# INDIVIDUALITY OF RESPONSES TO BILATERAL AND UNILATERAL VOLUME EQUATED HYPERTROPHIC STRENGTH TRAINING IN PHYSICALLY ACTIVE MEN

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## ABSTRACT

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*Purpose.* The purpose of the present study was to investigate possible differences between unilateral and bilateral strength training during ten weeks progressive hypertrophic strength training and six weeks detraining period. Furthermore, it was examined possible different individual responders in muscle hypertrophy during strength training and, how those different responders would behave during the detraining phase following the training period.

*Methods.* The present intervention included ten weeks of progressive hypertrophic resistance training followed by six weeks of detraining in healthy young men (n=24). Ultrasound was used to measure vastus lateralis (VL) muscle cross-sectional area (CSA) and triceps brachii (TB) thickness. Maximal bilateral and unilateral lower and upper body isometric forces as well as maximal dynamic unilateral and bilateral concentric maximum forces in leg and bench press were measured. The subjects trained three times per a week. The subjects received an individual example nutritional plan before the start of the intervention and they received protein and carbohydrate supplementation after every training session.

*Results.* VLCSA increased in the total group from 31.1±5.7cm to 35.1±5.0cm ($p=0.013$) and leg press 1RM from 154.6±24.9kg to 178.5±25kg ($p<0.0001$) after ten weeks of strength training. The subjects were split to three groups according to the increase in VLCSA: High responders >15% (n=10), Medium responders 15-5% (n=6) and Low responders <5% (n=8). High responders were the only ones to have a significant change in VLCSA from pre to mid, pre to post, pre to detraining 1, pre to detraining 2 and post to detraining 2. VLCSA increased in High responders by 22.3% ±7.0 during training and decreased by -9.3%±5.7 during detraining and 1RM by +20.5±12.5% and by -2.1±3.1%, respectively. The corresponding values for Medium responders were +8.7±6.0% and -4.7±3.3% for VLCSA, and +14.1±11.3% and -1.9±4.3% for 1RM, and for Low responders -2.3±5.9% and -0.5±8.4% for VLCSA and +12.6±11.5% and +0.19±3.4 for 1RM, respectively. All dynamic strength values were statistically significantly changed from pre to post for both groups. The unilateral group improved isometric strength statistically significantly from pre to post for isometric left and right leg, and also for bilateral leg press. The bilateral group only increased significantly in isometric bilateral leg press force from pre to post. The lean mass increases (total lean mass, arms, legs) were statistically significant for both groups.

*Conclusion.* High responders tend to lose VLCSA and dynamic bilateral strength somewhat faster than Low responders during the detraining phase. In addition, muscle hypertrophy in High responders was observed to appear already during earlier weeks of training. There were no statistically significant differences between the unilateral and bilateral groups in any variables. Both training types seem to lead to similar gains in terms of muscle hypertrophy and strength.



*Keywords:* hypertrophy, strength training, individuality, individual responders, high responders, low responders, unilateral, bilateral, isometric, maximum strength

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## ABBREVIATIONS AND DEFINITIONS

| | |
|---|---|
| AD | anterior deltoid |
| AL | activation level |
| BF | biceps femoris |
| C | cortisol |
| CSA | cross-sectional area |
| DEXA | dual-energy x-ray absorptiometry |
| DT | detraining |
| EMG | electromyography |
| MVC | maximal voluntary contraction |
| RF | rectus femoris |
| RM | repetition maximum |
| SHBG | sex hormone-binding globulin |
| T | testosterone |
| TB | triceps brachii |
| TT | total testosterone |
| US | ultrasound |
| UST | unilateral strength training |
| VL | vastus lateralis |
| VM | vastus medialis |
| W | watt |
| 1RM | one-repetition maximum |

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## 1. Introduction

Strength gains is mainly contributed by neural adaptations and hypertrophy adaptations. Neural adaptations are the main contributors to the early-observed strength improvement (Häkkinen et al., 1998). As the training proceeds the contribution of muscular hypertrophy will also take a more important role in strength gains. Hypertrophy becomes more important after the first three to five weeks (Moritani & DeVries, 1979). Hypertrophy can be defined as a protein content increase in a contractile part of a muscle fibre (McDonagh & Davies, 1984). It is well documented that systematic resistance training will induce growth and strength in skeletal muscle. Resistance training can also make functional and structural adaptations in the neuromuscular system. (Ahtiainen et al., 2003.) To induce adaptations muscle needs to be functionally overloaded. That can happen when muscle is required to contract more forcibly than in normal life. Resistance training induced changes can also be measured as an increase in a muscle cross-sectional area (e.g. MacDougall et al., 1979; Tesch 1987). Strength and muscle adaptations are dependent on the type, volume and intensity of the loading at the given time (Ahtiainen et al., 2003). Intensive resistance training is shown to increase force production and skeletal muscle mass (McDonagh & Davies, 1984). Hypertrophic strength training is characterized by somewhat submaximal loading and maximal repetitions, which means performing repetitions to concentric failure. To gain the maximal amount of lean body mass, it might be optimal to combine in the training program mechanical tension, muscle damage and metabolic stress, because they all play a role in a exercise-induced muscle growth. (Schoenfeld, 2010.)

The magnitude of these adaptations is highly depended on the individual`s responsiveness to training (Macdougall, 1986a). It is crucial to understand an individual's sensitivity to a certain type of resistance training program. It may enable tailored exercise program to really improve, for example, athletes performance. There are many other factors that can have an effect, for example: the intensity and duration of the training program, age and the background of the individual prior the program (Macdougall, 2003). This review is going to focus on

neuromuscular adaptations to strength training, bilateral and unilateral strength training, hypertrophic strength training and individuals' responsiveness to resistance training.

## 2. Neuromuscular adaptations to strength training

It is well documented that systematic strength training produces structural and functional adaptations in the body. Strength is one of these adaptations. Strength can be defined as the maximal, voluntary, isometric force (Enoka, 1988). Mechanisms that can explain increased strength through resistance training are muscle mass, contractile characteristics of the muscle, muscle architectural changes and neural factors (Gabriel, Kamen & Frost, 2006). This chapter is focusing on morphological and neural adaptations to strength training.

Strength evolves at the onset of the strength training mostly by neural factors with increasing contribution of hypertrophic factors as the strength training goes on (Häkkinen & Komi, 1983; Häkkinen, 1989). Changes in muscle size can be detectable after only three weeks of resistance training (Seynnes, 2007). Muscle hypertrophy can contribute to the strength gains earlier than previously thought (Seynnes, 2007). For example, Krieger (2010) suggest in his meta-analysis that some of the initial strength gains are because of hypertrophy. Untrained people can adapt much faster to resistance training then well-trained strength athletes. It seems that strength and hypertrophy adaptations are more limited in strength trained athletes than untrained people (Häkkinen, 1994a). Development and adaptations in strength-trained athletes depends on the type of training, intensity and loading (Häkkinen, 1989).

Changes in muscle size do not necessarily affect a change in strength. It might be a completely different phenomena. This is based on the weak correlation between the change in muscle size and the change in muscle strength after 20-24 weeks of training in untrained individuals (Ahtiainen et al., 2016). In addition, the evidence of the loss of muscle mass with detraining, yet a maintenance of muscle strength. Strength can be maintained during detraining up to four weeks without training (Häkkinen et al., 2000). Muscle fiber cross-sectional area declines rapidly during detraining especially in strength-trained athletes. Force production seems to decline more

slowly. (Mujika & Padilla, 2001.) It appears that one does not necessarily need a high propensity for skeletal muscle growth to increase strength and vice versa.

## 2.1 Morphological adaptations to strength training

When the strength training is first started the main contribute to strength are neural factors. As the training proceeds the contribution of muscular hypertrophy will also take a more important role in strength gains. Hypertrophy becomes more important after the first three to five weeks. Magnitudes of these changes vary due to the differences in training, muscle groups, subjects and methods. (Häkkinen & Komi, 1983; Moritani & DeVries, 1979.) To induce adaptations muscle needs to be functionally overloaded. That will happen when muscle is required to contract more forcefully than in normal life. It is well known fact that resistance training induce in a muscle an increase in cross-sectional fibre area (e.g. MacDougall et al., 1979; Tesch, 1987). One extreme example are bodybuilders.

Heavy-resistance exercise increases the myofibrillar protein synthesis in the exercised muscles (Chesley et al., 1992). When the training is repeated at regular intervals, there will be an increase in myofibrillar size and number. The magnitude of this change is highly depended on the individual`s responsiveness to training (Macdougall, 1986a). There are many other factors that affect the change, for example: the intensity and duration of the training program, age and the background of the individual prior the program. (Macdougall, 2003).

### 2.1.1 Changes in muscle CSA

Muscle is a postmitotic tissue and it does not go through significant cell replacement during its existence. Cell repair is needed to avoid apoptosis. This happens through the dynamic balance between muscle protein synthesis and degradation. Muscle hypertrophy and changes in the muscle cross-sectional area take place when protein synthesis exceeds protein breakdown. (Schoenfeld, 2010.)

Skeletal muscle hypertrophy will happen as an expansion of protein content of a pre-existing muscle fibre due to resistance training. It has been a widely accepted fact that heavy resistance strength training (HRST) induces a change in muscle CSA after relatively short time. The most used methods to mark these changes are magnetic resonance imaging (MRI) computed tomography and ultrasound. MRI is held the most accurate of these methods. In general, it can be said that MRI images of different soft tissue structures are more detailed images compared to other methods. Moreover, MRI can be used to calculate the cross-sectional are of individual muscles at several sites along the muscle's length. (Engstrom et al., 1991.) There are only a very limited number of studies done with computed tomography and ultrasound that have generated data for individual muscles (Ryushi et al., 1988; Sambrook, Rickards & Cumming, 1988).

Hypertrophic resistance training will cause an increase in cross-sectional areal of all fibre types. However, a greater relative hypertrophy happens in the type II muscle fibers (Tesch et al., 1985). They response easier to hypertrophy training. So, it seems that hypertrophy tend to happen more in type II muscle fibers. Häkkinen et al. (1981) reported that hypertrophy and atrophy happened more rapidly in type 2 fibers during a training intervention and detraining. The authors presented that type 2 fibers could be more plasticity and that's why react to faster to changes in stress environment. Longer studies like Häkkinen et al. (1981) have found significant hypertrophy in the both fiber type, whereas shorter intervention have frequently found only significant hypertrophy in type 2 fibers (Thorstensson et al., 1976; Houston et al., 1983).

Myofibrillar CSA will change as a result of strength training. MacDougall et al. (1980) reported a significant increase in myofibrillar CSA during six months of strength training. They also noted that there were an increase in myofibrils, because they longitudinal divide forming new myofibrils. However, they suggested that myofilament density was not affected by strength training and the density remain unchanged. Controversially, Phillips (2000) argues that there is growing myofibrillar protein density within the fiber before there is an increase in fiber diameter. Furthermore, that would affect the force generating capacity.

The mechanism behind myofibrils longitudinal split is the following. As myofibrillar size increases the A band and Z-disk will be influenced to a growing lateral pressure, which will cause Z-disk rupture if developed sufficiently. Z-disk rupture will lead the next Z-disk to split and this will go on until the entire myofibril has divided longitudinally. Figure 1 demonstrate Z-disk rupture and myofibrillar splitting. (Folland & Williams, 2007.)

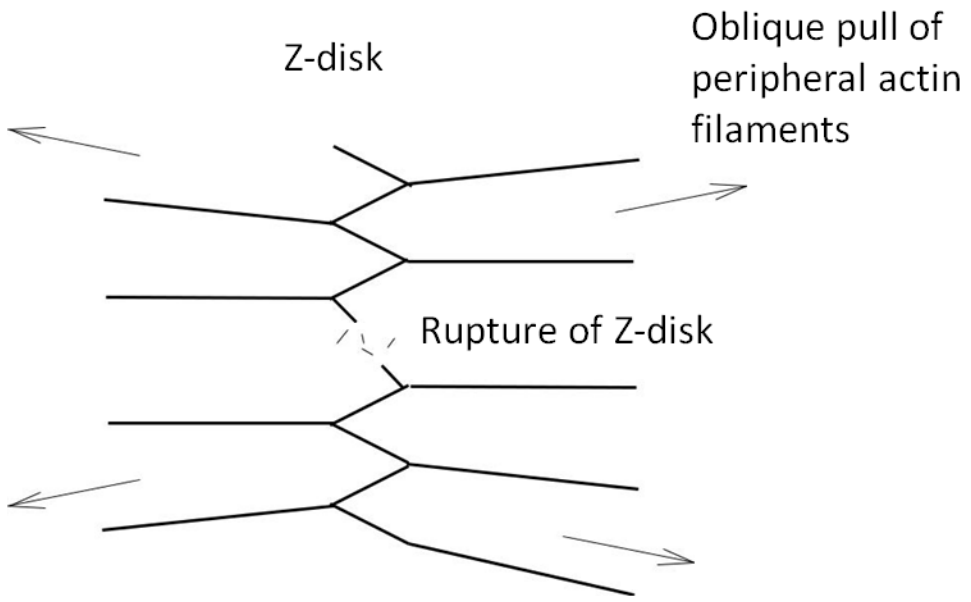


FIGURE 1: Myofibrillar splitting due to the oblique pull of peripheral actin filaments. (Folland & Williams, 2007).

In conclusion, it can be said that myofibrillar growth and proliferation are the main morphological changes that are responsible for hypertrophy. Myofibrillar proliferation is a result of Z-disk rupture and longitudinal division. That is a limiting and controlling factor in myofibrillar size. Also, myofibrillar regulation becomes easier because of the longitudinally division. (Follan & Williams, 2007.)

### 2.1.2 Satellite Cells and hypertrophy

In order for hypertrophy to happen, new contractile proteins must be made and integrated into the system. New contractile proteins are made when muscle protein synthesis exceeds muscle protein breakdown. Hypertrophy is thought to be mediated by the activity of satellite cells, which dwell between sarcolemma and the basal lamina (Hawke et al., 2001). Myonuclei can only attend to a certain volume of cytoplasmic material. For type 2 fibers the certain area tends to be about twice as high as for type 1 fibers. Satellite cells divide and proliferate to create new myonuclei. Satellite-cell proliferation might be a prerequisite for hypertrophy. (Folland & Williams, 2007.) In order to proliferate, satellite cells need to be aroused. Satellite cells becomes active, when a sufficient mechanical stimulus is given on muscle (Vierck et al., 2000). Satellite cells provide precursors for muscle tissue repair and growth (Schoenfeld, 2010). To sum up, satellite cells influence on hypertrophy in several different ways. Donating extra nuclei to muscle fibres grow the capacity to synthesize new contractile proteins. In addition, satellite cells retain their mitotic capability and helps a myonuclei to support muscle growth. Mitosis means the division of the nucleus. (Schoenfeld, 2010.) Finally, satellite cells coexpress to various myogenic regulatory factors that have big impact on muscle repair, regeneration and growth (Cornelison & Wold, 1997).

In humans, it has been shown that the number of satellite cells and myonuclei is higher in elite powerlifters than in untrained control group (Kadi et al., 1999; Kadi et al., 1999). Kadi & Thornell (2000) found that after resistance training satellite cells were activated. It seems that hypertrophy is dependent on the increased myonuclear content (Kadi et al., 1999). Proliferation of muscle satellite cells can occur as fast as four days within a single resistance training, moreover, a large increase in myofibrillar protein synthesis happen within 4 - 5 h of a single resistance training (Moore et al., 2005). These findings support the hypothesis that muscular hypertrophy can contribute to strength gains at the early stage of training and training can influence satellite cells.

## 2.2 Neural adaptations to strength training

The big role of the nervous system in strength gains can be notice in changes in muscle size and strength and the specificity of certain improvements in performance. Häkkinen et al. (1985) noticed that the strength gains surpass the changes in muscle size. Moreover, when subjects go through immobilization the loss of strength is usually greater than the muscle atrophy (Berg et al., 1997). Also, Duchateau & Hainaut (1987) found that strength declines faster than muscle size in healthy subjects. An increase in muscular strength without noticeable hypertrophy is the biggest evidence for neural adaptations in humans.

Strength gains can be achieved without participating in strength training routine. Imagined contractions and cross-education studies have shown that the nervous system has a huge part in strength gains. Strength increases can be made without repeated muscle activation. For example, with imaginary training strength increased after 20 training sessions even though electromyogram showed that there was no activity in the muscles during imagination training (Yue & Cole, 1992). The authors concluded that strength gains seem to result from the central motor programming and that the neural adaptations increase before muscle hypertrophy. In contrast, Herbert et al. (1998) found that imagined training increased strength but not significantly in versus to the control group. Furthermore Clark et al. (2014) found that imagined training, during immobilization with a surgical cast for an entire month, cut off by half the loss of strength in contrast to control group, who did not went through imagined training protocol. In summary, imagined contractions is an effective way to speed up the rehabilitation process, but is unlikely to be as effective as resistance training or real contractions. Imagined contractions demonstrate clearly that neural adaptations happen.

Several cross-education studies have shown that when one limb goes through a strength training program, the homologous muscles in the other limb have experienced a significant increase in muscle strength, even though there is a minimal activation during strength training program. Munn et al. (2004) noted in their meta-analysis that the contralateral effects of strength varies from -2.7 % to 21.6 % of initial strength and after pooling the data shows that unilateral strength

training produces modest increases in contralateral strength. Strength gains are also specific for the task. The tasks that requires more learning and are more complex are more influenced to specificity effect (Chilibeck et al., 1998).

### 2.2.1 Agonist and synergist activation and antagonist coactivation

To produce force the nervous system needs to activate main working muscle in the desired direction which is called agonist. To achieve more force, agonist is needed to activate more. There are three ways that contribute to agonist force production capability: motor unit recruitment, motor unit synchronization and motor unit firing frequency. Secondly, a synergist is needed to activate to help producing force. The synergist are doing the same task than agonist. The use of surface electromyographic (SEMG) is widely use to measure of muscle activity. SEMG is also use to measure strength gains because resistance training intervention is associated with an increase in the amplitude of SEMG activity (Gabriel, Kamen & Frost, 2006). SEMG activity anticipates an increase in neural drive and changes in SEMG activity impart changes in motor unit firing patterns. Neural adaptations might be very strong during early phase of the strength training (Gabriel, Kamen & Frost, 2006.) However, changes in agonist EMG activation level cannot fully explain early strength gain (Holtermann et al., 2005).

Agonist muscle activity results in a better limb movement, antagonist activity opposes that movement. When there is achieved a reduction in antagonist co-activation that will allow increased agonist muscle force. Thus, antagonist co-activation can't be completely removed because the joint integrity must be maintained. (Gabriel et al., 2006). Hypothesis is that the decreases activation of antagonist coactivation will allow higher agonist muscle strength, because contraction of the opposing muscle group will not disturb agonist work (Kamen, 1983). Carolan & Cafarelli (1992) work supports this and they concluded that there is a small decrease in antagonist coactivation during the early phase of training is a nonhypertrophic adaptation of the neuromuscular system in response to static resistance training of this type. However, Gabriel & Kroll (1991) found that when subjects did the fatiguing maximal isometric protocol intervention both agonist and antagonist SEMG activity increased. Increased antagonist

activation has also been noticed by Gabriel & Boucher (2000) and Gabriel, Basford & An (1997). To sum up, it seems that when the training goal is to become faster or stronger the antagonist coactivation seems to rise. It may be due to rising stiffness which may be beneficial when the stretch-shortening cycle is involved, for example, running.

There might be different activation strategy when aging. Häkkinen et al. (1998) found that elderly subjects had first higher antagonist coactivation, which could tell that they needed more stabilisation for the legs and when the training proceeded the antagonist coactivation was reduced. Moreover, they noticed that the different age group antagonist coactivation differed, even though the groups significantly increased their strength. The middle-aged groups remained unchanged while the older subjects exhibited reduction. In conclusion, the central nervous system will choose either force production or joint integrity. Higher joint stiffness can be achieved by increasing activation of the agonist and antagonist in situations of the uncertainty or unstable movement (Gabriel, Kamen & Frost, 2006). In addition, vertical stiffness is shown to increase with running velocity (Brughelli & Cronin, 2008) and higher speed of the movement and greater strength movements could imply rise of the antagonist activation. When the target of the training is simple and safe, for example, knee extension, the antagonist activation can be reduced safely.

### 2.2.2 Motor unit activation

As earlier told there are three ways that contribute to agonist force production capability: motor unit recruitment, motor unit synchronization and motor unit firing frequency. Humans are not able to fully activate muscle voluntarily (Dowling et al., 1994). That could be the result of limitations in motor unit recruitment or motor unit firing frequency. Knigth & Kamen (2001) used the interpolated twitch technique, which is commonly used to measure motor unit activation. During a maximal contraction, electricity is given to subjects to activate rest of the muscle fibres that were not activated during voluntary contraction. The authors noticed that there were 2 - 5 % additional force immediately after the stimulation. In addition, they noticed a 2 % increase in muscular activation throughout training.

Motor unit synchronisation means simultaneous activation of numerous motor units. This can result as an increase in force. It is another possible mechanism for increases in muscle strength, but it has not been completely demonstrated (Gabriel, Kamen & Frost, 2006). Semmler & Nordstrom (1998) researched motor unit discharges directly from musicians, weight-lifters and untrained people. They used isometric abduction of the index finger during low-force contraction. They noticed that motor unit synchronisation was higher whit training and the degree of motor unit synchronisation increased from untrained to musicians and finally to weight-lifters. Milner-Brown et al. (1975) findings supported this. They concluded that motor unit synchronisation increases with exercise training. There is a limited amount of evidence that resistance-training influence on motor unit synchronisation.

Motor unit firing frequency could explain for the rapid increases in muscular force during early phase of strength training (Gabriel, Kamen & Frost, 2006). The firing frequency will grow due to resistance training (Van Gutsem et al., 1998; Patten et al., 2001). In addition, it has been noted that elite weightlifters have higher firing frequencies than their non-training control group (Leong et al., 1999). Knigth & Kamen (2001) also found that there were no difference in motor unit firing rates when assessed at 10 % and 50 % of maximum voluntary contraction. That could mean that subjects are needed to be tested maximally to see differences in motor unit firing rate and that heavy-resistance training is needed to induce adaptations in motor unit firing frequency. Figure 2 tries to sum up the neural adaptations to strength training which occur at multiple sites.

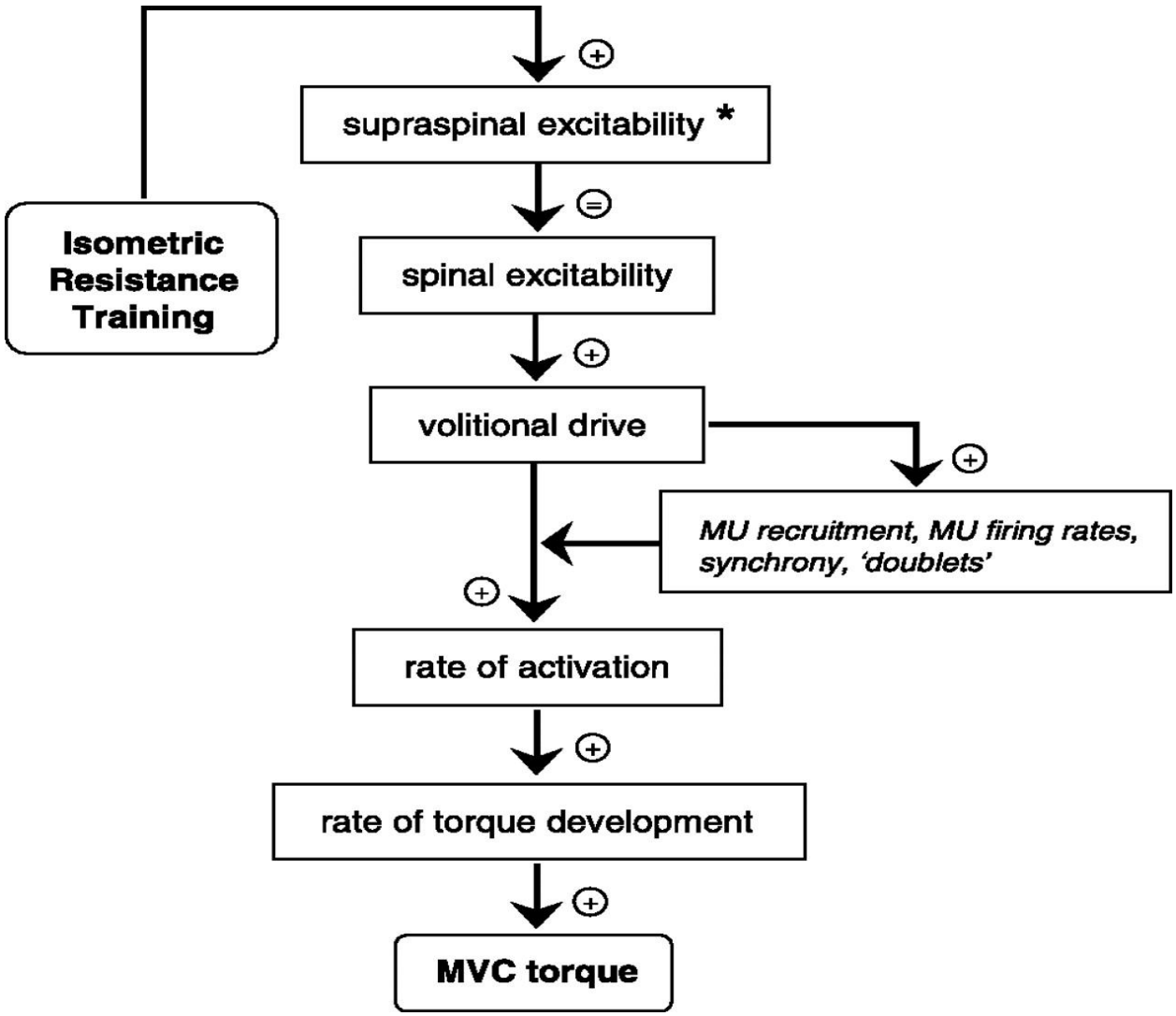


FIGURE 2: Summary of neural adaptations to isometric strength training. Neural adaptations occur at multiple sites along the pathway from the motor cortex to muscle. There were no change in "spinal excitability" in this study. (Del Balso & Cafarelli, 2007).

## 2.3 Detraining

Skeletal muscle tissue has an extraordinary plasticity and is able to adapt to variable states of neuromuscular activity. It will readjust to the reduced physiological stress during reduced use. (Mujika & Padilla, 2001.) Detraining is the phase when subjects do not train. During detraining, the decrease in muscle force is explained by the neural and muscular adaptations caused by the inactivity (Häkkinen & Komi, 1983). In figure 3 is presented changes in isometric force and static and dynamic surface electromyography (SEMG) with 16 weeks of training and eight weeks of detraining. Häkkinen et al. (1985) reported also that during 12-week detraining a huge decrease in maximal strength was correlated with the decrease in the maximum iEMG of the leg extensors after 24 weeks of progressive heavy resistance training

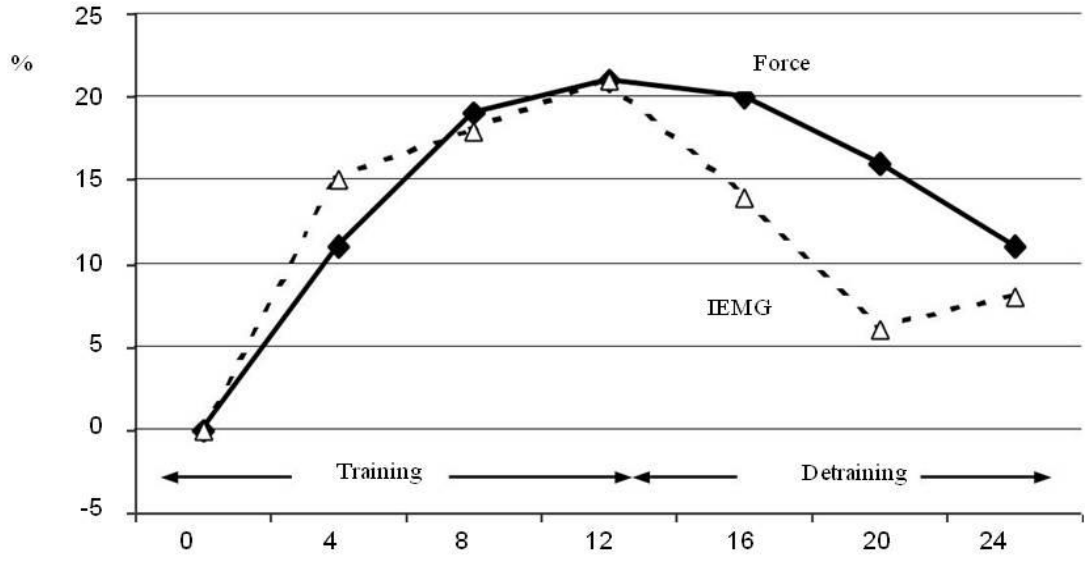


FIGURE 3: Changes in isometric force and SEMG with 16 weeks of training and 8 weeks detraining. (Adapted from Häkkinen & Komi, 1983).

It seems that strength can be maintained without training up to three or four weeks. After that it is gradually lost. Häkkinen et al. (2000) reported that beginners can take three weeks off of training without fear of losing strength. In addition they reported that short-term detraining led to only minor changes, while prolonged detraining resulted in muscle atrophy and decreased

strength. Izquierdo et al. (2007) reported that four week detraining may induce larger declines in muscle power output than in maximal strength after 16 weeks of resistance training. Children will also suffer from detraining process, because strength is lost during the detraining period (Faigenbaum et al., 1996). 60 days of unilateral strength training and 40 days of detraining will lead to decrease in muscle cross-sectional area (CSA), iEMG and maximum voluntary with a similar time course to that of training. In addition the kinetics of changes in CSA, force and neural drive during training and detraining are similar. (Narici et al., 1989.)

There are not much data about detraining on strength-trained athletes. Hwang et al., (2017) reported that trained men retain strength and muscle mass during a two week period of detraining. Hortobagyi et al. (1993) found that short-term detraining may specifically affect eccentric strength and the size of the Type II muscle fibers, leaving other aspects of neuromuscular performance uninfluenced. Muscle fiber CSA declines rapidly in strength and sprint athletes (Mujika & Padilla, 2001). In general, strength performance is easily retained for up to 4 wk of inactivity, but highly trained athletes' eccentric force and sport-specific power may suffer significant declines (Mujika & Padilla, 2001).

## 3. Unilateral and bilateral neuromuscular responses to resistance training

### 3.1 Unilateral and bilateral responses to resistance training

Unilateral strength training (UST) can mean any strength training movement which can be done with one limb. For example, one-leg squat, one-arm bench press, lunges, step-ups, one-arm row etc. Figure 4 demonstrate one style of unilateral squatting known as a Bulgarian split squat. UST can be integrated into a training program, but usually this kind of movements are only used when intention is to vary bilateral movements. Nonetheless, UST can be used to develop foundation of strength. Many movements in everyday life and in the sports field are performed entirely unilaterally. Jones et al. (2012) found that there was a similar testosterone response to bilateral squat and unilateral squat. The authors also discussed that there could be comparable neuromuscular and hormonal demands to bilateral squat and unilateral squat, even though the absolute work was less in unilateral squat. Moreover, unilateral or bilateral exercises do not seem to be a decisive factor for improving morphological adaptations and bilateral muscle strength in untrained women (Botton et al., 2015). It seems that unilateral training can be an effective tool when persuading muscular hypertrophy. Furthermore, unilateral and bilateral training can be equally effective for early phase strength and power improvements in untrained men and women (McCurdy et al., 2005). Häkkinen et al. (1996) found that both the unilateral and bilateral progressive heavy resistance strength training lead to major gains in maximal strength in middle-aged and elderly men and women. There were also an increase in voluntary neural activation of the trained muscles and increase in CSA in both groups.

For muscular adaptation point of view it seems that it does not matter if the movement is unilateral or bilateral. Staron et al. (1994) have investigated time course of muscular adaptations during eight weeks of resistance training. They noted that there were similar improvements in muscle cross-sectional area and relative maximal strength in both bilateral and unilateral leg press in men and women. To sum up it seems that both bilateral and unilateral training can be used to induce adaptations in the neuromuscular system.

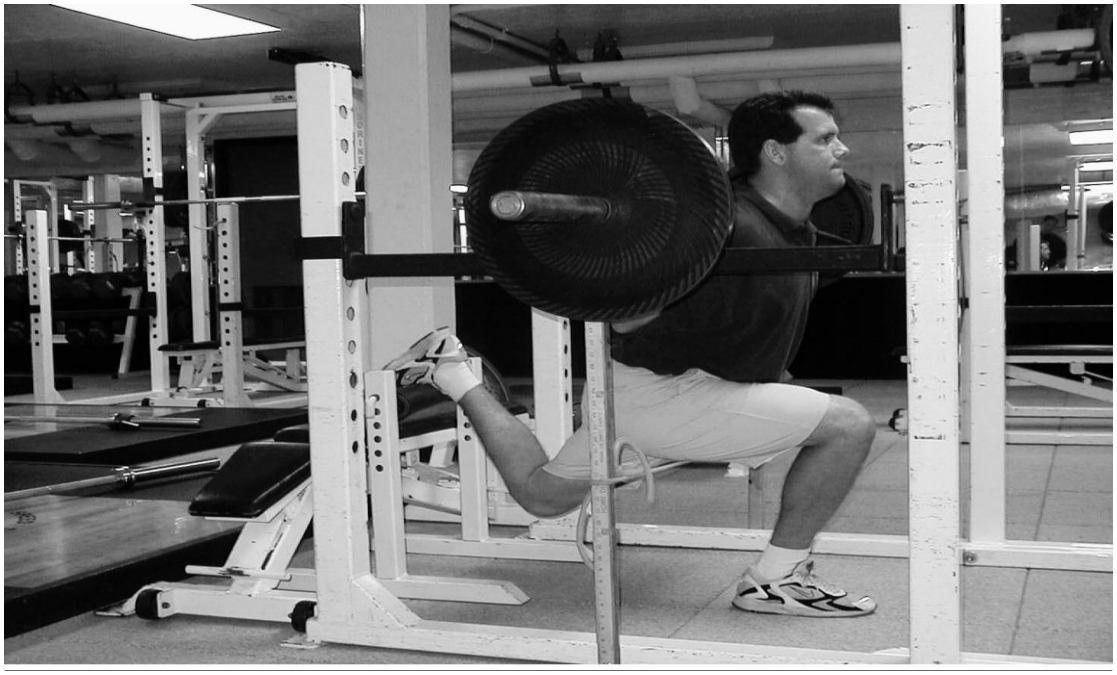

FIGURE 4: Unilateral squatting (Mccurdy et al, 2005).

### 3.2 Training specificity

There is a lot of evidence that increase in voluntary strength after resistance training are largely specific to the type of contraction and movement (e.g. Dons et al., 1979; Kanehisa & Miyashita, 1983a). In addition, specificity applies also to velocity of contraction (Kanehisa & Miyashita, 1983a) and angle of training (e.g. Meyers, 1967; Lindh, 1979). It seems that training specificity is a because of neural adaptations and this should always be taken into account.

Training with one limb or with two leg is no different in means of training specificity. Bilateral strength training induced bigger strength adaptations in bilateral strength whereas unilateral strength training tend to lead to greater unilateral strength (Häkkinen et al., 1996). Also Taniguchi (1997) found that bilateral resistance training improved strength production in bilateral condition. Researcher also found that strength production in the unilateral condition were increased more through unilateral strength training. Unilateral training induce local muscle hypertrophy only in the exercised limb and can happen without changes in systemic hormones

that play a significant role in the hypertrophic process (Wilkinson et al., 2006). On the other hand, Hubal et al. (2005) noticed statistically significant hypertrophy of the contralateral untrained arm.

### 3.3 Bilateral deficit

Bilateral deficit occurs when the sum of the strengths of right and left limbs are more than maximal voluntary strength of simultaneous bilateral contraction. Figure 5 shows theoretical representation of the bilateral deficit. Bilateral deficit has been noticed in many studies in both large and small muscle groups (Vandervoort et al., 1984; Secher et al., 1988; Howard & Enoka, 1991; Koh et al., 1993). A bilateral deficit has noticed for the first time in 1961 when Henry & Smith reported bilateral deficit in handgrip strength test. However, some studies have not shown bilateral deficit (Vandervoort et al., 1987; Häkkinen et al., 1996).

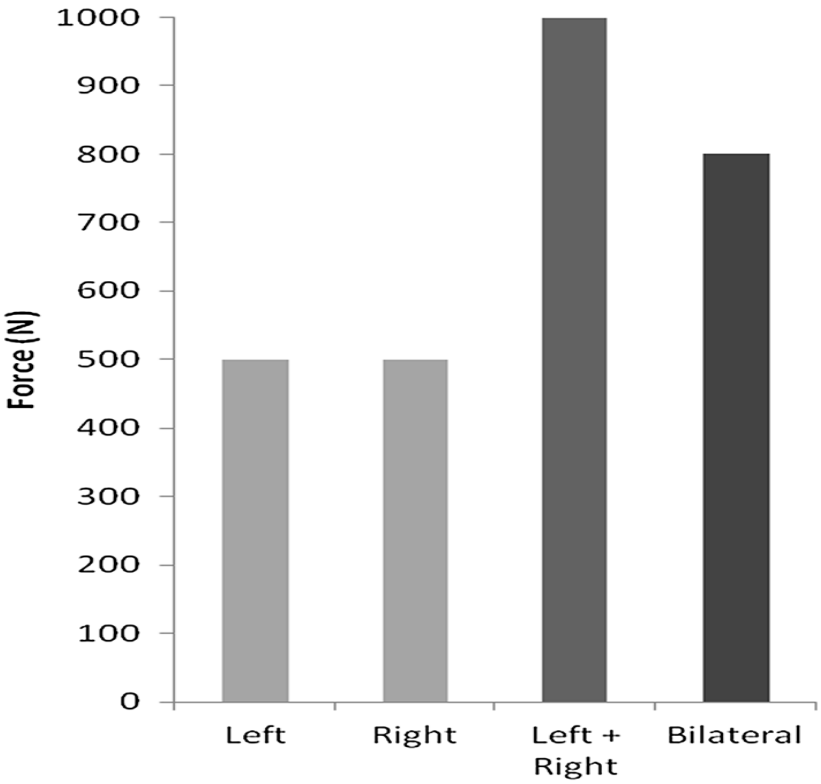


FIGURE 5: Theoretical representation of the bilateral deficit (Nijem & Galpin, 2014).

Some movement patterns show more frequently bilateral deficit than others do. Jakobi & Chilibeck (2001) report that combined knee and hip extension, for example, leg press and squat, show bilateral deficit more frequently than the one joint movement such as knee extension. The reason behind this might be that multiple joint exercise requires much more neural activation than the single joint exercises (Chilibeck et al., 1998). Moreover, Häkkinen et al. (1995) and (1996) did not observe a bilateral deficit during single joint movement that was a knee extension.

The cause of bilateral deficit is not completely clear, but is might caused by neural inhibition when contracting limbs simultaneously (Vandervoort et al., 1984; Kawakami et al., 1998). Jakobi & Chilibeck (2001) report that this phenomenon is most likely caused by descending drive between the cortical level and peripheral motor neuron. Bilateral training can reduce the bilateral deficit whereas unilateral training has minimal effect on the bilateral deficit (Janzen, Chilibeck & Davison, 2006). Strength trained athletes who use a lot of bilateral movements in their training such as squat have been reported to shown bilateral facilitation instead of a deficit (Howard & Enoka, 1991).

In some movements, some individuals may be able to produce more force in unilateral contraction. Given that information, unilateral training may be beneficial when attempting to build muscle mass (Jakobi & Chilibeck, 2001). On the other hand, the hormone responses to unilateral movement can be much smaller in some studies. In fact, unilateral training induced local muscle hypertrophy, which can occur without increase in endogenous anabolic hormone concentration (Wilkinson et al., 2006). Changes in anabolic hormones can affect to hypertrophic adaptations. Surprisingly Jones et al. (2012) found that there was a similar testosterone response to unilateral squat and bilateral squat.

In conclusion, bilateral deficit is a phenomenon that can take place in different movements. Many mechanisms have been proposed to be behind the phenomenon. Alteration of the nervous

system and higher-order neural inhibition from the central nervous system is one of the most commonly used explanation for bilateral deficit. (Skarabot et al., 2016.)

## 4. Individual responsiveness and adaptations to strength training

As earlier told the resistance training induces adaptations in the neuromuscular system. The size of the specific adaptations between individuals' varies a lot. The same training program will induce different kinds of adaptations in different people. Individual adaptations in endurance training is well known to affect many different factors differently. For example, blood pressure, blood lipids and insulin sensitivity (Bouchard et al., 2012). On the contrast, there is only a limited data on individual responsiveness to strength training.

People react differently to resistance training. All people are unique and some might develop very rapidly whereas others may develop more slowly. Different responsiveness of human being was first noted by Sheldon et al. in 1954 who found that people with different physiques had different abilities to gain morphological adaptations in response to training. These responses might be affected by gender, age, previous training history, physical activity level and endocrine status (Descheness & Kraemer, 2002). Gender is commonly held the largest factor in this case. Higher levels of anabolic hormones and greater amount of muscle mass will affect the level of responsiveness. Ivey et al. (2000) found that men gained more during a training intervention. However according Hubal et al. (2005) responses of muscle to hypertrophic training are not gender dependent. Ahtiainen et al. (2016) have reported similar results, but also that strength responses were similar between men and women at different ages.

Hubal et al. (2005) investigated 585 men and women and used very sensitive methods like magnetic resonance imaging during a 12-week resistance training intervention. Of those 585 subjects 232 subjects showed increase in the cross-sectional area of between 15-25 %, 10 subjects gained over 40 % and 36 subjects gained less than 5 %. There were high ranges in strength gains also. It can be deduced that there is large variation between responsiveness to a certain stimulus, even though Hubal et al. (2005) did not leveled volume between subjects.

Ahtiainen et al. (2016) noted also that between 287 subjects that they analyzed there were considerable inter-individual variation in both muscle size and strength adaptations. Some individuals responded favorably in muscle size and not strength, whereas others responded in strength but not size. According to researchers it is likely that nearly all will benefit from resistance training.

Montero & Lunby (2017) found that individuals that did not response to exercise training were able to make progress when the dose of exercise was increased. On the other hand, the study focuses to endurance and intervention was done with 60 minutes of cycling. If the participant didn't make any adaptations in six week, the non-responders in each group started a second 6-week program with higher dose of exercises per week. It would seem that non-responders need just more training to induce adaptations at least for endurance point of view. Churchward-Venne et al. (2015) reported also results that supported this idea. They found that all 110 participants made gains in at least one of measures (lean body mass, muscle fiber size, strength and physical function tests). In figure 6 is shown the gains in strength and muscle fiber size. They concluded that the level of responsiveness was strongly affected by the duration of the exercise intervention, with more positive responses when they trained more. This may indicate that "non-responders" needs just more specific training.

### 4.1 Individuals hypertrophic adaptations

There are plenty of evidence that resistance training does not induce adaptations in muscle size or only minimal adaptations (Bamman et al. 2007; Mitchell et al. 2013; Phillips et al. 2013). However, that can be because of the type and quality of intervention. Ahtiainen et al. (2016) analyzed 287 subjects who undergo resistance training intervention and noted that approximately 5 % mean increase in muscle size. It is more common to be a low responder to muscle size than muscle strength (Ahtiainen et al., 2016).

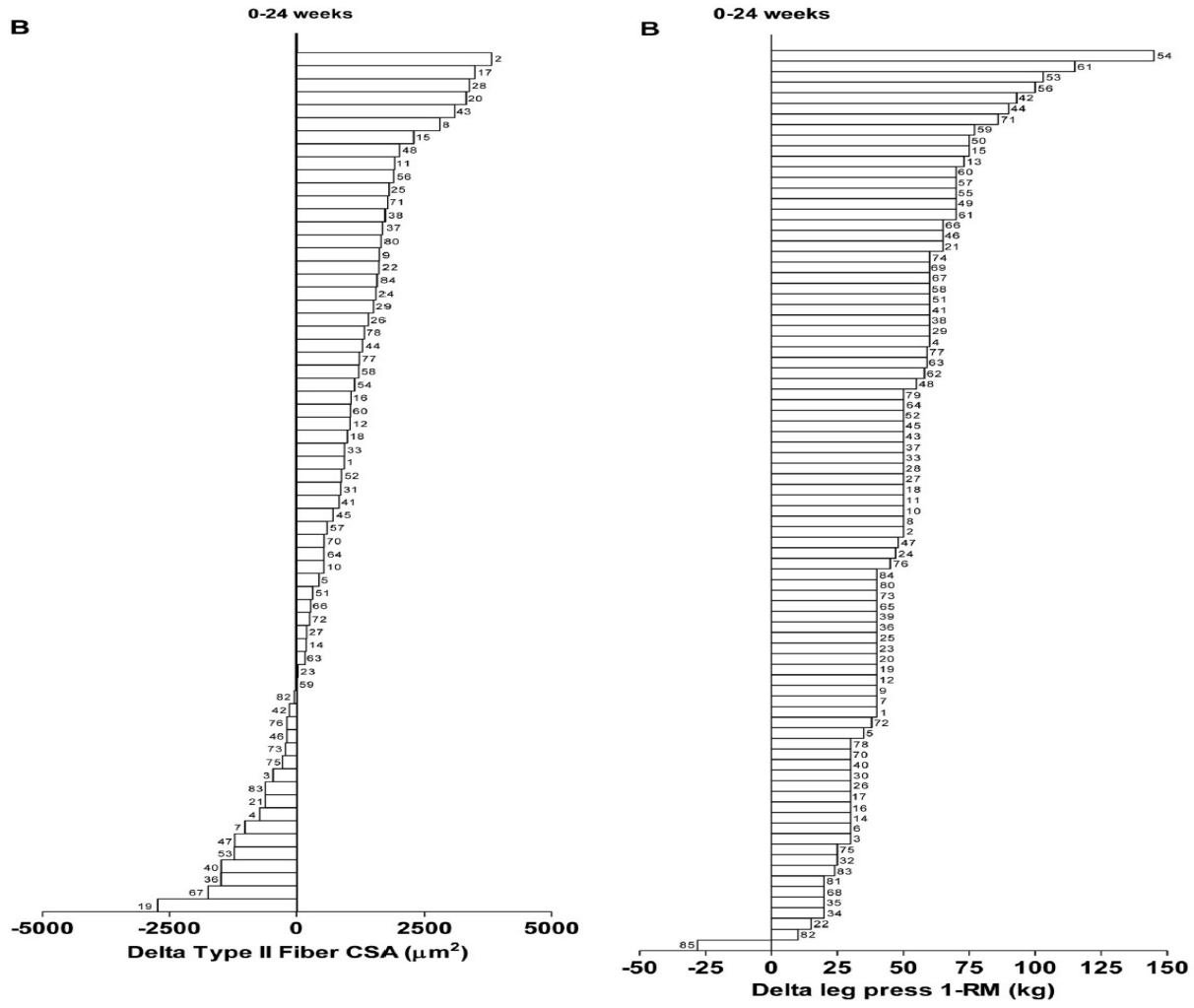


FIGURE 6: Almost everyone gained strength (right), but quite a few people's muscle fibers either didn't grow or actually shrank a bit (left) (Churward-Venne et al., 2015).

There are few differences why some gain more morphological adaptations than others. The expression of genes that code from growth factors associated with hypertrophy increase more post-exercise in the high responders (Bamman et al., 2007). In addition, high responders tend to add more myonuclei in their muscle fibers (Petrella et al., 2008). There is also a difference in the overall signaling pathways as the high responders´ cellular responses seems to be more like a growth response and in the non-responders more like a magnified inflammatory response (Thalacker-Mercer et al., 2013). Pro-inflammatory signaling increased post-training in everyone, but, however, the degree was higher in the non-responders (Thalacker-Mercer et al., 2013). Bamman et al. (2007) did 3 sets squats, leg press and knee extensions, three times per week and

all sets taken to failure. That could be a too much for a non-athletic person. There could be the body's protective system that inhibits the hypertrophy. Initial muscle damage inhibits hypertrophy at least when subjects first starts to exercise (Damas et al., 2016).

### 4.2 Individuals strength adaptations

It seems that the gains in strength during resistance training are as variable as hypertrophic adaptations and highly individual. As earlier told, Hubal et al. (2005) reported a high range of strength in their comprehensive study. Erskine et al. (2010) also noted that the changers in muscle force and physiological cross-sectional area varied substantially between individuals. They discussed that there is a possible greater inter-individual variability in specific tension response compared to that of maximal voluntary contraction as previously mentioned Ahtiainen et al. (2016) reported 21 % mean increase in muscle strength in their study. Churchward-Venne et al. (2015) found that all subjects, who were over 64 years old, except one, gained at least some strength. All data taken together it seems that nearly everyone will get stronger when they start to train, however, hypertrophic adaptations does not occur so easily and almost every study had some non-responders to hypertrophy. However, Erskine, Fletcher & Folland (2014) showed that resistance training induced muscle hypertrophy can explain notable proportions of the inter-individual changes in isometric and isoinertial strength. They based their conclusion on the correlations between the change in muscle volume and changes in isometric and isoinertial strength. It should be noted that they found only minor changes in neuromuscular activation, which could mean that there was a limited capacity for neural adaptations to resistance training in these subjects or the training was not sufficient

Narici et al. (1989) did 60 days of unilateral strength training and found that hypertrophy produced by strength training accounts for 40 % of the increase in force while the remaining 60 % seems to be attributable to an increased neural adaptations and maybe to changes in muscle architecture.

## 5. Hypertrophic strength training

Hypertrophy adaptations can be made with a wide range of different exercise programs. However, some programs might lead to greater hypertrophy than other programs. Repeated resistance training is shown to lead to hypertrophy in a wide variety of populations (Peterson, Rhea & Alvar, 2005). Many factors can be altered to change the outcome of the resistance training programs, for example load, intensity, velocity, volume, interest rest interval, type of movement, amount of muscle mass involved in the movement etc. Table 1 gives simple recommendations when progressing from novice lifter to advance lifter. This chapter is going to focus on finding the most optimal hypertrophy protocols.

TABLE 1: Recommendations for progression during hypertrophy training (Kraemer & Ratamess, 2004).

| | Novice | Intermediate | Advanced |
|---|---|---|---|
| Muscle action | ECC and CON | ECC and CON | ECC and CON |
| Exerc. selection | Single and multiple-joint | Single and multiple-joint | Single and multiple-joint |
| Exerc. order | Large < small muscles<br>Multi < single<br>High < low intensity | Large < small muscles<br>Multi < single<br>High < low intensity | Large < small muscles<br>Multi < single<br>High < low intensity |
| Loading | 60–70% 1 RM | 70–80% 1 RM | 70–100% 1 RM with emphasis 70–85% |
| Volume | 1–3 × 8–12 reps | Multi sets × 6–12 reps | Multi sets × 1–12 reps with emphasis 6–12 |
| Rest intervals | 1–2 min | 1–2 min | 2–3 min—heavy<br>1–2 min or less for others |
| Velocity | Slow to moderate | Slow to moderate | Slow, moderate, and fast |
| Frequency | 2–3 d·wk$^{-1}$ | 2–4 d·wk$^{-1}$ | 4–6 d·wk$^{-1}$ |

<, indicates the preceding exercise is to be performed before the succeeding exercise.

A common belief is that bodybuilding type of training would be the best way to induce muscular adaptations. Bodybuilding type of training tends to have more repetitions and shorter rest periods. It seems that bodybuilding (3 sets of 10 RM with 90 seconds rests) and powerlifting (7 sets of 3 RM with 3-mnute rests) volume-equated training induce similar changes in muscular size, but powerlifting is superior for gaining maximal strength (Schoenfeld et al., 2014). In that study, the volume was equated. Volume can be defined as total repetitions, sets and load performed in a one training session (Schoenfeld, 2010) There are a lot of evidences that volume

is a very important variable when target is to induce morphological adaptations. Krieger (2010) conducted a meta-analysis, which supported this hypothesis. When there were multiple sets, it was associated with 40 % greater hypertrophy effect size when comparing only to one set in both trained and untrained subjects. On the other hand, low volume strength training can also lead to hypertrophy (Carpinelli & Otto, 1998, Hass et al., 2000), but the acute endocrine responses are still depended on the volume and that have a big impact on hypertrophy. Gotshalk et al. (1997) compared one set to multiple set and measured acute hormonal response. The response was bigger with multiple sets and shorter rest periods. Schwab et al. (1993) noticed that only after fourth set of squat the testosterone significantly increased, which could indicate benefit of higher volume. There is also a large amount of evidence that higher volume produce greater growth hormone responses than a single set protocols (Mulligan et al., 1996). Ahtianen et al. (2003) found that trained individuals had greater acute growth hormone response compared to untrained people (figure 7), that would indicate that acute hormonal response have some part in muscle hypertrophy. They also found that changes in acute total testosterone response after the intervention correlated with muscle CSA ($r=0.76$, $P<0.05$).

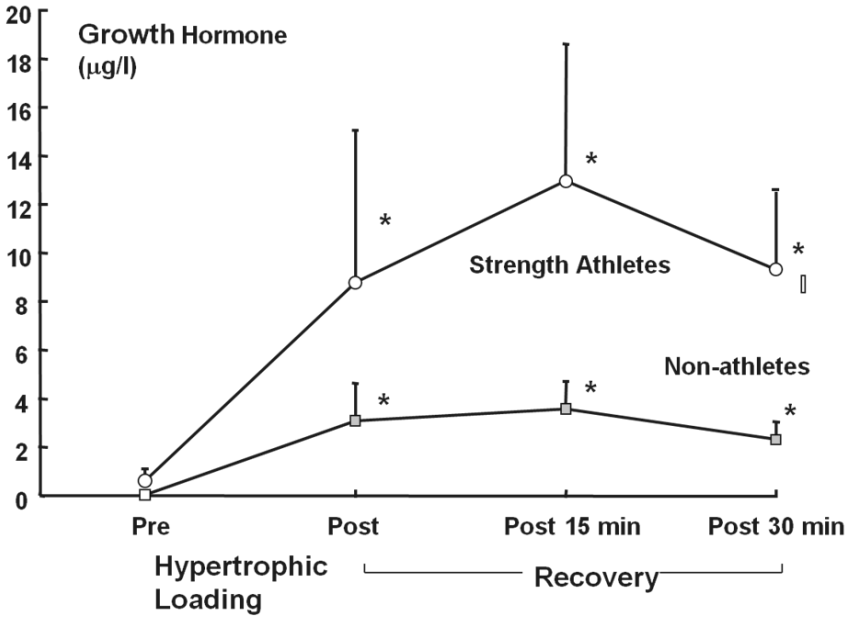


FIGURE 7: Acute hormone response after hypertrophic loading. Trained subjects demonstrated much larger inter-individual variation compared to untrained people. Adapted from Ahtiainen et al. (2003)

It is not clear why the higher-volume induce more hypertrophy, is it because of bigger muscle tension, metabolic stress or a muscle damage. However, one answer could be the acute endocrine responses. Schoenfeld et al. (2018) divided 34 men either low-volume group (one set), moderate-volume group (three sets) or high-volume group (five sets). All groups increased strength and endurance pre-to-post, with no significant between-group differences. Also the muscle size was increased in all groups, however the higher volume were seen to favor hypertrophy in elbow flexors, mid-thigh, and lateral thigh. Authors concluded that muscle hypertrophy follows a dose-response relationship and larger increase in muscle mass is achieved whit higher volume. Also Ralston et al. (2017) displayed in their meta-analysis that moderate to high weekly training volume are much more better for strength when compared to lower volumes. For trained subjects there are crossing results Marshall, McEwen & Robbins (2011) found higher volume better for strength, whereas Ostrowski et al. (1997) found no differences between low-. moderate- and high-volume training. It might be, that volume is crucially important for hypertrophy but not for strength. As was the case in previously mentioned Schoenfeld et al. (2018) study. Schoenfeld, Ogborn & Krieger (2017) found that hypertrophy clearly favors higher volume (figure 8).

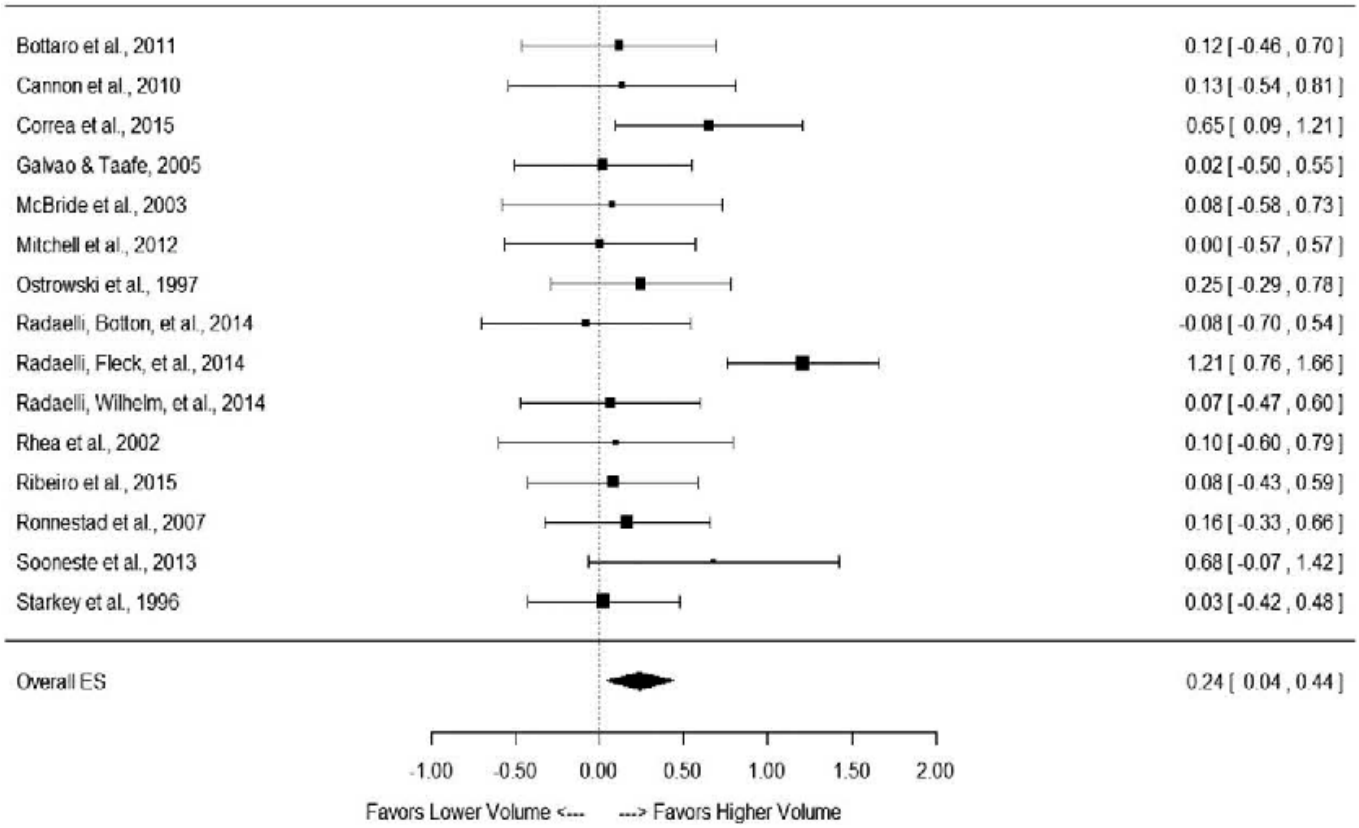


FIGURE 8: Schoenfeld, Ogborn & Krieger (2017) meta-analysis displayed that hypertrophy favors higher volume. The data shown are mean ±95% CI; the size of the plotted squares reflect each study's statistical weight. Abbreviations: ES (effect size.). Overall result is at the bottom.

Mechanical loading seems to be an effective way to induce hypertrophic response (Schiaffino et al., 2013). Mechanical loading stimulates protein synthesis in muscles (Seynnes, de Boer & Narici, 2007). Mechanical loading and stress alone can stimulate mTor (Hornberger et al., 2006). MTor, which is also known as mammalian target of rapamycin, is a pathway that is held particularly important to muscle anabolism. In addition to mechanical loading the metabolic stress can be important factor for enhancing hypertrophic response. There is some evidence that metabolic stress is an important factor and processes associated with fatigue contribute to the strength training stimulus (Rooney et al., 1994; Schott et al., 1995). In contrast Folland et al. (2002) did not find evidence that fatigue would be necessary for strength gains. In their study, they did not measured hypertrophy adaptations. Figure 9 shows the proposed mechanisms by which metabolic stress can affect to muscle hypertrophy. In conclusion, mechanical stress has

unquestionably a main role in post-exercise muscle growth, but there is a growing amount of evidence that metabolic stress also contributes to muscle hypertrophy (Schoenfeld, 2013). For achieving maximal hypertrophy, it is recommended to include both loading types in training programs.

To gain maximal amount lean body mass, it might be optimal to combine in the training program mechanical tension, muscle damage and metabolic stress. Because they all play a role in an exercise-induced muscle growth. (Schoenfeld, 2010).

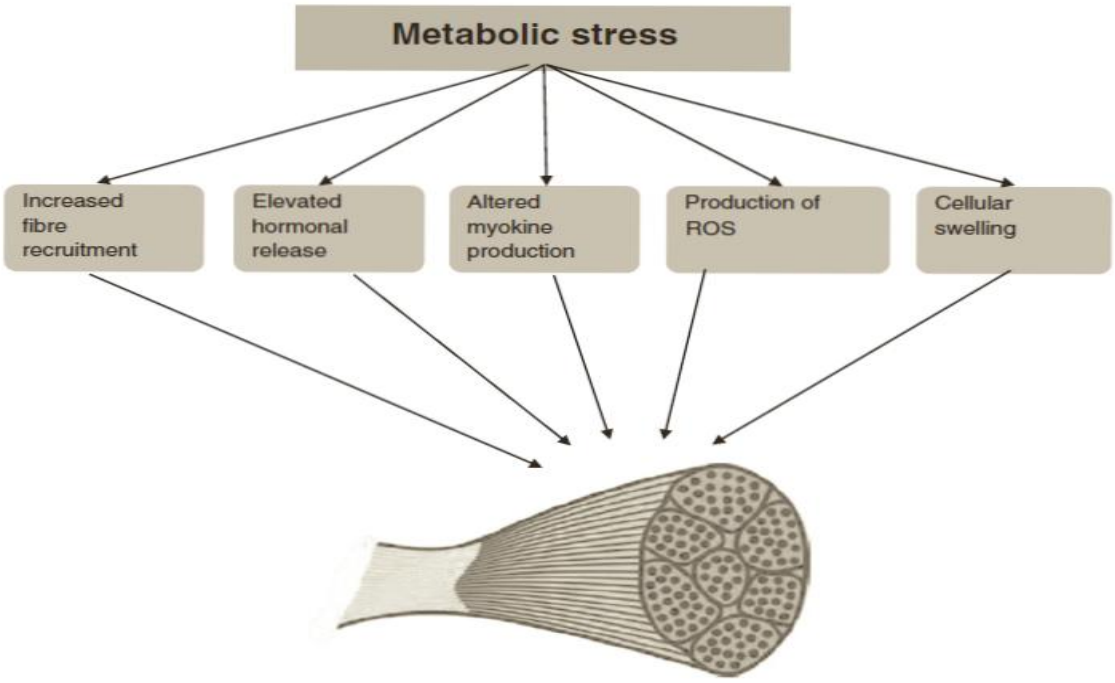


FIGURE 9: Mechanisms, which resistance training induced metabolic stress can influence to muscle hypertrophy. ROS stands for a reactive oxygen species. (Schoenfeld, 2013)

Training to muscular failure is a much used technique to maximize hypertrophic adaptations. Muscular failure can be defined as the point that muscles cannot produce necessary force to lift a given load (Schoenfeld, 2010). Training can be done to concentric failure or eccentric failure. Concentric failure is more commonly used. Training to failure may activate a bigger number of motor units (Willardson, 2007), and may be needed to maximize the hypertrophy (Willardson, 2007). In a fatigued state the lifter needs to recruit more motor units in order to continue activity. Training to failure may also grow metabolic stress, when activity is continued in anaerobic conditions. Bigger metabolic stress can elevate the hormonal release. Linnamo et al. (2005) noticed that 5 sets of 10 reps to failure significantly increased serum growth hormone in men and women. The same load not performed to failure did not increased significantly serum hormones. Also Ahtiainen et al. (2003) suggested that acute increases in serum testosterone concentrations due to a single resistance training session may have a major impact for training-induced muscle hypertrophy and strength development. It should be noted that training to failure is more demanding to the neuromuscular system and should be used periodized to avoid an overtrained state (Schoenfeld, 2010).

### 5.1 Resistance training frequency

Resistance training frequency has been an important target of research recently. Increases in muscle protein synthesis after heavy resistance training has been shown to last 24-48 hours after an resistance training session (Damas et al., 2015 & Damas et al., 2016). And that's why it has been rationalized that higher frequency would be optimal for hypertrophy. Barcelos et al. (2018) showed that there were similar results in muscle hypertrophy when trained two, three or five times per week in untrained subjects. Even though total training volume was higher for higher frequencies groups. When matching the volume, there has been similar results both in untrained and trained individuals (Benton et al., 2011; Brigatto et al., 2018; Candow & Burke, 2007).
Damas et al. (2018) did interesting within subject study in untrained people. One leg did resistance training five times per week, whereas the contralateral leg performed resistance training two or three times per week. The results showed a large intersubject variability, but there were no greater responses to muscle hypertrophy and strength with higher resistance training

frequencies . In fact there were no difference when manipulating frequency in 31.6 % of the subjects for muscle hypertrophy and 57.9 % for muscle strength. Higher frequency also meant higher volume in this study. Authors speculated that the total volume needed for maximal gains is individual-dependent and larger amount of volume could even impair gains in some subjects. In well-trained men Scoenfeld et al. (2015) showed that hypertrophy gains were bigger with higher weekly resistance training frequencies. Interestingly, there were no significant differences in maximal strength. Volume was equated between groups. McLester, Bishop & Guilliams (2000) reported that strength gains and hypertrophy favored higher frequencies, even though the results were not statistically significant. The subjects in that study were experienced with resistance training.

### 5.2 Periodizing hypertrophy training

To maximize hypertrophic adaptations training programs should target to produce significant metabolic stress while maintaining a decent amount of muscle tension. Repetition range can be to 6 to 12 reps per sets. Rest intervals should be low enough to produce metabolic stress, but high enough that enough mechanical tension can be done. Depending on the movement, but rest sets 60 - 90 seconds has been proposed. (Schoenfeld, 2010.) On the other hand, Ahtiainen et al. (2005) did not found any difference between two or five-minute rest sets. There should be variation in exercise selection to ensure maximal stimulation of all muscle fibres. Multiple sets should be used and some sets should be carried to the concentric failure. Concentric contraction should be done as fast as possible and eccentric contraction much slower (2-4 seconds). Volume should progressively be increased over a given time to ensure progression in mechanical tension. Brief tapers or cessation from training can be included into programs. (Schoenfeld, 2010.)

The periodization and its effects on hypertrophy is not really well examined. Stone, O'Bryant & Garhammer (1981) studied periodized and non-periodized programs and found the periodized program much better for hypertrophy. On the other hand the hypertrophy was measured with under water weighing, which sets the results to a new light. Baker, Wilson & Carolyn (1994) found no differences between periodized and non-periodized programs. Hypertrophy was

measured with skin fold measurements. Whereas Monteiro et al. (2009) found difference, not statistically significant, in favor of periodized programs. Again the methods were questionable, because the hypertrophy was measured with skin fold test.

There is only one study that has evaluated should hypertrophy training programs be periodized (Grgic et al., 2017). In that systematic review authors found that both periodized and nonperiodized training programs can be used to achieve muscular hypertrophy and that similar hypertrophy effect was gained using either approach. In addition, the authors speculated that there is no differences between linear periodization and nonperiodization in untrained individuals. However, current evidence in insufficient to say whether periodization aproach should be used in trained individuals. Fu et al. (2017) compared group who trained for 4-days per week and group who trained 2-days per week. Their weekly volume was equated and the subjects were trained males. They trained for six week and after that both groups improved performance, but only the 2-days group increased upper body hypertrophy and improved body composition. These results indicates that for hypertrophy trained males would need higher volume dose per one training session. The authors discussed that higher volume generates bigger metabolic stress, which is an important stimulus for anabolic response to happen. Moreover, high volume routines have associated with greater acute post increase of testosterone and growth hormone.

Periodized programs will work better than non-periodized programs for strength (e.g. Ahmadizad et al., 2014; Monteiro et al., 2009; Willoughby, 1993). When strength is raised there could be possibility of larger gains also in hypertrophy, mainly because of the greater mechanical tension aroused by increased forces, but this is yet to be proven.

## 6. Purpose of the study

- What kind of individual differences are there in strength training adaptations between subjects? Can we find (fast) responders or low-responders?
- How do the different responder groups behave during the detraining phase?
- Are there differences in strength gains between the bilateral and unilateral groups? Differences in hypertrophy and/or in maximal muscle activation (surface EMG) of the trained muscles?
- To what extent do the strength gains are due to changes in maximal muscle activation or hypertrophic adaptations?

### 6.1 Research hypotheses

From the current literature, it seems clear that individual human beings responds differently to strength training. However, as far as it is known there are no studies about responders' behavior during the detraining phase. We have no information about that, and it can be assumed that there will be some kinds of individual differences during the detraining. Regarding to the unilateral and bilateral training, there is some conflicting evidence. In some cases, unilateral training is as effective as bilateral training, but sometimes not. It is assumed that they are equally effective to build muscle and strength, if the volume is matched between the groups.

The hypotheses were as follows:

1. There will be different individual responders and we can divide them into different groups.

2. Individual responders will also behave differently during detraining. Some subjects might lose muscle mass or strength faster than the other ones.

3. Both unilateral and bilateral training can be used to build muscle mass.

## 7. Methods

### 7.1 Subjects

Twenty-six healthy young man aged between 19-30 from the city of Jyväskylä were recruited to participate in the study. Recruitment was done through advertisements. Ads were placed to different places around the university campus. Ads were published in a local newspaper, websites of the University of Jyväskylä, on the social media and on the university staff- and student e-mail lists.

The exclusion criteria included cardiovascular diseases, problems with the respiratory system, impaired musculoskeletal and /or endocrine functions, diabetes, or any other condition that may limit performing the testing or training intervention. Subjects needed to be recreationally active and they could not have systematic or progressive strength training background. The great amount of endurance training was also an exclusion criteria.

All recruited participants attended a screening for resting ECG and resting blood pressure. Furthermore, they were interviewed about their general health and motivation towards the study. Cardiologist went through the participants' data before they were given a position as a subject. Overall 32 participants went through pre-screening, from those participants, 26 subjects started the study. Each subject was informed of all potential risks and discomforts of the study, and the possibility to drop out from the research project at any time. After that, they signed an informed consent document. Two subjects dropped out from the study because of the health problems. Table 2 presents anthropometrics of subjects in each group. The study was conducted according to the declaration of Helsinki. The Ethics Committee of the University of Jyväskylä approved the study.

TABLE 2: Physical characteristics of the subjects.

| Group | n | Age (years) | Height (cm) | Weight (kg) |
|---|---|---|---|---|
| **Unilateral** | 13 | 24.9 (±4.1) | 179 (±4.6) | 75.2 (±9.8) |
| **Bilateral** | 13 | 24.2 (±3.5) | 182 (±9.2) | 78.8 (±10.2) |

### 7.2 Experimental design

In order to study the effects of unilateral and bilateral training and detraining a total of an 18-week long intervention plan was created.

The study started in September 2017 and ended in February 2018. The study included two measurement points before the intervention. The actual intervention was ten weeks of progressive hypertrophic resistance training and six weeks of detraining (Figure 10).

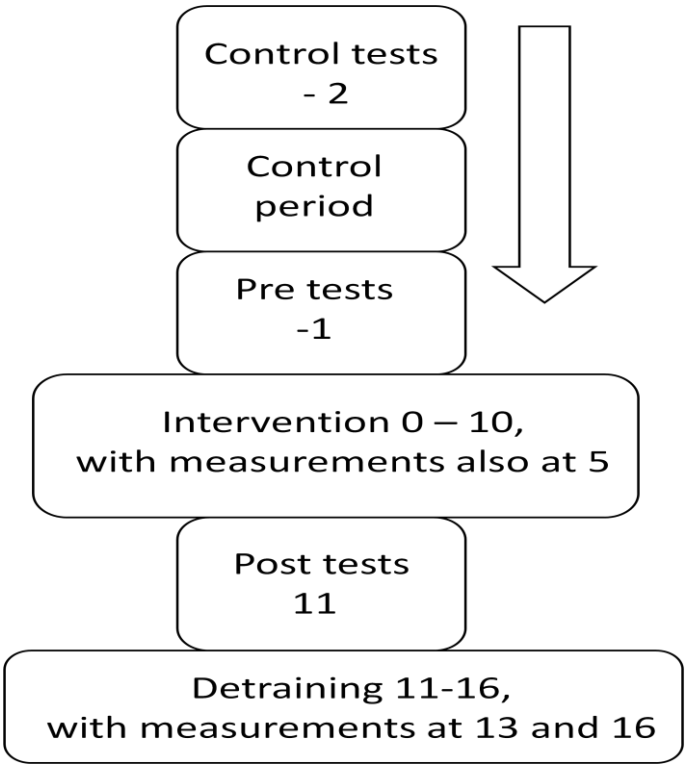


FIGURE 10: Overview of the experimental design of the study. The numbers refer for weeks.

Subjects were tested both bilaterally and unilaterally all other times except at the midtests. Tests in mid tests was only conducted according to subjects own group's training. In order to minimize the bilateral training for unilateral group and vice versa. That was because we wouldn't want them to do nothing else than their own group movements. The testing sessions for the individual subjects were performed at the same time of day during the study. Bilateral deficit was calculated from isometric leg press according Howard & Enoka (1985). After that the subjects were divided into two groups according their bilateral strength level and the bilateral deficit. During the intervention the subjects trained only either unilaterally or bilaterally. Table 3 presents both groups strength levels and bilateral deficit before the intervention.

The measurement order in the testing situation was always the same. The subjects started with left leg/arm and then did right leg/arm and finally the bilateral version. The order was following: Inbody, Ultrasound, preparing the subject and putting electrodes on, warm-up, isometric leg press, isometric knee extension, electrical stimulation, isometric bench press, counter movement jumps, dynamic bench press and dynamic leg press. The testing situation for individual subject was always conducted at the same time of day during the experimental period.

TABLE 3: Maximum bilateral isometric strength and bilateral deficit of the both group before the intervention.

| **Group** | **Maximum bilateral isometric strength** | **Bilateral deficit** |
|---|---|---|
| **Unilateral** | 2968 N (±513) | -15.2 % (±13.1) |
| **Bilateral** | 2951 N (±658) | -15.2 % (±4.9) |

### 7.3 Intervention

The intervention lasted 10 weeks and subjects trained three times per week. Training took place on Wednesday, Friday and Sunday. We had some flexibility with training times and if the subject couldn't participate on those days, extra training session were reorganized. Overall we had 30 training sessions during the intervention. Average participation times in training sessions were 29.1±0.93. The mean participation rate was 96.9 %. Only one person missed more than two times trainings during the whole intervention. All of the intervention's training sessions were supervised by an expert who was always someone from the study group.

The subjects received protein and carbohydrate supplementation after every training session. They were given protein bar, which included 203 kcal, 7 g of fat, 20.1 g of carbohydrate and 19.6 g of protein per one bar. They were also given an individual example of the nutritional plan before the training intervention, and they were advised to follow it during the intervention. Implementation of the nutritional plan was not controlled.

The training program consisted three medium weeks, four hard weeks and then again three medium weeks. The volume of training increased over the first seven weeks, after that the volume remained approximately the same and the intensity rose. Training volume was equated between the groups. Volume equating is not a simple thing to do in unilateral and bilateral training. The best solution was that the unilateral group did the same amount of reps per leg than bilateral group. For example, if the bilateral group did 5x10x70% of their bilateral maximum, the unilateral group did on their left leg 5x10x70% of their unilateral left leg maximum and on their right leg 5x10x70% of their unilateral right leg maximum. The absolute loads between the legs could naturally be slightly different.

Rest time between the main movements was 3 minutes and between the accessory movements 60s. The unilateral group had 2-3 min rest between limbs in main movements. They did accessory movements without the rest when they changed limbs. Rest time between the sets stayed the same during the whole intervention. The first session of the week, the subjects did

dynamic leg press 5x10xRM, the weights were determined so that failure occurred during that training. The rest of the reps were carried out by forced repetitions, when needed (Figure 11). On the same session they did isometric knee extension (90 degree) 5 seconds of work and 15 seconds of rest. They did it three times and then had 90 seconds of rest. The same thing was done with isometric knee flexors.

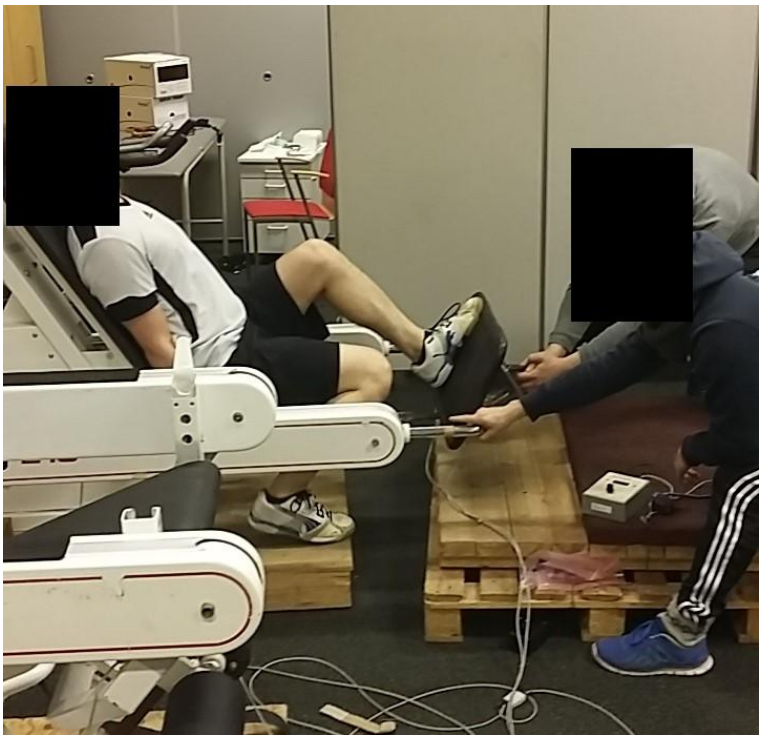

FIGURE 11: 5x10RM was carried out by using the forced repetitions, if the subjects were not able to perform the last rep(s) of the sets by themselves.

After the mid test the isometric bench press was added to the program and was done in the same fashion as the others. They did also lat pulldown and some core exercises was added to that training. The results of the isometric training was always shown to the participant and he was then encouraged to go over the previous number. This was done to ensure the progression of isometric training. Isometric training covered approximately 5 % of the whole volume of the intervention. On the second training of the week, the subjects started from the legs and moved on to the upper body. On the third session of the week it was vice versa to minimize the order effect.

The training program consisted leg press and bench press three times per week. Bench pressing (figure 12 A) was done in smith, because the unilateral group would not been able to do it with free weights. Unilateral leg pressing and bench pressing can be seen in figures 12 B and 13. The training program included also knee extension, knee flexion, dumbell benchpress, seated french press, elbow flexion and extension movements, horizontal row and core movements, all done either bilaterally or unilaterally. The tempo of the movements was controlled by the supervisor. The few comprehensive examples from the training program can be found in the attachments.

FIGURE 12 A & B: On the left (12 A) bilateral bench press training is going on and on the right (12 B) a subject is doing unilateral bench press training.

FIGURE 13: Subjects trained both horizontal and more upright leg press to ensure different training angles during intervention. The subject is performing unilateral leg press in the upright leg press.

### 7.4 Data collection and analyses

#### 7.4.1 Anthropometric and muscle mass measurements

**Body mass and height.** Body mass was measured every second week during the intervention with the electronic scale. Body height were measured with the wall-mounted measurement scale.

**Whole body composition and lean body mass.** Dual-energy X-ray Absorptiometry (DXA) (LUNAR Prodigy Advance, GE Medical Systems, Madison, USA) was used to measure whole body composition and lean mass and those changes after the intervention. DXA were done only at pre and post of the intervention, because of the adverse radiation. Software's general recommendations were used to isolate legs and arms from the trunk (enCORE 2005, version 9.3). The legs were secured by using Styrofoam and elastic straps and the arms by rice bags to prevent any movement during the scan. The subjects came to DXA-scan in a fasted state and they had been 24 hours without training. They could have one cup of water in the morning, before the scan. Prior the measurement, all metal objects were removed from the subject and subjects were instructed to be in their underwear's. The same investigator performed all measurements and analyses.

Bioelectrical impedance (InBody 720 body composition analyzer, Biospace Co. Ltd, South Korea) measurements were done more frequently than DXA, overall six times. Subjects stood on the device with the arms abducted little a bit to side to ensure that the arms and trunk would not were in contact.

**Muscle cross-sectional area.** Vastus lateralis cross-sectional area (CSA) was assessed using B-mode axial-plane ultrasound (model SSD-a10, Aloka Co Ltd, Japan). Subjects laid supine with the legs strapped to polystyrene moulds (Figure 14 A). Anatomical landmarks for CSA determination were measured from the middle section between the joint space on the lateral side of the knee and to the greater trochanter. 40 % of femur length was marked and line was drawn from the lateral to medial diaphysis of the right tight. A 10 MHz linear-array probe (60 mm

width) was moved very slowly and continuously manually along the marked line. A custom made probe support was used to assure perpendicularity and the extended-field was on. Great care was taken to not compress the muscle tissue. Ultrasound images were combined automatically to a panorama view in a device. Three panoramic CSA were measured and the mean of those was used in analyses. The CSA were then determined with Image-J - program (version 1.37, National Institute of Health, USA). Within Image-J the analyze was done with polygon selection - tool, which enabled manual tracing along the border of the vastus lateralis muscle. The investigator followed the inner line of fascia and when the fascia was not seen, the predicted route was chosen according previous images. Great care was used to complete analyses. The same investigator performed all measurements and analyses.

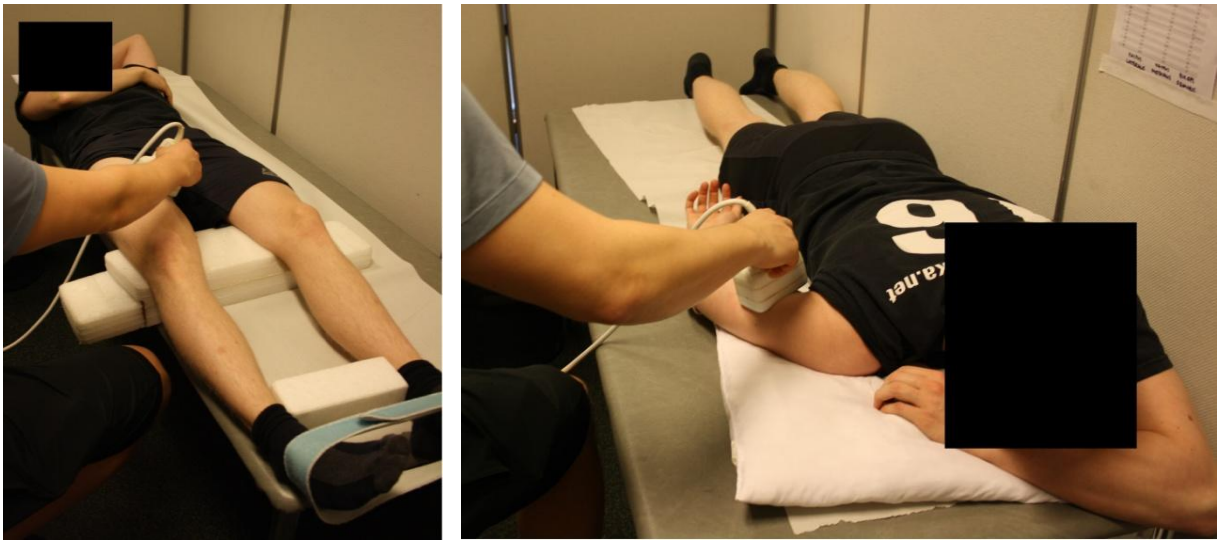

FIGURE 14 A & B: On the left (A) the investigator is measuring vastus lateralis' CSA and on the right (B) measuring triceps brachii thickness with the ultrasound.

Triceps brachii thickness was obtained when the subjects laid prone. Anatomical landmarks for triceps brachii thickness determination were measured from acromion to medial condyle of humerus. 50% of the length were marked and from that point the measurements were made (Figure 14 B). Thickness was measured as the distance from superficial layer of fascia and deep aponeurose. Three images were measured and those mean was used in analyses.

**Echo intensity.** Echo intensity was assayed by mean grayscale analysis using the standard histogram function in Image-J. The mean echo intensity was used in analyses. It's number between 0 and 255, where complete black is 0 and complete white is 255. In vastus lateralis the echo intensity as determined from the same area as CSA. While in triceps brachii the echo intensity was assayed in the same area as the thickness. This procedure has some proves that it is reliable (Harris-Love et al., 2016; Caresio et al., 2014; Bartolomei et al., 2017).

### 7.4.2 Muscle activity and electrical stimulation measurements

**Electromyography (EMG).** Muscle activity was recorded during the isometric strength testing from the agonist muscles vastus lateralis (VL) and vastus medialis (VM) of the right leg. Skin was prepared by shaving, scraping and disinfecting. After that, the electrodes were placed according to SENIAM guidelines (Hermens et al., 2000). On the first time, the positions of the electrodes were marked on the skin by ink dots to ensure always the same location of electrodes in each test during the study. Electrodes were bipolar Ag/AgCl electrodes with 5 mm diameter and 20 mm inter-electrode distance.

During the measurements the raw signals were amplified (500 gain) at a bandwidth of 10–500 Hz, the sampling frequency were 3000 Hz. After that, the signals went through transportable pack to the receiving box (Telemyo 2400R, Noraxon, Scottsdale, USA), and then to an AD converter (Micro1401, Cambridge Electronic Design, UK) and recorded by Signal 4.04 software (Cambridge Electronic Design, UK). EMG signals were analyzed by a customized script. Maximum values were obtained from the contraction time period of 500-1500 ms.

**Quadriceps muscle electrical stimulation.** Constant current stimulator (Digitimer Stimulator Model DS7AH; Digitimer Ltd., United Kingdom) was used to stimulate the quadriceps muscle. Four, galvanically paired electrodes (6.98 cm V-trodes, Mettler Electronics Corp, USA) were placed on the proximal and middle regions of the quadriceps muscle, so that they would cover up muscle CSA as much as possible. Figure 15 A & B demonstrate the placement of electrodes and the set-up for resting stimulation. Skin under the electrodes was shaved and disinfected.

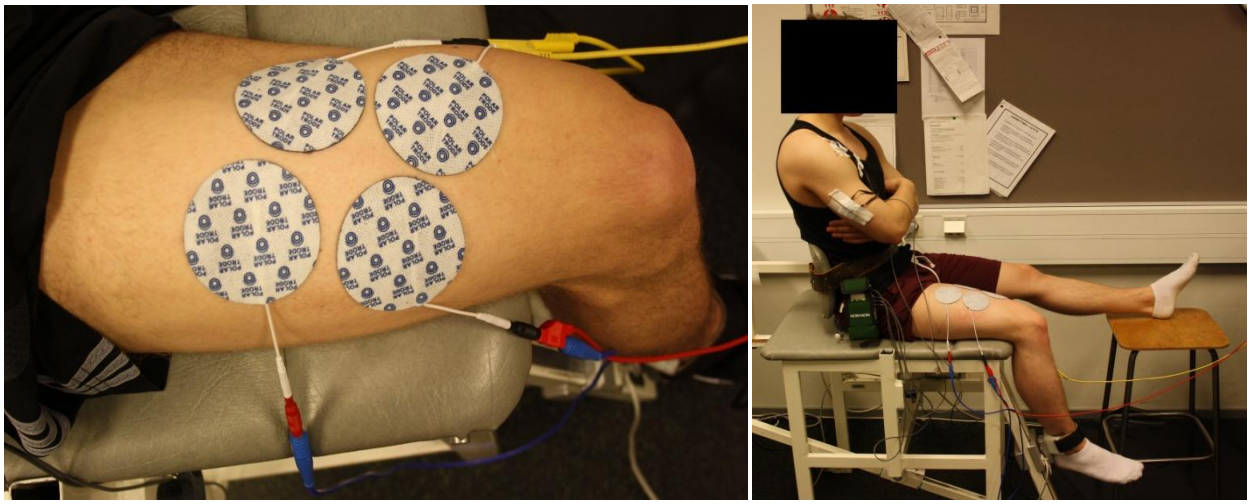

FIGURE 15 A & B: On the left (A) is the placement of electrodes on the skin of the right quadriceps muscle. On the right (B) is the set-up for resting stimulation.

Resting stimulation was done first. The subjects sat on the custom made chair with the knee angle of 107. They were strapped in and the left leg was placed on a chair, so that it could be relaxed. Hands were crossed in the lap. Single 1ms pulses were given by a constant-current stimulator until a force plateau was found. After that, the maximum voluntary contraction were produced and during that an additional 25 % of stimulation was added to the identified current. During the MVC hands were instructed to keep on the side of the bench. So the stimulation was given during the plateau of peak torque and then one more pulse 2 sec after contraction to assess voluntary activation. The subjects were given three trials and one minute rest between them. Voluntary activation level (AL) was calculated according Bellemare & Bigland-Ritchie (1984): Activation level %= [1 – (Pts/Pt)] × 100, where Pts is the difference between the voluntary torque and the stimulation helped torque, and Pt is the resting twitch after the maximum voluntary contraction.

### 7.4.3 Blood samples

Blood samples were collected from antecubital vein via sterile techniques. Blood samples were drawn into serum tubes (Venosafe, Terumo Mediacl Co., Leuven, Belgium) by a qualified lab technician. Resting serum blood samples were obtained in the morning in the fasted state for the determination of basal hormone concentrations. The subjects fasted approximately twelve hours. The subjects could drink a class of water before coming to the blood collection. All food and other liquids were prohibited. The collected blood were held for 15 min at room temperature before they were centrifuged for 10 minutes at the speed of 3500 rpm (Megafuge 1.0R, Heraeus, Germany). Serum samples were then put it in the refrigerator (-80°C) and stored for future analysis. Serum testosterone (TT), serum cortisol (C), serum growth hormone (GH) and serum sex hormone binding globulin (SHGB) were analyzed from the samples. Analyzes were accomplished by immunomeric chemiluminescence techniques (Immulite 2000) and hormone specific immunoassay kits (Immulite, Siemens, Illinois, USA)). Analytical sensitivity was 0.01 ng/mL for growth hormone, 0.5 nmol/L for total testosterone, 0.02 nmol/L for SHBG, 5.5 nmol/L for cortisol and 0.05 mlU/mL for LH. Intra- and Inter-assay reliability (CV%) were within acceptable limits (Total testosterone = 8.3%, Cortisol = 6.1%, SHBG = 2.5% and LH = 3.6%).

### 7.4.4 Isometric strength testing

**Leg extension.** Maximal bilateral and unilateral isometric leg extensors strength was measured on the custom-built horizontal leg press (Department of Biology of Physical Activity, University of Jyväskylä) at a knee angle of 107 ° (Figure 16 A and B). The subjects were instructed to push "as fast and hard as possible" and maintain their force levels as long as they receive encouraging (approximately three seconds). They were also instructed to keep their back and pelvis in contact with the bench throughout the movement and also to grasp the handles in the side. Subjects has at least three trials, but if the force rose more than five percentage, they were given another trial.

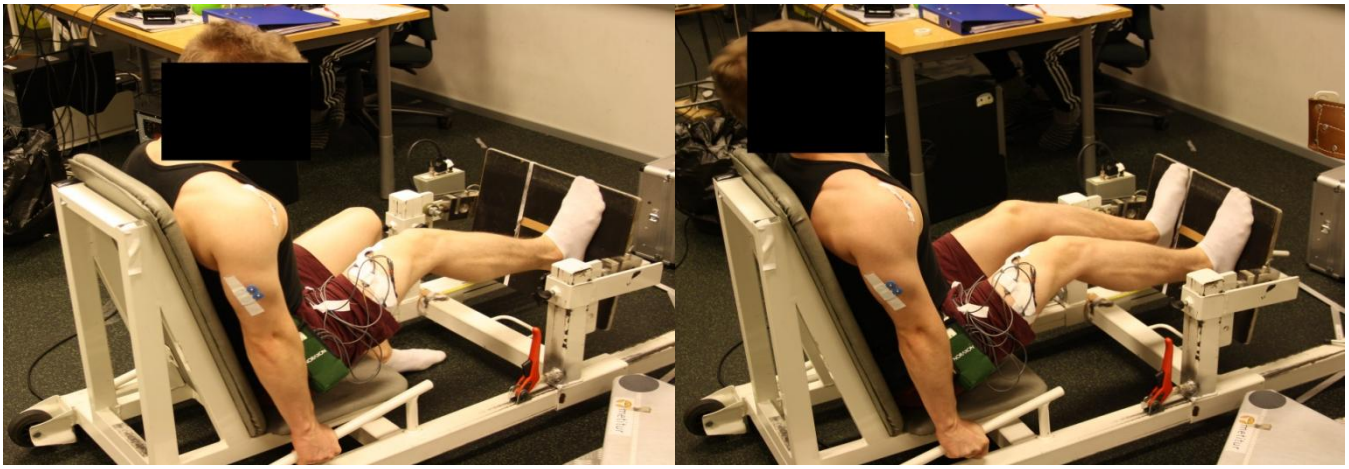

FIGURE 16 A & B: A: On the left, unilateral isometric leg extension testing. B: On the right, bilateral isometric leg extension testing.

**Upper body extension.** Elbow and shoulder joints unilateral and bilateral isometric extension strength were measured with isometric bench press. Subjects laid in a custom made bench (Department of Biology of Physical Activity, University of Jyväskylä) and were tied up in a bench from the upper body, so that any lateral movement wouldn't happen. Elbow angle was set to 90° (Figure 17) Subjects were instructed to fill up the lungs and press just a little bit, because there were some looseness in the device. They were given at least three trials, but if the force rose more than five percentage, they were given another trial.


FIGURE 17: Unilateral maximum isometric bench press testing.

### 7.4.5 Dynamic strength and power testing

**Bench press.** Bench press was done with the same width than isometric bench press. The investigator placed subjects hands with a tape measure to the bar, so that the elbow angle would be 90°. Subjects were instructed to lower the weights evenly and lift the bar after the voice command. The stop on the chest was one second. The weight was progressively raised using 2.5kg increments, until the subjects could no longer lift the load. Figure 18 demonstrates the stop position of the bar.


FIGURE 18: The stop position of the dynamic bench press testing.

**Bench press power (W).** Power was measured after the maximum bench press tests. It was done with the 30% load from 1RM. Myotest Performance Measuring System (Myotest, SA, Sion, Switzerland) instrument was mounted to a barbell to measure the force and power production in the bench press exercise. When fixed on the bar in the vertical axis, the Myotest has been proved to be a valid field instrument for measuring force and power in commonly used exercise movements (Comstock et al., 2011). Myotest made a sound mark, and from that the subject laid the barbell above the chest. From the second sound mark from the machine, the subject pushed the barbell up as fast as he could. Three trials were performed and the best was used in analyses. If the result improved more than five percentage, they were allowed extra trial.

**Leg press.** Maximal unilateral and bilateral concentric maximum force was measured in the leg press (David 210, David Health Solutions Ltd, Helsinki, Finland). Subjects' starting position was seated in the device with the knee angle of 60°. There they started with a concentric press. They were required to lift the load to a fully extended position. The weight was progressively raised using 5kg increments, until the subjects could no longer lift the load. Figure 19 demonstrates the starting position.

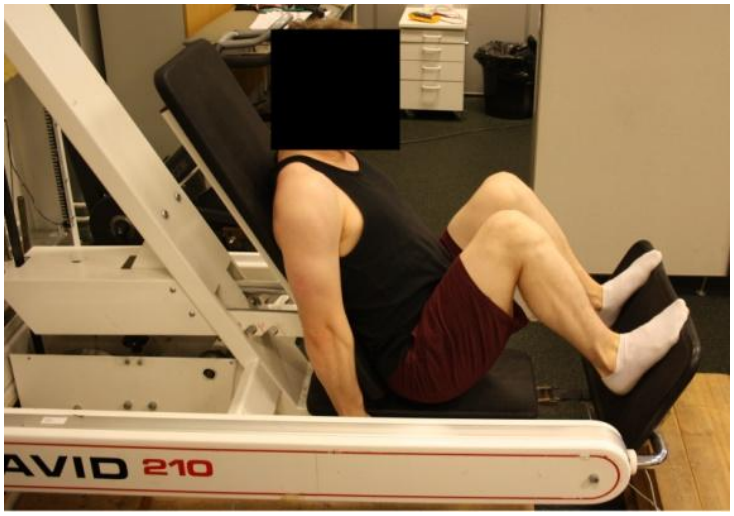


FIGURE 19: The starting position of dynamic leg press test.

### 7.5 Statistical methods

Standard statistical analyses were used for descriptive variable. Those were means and standard deviations (SD). Normal distributions were determined through the Shapiro-Wilk test and acceptable levels of skewness and kurtosis was also checked. All dependent variables were evaluated by using a two-way analysis of variance (Anova) with repeated measures. When a significant F-value was found using an ANOVA with repeated measures with a Greenhouse-Geisser correction, the post hoc tests using the Bonferroni correction was used to locate the pair-wise differences. Differences between groups were analyzed by paired T-test. SPSS Statistics version 24 (IBM corp., New York, NY, USA) was used for statistical analyses. For all tests, the alpha level was set at $p \leq 0.005$.

## 8. Results

Table 4 presents the ultrasound results of the intervention and Table 5 shows the dynamic strength results. Table 6 displays the changes in lean masses. Table 7 & 8 presents neural and isometric adaptations to the intervention. Table 9 displays the changes in serum hormone concentrations during the intervention.

TABLE 4: Ultrasound results (mean and SD) for VL and TB. On the left there are variables and the groups. The upper part shows pre-post changes and post to detraining changes.

| **Muscle CSA and TB** | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** | **POST** | **DT +6WK** | **Δ% Post to DT +6wk** | **SD** | **p-value** |
| **VL CSA ($cm^2$)** | | | | | | | | | | |
| All subjects | 32.1 | 35.1 | 10.7 | 12.5 | p=0.013 | 35.1 | 33.2 | -4.9 | 7.3 | - |
| Bilateral group | 32.5 | 36.1 | 12.8 | 13.5 | - | 36.1 | 33.9 | -5.8 | 9.3 | - |
| Unilateral group | 31.7 | 33.9 | 10.2 | 10.0 | - | 33.9 | 32.6 | -4.0 | 5.4 | - |
| **TB Thickness (cm)** | | | | | | | | | | |
| All subjects | 3.8 | 4.2 | 12.6 | 14.2 | p=0.001 | 4.2 | 3.8 | -9.4 | 8.0 | >0.0001 |
| Bilateral group | 3.8 | 4.1 | 9.0 | 8.1 | p=0.01 | 4.1 | 3.9 | -6.3 | 8.0 | - |
| Unilateral group | 3.8 | 4.2 | 17.9 | 19.3 | p=0.037 | 4.2 | 3.7 | -12.2 | 7.2 | p=0.001 |

TABLE 5: Dynamic strength results (mean and SD). On the left there are variables and the groups. The upper part shows pre-post changes and post to detraining changes.

| **Strength** | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** | **POST** | **DT +6WK** | **Δ% Post to DT +6wk** | **SD** | **p-value** |
| **Bilateral leg press (kg)** | | | | | | | | | | |
| All subjects | 155 | 179 | 16.3 | 11.8 | p<0.0001 | 179 | 176 | -1.2 | 3.7 | - |
| Bilateral group | 154 | 180 | 17.6 | 10.1 | p<0.0001 | 180 | 178 | -0.9 | 4.3 | - |
| Unilateral group | 156 | 177 | 14.7 | 13.9 | p=0.007 | 177 | 174 | -1.6 | 3.1 | - |
| **Left leg - leg press (kg)** | | | | | | | | | | |
| All subjects | 81 | 100 | 34.3 | 43.3 | p<0.0001 | 100 | 100 | -0.4 | 5.6 | - |
| Bilateral group | 78 | 98 | 37.3 | 56.4 | p=0.003 | 98 | 99 | 1.4 | 5.5 | - |
| Unilateral group | 84 | 103 | 30.4 | 17.7 | p=0.001 | 103 | 101 | -2.7 | 5.0 | - |
| **Right leg - leg press (kg)** | | | | | | | | | | |
| All subjects | 86 | 104 | 23.3 | 12.3 | p<0.0001 | 104 | 103 | -1.7 | 5.8 | - |
| Bilateral group | 84 | 99 | 18.4 | 10.7 | p<0.0001 | 99 | 99 | 0.00 | 6.1 | - |
| Unilateral group | 87 | 111 | 29.0 | 12.0 | p<0.0001 | 111 | 107 | -3.8 | 4.8 | - |
| **Bench press (kg)** | | | | | | | | | | |
| All subjects | 68 | 79 | 17.5 | 8.4 | p<0.0001 | 79 | 76 | -7.0 | 20.4 | p=0.024 |
| Bilateral group | 63 | 76 | 20.9 | 8.3 | p<0.0001 | 76 | 73 | -3.0 | 5.8 | - |
| Unilateral group | 74 | 83 | 13.4 | 6.9 | p=0.003 | 83 | 78 | -11.7 | 29.7 | - |

TABLE 6: DEXA results. The measurements were conducted only pre and post. On the left there are variables and the groups. The upper part shows pre-post changes (means and SD for relative changes).

| **DEXA results** | | | | | |
|---|---|---|---|---|---|
| | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** |
| **Total lean mass (g)** | | | | | |
| All subjects | 59242 | 62110 | 4.9 | 2.6 | p<0.0001 |
| Bilateral group | 59790 | 63022 | 5.4 | 2.5 | p<0.0001 |
| Unilateral group | 58595 | 61032 | 4.3 | 2.8 | p<0.0001 |
| **Arms lean mass (g)** | | | | | |
| All subjects | 7668 | 8168 | 6.9 | 4.7 | p<0.0001 |
| Bilateral group | 7598 | 8143 | 7.2 | 2.9 | p<0.0001 |
| Unilateral group | 7752 | 8198 | 6.5 | 6.3 | *p*=0.009 |
| **Legs lean mass (g)** | | | | | |
| All subjects | 20479 | 21186 | 3.4 | 2.4 | p<0.0001 |
| Bilateral group | 20938 | 21773 | 4.0 | 2.8 | p<0.0001 |
| Unilateral group | 19936 | 20493 | 2.8 | 1.6 | p<0.0001 |

TABLE 7: Voluntary activation (%) and isometric lower body strength. On the left there are variables and the groups. The upper part shows pre-post changes and post to detraining changes (means and SD for relative changes).

| **Voluntary activation (%) and isometric strength** | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** | **POST** | **DT +6WK** | **Δ% Post to DT +6wk** | **SD** | **p-value** |
| **Activation level of quadriceps muscle (%)** | | | | | | | | | | |
| All subjects | 92.3 | 90.7 | -2.0 | 4.4 | - | 90.7 | 94.3 | 4.2 | 4.6 | p=0.001 |
| Bilateral group | 90.1 | 88.8 | -2.0 | 4.2 | - | 88.8 | 93.1 | 5.2 | 4.4 | p=0.020 |
| Unilateral group | 94.6 | 92.7 | -2.0 | 4.8 | - | 92.7 | 95.5 | 3.2 | 4.8 | - |
| **Isometric bilateral leg press (N)** | | | | | | | | | | |
| All subjects | 2901 | 3407 | 17.3 | 16.6 | p<0.0001 | 3407 | 3230 | -4.4 | 9.7 | p=0.018 |
| Bilateral group | 2893 | 3384 | 15.6 | 17.5 | p=0.003 | 3384 | 3215 | -3.6 | 12.8 | - |
| Unilateral group | 2911 | 3434 | 19.3 | 16.2 | p=0.002 | 3434 | 3247 | -5.3 | 3.9 | p=0.006 |
| **Isometric left leg leg press (N)** | | | | | | | | | | |
| All subjects | 1715 | 1908 | 11.6 | 15.0 | p=0.002 | 1908 | 1847 | -2.8 | 7.5 | - |
| Bilateral group | 1775 | 1870 | 5.3 | 10.2 | - | 1870 | 1818 | -2.0 | 9.3 | - |
| Unilateral group | 1643 | 1954 | 19.1 | 16.6 | p=0.008 | 1954 | 1880 | -3.6 | 5.0 | p=0.030 |
| **Isometric right leg leg press (N)** | | | | | | | | | | |
| All subjects | 1731 | 1899 | 9.9 | 11.1 | p=0.001 | 1899 | 1842 | -2.7 | 8.9 | - |
| Bilateral group | 1759 | 1852 | 5.4 | 11.3 | - | 1852 | 1837 | -0.6 | 8.8 | - |
| Unilateral group | 1698 | 1953 | 15.3 | 8.3 | p=0.001 | 1953 | 1847 | -5.1 | 8.6 | - |

TABLE 8: Isometric upper body strength. On the left there are variables and the groups. The upper part shows pre-post changes and post to detraining changes (means and SD for relative changes).

| | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** | **POST** | **DT +6WK** | **Δ% Post to DT +6wk** | **SD** | **p-value** |
|---|---|---|---|---|---|---|---|---|---|---|
| **Isometric bench press - left arm (N)** | | | | | | | | | | |
| All subjects | 357 | 390 | 9.8 | 13.5 | p=0.006 | 390 | 377 | -0.6 | 14.7 | - |
| Bilateral group | 347 | 372 | 7.9 | 10.4 | - | 372 | 373 | 0.5 | 17.2 | - |
| Unilateral group | 370 | 412 | 12.1 | 16.8 | - | 412 | 383 | -2 | 11.5 | - |
| **Isometric bench press - right arm (N)** | | | | | | | | | | |
| All subjects | 364 | 427 | 18.2 | 15.5 | p<0.0001 | 427 | 419 | -2.2 | 9.2 | - |
| Bilateral group | 351 | 409 | 17.6 | 11.4 | p<0.0001 | 409 | 398 | -2.4 | 9.2 | - |
| Unilateral group | 381 | 449 | 18.8 | 20 | - | 449 | 444 | -1.8 | 9.7 | - |
| **Isometric bilateral bench press (N)** | | | | | | | | | | |
| All subjects | 745 | 879 | 19 | 13.4 | p<0.0001 | 879 | 847 | -2.1 | 6.2 | - |
| Bilateral group | 711 | 837 | 19.4 | 13.9 | p<0.0001 | 837 | 828 | -0.8 | 7 | - |
| Unilateral group | 788 | 928 | 18.5 | 13.5 | p=0.018 | 928 | 871 | -3.8 | 4.7 | - |

TABLE 9: Serum hormone concentrations during the intervention. On the left there are variables and the groups. The upper part shows pre-post changes and post to detraining changes (means and SD for relative changes).

| **Hormone concentrations** | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** | **POST** | **DT +6WK** | **Δ% Post to DT +6wk** | **SD** | **p-value** |
| **Testosterone** | | | | | | | | | | |
| All subjects | 17.8 | 18.6 | 7.1 | 33.0 | - | 18.6 | 17.8 | -0.4 | 23.4 | - |
| Bilateral group | 15.9 | 16.9 | 11.1 | 44.3 | - | 16.9 | 16.4 | 2.9 | 29.7 | - |
| Unilateral group | 20.2 | 20.7 | 2.4 | 10.7 | - | 20.7 | 19.4 | -4.4 | 13.3 | - |
| **Testosterone /SHBG -ratio** | | | | | | | | | | |
| All subjects | 0.6 | 0.7 | 12.7 | 47.0 | - | 0.7 | 0.7 | 12.2 | 29.7 | - |
| Bilateral group | 0.6 | 0.8 | 17.9 | 61.4 | - | 0.8 | 0.9 | 19.0 | 36.9 | - |
| Unilateral group | 0.5 | 0.6 | 6.4 | 21.5 | - | 0.6 | 0.6 | 4.1 | 16.3 | - |
| **Testosterone /Cortisol -ratio** | | | | | | | | | | |
| All subjects | 0.043 | 0.049 | 23.7 | 66.9 | - | 0.049 | 0.045 | 7.710 | 52.1 | - |
| Bilateral group | 0.038 | 0.046 | 32.0 | 84.1 | - | 0.046 | 0.041 | 12.067 | 62.9 | - |
| Unilateral group | 0.048 | 0.053 | 13.9 | 40.2 | - | 0.053 | 0.049 | 2.562 | 38.1 | - |

## 8.1 Anthropometrics and muscle mass

### 8.1.1 DEXA

No significant changes occurred in total mass (Figure 20). Changes in the total lean mass (Figure 21 A) were statistically significant for both groups ($p<0.0005$). The arms lean mass (Figure 22 A) were also statistically significant for the unilateral ($p=0.009$) and the bilateral group ($p<0.0005$). The changes in the legs lean mass (Figure 22 B) were statistically significant for both groups ($p<0.0005$).

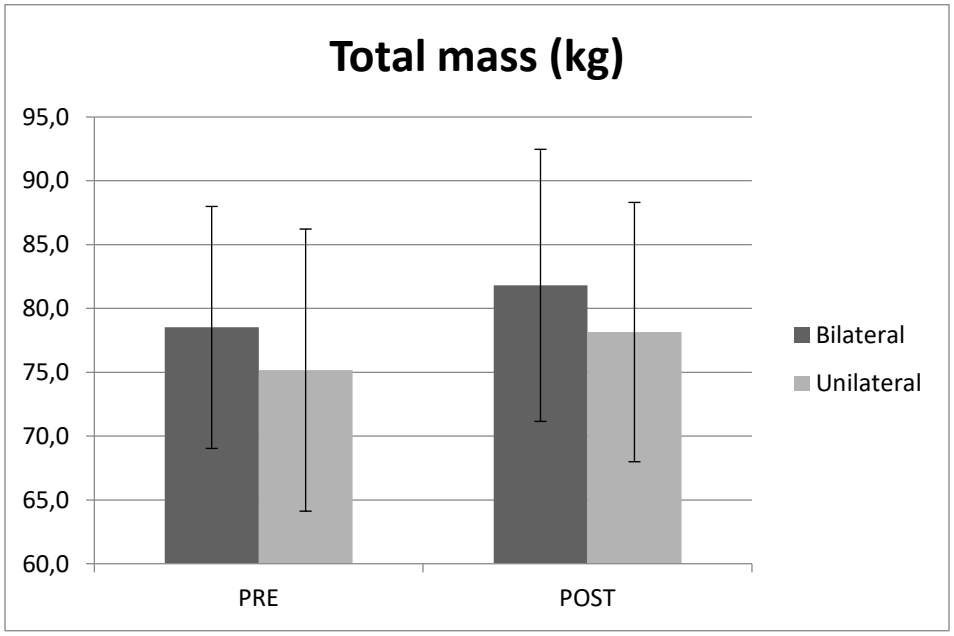


FIGURE 20: Total mass from pre to post.

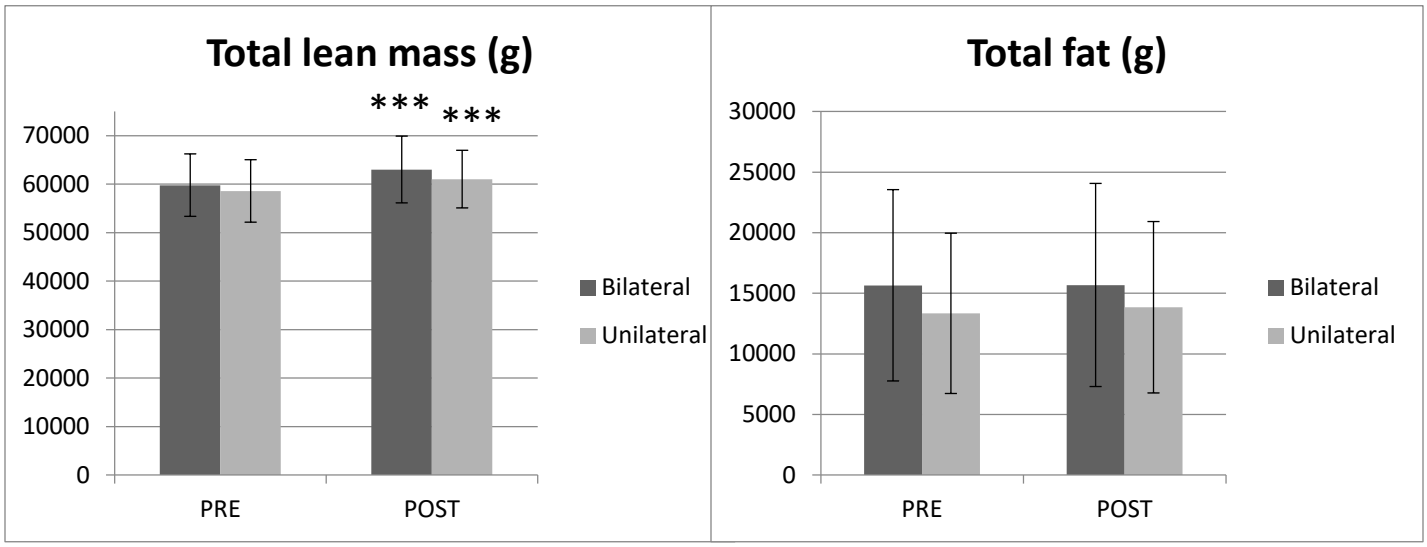


FIGURE 21 A & B: Total lean mass on the left (A) and fat mass on the right (B)

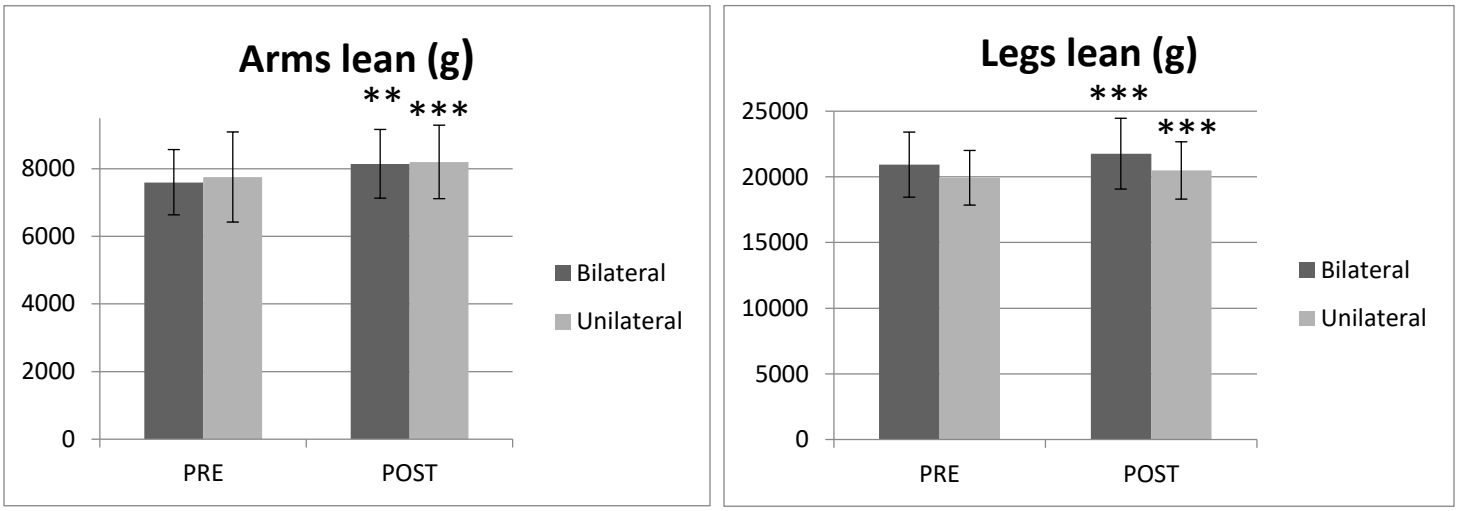


FIGURE 22 A & B: Arms lean mass on the left (A) and legs lean mass on the right (B).

### 8.1.2 Vastus lateralis CSA and triceps brachii thickness

Changes in VL CSA were not statistically significant for either of the groups (Figure 23). Triceps brachii changes were statistically significant for both groups from pre to post (Bilateral p=0.01 and unilateral p=0.037). There were significant changes also for the unilateral group from post to detraining 3wk (p=0.029) and for post to detraining 6wk (p=0.006) (figure 24).

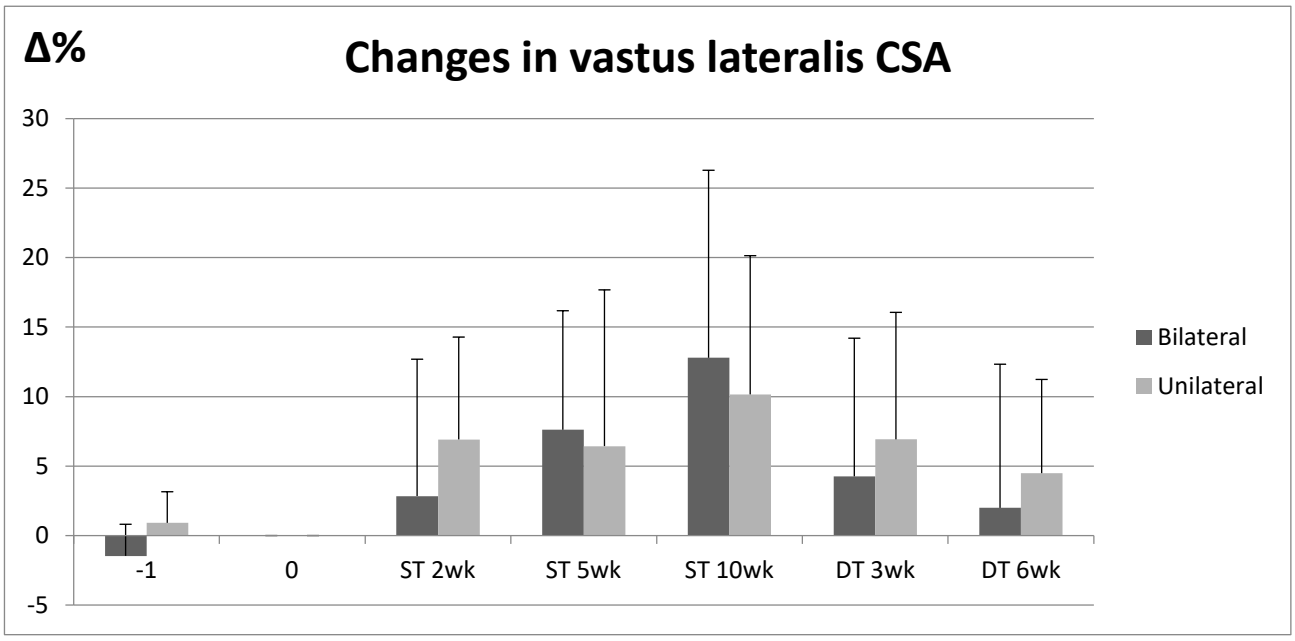


FIGURE 23: Changes in vastus lateralis CSA (means and SD for relative changes) during the whole intervention.

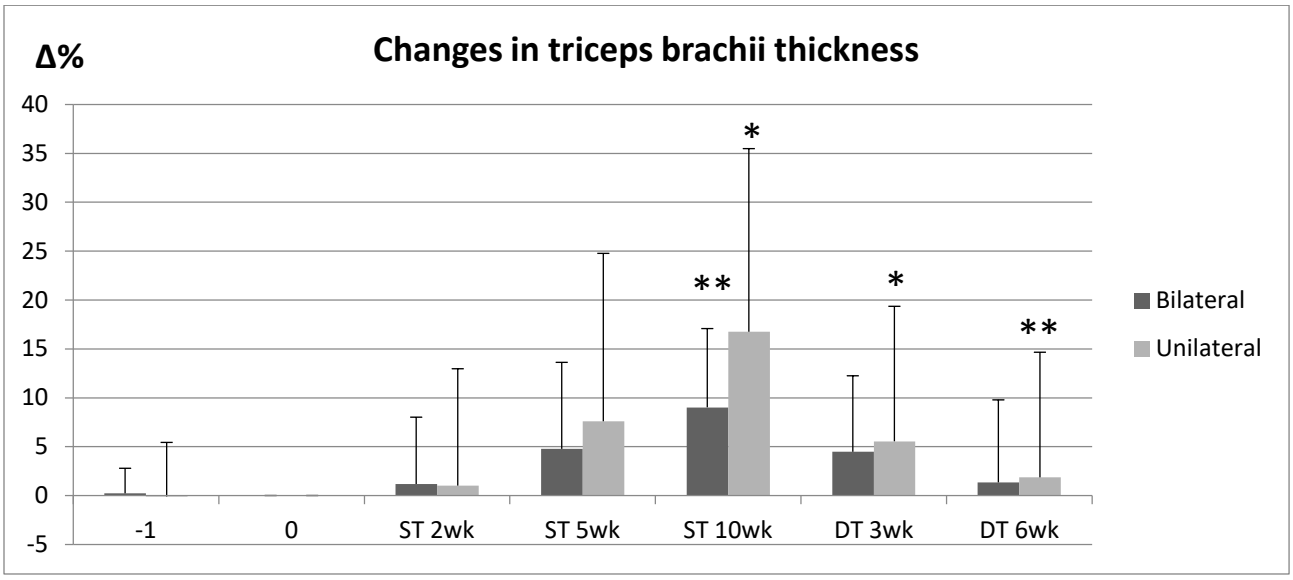


FIGURE 24: Changes in triceps brachii thickness (means and SD for relative changes) during the intervention.

### 8.1.3 Individual responders to hypertrophy

Mean VL CSA change for the total group of subjects is displayed in figure 25 and individual data is shown in figure 26. VL CSA changes were significant from pre to post (p=0.046).

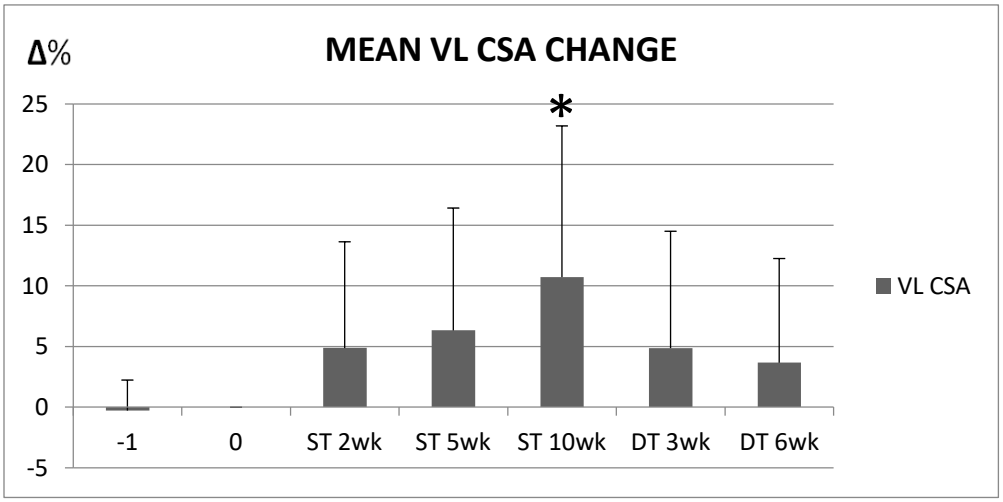


FIGURE 25: Changes in VL CSA (means and SD for relative changes) for the total group of subjects during the intervention.

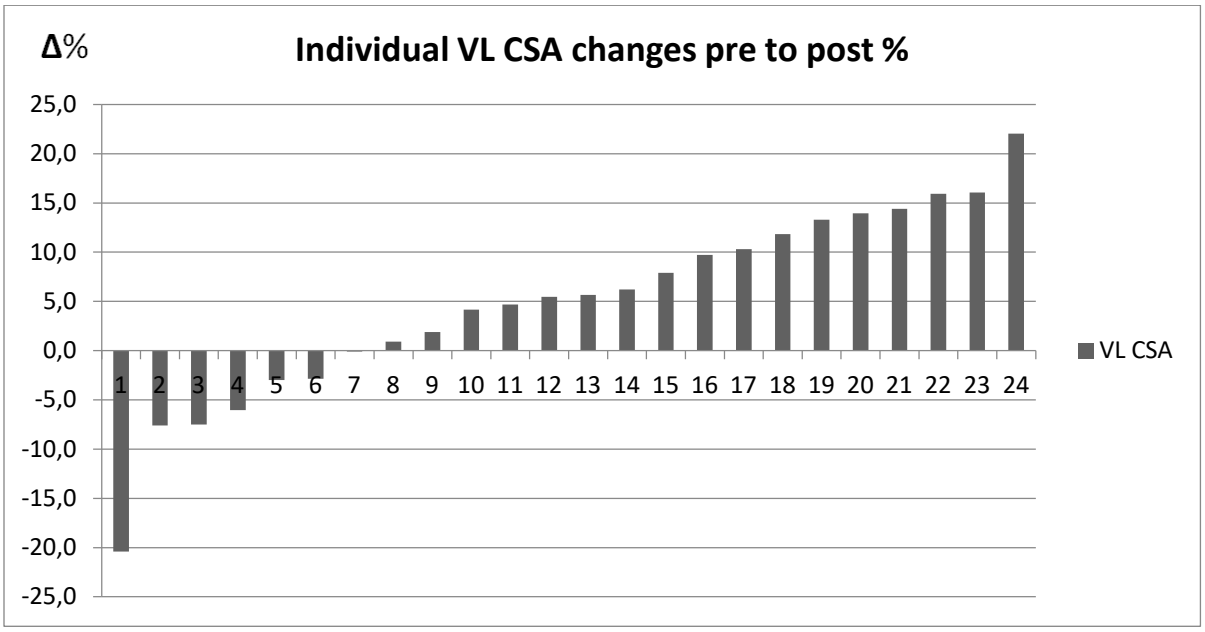


FIGURE 26: Individual data for each subject for VL CSA delta changes from pre to post.

After the intervention, it was possible to identify three different subgroups. Subjects were split to three groups according to the growth of the VL CSA during the 10-week training period.

High responders >15%, (n=10)

medium reponders 15-5% (n=6)

Low responders <5% (n=8)

High responders were the only ones to have a significant change in VL CSA from pre to mid (p=0.003), pre to post-training (p=0.001), pre to detraining 1 (p=0.007), pre to detraining 2 (p=0.002), and post-training to detraining 2 (figure 27). The subgroups changes in dynamic bilateral leg press are shown in figure 28. High responders to hypertrophy increased strength statistically significantly from pre to mid (p=0.010), pre to post (p=0.028), pre to detraining 1 (p=0.033). Medium responders to hypertrophy did not increase statistically significantly. Low responders did not achieved statistically significant change in VL CSA and actually their mean VL CSA dropped from 37.6 (±5.2) to post value of 36.8 (±6.1). Low responders to hypertrophy, on the other hand, increased strength statistically significantly from pre to mid (p=0.023), pre to detraining 1 (p=0.029) and from pre to detraining 2 (p=0.002).

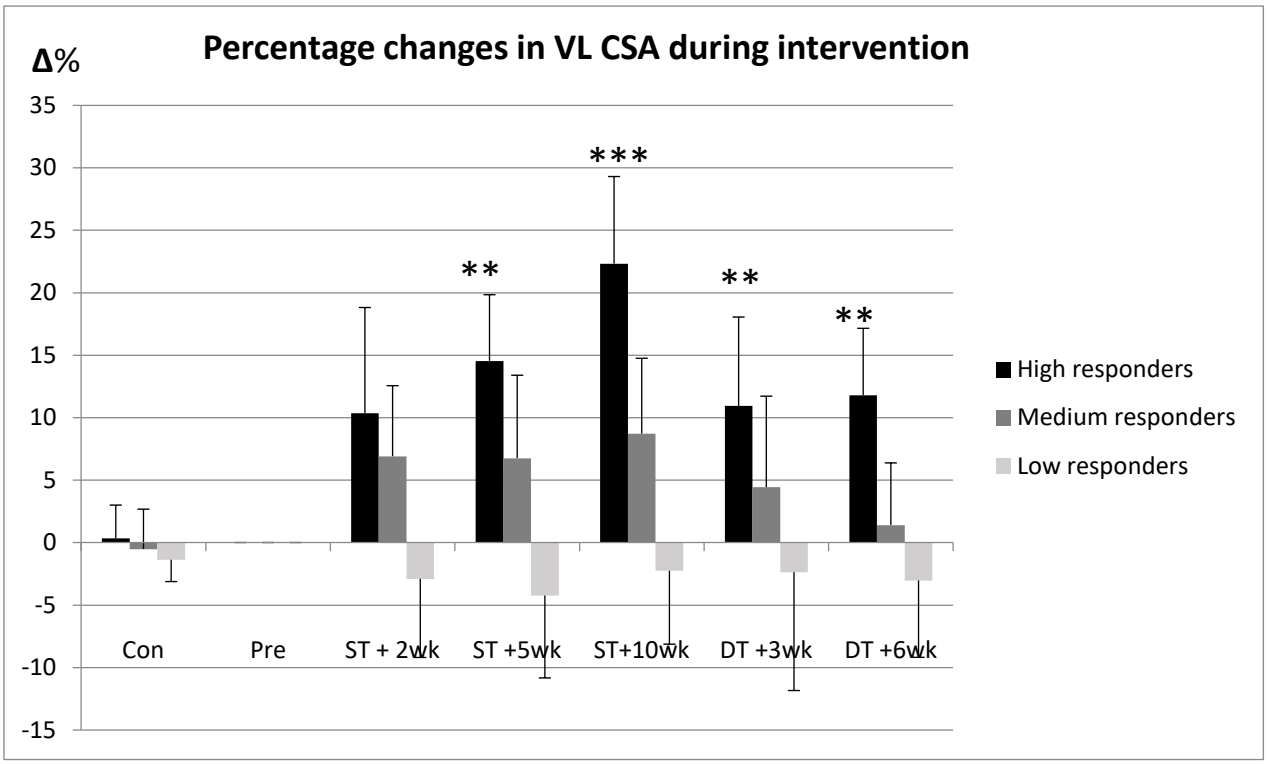


FIGURE 27: Three different subgroup and their VL CSA changes (means and SD for relative changes) during the intervention.

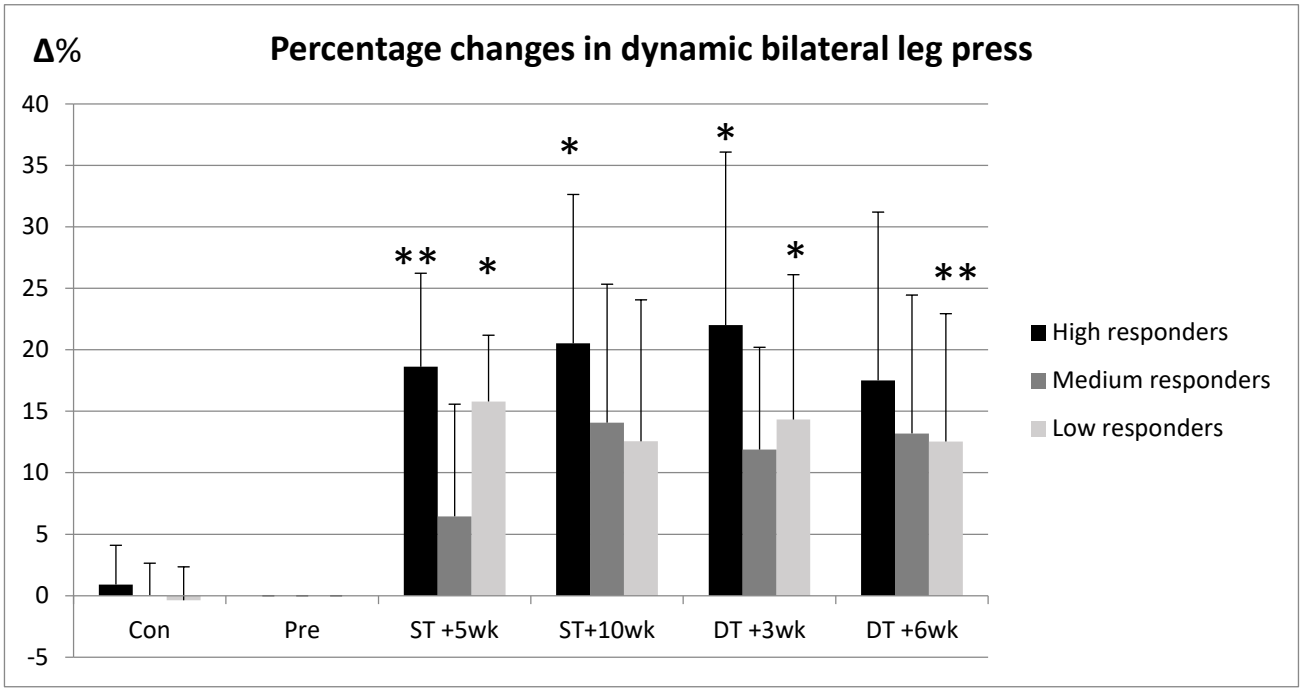


FIGURE 28: Changes (means and SD for relative changes) in dynamic strength in different responder groups to hypertrophy.

Table 10 provides changes in VL CSA and dynamic bilateral leg press to different subgroups during the detraining after the strength training period.

TABLE 10: Changes (means and SD for relative changes) in the responder groups from pre to post and post to detraining +6wk.

| | **Pre to Post Changes %** | | **Post to DT +6WK Changes %** | |
|---|---|---|---|---|
| **VL CSA** | **Mean** | **SD** | **Mean** | **SD** |
| High responders | +22.3 | 7.0 | -9.3 | 5.6 |
| Medium responders | +8.7 | 6.0 | -4.7 | 3.2 |
| Low Responders | -2.3 | 5.9 | -0.5 | 8.4 |
| **DYNAMIC BILATERAL LEG PRESS** | **Mean** | **SD** | **Mean** | **SD** |
| High responders | +20.5 | 12.1 | -2.1 | 3.1 |
| Medium responders | +14.1 | 11.3 | -1.9 | 4.2 |
| Low Responders | +12.6 | 11.5 | +0.1 | 3.4 |

Correlations between the strength and hypertrophy were the following in different time points: Control: 0.546, p=0.006*, Pre: 0.452, p=0.026*, ST +5wk: 0.332, p=0.268, ST +10wk: 0.174, p=0.417, DT +3wk: 0.323, p=0.132, DT +6wk: 0.486 p=0.025*.

Strength and hypertrophy correlated with each other statistically significantly in the control measurements, in the pre measurements and again in measurements at detraining +6wk.

### 8.2 Electrical stimulation, activation level and muscle activity

#### 8.2.1 Activation level from electrical stimulation

The mean scores for the changes in the electric stimulation activation level were not statistically significantly different for the unilateral group, but for the bilateral group they were statistically significantly different (figure 29). The results for the bilateral group were significantly different from each other at post to DT +6wk (p=0.020). Figure 30 presents the voluntary activation level data for the whole group as one. There was a statistically significant change from post to detraining 2 (p=0.003).

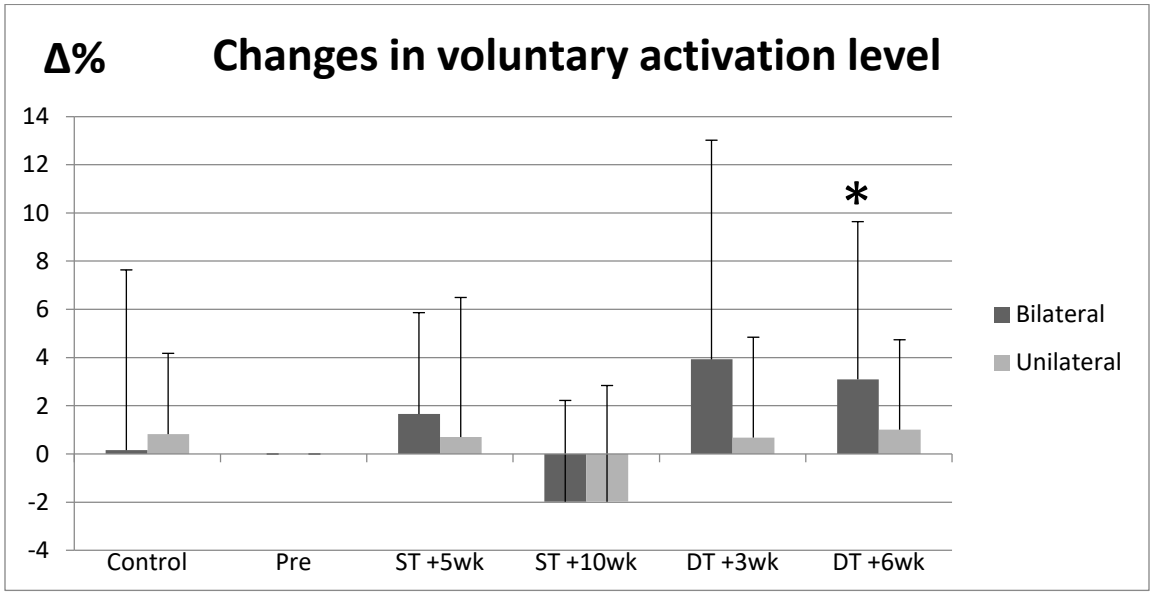


FIGURE 29: Changes (means and SD for relative changes) in the right leg's quadriceps voluntary activation level.

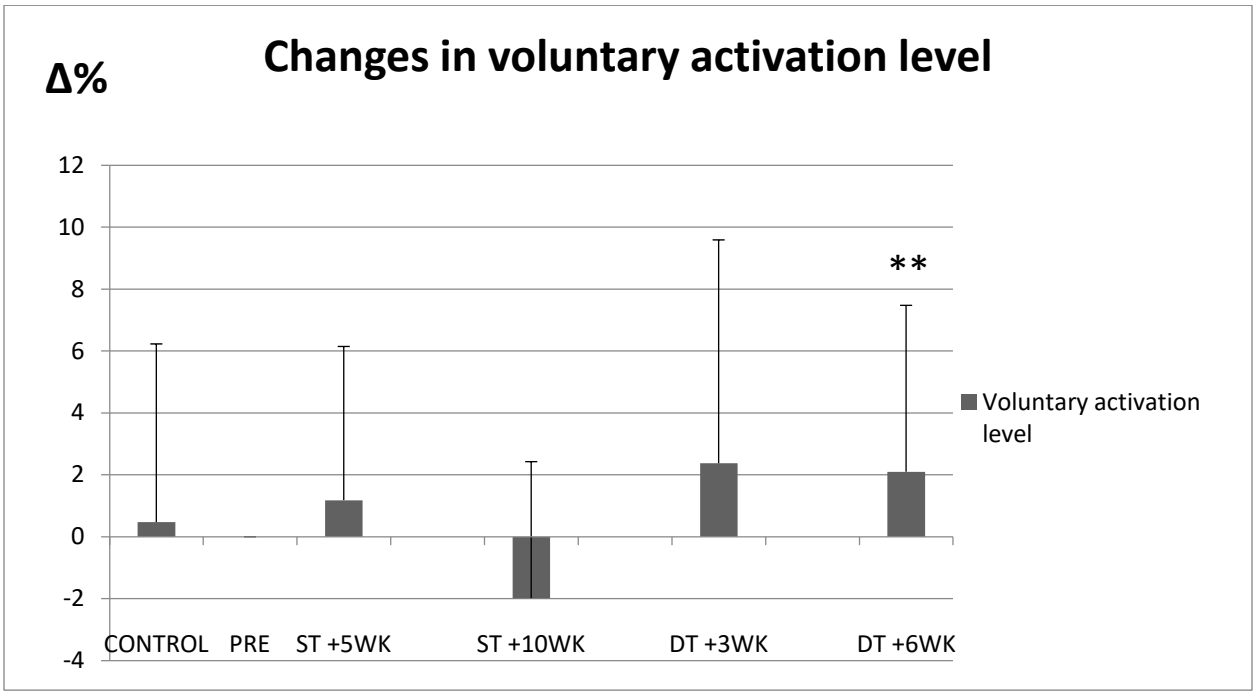


FIGURE 30: Changes (means and SD for relative changes) in voluntary activation level for the whole group.

When looking at the different responder subgroups to hypertrophy, the high responders lost most activation level after the 10 weeks of strength training (-3.18, ± 5.84) compared to medium responders (-1.16, ± -1.16) or low responders (-1.25, ± 3.61) (Figure 31).

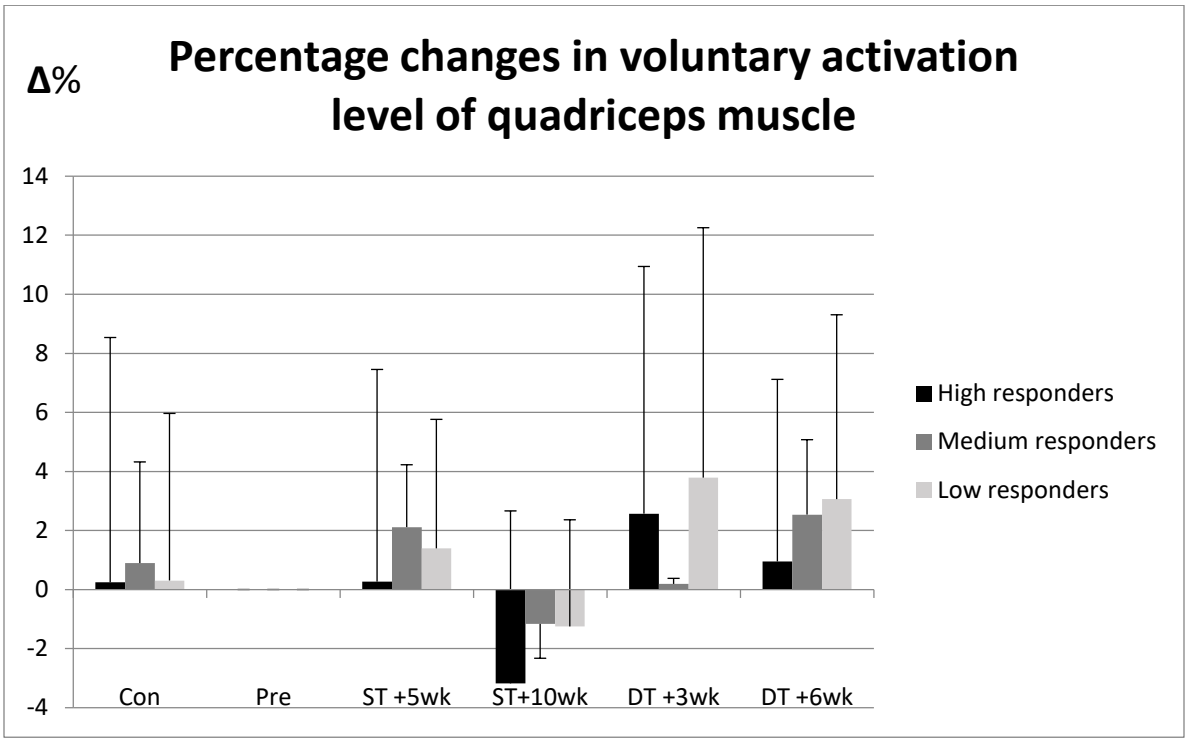


FIGURE 31: Different responder groups' activation level (means and SD for relative changes) during the training and the detraining.

There were negative but nonsignificant correlations between VLCSA and AL.

Control: -0.28, p=0.219, Pre:-0.27, p=0.237, Mid: -0.357, p=0.103, Post: -0.25, p=0.249, DT1: -0.117, p=0.594, DT2: -0.308, p=0.186.

### 8.2.2 Muscle activity

Muscle activity (EMG) in the isometric leg press was measured unilaterally and bilaterally. Table 11 summarizes the VL and TB results during the intervention. VL muscle activity is displayed in figures 32 and 33. VM muscle activity in unilateral and bilateral leg press is shown in figures 34 and 35.

TABLE 11: Changes in VL and TB EMG during the intervention.

| | **Pre to Post Changes %** | | **p-value** | **Post to DT +6WK Changes %** | | **p-value** |
|---|---|---|---|---|---|---|
| **VL activity in unilateral leg press** | **Mean** | **SD** | | **Mean** | **SD** | |
| Bilateral group | +32.8 | 20.8 | p=0.008 | -15.4 | 16.4 | |
| Unilateral group | +45.3 | 52.8 | | -25.8 | 24.4 | |
| **VL activity in bilateral leg press** | | | | | | |
| Bilateral group | +27.2 | 22.8 | | -6.2 | 10.5 | |
| Unilateral group | +34.7 | 18.1 | p=0.001 | -20.5 | 24.4 | |
| **TB activity in unilateral bench press** | | | | | | |
| Bilateral group | +8.2 | 38.9 | | -40.2 | 40.2 | p=0.01 |
| Unilateral group | +18.4 | 28.8 | | -0.4 | 37.5 | |
| **TB activity in bilateral bench press** | | | | | | |
| Bilateral group | +12.4 | 52.9 | | -39.3 | 29.3 | p=0.013 |
| Unilateral group | +5.5 | 28.2 | | -21.1 | 41.4 | |

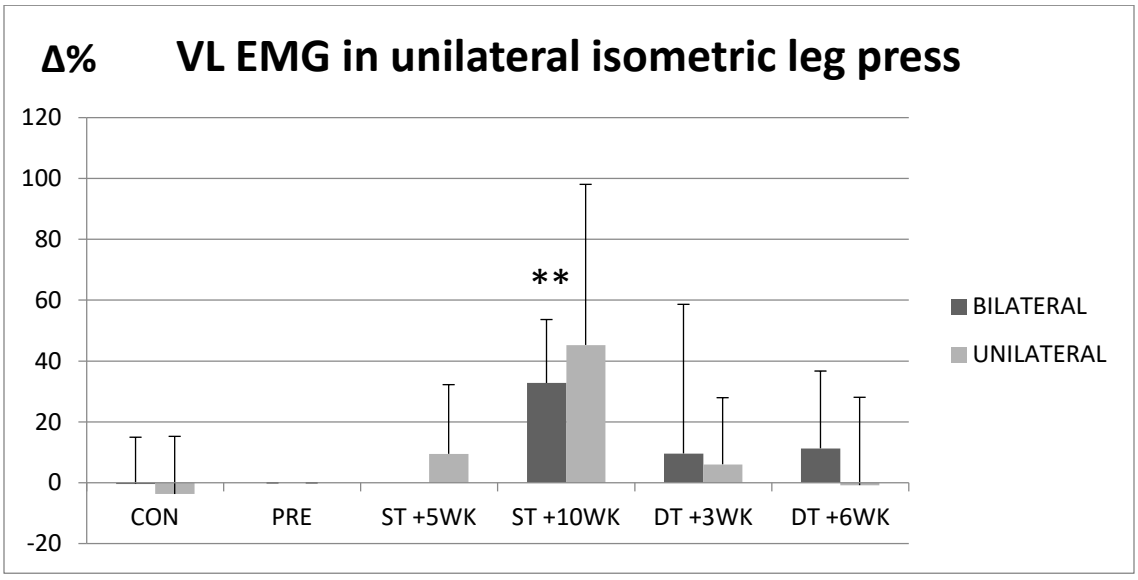


FIGURE 32: Changes (means and SD for relative changes) in VL muscle activity in the unilateral isometric leg press during the intervention. Tests in ST +5WK was only conducted according to subjects own group's training. In order to minimize the bilateral training for the unilateral group and vice versa.

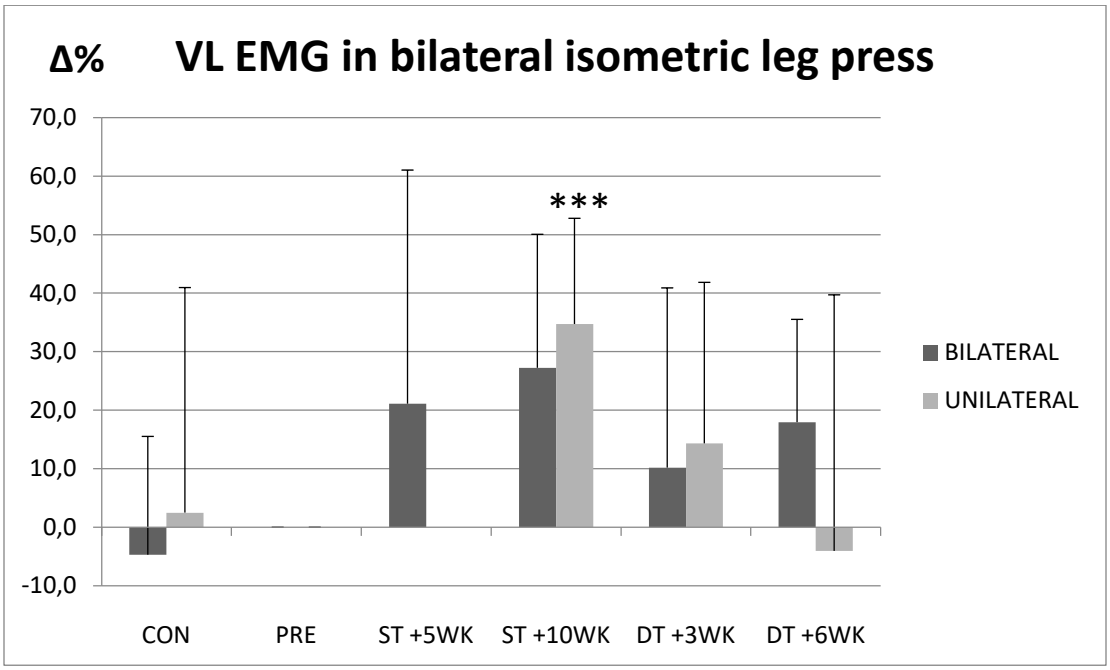


FIGURE 33: Changes (means and SD for relative changes) in VL muscle activity in bilateral isometric leg press during the intervention.

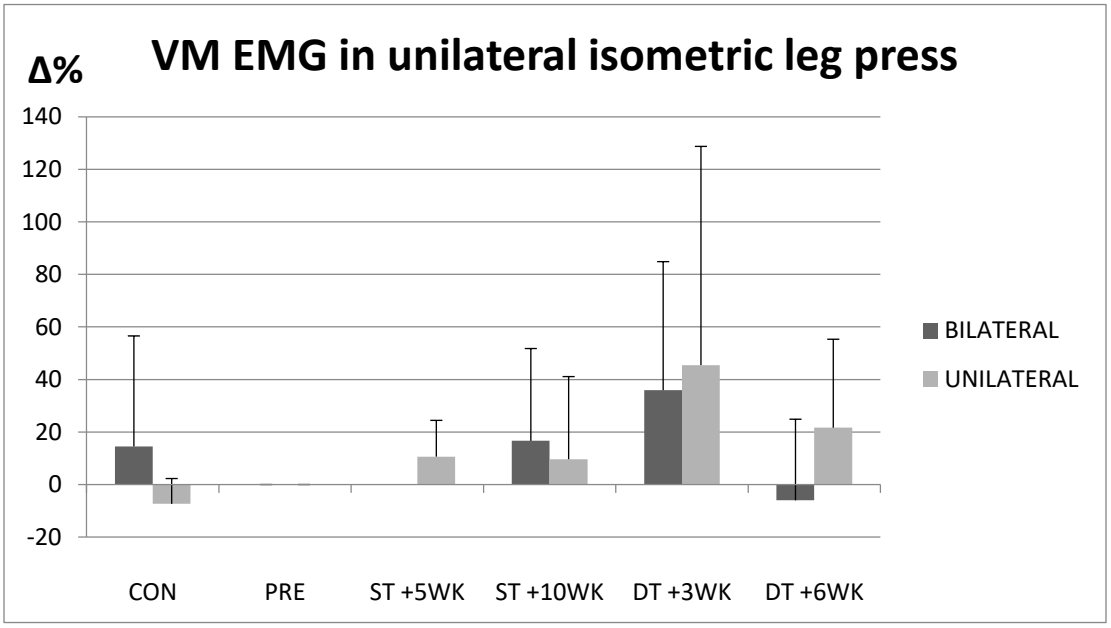


FIGURE 34: Changes (means and SD for relative changes) in right quadricep vastus medialis muscle activity in the unilateral isometric leg press.

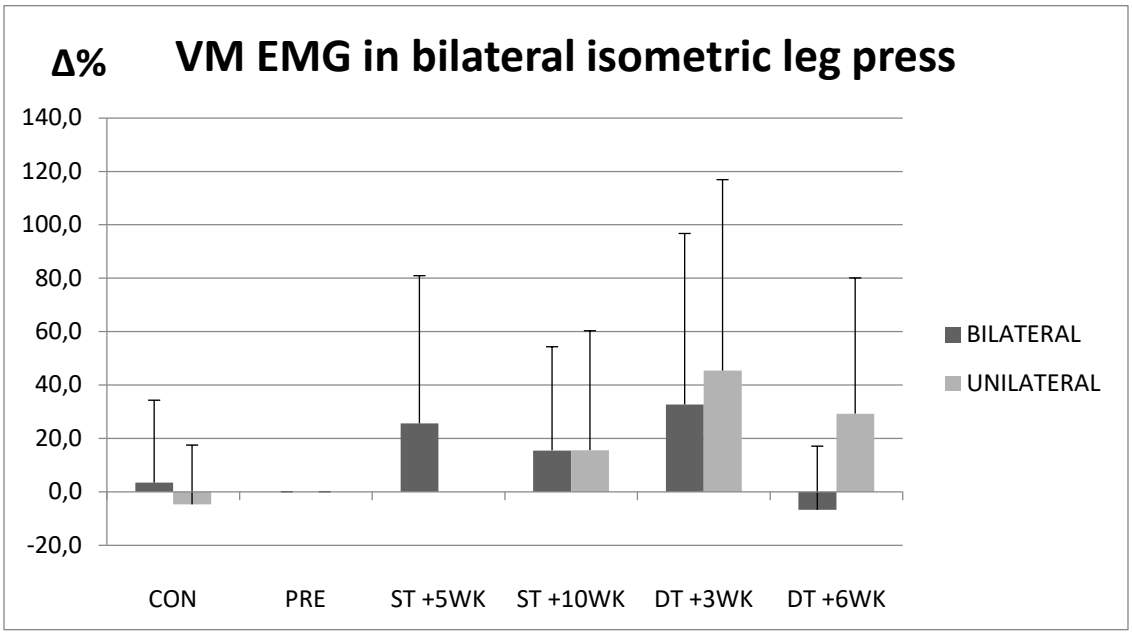


FIGURE 35: Changes (means and SD for relative changes) in right quadriceps vastus medialis muscle activity in bilateral isometric leg press.

For the unilateral bench press, the bilateral group made statistically significant changes in TB muscle activity from pre to DT2 (0.26 ± 0.14) (p=0.01) and post to DT2 (p=0.01) (figure 36). There were similar changes for AD (figure 37): Pre to DT2 (p=0.025) and post to DT2 (p=0.048). The unilateral group did not have any statistically significant changes in their muscle activity neither in the unilateral nor bilateral bench press. For the bilateral bench press the bilateral group's TB muscle activity statistically significantly changed from post to DT2 (p=0.013) (figure 38). There were no statistically significant changes in AD's muscle activity in the bilateral bench press in either of the groups (figure 39).

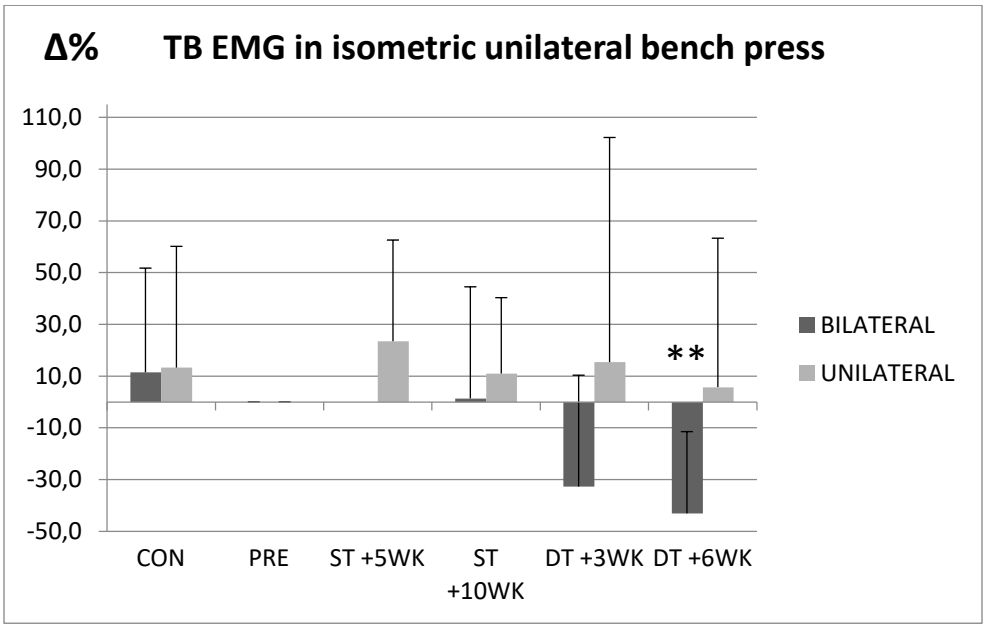


FIGURE 36: Changes (means and SD for relative changes) in muscle activity of triceps brachii during the unilateral right arm bench press.

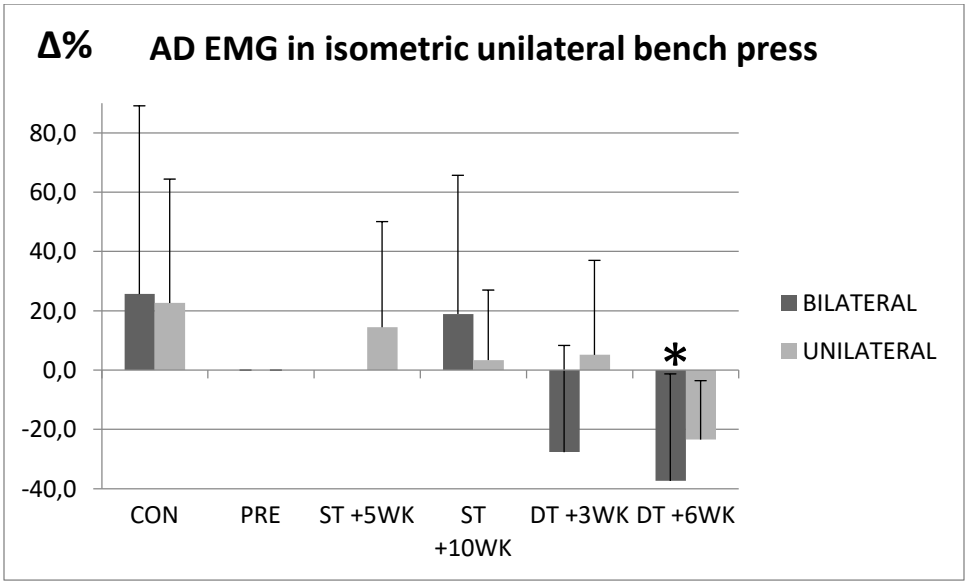


FIGURE 37: Changes (means and SD for relative changes) in muscle activity of anterior deltoid during the unilateral right arm bench press.

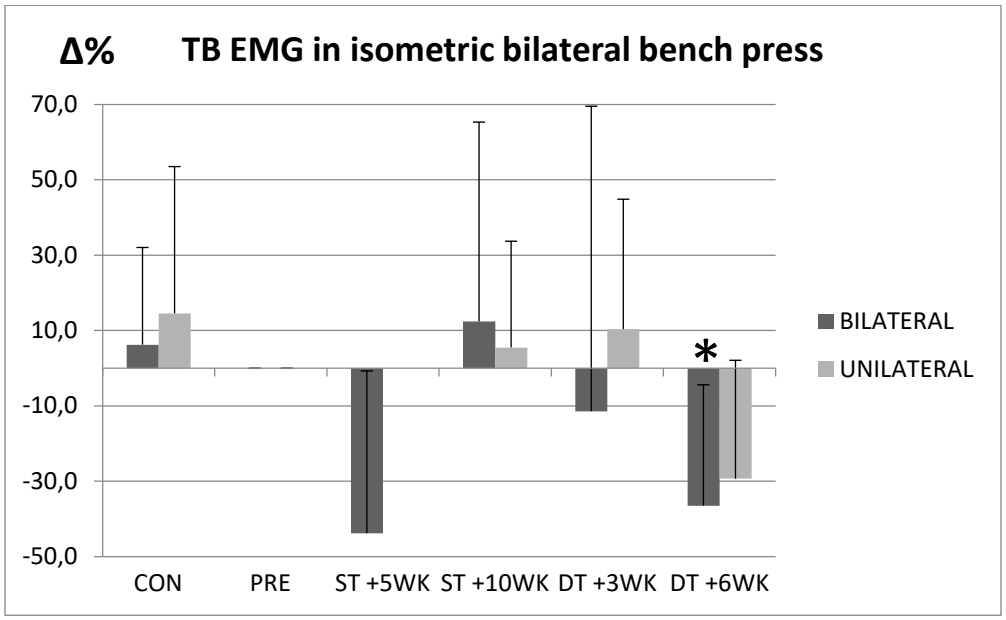


FIGURE 38: Changes (means and SD for relative changes) in muscle activity of TB during the bilateral bench press.

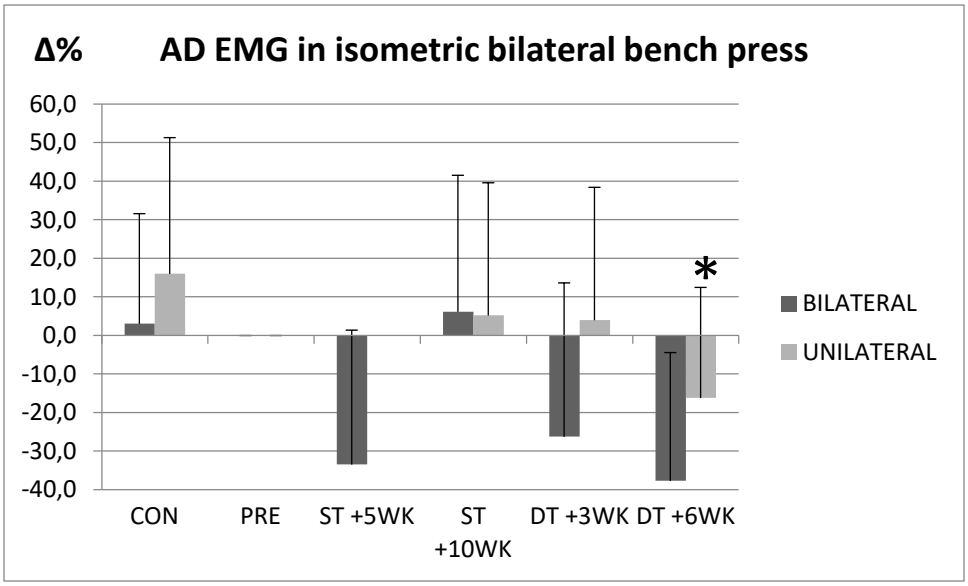


FIGURE 39: Changes (means and SD for relative changes) in muscle activity of AD during the bilateral bench press.

### 8.3 Basal serum hormone levels

There were no statistically significantly changes between any time points in any of the measured hormones. Figures 40, 41 and 42 present hormonal concentrations for testosterone, testo/SHBG - ratio and cortisol.

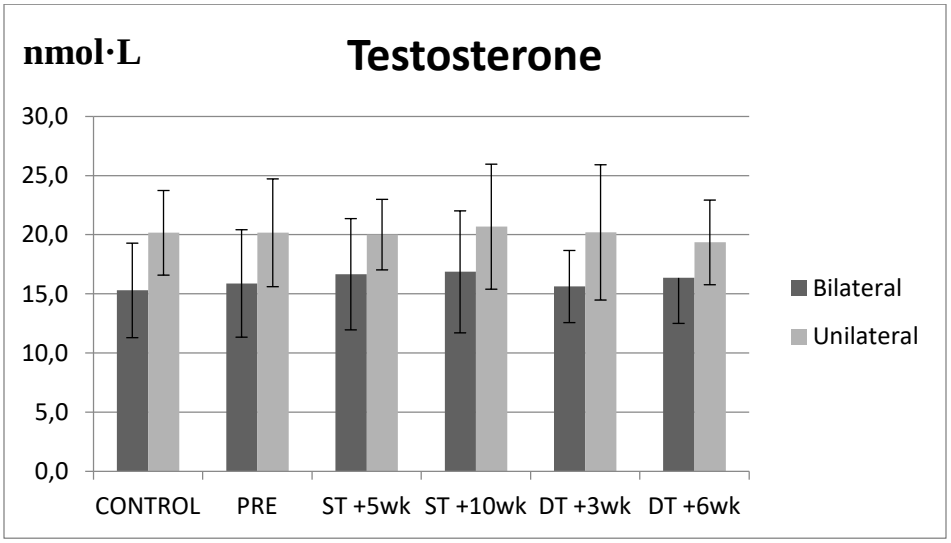


FIGURE 40: Absolute values for testosterone during the intervention.

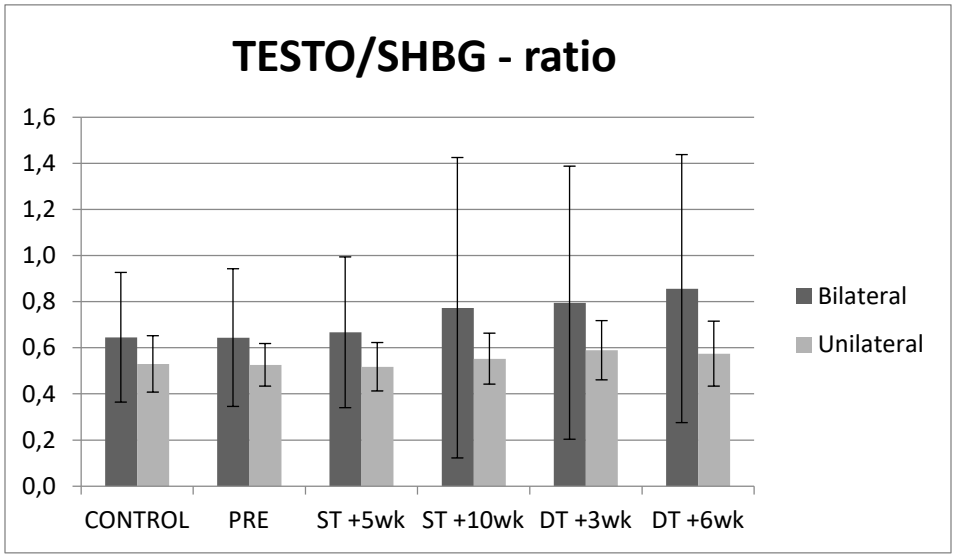


FIGURE 41: Testo/SHBG - ratio during the intervention.

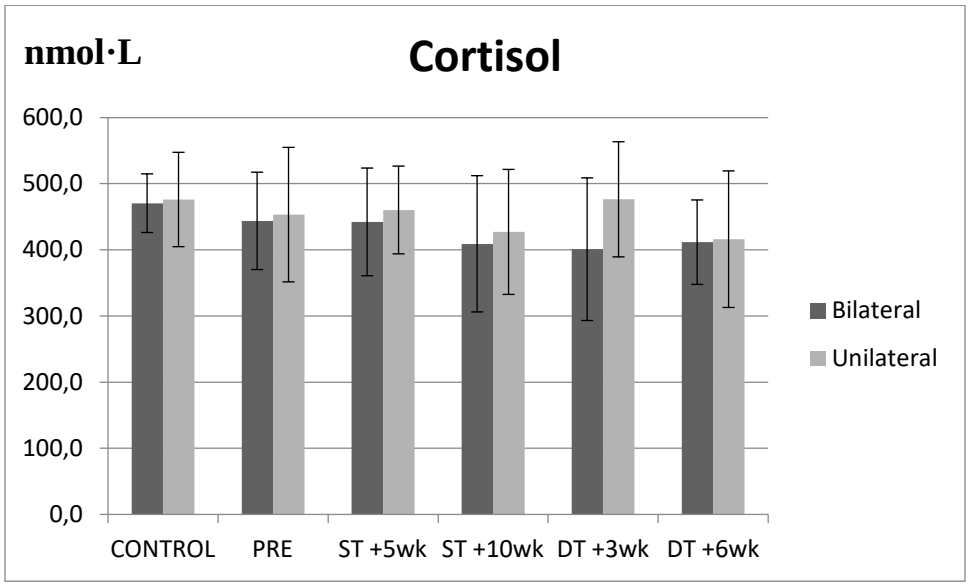


FIGURE 42: Absolute values for cortisol during the intervention.

### 8.4 Isometric strength

The unilateral group improved isometric strength statistically significantly from pre to post for isometric left leg - leg press ($p=0.04$), isometric right leg - leg press ($p=0.006$), and also for bilateral leg press from pre to post ($p=0.02$) and post to detraining +6wk ($p=0.008$). Whereas the bilateral group improved in isometric bilateral leg press from pre to mid ($p=0.024$) and pre to post ($p=0.014$) (table 12 & 13). Figure 43 demonstrates the changes in isomeric bilateral leg press during the entire intervention. The unilateral group was tested only unilaterally in the mid tests, while the bilateral group did only the bilateral tests in the mid tests.

The unilateral group improved upper body isometric strength tests statistically significantly only in bilateral bench press pre to post $p=0.018$. Whereas the bilateral group improved right arm isometric bench press from pre to post ($p<0.0001$) and bilateral isometric bench press ($p<0.0001$) (figure 44). Changes in isometric bilateral bench press are presented in figure 45.

TABLE 12: The results of the lower body isometric strength tests during the intervention.

| **Isometric tests:** | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** | **POST** | **DT +6WK** | **Δ% Post to DT +6wk** | **SD** | **p-value** |
|---|---|---|---|---|---|---|---|---|---|---|
| **Isometric bilateral leg press (N)** | | | | | | | | | | |
| All subjects | 2901 | 3407 | 17.3 | 16.6 | p<0.0001 | 3407 | 3230 | -4.4 | 9.7 | p=0.018 |
| Bilateral group | 2893 | 3384 | 15.6 | 17.5 | p=0.003 | 3384 | 3215 | -3.6 | 12.8 | - |
| Unilateral group | 2911 | 3434 | 19.3 | 16.2 | p=0.002 | 3434 | 3247 | -5.3 | 3.9 | p=0.006 |
| **Isometric left leg leg press (N)** | | | | | | | | | | |
| All subjects | 1715 | 1908 | 11.6 | 15.0 | p=0.002 | 1908 | 1847 | -2.8 | 7.5 | - |
| Bilateral group | 1775 | 1870 | 5.3 | 10.2 | - | 1870 | 1818 | -2.0 | 9.3 | - |
| Unilateral group | 1643 | 1954 | 19.1 | 16.6 | p=0.008 | 1954 | 1880 | -3.6 | 5.0 | p=0.030 |
| **Isometric right leg leg press (N)** | | | | | | | | | | |
| All subjects | 1731 | 1899 | 9.9 | 11.1 | p=0.001 | 1899 | 1842 | -2.7 | 8.9 | - |
| Bilateral group | 1759 | 1852 | 5.4 | 11.3 | - | 1852 | 1837 | -0.6 | 8.8 | - |
| Unilateral group | 1698 | 1953 | 15.3 | 8.3 | p=0.001 | 1953 | 1847 | -5.1 | 8.6 | - |

TABLE 13: The upper body isometric strength results during the intervention.

| **Isometric tests:** | **PRE** | **POST** | **Δ% Pre to post** | **SD** | **p-value** | **POST** | **DT +6WK** | **Δ% Post to DT +6wk** | **SD** | **p-value** |
|---|---|---|---|---|---|---|---|---|---|---|
| **Isometric bench press - left arm (N)** | | | | | | | | | | |
| All subjects | 357 | 390 | 9.8 | 13.5 | p=0.006 | 390 | 377 | -0.6 | 14.7 | - |
| Bilateral group | 347 | 372 | 7.9 | 10.4 | - | 372 | 373 | 0.5 | 17.2 | - |
| Unilateral group | 370 | 412 | 12.1 | 16.8 | - | 412 | 383 | -2.0 | 11.5 | - |
| **Isometric bench press - right arm (N)** | | | | | | | | | | |
| All subjects | 364 | 427 | 18.2 | 15.5 | p<0.0001 | 427 | 419 | -2.2 | 9.2 | - |
| Bilateral group | 351 | 409 | 17.6 | 11.4 | p<0.0001 | 409 | 398 | -2.4 | 9.2 | - |
| Unilateral group | 381 | 449 | 18.8 | 20.0 | - | 449 | 444 | -1.8 | 9.7 | - |
| **Isometric bilateral bench press (N)** | | | | | | | | | | |
| All subjects | 745 | 879 | 19.0 | 13.4 | p<0.0001 | 879 | 847 | -2.1 | 6.2 | - |
| Bilateral group | 711 | 837 | 19.4 | 13.9 | p<0.0001 | 837 | 828 | -0.8 | 7.0 | - |
| Unilateral group | 788 | 928 | 18.5 | 13.5 | p=0.018 | 928 | 871 | -3.8 | 4.7 | - |

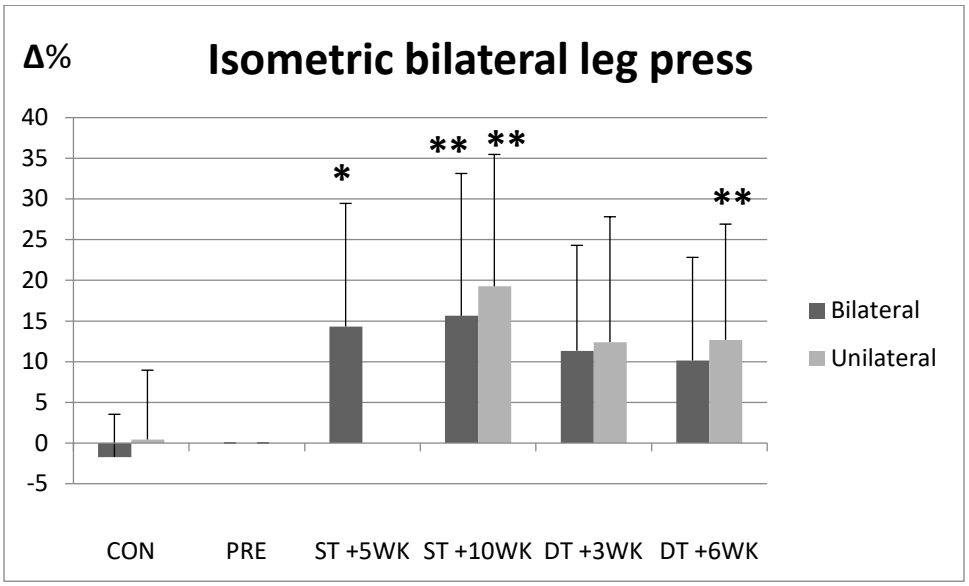


FIGURE 43: Changes (means and SD for relative changes) in the isometric bilateral leg press during the intervention.

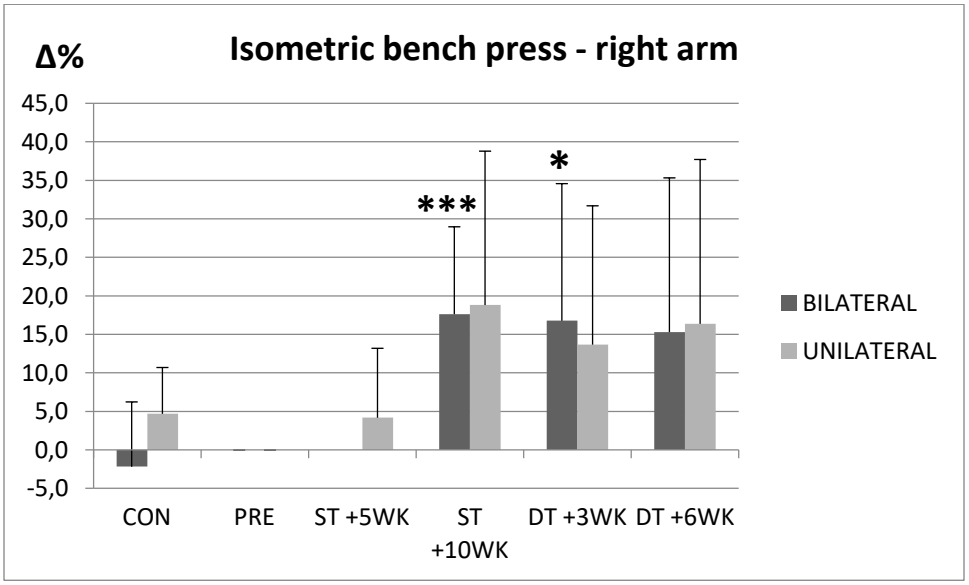


FIGURE 44: Changes (means and SD for relative changes) in the unilateral isometric bench press strength for the right arm.

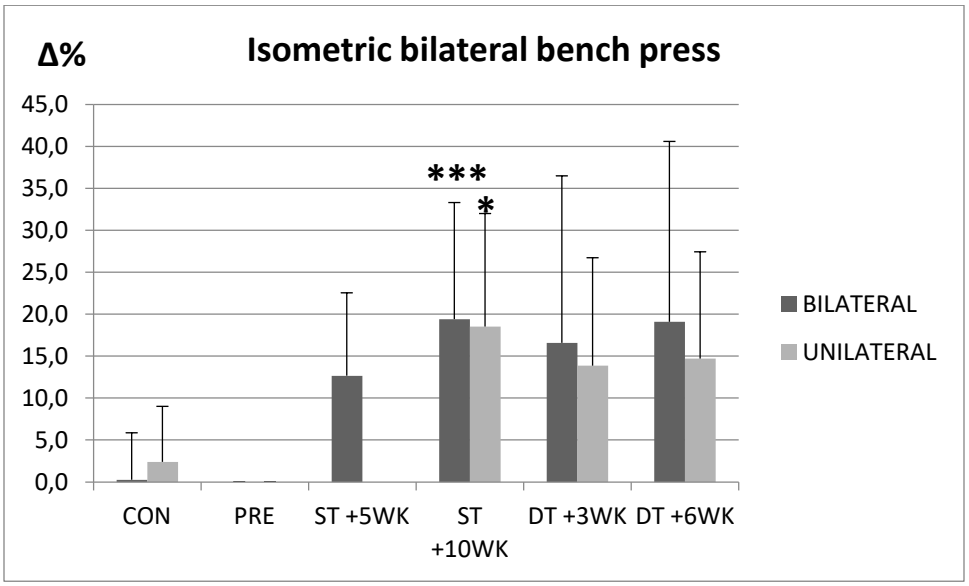


FIGURE 45: Changes (means and SD for relative changes) in the isometric bilateral bench press strength during the intervention.

## 8.5 Dynamic strength

All dynamic strength values changed statistically significantly from pre to post for both groups. Figures 46-49 show the changes in the dynamic strength during the intervention. The bilateral group improved statistically significantly bench press power from pre to post p=0.013 (figure 50).

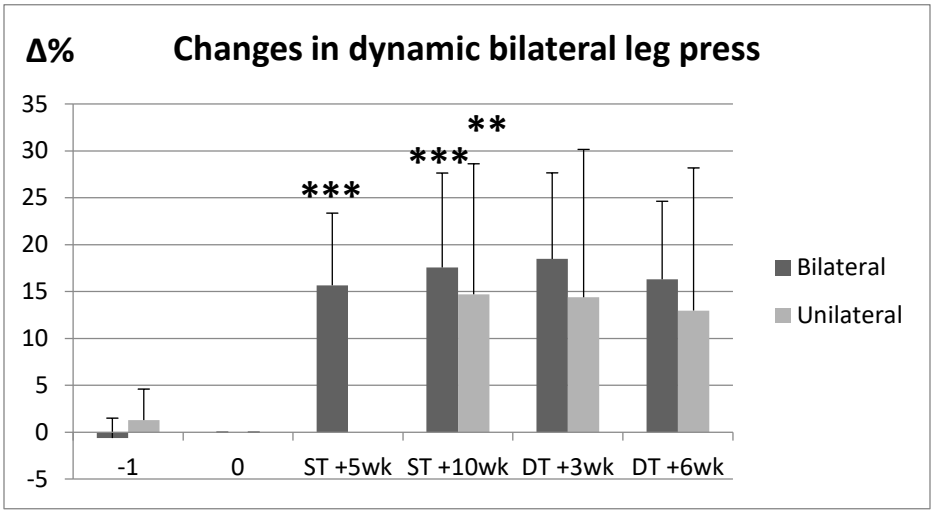


FIGURE 46: Changes (means and SD for relative changes) in the dynamic bilateral leg press during the entire intervention.

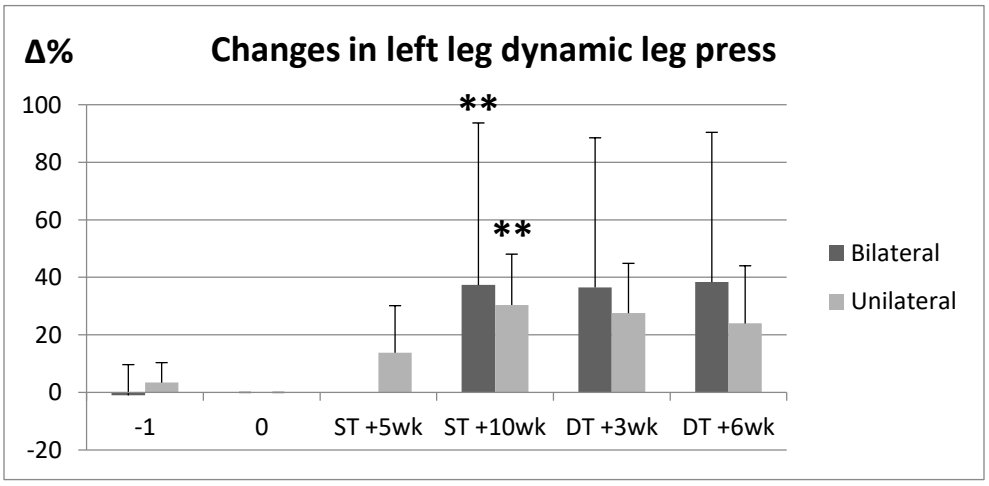


FIGURE 47: Changes (means and SD for relative changes) in left leg strength during the intervention.

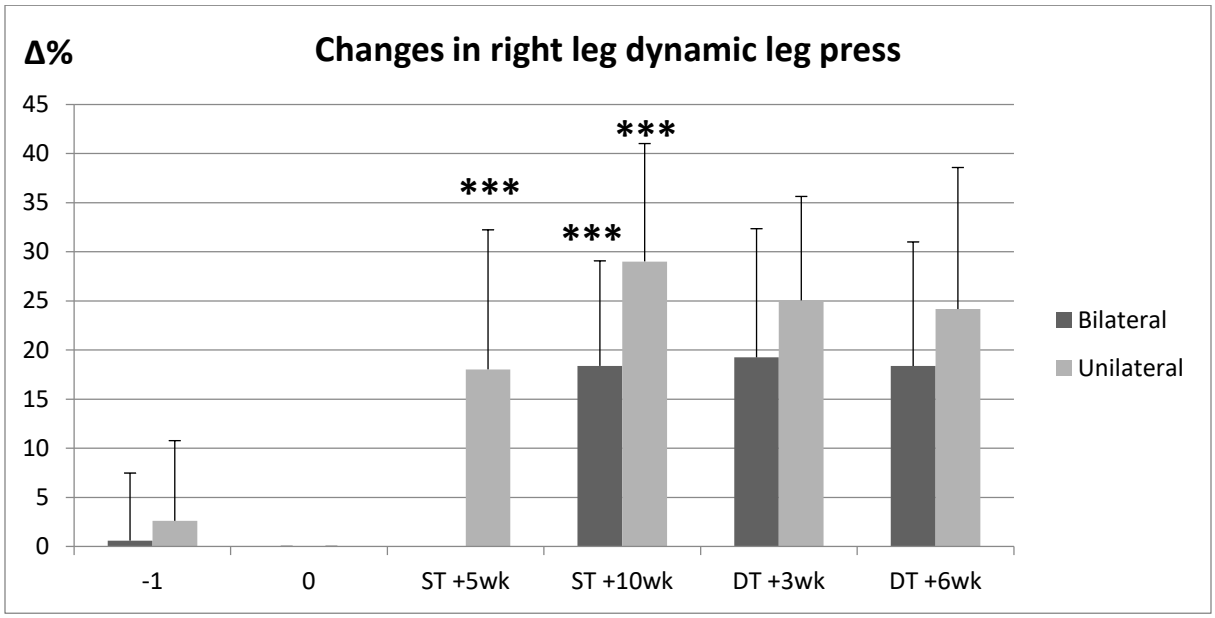


FIGURE 48: Changes (means and SD for relative changes) in right leg strength during the intervention.

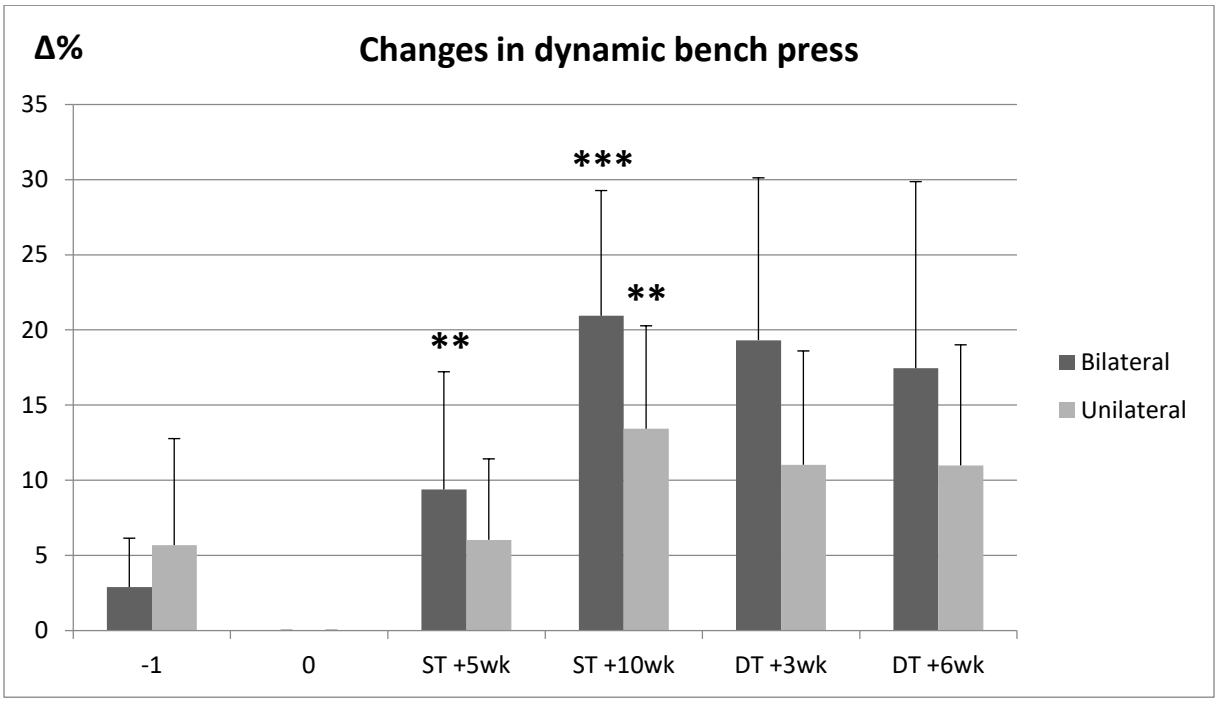


FIGURE 49: Changes (means and SD for relative changes) in bilateral bench press during the intervention.

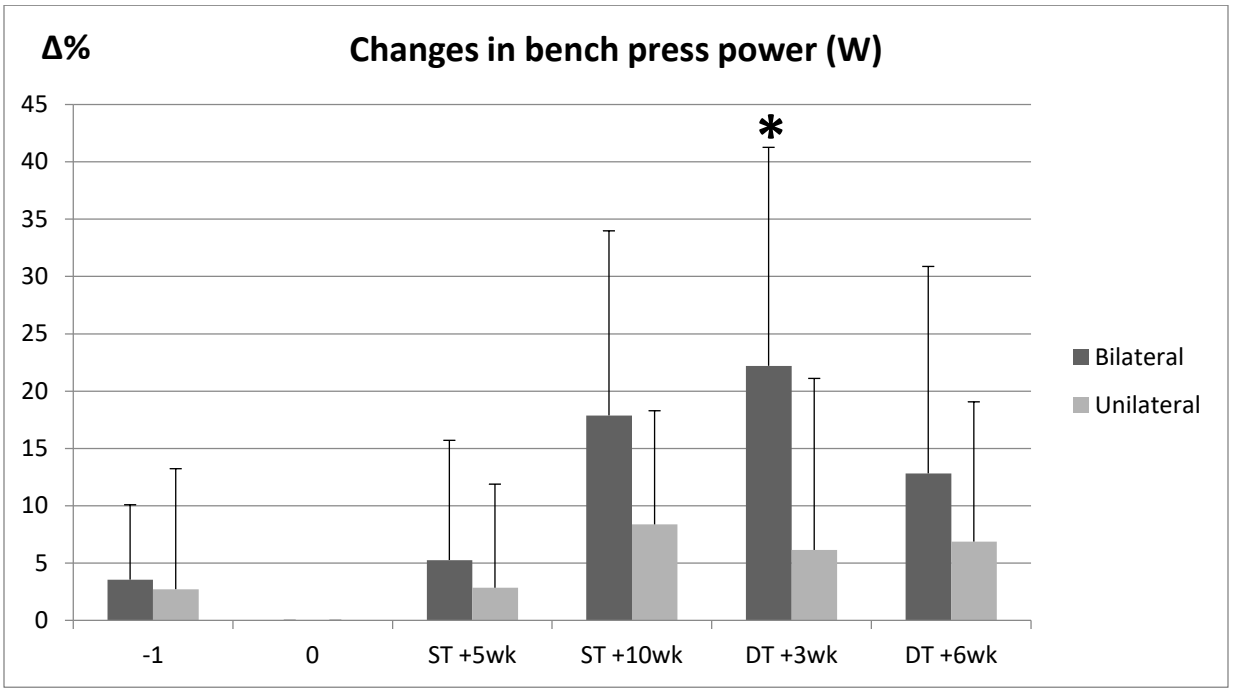


FIGURE 50: Changes (means and SD for relative changes) in bench press power during the intervention.

### 8.6 Comparison of the groups and bilateral deficit for the legs

There were no statistically significant differences between the unilateral and the bilateral groups in any of measured variables. In the beginning of the intervention bilateral deficit for the legs was -15.2 (±13.1) for the unilateral group and -15.2 (±4.88) for the bilateral group. Changes in bilateral deficit during the intervention are displayed in table 14. There were no statistically significant differences between the groups. However, the bilateral group's bilateral deficit changed statistically significantly from Pre to post ($p<0.0005$), pre to DT +3WK ($p=0.045$), pre to DT + 6WK ($p=0.042$).

TABLE 14: Bilateral deficit (means and SD) for the legs presented in different time points.

| **BILATERAL DEFICIT** | | | | |
|---|---|---|---|---|
| | | **Mean** | **SD** | **N** |
| **UNILATERAL** | PRE | -13.1 | 7.5 | 11 |
| | POST | -12.1 | 4.3 | 11 |
| | DT +3WK | -15.0 | 5.2 | 11 |
| | DT + 6WK | -12.7 | 7.4 | 11 |
| **BILATERAL** | PRE | -18.2 | 6.9 | 13 |
| | POST | -9.2 | 5.9 | 13 |
| | DT +3WK | -13.1 | 8.5 | 13 |
| | DT + 6WK | -11.3 | 10.9 | 13 |

## 9. Discussion

The present study included ten weeks of progressive hypertrophic resistance training followed by six weeks of detraining. In all dynamic strength tests statistically significantly increases took places from pre to post for both groups, indicating that our intervention was highly effective. After the strength training period, VL CSA increased in the total group from 31.1±5.7cm to 35.1±5.0cm. The subjects were split to three groups according to the increase in VL CSA: High responders >15% (n=10), Medium responders 15-5% (n=6) and Low responders <5% (n=8).

Our data indicates that high responders gained muscle mass already during the firsts two weeks of strength training. High responders gained +10.4 % (±8.4) in their VL CSA and even medium responders gained +6.9 % (±5.6). Even though the individual variation was rather large, the possibility to gain muscle sooner than usually proposed seems thus to be possible. The proposed late stage hypertrophy theory by, for example, Moritani & DeVries (1979), that hypertrophy will come important only after three to five weeks, was not supported by our intervention. The highest individual gain was from 29.9 cm to 36.4 (21.3%) cm only in two weeks. However, we measured hypertrophy only from one muscle (VL), the whole quadriceps hypertrophy was not measured. The results must therefore be interpreted carefully. Nonetheless, these individual findings underline the different individual adaptation capability of the neuromuscular system during well design strength training. Also Norrbrand et al. (2018) and Illera-Dominiguez et al. (2018) detected early adaptations in skeletal muscle size. Illera-Dominiguez et al. (2018) found in their study that only after 14 days the changes in the quadriceps CSA was 5.5% ± 1.9%. In both studies, the scans were performed after ≥96 h of recovery after training sessions to avoid muscle swelling. Krentz & Farthing (2010) reported significant changes in biceps brachii thickness after only eight days measured by ultrasound, but the tests were conducted only after 48 h after the hard strength session and there could be some muscle swelling. In addition, there was a decrease in strength, which would indirectly indicate that there were muscle damages still in the muscle, and that would cause swelling (Paulsen et al., 2012). In addition, Damas et al. (2016) have noted that early gains in hypertrophy are because of edema-induced muscle swelling in untrained individuals. In the present study we performed ultrasound scans after ≥48 h of the

heavy strength training session, but we used the echo intensity method to measure, if there were any muscle swelling in the muscles. Echo intensity scans revealed that there were no statistically significant changes after the first two weeks of strength training. This suggests that the present results would indicate that real muscle hypertrophy has happened. However, there was the decrease of - 3.1 % (±5.5) in echo intensity from pre to wk 2, although not significant, meaning possibly some muscle swelling may have taken place. Thus, the results should be interpreted with care. Interestingly, pre to post changes in echo intensity was + 4.4 (±7.4), not significant, but denoting that the muscle swelling decreased during the training program. The subjects may have accustomed to the strength training rather well during the present time period.

In addition, the data from our study indicates that high responders tend to lose muscle mass and strength a little bit faster than low responders during the detraining phase. High responders were the only ones to have a significant change in VLCSA from pre to mid, pre to post, pre to detraining 1, pre to detraining 2 as well as post to detraining 2. To the best of our knowledge, these results are unique and probably published for the first time that the different responder groups demonstrated different degree of muscle mass loss during the detraining phase.

The present study additionally showed that the activation level (AL) in high responders decreased most (-3.2% ± 5.8 compared to low responders -1.3% ± 3.6) during the strength training period (figure 32). This indicates that the neuromuscular system of the high responders was most tired after the strength training period. The training program could have suited the needs of high responders best, and they gained muscle mass quite a lot. For the low responders the training program utilized in the study could have been not that suitable. Damas et al. (2018) found that manipulation of resistance training frequency can alter individual responsiveness to strength training. Other factors, such as nutritional intake, daily rhythm, sleep habits, genetic environment, stress, etc., could also affect ability of subjects to adapt to strength training. Thus, changing the training factors might have had an impact to our results. Our training frequency was the same during the whole intervention and that could fit better for some and not that much for others. Furthermore, it must be taken into the consideration that the results reflect the adaptive capacity of individuals at a given time. Subjects did or did not respond to our intervention

training program, but they might behave differently, if the intervention would be repeated. (Pickering & Kiely, 2018.)

In the total group of subjects there was a small negative correlation between the baseline values and the percentage changes after the intervention both in the case of muscle hypertrophy (-0.09, p=0.65) and dynamic 1RM bilateral leg press strength (-0.07, p=0.72). These results indicate moderately that those with lower baseline values did gain somewhat more, but the correlation was very low and not significant. This means that baseline values did not affected to which responder group the individual subject ended.

There were statistically significant correlations between muscle size and strength at control, pre and again at detraining +6wk, but not during intervention at ST+5wk, ST+10wk and neither at DT+3wk. This suggests that the present hypertrophy strength training period diminished the correlation between those two variables. Other researchers have found that force relative to CSA only increases when the muscle fibers did not show hypertrophy (Widrick et al., 2002; Trappe et al., 2000; Pansarasa et al., 2009; Parente et al., 2008). However, in those studies the force relative to fiber diameter increased. Ahtianen et al. (2016) found that the correlation between quadriceps size and leg press strength was only r=0.157 (males and females, aged between 19 to 78 years, n = 287). Our data supports this, since in the post tests the correlation was r=0.174. Erskine et al. (2010) had similar results, when the correlation was r=0.14 or r=0.15, depending on how strength and muscle mass were measured. In addition, Hubal et al. (2005) measured a correlation of r=0.23. Overall, it seems that in untrained people hypertrophy can explain to a rather low extend of the strength gains during initial weeks of training. However, for strength trained athletes muscle size and strength have correlated strongly, when Baker et al. (1994) measured correlation of r=0.59 and Appleby et al. (2012) in turn measured correlation of r=0.692. On the other hand, Zourdos et al. (2015) noted a large increase in strength with no changes in the hypertrophy in trained powerlifters. Further, Siahkouhian & Hedayatneja (2010) found that the correlation between lean body mass and strength ranged from r=0.84 - 0.90 in elite weightlifters. Thus, muscle size may become more predictive of strength, when training status will mature but that does not explain all strength gains, because there are other factors like lateral

force transmission, voluntary activation, tendon stiffness, load-specific coordination, synergist activation, antagonist coactivation, muscle fascicle length, pennation angle, muscle moment arm length, myofilament packing density etc. Bickel et al. (2011) observed that subjects maintained their strength during the 32-week long detraining even though decreases in muscle CSA took place. However, subjects did some training during that period. Their training volume was one-third or one-ninth of what was their volume in the training period. We detected similar results when strength decreased only by -1.2 % ± 3.7, whereas VL CSA decreased by -4.9 % ± 7.3. In addition, Häkkinen et al. (2000) found that strength remained elevated during the 3-week detraining period despite the decrease in muscle CSA.

To sum up, strength may be easier to maintain compared to muscle CSA. Some authors have suggested that these adaptations are largely independent of each other (Buckner et al., 2016). More studies are needed to better understanding of this phenomenon.

**Unilateral and bilateral training.** There were no statistically significant differences between the unilateral and the bilateral groups in terms of hypertrophy and strength. Our researcher team was not able to identify differences between the mentioned groups. Both training types led to similar results in terms of hypertrophy and strength in our physical active male subjects. McCurdy et al. (2005) have observed similar results when compared unilateral and bilateral training in untrained population. Also Janzen et al. reported (2006) that there were no differences in lean body mass between unilateral and bilateral training groups in post-menopausal women. In addition, Speirs et al. (2016) found that unilateral and bilateral training is equal for lower-body strength, 40-m speed, and change of direction in academy level rugby players. Unilateral strength training (UST) can be used as a part of hypertrophy training and it is as effective to build muscle as bilateral training, contrary what the general belief is. Häkkinen et al., (1996) noted also that CSA did not differ significantly between bilateral and unilateral groups after 12-weeks, when elderly subjects trained for 12-weeks. However, they noticed that increased in the averaged maximum EMG, during the bilateral contractions, were larger in the bilateral group, than in the unilateral group, and the increase in unilateral contractions were significant only for the unilateral group, indicating that there were neural specificity between the bilateral and the

unilateral training. Neural specificity is even more emphasized when there were no differences in CSA between unilateral and bilateral groups. (Häkkinen et al., 1996.)

Subjects recovered faster from bilateral training than from unilateral training. The bilateral group's AL rose statistically significantly from post to DT+6wk. The unilateral group's results did not rise significantly. In contrast, Keitaro et al. (2010) found that AL didn't change during a 3-month detraining period. In the present study, the decrease of AL during the intervention and the rise of the AL during detraining would indicate that our hypertrophy strength training was really taxing for the neuromuscular system, maybe even too taxing. However, it seems that the unilateral training may be more demanding for the neuromuscular system and recovery from the unilateral training would take more time than recovery from the bilateral training.

Both groups improved isometric bilateral leg press statistically significantly. The unilateral group actually improved somewhat more (19.3%, p=0.002) isometric bilateral leg press than the bilateral group (15.6%, p=0.003), even though the unilateral group did not perform any bilateral activity during the whole intervention, in contrast to what Häkkinen et al. (1996) found. In their study, the bilateral group's average relative increase in bilateral leg press was greater than unilateral group's average relative increase.

The increases in the isometric unilateral leg presses were both statistically significant for the unilateral group. The bilateral group made only minor improvements in the unilateral leg strength tests and those were not significant. Actually, the unilateral group increased their result in all isometric leg press testes, while the bilateral group improved their result only in the isometric bilateral leg press test indicating that unilateral training may have had larger crossover to different strength tests. Rutherford & Jones (1986) concluded that the ability to lift weights depends on an ability to coordinate other muscle groups which are involved in the movement. The unilateral group seems to have been making a bigger effort to stabilize the leg during the unilateral movements. That might lead to better coordination of involved muscles and better strength performance.

Strength training using unilateral bench press actions seemed to be a good method for developing isometric and dynamic strength in the bench press. The unilateral group improved statistically significantly isometric bilateral bench press by 18.5 % ± 13.5 from pre to post. In turn, the bilateral group improved also statistically significantly bilateral bench press, augmenting 19.4 % ± 13.9 from pre to post. In the testing situation the subjects were tied to the bench, so they could not twist their bodies, but during the training that was not utilized. Patterson et al. (2015) noticed that unilateral dumbbell bench pressing has larger range of motion in comparison to bilateral conditions. Thus, unilateral training could have been so effective, because there could have been a greater range of motion, when unilateral dumbbell bench pressing was utilized in comparison to bilateral group. The unilateral group trained with equal volume, compared to the bilateral group, but the use of greater range of motion could have led to bigger neuromuscular respond. The unilateral group improved their unilateral left arm results by 12.1 % ± 16.8, while the bilateral group improved 7.9% ± 10.4, neither result was significant. In the right arm test the bilateral group improved statistically significantly from pre to post, whereas the unilateral group did not improve significantly, even though they had larger improvement.

To the best of our knowledge, this was the first time that the unilateral bench pressing was trained and tested for a prolonged time period. It can be said that this unique way of training and testing isometric bench pressing was a success. The unilateral group improved their dynamic bilateral bench press by 13.4% ± 6.9 from pre to post and that, by not doing any bilateral movements. However, the corresponding values of the bilateral group was 21.0% ± 8.3, demonstrating much larger increases. In addition, only the bilateral group improved bench press power from pre to post statistically significantly, while the unilateral group achieved only minor improvements in their bench press power (figure 51). This suggests that, for power training, bilateral bench pressing seems to be superior. This could be because of the complexity of unilateral bench pressing. It may be difficult to focus on creating power, when your body is in an unstable position, whereas it could be much easier for the bilateral group, perhaps due to a more balanced body position. Anderson & Behm (2004) have suggested that instability would lower maximal force, however, Goodman et al. (2008) showed that there were no differences in strength and muscle activation when bench press was done on a stable or on a unstable surface. It

is possible that, because we tested bilaterally, the results benefited the bilateral group more. The results could be different, if it would have been possible to measure unilateral upper body power.

The gains in the bilateral group were lost quite fast in the dynamic bench press. After the detraining the bilateral group had lost more strength than the unilateral group. For the bench press, high frequency and only short pauses from the training can be recommended, if the goal is maximize the bench press result. The drop -7.0 ± 20.4 from post to detraining 2 was statistically significant for all subjects. This indicates that bench press needs to done more frequently to gain or to maintain strength. Upper body muscle loss (TB) was also statistically significant for the whole group of subjects after the detraining (-9.4 ± 8.0). Walking and everyday activity may preserve some strength in the legs, but the normal life was not able to preserve the gains in the upper body.

The bilateral deficit changed statistically significantly for the bilateral group from pre (-18.2 ± 6.9) to post (-9.2 ± 5.9), while in the unilateral group results it remained quite unchanged. Janzen et al. (2006) reported similar results. Bilateral training decreased the bilateral deficit in post-menopausal women and unilateral training had only a minimal effect on the bilateral deficit. This suggests that bilateral training can decrease the bilateral deficit, and lead in highly trained bilateral athletes to bilateral facilitation (bilateral strength would be greater than the summed unilateral strengths) (Howard & Enoka, 1991; Taniguchi, 1997). Thus, this phenomenon can be modified with the proper bilateral training. The values of the unilateral group did not change statistically significantly, suggesting that that bilateral deficit cannot be changed with unilateral training. This can be seen as an evidence for a training specificity. Rutherford & Jones (1986) reported that strength tasks improved with learning, so it might be possible that the subjects are not familiar to performing maximal bilateral strength tests. Familiarization has been used to decrease the bilateral deficit (Secher et al., 1988). It has been suggested that higher-order neural inhibition is one of the reasons for the bilateral deficit (Ferbert et al., 1992; Ohtsuki, 1983). This inhibition seems to decrease with bilateral training.

There were some interesting statistically significant changes in legs muscle activity. VL activity in the unilateral leg press was statistically significantly increased in bilateral group from pre to post, but not in the unilateral group, even though the unilateral group has larger increase. The same thing happened in the bilateral leg press, when the unilateral group increased significantly from pre to post, but the bilateral group did not. However, the magnitude of changes was almost similar in both movements in both groups, but the standard deviation was larger in another group enabling statistically significant changes.

For TB, there were statistically significant changes in both groups from post to detraining 2, indicating that the quick loss of dynamic bench press strength is also associated with the loss of muscle activity. There were similar changes in the muscle activity of AD. The decrease from post to detraining 2 was statistically significant for the unilateral group and for the whole group. This would be another evidence that bench press need higher frequency than lower body training. There were also some quite interesting findings from pre to post. In the bench press muscle activity seems to be task dependent, even though both groups improved their muscle activity. Higher results were always obtained by the group which also trained as they were tested. For example, the unilateral group improved muscle activity in the unilateral bench press more than the bilateral group, while in the bilateral bench press the situation was vice versa. In addition, the unilateral group seemed to retain their muscle activity in both ways a little bit better than the bilateral group. Häkkinen et al. (1996) showed a specificity effect in leg press 1RM, so that bilaterally trained subjects increased their bilateral 1RM strength significantly greater than unilaterally trained subjects. In the unilaterally 1RM strength the situation was vice versa. Unilaterally trained subjects increased their unilateral 1RM more than bilaterally trained subjects.

There were no major new findings in basal concentrations of serum hormones suggesting that the basal concentration levels may not be altered with strength training. However, the standard deviation for the testo/shbg ratio increased vastly, when the intervention reached 10 weeks. This indicates very individual hormonal respond to hypertrophic strength training. Ahtiainen et al. (2003) reported similar results, when there were no significant changes in basal serum

concentrations when untrained and strength-trained men trained for 21-weeks. However, they noticed that the basal testosterone and free testosterone increased during the first 14 weeks and decreased from week 14 to week 21 in strength-trained men. The volume of the training increased during the first 14 weeks and decreased during the second part of the intervention. Also Häkkinen et al. (1987 & 1988) showed that volume/intensity of strength training affected to the serum testosterone concentrations. These findings suggest that serum testosterone concentrations can differ with regard to the volume of the strength training and can be an important factor for strength development in strength-trained men. Häkkinen et al. (1985c) found also that the changes in strength and in hormonal balance correlated significantly during the later stressful training weeks of the prolonged training intervention and during the detraining period. They concluded that the levels of biologically active unbound testosterone may be important for trainability. Ahtiainen et al. (2003) found also a correlation between testosterone concentrations and the changes in isometric strength in strength-trained men. For untrained people Ahtianen et al. (2003) suggest that they may gain strength and muscle mass without the changes in the basal serum testosterone concentrations. We found that serum testosterone concentrations increased steadily (mid = 4.4 ± 19.0, post = 6.3 ± 44.3, after detraining = -0.4 ± 23.4). We also conclude that untrained people can gain strength and muscle mass without major changes in the basal serum testosterone concentrations.

**Limitations.** The present study had several limitations that must be considered when attempting to draw evidence-based conclusions. Firstly, the low sample size of in all 24 participants was a limitation. Furthermore, the strength training intervention lasted only 10 weeks and although this period was sufficient to achieve significant increases in muscular strength and hypertrophy for both groups, it is possible that results between the groups could have diverged with a longer intervention protocol. Secondly, the measurements of TB thickness were obtained only at the middle portion of the muscle. Although this region is often used as a proxy of overall growth of a given muscle, research indicates that hypertrophy manifests in a regional specific manner, with greater gains sometimes observed at the proximal and distal aspects (Wakahara et al., 2012). The possibility therefore exists that differential changes in proximal or distal MT may have occurred, which would have left undetected in our protocol.

It is also important to highlight that we gave protein and carbohydrate supplementation to the subjects, and the participants also received an individual diet plan before the intervention began. However, the following of the diet was not controlled in any way. The diet is an important part of any kind of strength training and can heavily influence the results of the intervention (Cermak et al., 2012; Schoenfeld et al., 2013; Nissen & Sharp, 2003).

Finally, the findings of our study are specific to young physically active men and, therefore, cannot be generalized to other populations. Future research is required to determine the optimal training intensity, volume, exercise selection and programming for different responder groups.

**Practical implications.** Unilateral strength training seems to have a larger crossover and skill transfer than bilateral training. It can be a useful tool also in sports training. In addition, it can be used as a part of hypertrophy training and is at least as an effective tool for hypertrophy as bilateral training is. Finally, there were a lot of different responders to our strength training program. Some responded well to our intervention, whereas others did not. It is important to remember that all programs will not work for all and individual tailoring is needed when building a strength/hypertrophy training program.

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## 10. Conclusions

The present study included ten weeks of progressive hypertrophic resistance training followed by six weeks of detraining. 26 male subjects (age 24.6±3.8 yrs, height 180.7±7.3 cm, weight 77.0±10 kg) started the intervention and 24 subjects finished the whole intervention.

After the strength training period, we were able to identify three different responder groups for hypertrophic strength training. Our results indicate that High responders in muscle CSA tend to lose VL CSA and dynamic bilateral strength somewhat faster than Low responders during the detraining phase. Muscle hypertrophy in High responders was observed to appear already during earlier weeks of strength training. The results highlight the different adaptation capacities of different individuals. In addition, it expresses the need of individual tapering and detraining programs for maximum development in hypertrophy and maximal strength in the long term

There were no statistically significant differences between the unilateral and bilateral groups. Both training types will lead to similar results in terms of hypertrophy and strength in physical active young men. Unilateral strength training is as an efficient way to build muscle and strength as the bilateral strength training is.

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## Attachments

The few examples from the training program can be found attached.

| Intervention training program | Week 1 |
|---|---|
| **Session 1** | |
| | Dynamic leg press 5x10xRM |
| | Isometric knee extensors (90deg.) 2x60s(5s work + 15s rest) |
| | Isometric knee flexors (90deg.) 2x60s(5s work + 15s rest) |
| | Three rounds of core work |
| **Session 2** | |
| Leg press: Bil. + Uni | 3x10x70% |
| Knee ext.: Bil. + Uni | 3x12x60% |
| Knee flex. Laying down: Bil. + Uni | 3x12x60% |
| Dumbell benchpress: Bil. + Uni | 3x10x50% from BP |
| Seated french press with DB: Bil. + Uni. | - |
| Horizontal row with narrow grip: Bil. + Uni | 3x12x60% |
| Plank + isometric back extension | 2x45 s + 10 |
| **Session 3** | |
| Bench press done in smith: Bil. + Uni | 3x10x70% |
| Horizontal row with narrow grip: Bil. + Uni | 3x12x60% |
| Zotmann curl with dumbell: Bil. + Uni | 3x12x60% |
| Leg press: Bil. + Uni | 3x10x70% |
| Knee extensors : Bil. + Uni | 3x12x60% |
| Knee flexors: Bil. + Uni | 3x12x60% |
| Seated abdominals | 2x10 |

| | Week 6 |
|---|---|
| **Session 1** | |
| | Isometric leg press testing |
| | isometric knee extensors testing |
| | Dynamic leg press 5x10xRM |
| | Isometric leg press testing |
| | isometric knee extensors testing |
| | Isometric knee extensors (90deg.) 2x60s(5s work + 15s rest) |
| | Isometric bench press (90deg.) 2x60s(5s work + 15s rest) |
| | Knee flexors Bil. + Uni. 3x12x70% |
| | Lat pulldown Bil. + Uni. 3x12x70% |
| | Superman - core exercise 3x10 |
| **Session 2** | |
| Leg press: Bil. + Uni | 5x4x90% |
| Knee ext.: Bil. + Uni | 3x10x70% |
| Stiff legged deadlift with DB: Bil. + Uni | 3x10x70% |
| Dumbell benchpress with stop: Bil. + Uni | 3x8x72,5% |
| Seated french press with db: Bil. + Uni. | 4x8x70% |
| Horizontal row with wide grip: Bil. + Uni | 3x10x70% |
| Seated abdominals | 3x15 |
| **Session 3** | |
| Bench press done in smith: Bil. + Uni | 5x4x90% |
| Seated overhead press in machine Bil. + Uni. | 3x10x50% |
| Zotmann curl with dumbell: Bil. + Uni | 2x15x50% |
| Row with dumbell in incline bench: Bil. + Uni. | 3x10x70% |
| Triceps push down: Bil. + Uni | 3x10x70% |
| Leg press one and half rep. Bil. + Uni | 3x6x60% |
| Knee flexors: Bil. + Uni | 3x10x70% |
| Side plank + Dynamic back extensions | 3x30 s per side + 15 |

| | Week 8 |
|---|---|
| **Session 1** | |
| | Dynamic leg press 5x10xRM |
| | Isometric knee extensors (90deg.) 2x60s(5s work + 15s rest) |
| | Isometric bench press (90deg.) 2x60s(5s work + 15s rest) |
| | Knee flexors 3x12x70% |
| | Lat pulldown 3x12x70% |
| | Superman - core exercise 3x10 |
| **Session 2** | |
| | Leg press max tests (New training weights): |
| Leg press: Bil. + Uni | 4x6x80% |
| Knee ext.: Bil. + Uni | 3x6x80% |
| Stiff legged deadlift with DB: Bil. + Uni | 3x6x80% |
| Dumbell benchpress with 6s eccentric phase: Bil. + Uni | 4x6x80% |
| Dumbell incline benchpress: Bil. + Uni | 2x12x65% |
| Horizontal row with narrow grip: Bil. + Uni | 3x6x80% |
| Plank + isometric back extensions 5kg | 3x45 s + 10 |
| **Session 3** | |
| | Bench press max tests (New training weights): |
| Bench press done in smith: Bil. + Uni | 4x6x80% |
| Biceps curl with dumbell: Bil. + Uni | 3x12x50% |
| Seated overhead press in machine Bil. + Uni. | 2x10x50% |
| Triceps push down 1s hold in lock position: Bil. + Uni | 3x6x80% |
| Leg press with 6s eccentric phase: Bil. + Uni | 4x6x70% |
| Knee extensors: Bil. + Uni | 3x6x80% |
| Side plank + Dynamic back extensions | 3x30 s per side + 10 |