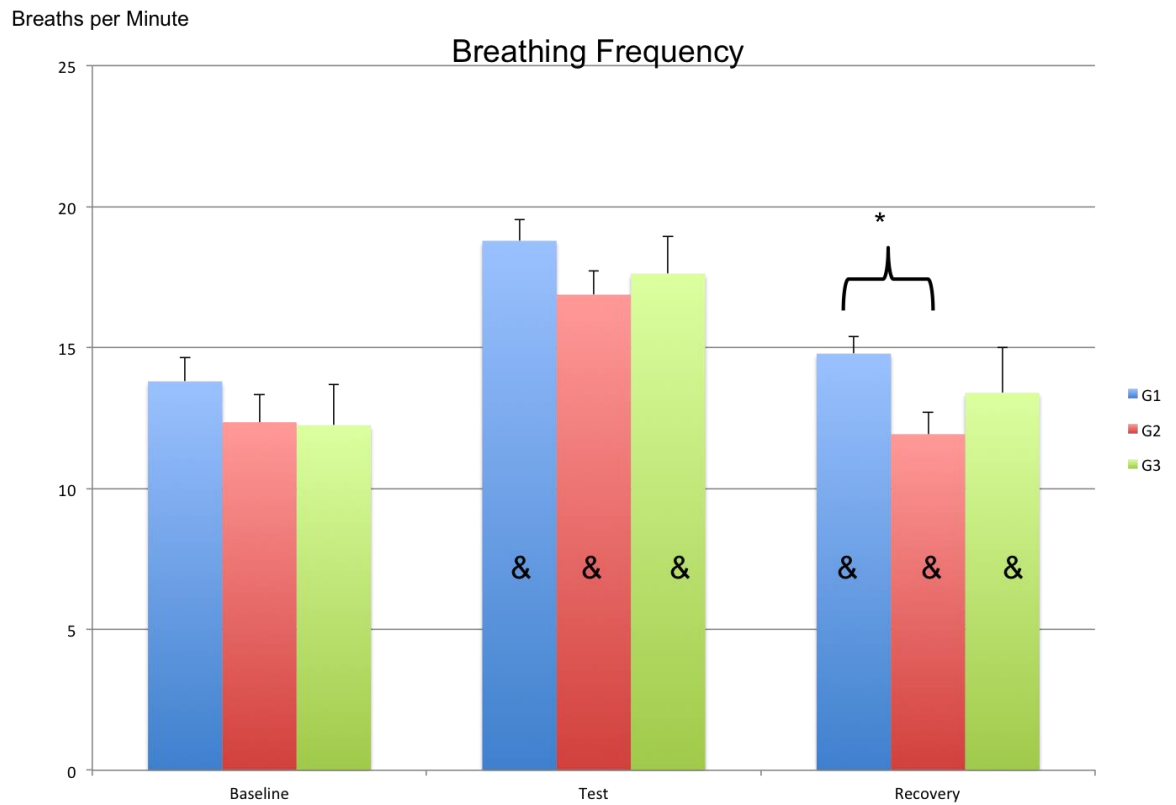


FIGURE 22. Breathing Frequency measured for all the different three Groups (G). & =  $p < 0.05$  compared to previous phase's measurement, \* =  $p < 0.05$  when compared to another group.

### 8.7.1 Relative change

No differences were found.

### 8.7.2 Absolute Change

No differences were found.

## 8.8 Summary of Statistically Significant Differences in Biomarkers

For clearness it makes sense to list the differences in a one table.

TABLE 5.. All the statistically significant differences between groups for biomarkers. Electrodermal Activity (EDA), Heart Rate (HR), Systolic Blood Pressure (SBP) Relative Change (RC), Absolute Change (AC), Breathing Frequency (BF). 1 = Baseline, 2 = Test, 3 = Recovery.

Variable	Mean of the Group and Standard Deviation			Difference between groups. - = $p \geq 0.05$ , * = $p \leq 0.05$ , *** = $p \leq 0.001$		
	Group 1	Group 2	Group 3	1->2	1->3	2->3
EDA Baseline	2.886±2.072	0.647±0.414	0.845±0.987	*	*	-
EDA Test	7.176±3.629	2.635±1.259	2.084±1.45	*	***	-
EDA Recovery	5.654±3.794	3.534±1.964	2.226±1.871	-	*	-
EDA RC 1-3	251.445±238	688.004±395	482.975±363	*	-	-
EDA AC 1-2	4.291±3.04	1.989±1.06	1.238±0.996	*	*	-
EDA AC 2-3	-1.522±3.353	0.899±1.398	0.143±1.716	*	-	-
HR Baseline	65.356±7.017	57.161±7.452	79.233±11.7	*	*	***
HR Test	72.44±6.78	66.126±13.057	88.579±13.773	-	*	*
HR Recovery	64.513±5.877	55.373±5.436	76.781±11.150	*	*	***
SBP RC 1-2	99.093±7.633	105.756±5.581	104.014±7.203	*	-	-
SBP RC 1-3	97.317±4.372	97.966±3.103	101.607±3.193	-	*	-
SBP AC 1-2	-2±10.333	7.25±6.628	5.625±10.663	*	-	-
SBP AC 2-3	-1.571±9.104	-10.125±8.593	-3.625±10.528	*	-	-
SBP AC 1-3	-3.571±6.085	-2.875±4.086	2±3.928	-	*	-
DBP RC 1-2	102.464±8.871	97.568±7.228	91.919±13.959	-	*	-
DBP AC 1-3	-1.643±3.855	-1.125±3.091	-4.125±5.41	-	*	-
BF Recovery	14.783±2.232	11.925±2.209	13.4±4.524	*	-	-
Cortisol Baseline	0.1264±0.054	0.2438±0.158	0.295±0.224	*	-	-
Cortisol Test	0.126±0.064	0.279±0.181	0.301±0.19	*	*	-
Cortisol Recovery	0.117±0.055	0.325±0.26	0.269±0.163	*	*	-
Cortisol RC 1-3	0.934±0.23	1.36±0.73	1.08±0.36	*	-	-
Cortisol AB 2-3	-0.009±0.049	0.073±0.107	-0.033±0.066	*	-	*
Cortisol AB 1-3	-0.009±0.037	0.104±0.109	-0.026±0.081	*	-	*

## 9 DISCUSSION

Purpose of this study was to see how different kind of training background affects the biomarkers during mentally induced stress. We are most interested about the differences between strength and endurance athletes, as it has been clearly shown earlier that physical activity has positive effects to person's ability to cope with stress (Huang et al. 2013; Salmon 2001; Forcier et al. 2006; Ensel 2004). Also we are interested to see when strength and endurance athletes react differently when compared to sedentary group. We shall first look at the general findings of the study and then look differences between athlete groups and different reactions for athlete groups compared to non-athlete group.

In general it was shown that the measured PSS stress scale or subjects' own estimate of the stress level shows no differences between the groups so the experience can be assumed to be similar. In earlier studies it was recorded that physically active people had their own stress reaction lower than sedentary (Nguyen-Michel et al 2006; Klaperski et al. 2013) but we didn't see this in this study, as there wasn't differences between the groups. This can be the cause of the small amount of participants and the inflicted psychological stress wasn't high enough. All the subjects felt at least some amount of mental stress from the test as per their own estimate of the level of stress. So it could be presumed that from psychological point of view the mental stress by the video test was achieved. It was no surprise here that sedentary group was less active than athlete groups. Athletes do train more than non-athletes in general. Looking at the reaction of the biomarkers; HR is elevated for every group at the test phase compared to baseline or recovery level, so this can be thought as a sign of stressful reaction at the test (Huang et al. 2013; Klaperski et al. 2013). Similar kind of result can be seen in the EDA as the test phase has higher results for all the groups when compared to baseline results.

## 9.1 Differences Between Endurance and Strength Athletes

There are differences in weight, BMI, fat %, muscle mass between athlete groups. These can be easily explained and are aligned with previous findings. Endurance athletes tend to have less fat in their body where as strength athletes have more muscle mass and more fat tissue. (Santos et al. 2014; Kanehisa, & Fukunaga 1998)

HR varies between strength and endurance athlete groups in every stage of the study. Endurance athletes have lower HR in every step. This is very common for endurance athletes (Spalding et al. 2004). During their training their heart gets stronger and this leads to lower HR as their blood circulation system is more efficient due to more positive train related stress to it (Jones & Carter 2000). As no statistically significant changes were found when looking at the absolute or relative changes could be found, it is thought in this study that HR isn't a key variable to be analyzed here. It would have been expected to see a difference in the change of HR between the groups (Spalding et al. 2004). Interesting difference can be seen at the change of the absolute values from baseline to recovery and test to recovery test phases. In both of these changes the endurance athletes' cortisol levels rise where as strength and non-athletes levels go down.

Cortisol level means show no difference between groups during the study. Endurance athlete's cortisol levels are rising during the study where as other groups are reducing the level of cortisol in the recovery phase compared to test phase. Non-athlete group has statistically significant difference is all the phases compared to endurance and strength athletes if the outlier with the value 0.04 at baseline phase is dropped from strength athlete group. Otherwise the significant difference isn't valid between non-athletes and strength athletes at the baseline where as it is at all the other places. Cortisol levels of athletes are higher from the start compared to the non-athletes. It has been reported lower changes in cortisol levels for physically active individuals compared to sedentary individuals (Huang et al. 2013; Klaperski et al. 2013).

## **9.2 Different Reactions for Endurance and Strength Athletes Compared to Non-athletes**

When looking for different kind of results between the different types of athletes it is not only the differences in the results between the groups but also if there are different kinds of results when compared to non-athletes, can we learn something interesting. In EDA we can see that the baseline of non-athletes is much higher compared to athletes. Also EDA keeps rising for the athletes where as for the non-athletes it starts to decline after the test. In EDA the relative change of the endurance athletes' change between baseline and recovery is 688%, which is significantly higher than the other groups but only statistically significant when compared to non-athletes, which indicates that the change of EDA is strong for both athlete groups. HR has one difference when looking at the responses from non-athlete vs. athletes; at the test phase the difference between endurance athletes and non-athletes isn't significant where as the strength athletes' HR is much higher compared to the other groups.

There are few interesting discoveries when looking at the BP behavior of the groups. From baseline to test phase the non-athletes' systolic BP decreases, where the athletes increases. Systolic BP then lowers for all the others but for the strength athletes it rises from baseline to recovery. Diastolic BP then decreases in absolute values greatest for the strength athletes from baseline to recovery.

When looking at the BF endurance athletes have much lower frequency. This could be explained by the fact that endurance athletes tend to have more trained and efficient respiratory system than other athletes and non-athletes (Jones & Carter 2000). As discussed earlier in the chapter 9.1.4 the difference in cortisol behavior is that endurance athletes' levels keep on increasing during the test where as the other groups start to decrease after the test in the recovery phase.

### 9.3 Conclusion

Key findings of this study are from the author's point of view:

1. The behavior of the cortisol. It is interesting that the baseline levels are so much higher for the athletes when compared to non-athletes. Also the different behavior of the two types of athletes raises interesting questions about what is behind of this change.
2. EDA levels were higher for non-athletes before and after the mental stress test.
3. Systolic BP decreases for the non-athletes during the test and for strength athletes the systolic BP is higher in the recovery phase than in the baseline phase.

Research question 1 was about could the mental stress caused by this study be seen in the physiological biomarkers. We expected to see based on literature that HR, cortisol, BP and EDA would be elevated (Gerra 2001; Noto et al. 2005; Salmon 2001). HR and EDA were elevated for all the groups in the test phase, but BP or cortisol wasn't systematically elevated for all groups. Based on almost all biomarkers we can say that mental stress caused by the test phase was able to produce a measurable result in physiological biomarkers.

Research question 2 was about the differences between the groups. We expected to see lower changes in BP, HR, cortisol and EDA between the physically active and sedentary groups (Spalding et al. 2004; Salmon 2001; Forcier et al. 2006; Huang et al. 2013). Lower reaction in SBP was expected for endurance group when compared to sedentary and strength groups. What we actually found was that the SBP reactivity was lower for sedentary group when compared to physically active groups. Whereas in DBP a lower response was found for strength athletes, when compared to sedentary group. In HR changes we couldn't find differences between groups, which was a surprise; literature is quite unanimous about lower HR change for physically active individuals compared to sedentary groups. EDA reaction was lower for both of the physically active groups when

compared to sedentary group. Cortisol change measurements didn't show expected differences between the physically active and sedentary groups. We were able to see expected and unexpected differences between the groups, which can be used to argue that training background has an effect on mental stress related physiological biomarkers.

Limitations of this study are the amount of test subjects. It was earlier discussed that one outlier influences the results already so much that statistically significant results are lost. Also the test group is very heterogeneous. It would also help to have more extensive background questionnaire used so that the current status of the person was known, now it could be so that the person has a very intensive periodization phase ongoing and this could have an effect to results. All the athletes were recruited based on their own estimate that they are endurance or strength athletes, it would make sense to have some tests made to have measurable units for athlete's level. One option would be to recruit subjects from national level competitions, and then there would be clear measurable results to prove that the training has lead to significant results.

Future studies could be focused to understand the root causes behind the aforementioned key points. Also as it was discussed in the literature the role of myokines would be highly interesting to study. A study with similar kind of psychological stress to see how the level of myokines change between different groups would bring probably interesting findings to this study. Due to the limitations of hormone measurement in this study we couldn't measure other hormones on top of cortisol. Also one very interesting study would be to involve an intervention where we would first measure the baseline for strength athletes, endurance athletes and non-athletes. Then we would have an intervention where the strength athletes would have endurance training and endurance athletes strength training to see if the biomarkers would then change during mental stress exercises. Test groups should also be more homogenous. It could also be a good idea to have more stressful situation as a method of causing stress to study group. Accuracy of BP measurement was sufficient but it would bring more information to have the BP measured continuously during the test.



## REFERENCES

- Agudelo, L. Z., Femenía, T., Orhan, F., Porsmyr-Palmertz, M., Goiny, M., Martinez-Redondo, V., ... & Pettersson, A. T. 2014. Skeletal muscle PGC-1 $\alpha$ 1 modulates kynurenine metabolism and mediates resilience to stress-induced depression. *Cell*, 159(1), 33-45.
- Aldana, S. G., Sutton, L. D., Jacobson, B. H., & Quirk, M. G. 1996. Relationships between leisure time physical activity and perceived stress. *Perceptual and motor skills*, 82(1), 315-321.
- Aubert, A., E., Seps, B. & Beckers, F. 2003. Heart rate variability in athletes. *Sports Med.* 2003;33(12):889-919.
- Armstrong, L. E., & VanHeest, J. L. 2002. The unknown mechanism of the overtraining syndrome. *Sports Medicine*, 32(3), 185-209.
- Baracos, V., & Kazemi-Bajestani, S. M. R. 2013. Clinical outcomes related to muscle mass in humans with cancer and catabolic illnesses. *The international journal of biochemistry & cell biology*, 45(10), 2302-2308.
- Bartholomew, J., B., Stults-Kolehmainen, M., A., Elrod, C., C. & Todd, J., S. 2008. Strength gains after resistance training: the effect of stressful, negative life events. *J Strength Cond Res.* 2008 Jul;22(4):1215-21.
- Björntorp, P. 2001. Do stress reactions cause abdominal obesity and comorbidities?. *Obesity reviews*, 2(2), 73-86.

- Bretland, R. J., & Thorsteinsson, E. B. 2015. Reducing workplace burnout: the relative benefits of cardiovascular and resistance exercise. *PeerJ*, 3, e891.
- Brotman, D., J., Golden, S., H. & Wittstein, I., S. 2007. The cardiovascular toll of stress. *The Lancet*, 370(9592), 1089-1100.
- Campbell, W. W., Crim, M. C., Young, V. R., & Evans, W. J. 1994. Increased energy requirements and changes in body composition with resistance training in older adults. *The American journal of clinical nutrition*, 60(2), 167-175.
- Carroll, T. J., Riek, S., & Carson, R. G. 2001. Corticospinal responses to motor training revealed by transcranial magnetic stimulation. *Exercise and sport sciences reviews*, 29(2), 54-59.
- Carson, R. G. 2006. Changes in muscle coordination with training. *Journal of Applied Physiology*, 101(5), 1506-1513.
- Chun-Jung, H., Heather E., W., Michael, C., Z. & Edmund, O., A. 2013. Cardiovascular reactivity, stress, and physical activity. *Frontiers in Physiology* 4, Article 314
- Clays, E., De Bacquer, D., Crasset, V., Kittel, F., De Smet, P., Kornitzer, M., ... & De Backer, G. 2011. The perception of work stressors is related to reduced parasympathetic activity. *International archives of occupational and environmental health*, 84(2), 185-191.
- Clow, A., & Hucklebridge, F. 2001. The impact of psychological stress on immune function in the athletic population. *Exercise immunology review*, 7, 5-17.
- Cooper, R., Kuh, D., & Hardy, R. 2010. Objectively measured physical capability levels and mortality: systematic review and meta-analysis. *Bmj*, 341.

- Cotman, C. W., Berchtold, N. C., & Christie, L. A. 2007. Exercise builds brain health: key roles of growth factor cascades and inflammation. *Trends in neurosciences*, 30(9), 464-472.
- Crews, D. J., & Landers, D. M. 1987. A meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. *Medicine & Science in Sports & Exercise*.
- Damas, F., Phillips, S., Vechin, F. C., & Ugrinowitsch, C. 2015. A Review of Resistance Training-Induced Changes in Skeletal Muscle Protein Synthesis and Their Contribution to Hypertrophy. *Sports Medicine*, 45(6), 801-807.
- Dodge, R., Daly, A. P., Huyton, J., & Sanders, L. D. 2012. The challenge of defining wellbeing. *International Journal of Wellbeing*, 2(3).
- Doll H., A., Petersen, S., E., K. & Stewart-Brown S., L. 2000. Obesity and physical and emotional wellbeing: Associations between body mass index, chronic illness, and the physical and mental components of the SF-36 questionnaire. *Obesity Research* 2000; 8: 160-170.
- Duman, C. H., Schlesinger, L., Kodama, M., Russell, D. S., & Duman, R. S. 2007. A role for MAP kinase signaling in behavioral models of depression and antidepressant treatment. *Biological psychiatry*, 61(5), 661-670.
- Enoka, R., M. 2008. *Neuromechanics of Human Movement*. Fourth Edition. Human Kinetics. USA
- Ensel, W., M., & Lin, N., 2004. Physical fitness and the stress process. *Journal of Community Psychology*, 32(1), 81-101.

- Esch, T., Stefano, G. B., Fricchione, G. L. & Benson, H. 2002. Stress-related diseases – a potential role for nitric oxide. *Med Sci Monit* 8(6), 103-118
- Esch, T., Stefano, G. B., Fricchione, G. L. & Benson, H. 2002. Stress in cardiovascular diseases. *Medical Science Monitor*, 8(5), RA93-RA101.
- Fogelholm M., Malmberg J., Suni J., Santtila M., Kyröläinen H., Mäntysaari M., Oja P. 2006. SIVAQ - Single-Item Question on Leisure-Time Vigorous Physical Activity International Physical Activity Questionnaire: Validity against fitness. *Med. Sci. Sports Exerc.*, 38: 753–760.
- Forcier, K., Stroud, L. R., Papandonatos, G. D., Hitsman, B., Reiche, M., Krishnamoorthy, J., & Niaura, R. 2006. Links between physical fitness and cardiovascular reactivity and recovery to psychological stressors: A meta-analysis. *Health Psychology*, 25(6), 723.
- Gardiner, P. F. 2006. Changes in  $\alpha$ -motoneuron properties with altered physical activity levels. *Exercise and sport sciences reviews*, 34(2), 54-58.
- Gardiner, P., Dai, Y., & Heckman, C. J. 2006. Effects of exercise training on  $\alpha$ -motoneurons. *Journal of Applied Physiology*, 101(4), 1228-1236.
- Gerra, G., Zaimovic, A., Mascetti, G. G., Gardini, S., Zambelli, U., Timpano, M., ... & Brambilla, F. 2001. Neuroendocrine responses to experimentally-induced psychological stress in healthy humans. *Psychoneuroendocrinology*, 26(1), 91-107.
- Gianaros, P. J., Jennings, J. R., Sheu, L. K., Greer, P. J., Kuller, L. H., & Matthews, K. A. 2007. Prospective reports of chronic life stress predict decreased grey matter volume in the hippocampus. *Neuroimage*, 35(2), 795-803.

- Gleeson, M. 2006. *Immune Function in Sport and Exercise: Advances in Sport and Exercise Science Series*. First Edition. Churchill Livingstone. China.
- Hakkinen, K., & Pakarinen, A. 1991. Serum hormones in male strength athletes during intensive short term strength training. *European journal of applied physiology and occupational physiology*, 63(3), 194-199.
- Hakkinen, K., Pakarinen, A., Kraemer, W. J., Newton, R. U., & Alen, M. 2000. Basal concentrations and acute responses of serum hormones and strength development during heavy resistance training in middle-aged and elderly men and women. *Journals of Gerontology-Biological Sciences and Medical Sciences*, 55(2), B95.
- Henelius, A., Sallinen, M., Huotilainen, M., Müller, K., Virkkala, J., & Puolamäki, K. 2014. Heart rate variability for evaluating vigilant attention in partial chronic sleep restriction. *Sleep*, 37(7), 1257.
- Hicks, A. L., Martin, K. A., Ditor, D. S., Latimer, A. E., Craven, C., Bugaresti, J., & McCartney, N. 2003. Long-term exercise training in persons with spinal cord injury: effects on strength, arm ergometry performance and psychological well-being. *Spinal cord*, 41(1), 34-43.
- Hortobágyi, T. 2005. Cross education and the human central nervous system. *Engineering in Medicine and Biology Magazine, IEEE*, 24(1), 22-28.
- Huang C., J., Webb, H., E., Zourdos, M.,C., Acevedo, E., O. 2013. Cardiovascular reactivity, stress, and physical activity. *Front Physiol*. 2013 Nov 7;4:314. doi: 10.3389/fphys.2013.00314.

Hynynen, E., 2011a. Heart rate variability in chronic and acute stress : with special reference to nocturnal sleep and acute challenges after awakening. Ph.D. Thesis. University of Jyväskylä. Studies in sport, physical education and health 163. ISBN: 978-951-39-4207-6

Hynynen, E., Konttinen, N., Kinnunen, U., Kyröläinen, H., & Rusko, H. 2011b. The incidence of stress symptoms and heart rate variability during sleep and orthostatic test. *European journal of applied physiology*, 111(5), 733-741.

Jansen, A. S., Van Nguyen, X., Karpitskiy, V., Mettenleiter, T. C. & Loewy, A. D. (1995). Central command neurons of the sympathetic nervous system: basis of the fight-or-flight response. *Science*, 270(5236), 644-646.

Jones, A., M. & Carter, H. 2000. The Effect of Endurance Training on Parameters of Aerobic Fitness. *Sports Med* 2000 Jun; 29 (6): 373-386

Kalman, D., Feldman, S., Martinez, M., Krieger, D. R., & Tallon, M. J. 2007. Effect of protein source and resistance training on body composition and sex hormones. *Journal of the International Society of Sports Nutrition*, 4(1), 1-8.

Kanehisa, H., Ikegawa, S., & Fukunaga, T. 1998. Body composition and cross-sectional areas of limb lean tissues in Olympic weight lifters. *Scandinavian journal of medicine & science in sports*, 8(5), 271-278.

Kanzleiter, T., Rath, M., Görgens, S. W., Jensen, J., Tangen, D. S., Kolnes, A. J., ... & Eckardt, K. 2014. The myokine decorin is regulated by contraction and involved in muscle hypertrophy. *Biochemical and biophysical research communications*, 450(2), 1089-1094.

Kell, R., T., Bell, G. & Quinney, A. 2001. Musculoskeletal Fitness, Health Outcomes and Quality of Life. *Sports Med* 2001; 31 (12): 863-873

Kettunen, O. 2015. Effects of physical activity and fitness on the psychological wellbeing of young men and working adults: associations with stress, mental resources, overweight and workability. Ph.D. Thesis. University of Turku. Department of Physical Activity and Health. ISBN 978-951-29-6031-6.

Kirschbaum, C., Prussner, J. C., Stone, A. A., Federenko, I., Gaab, J., Lintz, D., ... & Hellhammer, D. H. 1995. Persistent high cortisol responses to repeated psychological stress in a subpopulation of healthy men. *Psychosomatic medicine*, 57(5), 468-474.

Klaperski, S., von Dawans, B., Heinrichs, M. & Fuchs, R. 2013. Does the level of physical exercise affect physiological and psychological responses to psychosocial stress in women? *Psychology of Sport and Exercise* 14, 266-274

Kraama, L., 2013. Effects of eight weeks physical training on physical performance and heart rate variability in children. Ph.D. Thesis. University of Jyväskylä. Department of Biology of Physical Activity. <http://urn.fi/URN:NBN:fi:jyu-201304291522>

Kraemer, W. J., & Ratamess, N. A. 2005. Hormonal responses and adaptations to resistance exercise and training. *Sports Medicine*, 35(4), 339-361.

Kreider, R., B., Fry, A., C. & O'toole, M. L. 1998. *Overtraining in Sport*. Human Kinetics. USA

Lazarus, R. S. 1993. Coping theory and research: past, present, and future. *Psychosomatic medicine*, 55(3), 234-247.

Lee M. & Carroll T., J. 2007. Cross education: possible mechanisms for the contralateral effects of unilateral resistance training. *Sports Med.* 2007;37(1):1-14.

- Lieber, R., L. & Fridén, J. 2000. Functional and clinical significance of skeletal muscle architecture. *Muscle & nerve*, 23, 1647-1666.
- Liston, C., Miller, M. M., Goldwater, D. S., Radley, J. J., Rocher, A. B., Hof, P. R., ... & McEwen, B. S. 2006. Stress-induced alterations in prefrontal cortical dendritic morphology predict selective impairments in perceptual attentional set-shifting. *The Journal of Neuroscience*, 26(30), 7870-7874.
- Lundberg, U. 2005. Stress hormones in health and illness: the roles of work and gender. *Psychoneuroendocrinology*, 30(10), 1017-1021.
- Maina, G., Bovenzi, M., Palmas, A., & Filon, F. L. 2009. Associations between two job stress models and measures of salivary cortisol. *International archives of occupational and environmental health*, 82(9), 1141-1150.
- McArdle, W.D., Katch, F.I. & Katch, V.L. 2014. *Exercise Physiology. Eight Edition.* Lippincott Williams & Wilkins. China.
- McEwen, B.S. 1998. Protective and damaging effects of stress mediators. *N. Engl. J. Med.* 338: 171–179.
- Nguyen-Michel, S., T., Unger, J., B., Hamilton J. & Spruijt-Metz, D. 2006. Associations between physical activity and perceived stress/hassles in college students. *Stress Health*. 2006;22(3):179–88.
- Nienstedt, W., Hänninen, O., Arstila & A., Björkqvist, S-E. 2009. *Ihmisen Fysiologia ja Anatomia. 18 painos.* Werner Söderström Osakeyhtiö.



Norris, R., Carroll, D., & Cochrane, R. 1990. The effects of aerobic and anaerobic training on fitness, blood pressure, and psychological stress and well-being. *Journal of psychosomatic research*, 34(4), 367-375.

Noto, Y., Sato, T., Kudo, M., Kurata, K., & Hirota, K. 2005. The relationship between salivary biomarkers and state-trait anxiety inventory score under mental arithmetic stress: a pilot study. *Anesthesia & Analgesia*, 101(6), 1873-1876.

Oken, B. S., Chamine, I., & Wakeland, W. 2015. A systems approach to stress, stressors and resilience in humans. *Behavioural brain research*, 282, 144-154.

Payne, P., & Crane-Godreau, M. A. 2015. The preparatory set: a novel approach to understanding stress, trauma, and the bodymind therapies. *Frontiers in human neuroscience*, 9.

Perna, F. M., Schneiderman, N., & LaPerriere, A. 1997. Psychological stress, exercise and immunity. *International journal of sports medicine*, 18, S78-83.

Pichot, V., Bourin, E., Roche, F., Garet, M., Gaspoz, J. M., Duverney, D., ... & Barthélémy, J. C. 2002. Quantification of cumulated physical fatigue at the workplace. *Pflügers Archiv*, 445(2), 267-272.

Pike, J. L., Smith, T. L., Hauger, R. L., Nicassio, P. M., Patterson, T. L., McClintick, J., ... & Irwin, M. R. 1997. Chronic life stress alters sympathetic, neuroendocrine, and immune responsivity to an acute psychological stressor in humans. *Psychosomatic Medicine*, 59(4), 447-457.

Porges, S. W., 1995. Cardiac Vagal Tone: A Physiological Index of Stress. *Neuroscience & Biobehavioral Reviews* Volume 19, Issue 2, Summer 1995.

- Pruessner, J., C., Hellhammer, D., H., & Kirschbaum, C. 1999. Burnout, perceived stress, and cortisol responses to awakening. *Psychosomatic medicine*, 61(2), 197-204.
- Ritvanen T., Louhevaara V., Helin P., Väisänen S. & Hänninen O. 2005. Responses of the autonomic nervous system during periods of perceived high and low work stress in younger and older female teachers. *Appl Ergon*. 2006 May;37(3):311-8. Epub 2005 Sep 19.
- Rosmond, R. 2002. Stress induced disturbances of the HPA axis: a pathway to Type 2 diabetes? *Med Sci Monit*, 2003; 9(2): RA35-39
- Salmon, P. 2001, Effects of physical exercise on anxiety, depression, and sensitivity to stress: a unifying theory. *Clinical psychology review*, 21(1), 33-61.
- Santos, D. A., Dawson, J. A., Matias, C. N., Rocha, P. M., Minderico, C. S., Allison, D. B., ... Silva, A. M. 2014. Reference Values for Body Composition and Anthropometric Measurements in Athletes. *PLoS ONE*, 9(5), e97846. <http://doi.org/10.1371/journal.pone.0097846>
- Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. 2001. Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences*, 98(8), 4770-4775.
- Schnohr P., Kristensen T., S., Prescott E. & Scharling H. 2005. Stress and life dissatisfaction are inversely associated with jogging and other types of physical activity in leisure time – The Copenhagen city heart study. *Scandinavian Journal of Medicine and Science in Sports* 2005; 15: 107-112.

- Schnyder, S., & Handschin, C. 2015. Skeletal muscle as an endocrine organ: PGC-1 $\alpha$ , myokines and exercise. *Bone*, 80, 115-125.
- Segerstrom, S. C., & Miller, G. E. 2004. Psychological stress and the human immune system: a meta-analytic study of 30 years of inquiry. *Psychological bulletin*, 130(4), 601.
- Selye, H. 1950. Stress and the general adaptation syndrome. *Br Med J*. 1950 Jun 17; 1(4667): 1383–1392
- Semmler, J. G., & Enoka, R., M. 2000. Neural contributions to changes in muscle strength. In V.M. Zatsiorsky (Ed.), *Biomechanics in sport*. Oxford, UK: Blackwell Science.
- Sothmann, M., S., Buckworth, J., Claytor, R.,P., Cox R.,H., White-Welkley, J.,E. & Dishman, R., K. 1996. Exercise training and the cross-stressor adaptation hypothesis. *Exerc. Sport Sci. Rev.* 1996; 24: 267–87.
- Spalding, T. W., Lyon, L. A., Steel, D. H., & Hatfield, B. D. 2004. Aerobic exercise training and cardiovascular reactivity to psychological stress in sedentary young normotensive men and women. *Psychophysiology*, 41(4), 552-562.
- Steptoe, A. 2000. Stress, social support and cardiovascular activity over the working day. *International Journal of Psychophysiology*, 37(3), 299-308.
- Strickland, J. C., & Smith, M. A. 2014. The anxiolytic effects of resistance exercise. *Frontiers in psychology*, 5.
- Ströhle, A. 2009. Physical activity, exercise, depression and anxiety disorders. *Journal of neural transmission*, 116(6), 777-784.

Stults-Kolehmainen, M., A. & Sinha, R. The Effects of Stress on Physical Activity and Exercise. 2014. *Sports Med.* 2014 Jan; 44(1): 81–121.

Stults-Kolehmainen, M., A., Bartholomew, J., B. & Sinha, R. 2014. Chronic Psychological Stress Impairs Recovery of Muscular Function and Somatic Sensations Over a 96-Hour Period. *Journal of Strength & Conditioning Research*: July 2014 - Volume 28 - Issue 7 - p 2007–2017

Stunkard A., L., Faith, M., S & Allison K., C. 2003. Depression and obesity. *Biological Psychiatry* 2003; 54: 330-337.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. 1996. Heart Rate Variability, Standards of Measurement, Physiological Interpretation and Clinical Use. *Circulation*, 93, 1043 – 1052.

Taylor, S., Jaques, N., Chen, W., Fedor, S., Sano, A., & Picard, R. (2015, August). Automatic identification of artifacts in electrodermal activity data. In *Engineering in Medicine and Biology Society (EMBC), 2015 37th Annual International Conference of the IEEE* (pp. 1934-1937). IEEE.

Topouchian, Jirar, et al. 2011 "Validation of four automatic devices for self-measurement of blood pressure according to the international protocol of the European Society of Hypertension." *Vascular health and risk management* 7.

Toppinen-Tanner, S. & Ahola K. 2012. *Kaikkea stressistä*. Tallinna Raamatutrükikoja OÜ. Estonia

Tsatsoulis, A. & Fountoulakis, S. 2006. The Protective Role of Exercise on Stress System Dysregulation and Comorbidities. *Ann. N.Y. Acad. Sci.* 1083: 196–213

- Tsutsumi, T., Don, B. M., Zaichkowsky, L. D., & Delizonna, L. L. 1997. Physical fitness and psychological benefits of strength training in community dwelling older adults. *Applied human science*, 16(6), 257-266.
- Twenge, J. M. (2000). The age of anxiety? The birth cohort change in anxiety and neuroticism, 1952–1993. *Journal of personality and social psychology*, 79(6), 1007.
- Vrijkotte, T. G., Van Doornen, L. J., & De Geus, E. J. 2000. Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. *Hypertension*, 35(4), 880-886.
- Vuori, I., Taimela, S. & Kujala U. 2013. *Liikuntalääketiede*. 3.-6. Edition. Kustannus Oy Duodecim. Finland
- Vyas, A., Mitra, R., Rao, B. S., & Chattarji, S. 2002. Chronic stress induces contrasting patterns of dendritic remodeling in hippocampal and amygdaloid neurons. *The Journal of Neuroscience*, 22(15), 6810-6818.
- Weijs, P. J., Looijaard, W. G., Dekker, I. M., Stapel, S. N., Girbes, A. R., Oudemans-van Straaten, H. M., & Beishuizen, A. 2014. Low skeletal muscle area is a risk factor for mortality in mechanically ventilated critically ill patients. *Crit Care*, 18(1), R12.
- Wideman, L., Weltman, J. Y., Hartman, M. L., Veldhuis, J. D., & Weltman, A. 2002. Growth hormone release during acute and chronic aerobic and resistance exercise. *Sports Medicine*, 32(15), 987-1004.
- World Health Organization. 2015. Mental disorders. Fact sheet N°396. Updated October 2015. Available at: <http://www.who.int/mediacentre/factsheets/fs396/en/>