Introduction
Resistance training induces adaptations in the neuromuscular system. However, the magnitude of specific adaptations between individuals varies noticeably. Different responsiveness of human beings was first noted (39) in 1954, when it was 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, training history, physical activity level, and the endocrine status (13). However, data from the HERITAGE study presents that age, gender, and race have only a minor impact on interindividual differences in training responses. On the other hand, training history, environment, and genetic factors might have a greater influence on the magnitude of adaptations (9).

Resistance training has been shown to induce adaptations of various scales (3,22). In the largest study to date with the total number of 585 subjects, 232 subjects showed increases in the cross-sectional area (CSA) of trained elbow flexor muscles between 15 and 25%, 10 subjects gained more than 40%, and 36 subjects gained less than 5% (22). There were high ranges in strength gains as well. It can be concluded that there is a large variation between responsiveness to a certain stimulus. Additionally, Ahtiainen et al. (3) found considerable interindividual variation (n = 287) in both muscle size and strength adaptations. Some individuals responded favorably by gaining muscle size but not strength, whereas others responded in strength but not size. They also noted that 30% of subjects were low responders to lower-body hypertrophy, but only 7% were low responders to strength adaptations. It is more common to be a low responder to muscle size than muscle strength (3).

Skeletal muscle tissue has extraordinary plasticity and can adapt to variable states of neuromuscular activity. It will readjust to reduced physiological stress during a reduced use of muscles (35). Detraining is the phase when subjects do not train. During detraining, the decrease in muscle force is explained by both neural and muscular adaptations caused by the inactivity period (35). Dedraining is the phase when subjects do not train. During detraining, the decrease in muscle force is explained by both neural and muscular adaptations caused by the inactivity period (24). Hakkinen et al. (25) reported that after 24 weeks of resistance training, a 12-week detraining period led to the great decrease in maximal strength, and individual strength decreases correlated with individual decreases in the maximum Integrated electromyography (cEMG) of the leg extensor muscles. It seems that beginners may maintain their maximal strength without training up to 2 or 3 weeks and that short-term detraining will lead only to minor changes, whereas prolonged detraining resulted also in muscle atrophy and further decreases in strength (26). Moreover, 4 weeks of detraining may induce larger declines in muscle power output than in maximal strength after 16 weeks of resistance training (31). After 60 days of unilateral strength training, 40 days of detraining led to decreases in muscle CSA, maximal muscle iEMG, and maximum voluntary force with about a

Key Words: strength training, hypertrophy, high and low responders, detraining
similar time course compared with the training period. In addition, the kinetics of changes in CSA, force, and neural drive during training and detraining seem rather similar (36).

Strength-trained men can retain strength and muscle mass during a 2-week period of detraining (23). Short-term detraining may specifically affect eccentric strength and the size of the type II muscle fibers, leaving other aspects of neuromuscular performance uninfluenced (21). Muscle fiber CSA declines rapidly in strength and sprint athletes (35). In general, strength performance may be retained for up to 3–4 weeks of inactivity, but highly trained athletes’ eccentric force and sport-specific power may suffer significant declines (35). It is possible that different responders might react differently to a detraining phase.

It could be concluded that human beings can respond rather individually to strength training. However, less is known about responders’ adaptations to the following detraining phase. The purpose of the present study was to investigate whether subgroups of different individual responders could be observed in muscle hypertrophy and strength during 10 weeks of progressive hypertrophic strength training and how those different responders would behave during the detraining phase following the training period.

**Methods**

**Experimental Approach to the Problem**

To study whether different responders can be observed during the training and detraining periods, a total of a 16-week intervention was designed. The study included 2 measurement points before the intervention. The actual intervention included 10 weeks of progressive hypertrophic resistance training and 6 weeks of detraining. The control measurements were performed first. Thereafter, a 1-week control period (with no strength training) took place, and the measurements were repeated at the pretests. The control period was used as a reference in the figures and tables for the measurement error size. Thereafter, the strength training period started lasting for 10 weeks, and the midtests were performed at week 5. The postmeasurements were performed after the training period. Thereafter, the detraining period started lasting for 6 weeks. During the detraining period, the measurements were performed also in the middle of the detraining (at week 13) and after the end of the detraining at week 16 (Figure 1). The measurement sessions for individual subjects were performed at the same time of day during the study period.

After the strength training intervention, the subjects were split into 3 different subgroups: high responders (HR), medium responders (MR), and low responders (LR). The level of responsiveness was determined based on how much muscle growth took place in vastus lateralis CSA (VLCSA). Panoramic ultrasonography was used to measure VLCSA.

**Subjects**

Twenty-six healthy young men (age, 19–30 years) from the city of Jyväskylä, Finland, were recruited to participate in the study. Recruitment was done through advertisements in a local newspaper, websites, and bulletin boards of the University of Jyväskylä, on the social media and the University of Jyväskylä 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 measurements or training intervention. Subjects needed to be recreationally physically active but without a systematic strength training background. The subject was considered physically active if they had moderate activity weekly. Performing endurance training or team sports more than once a week was also an exclusion criterion. In addition, subjects were advised not to participate in any endurance or team sports activities during the intervention.

All recruited subjects attended a screening session for resting electrocardiogram (ECG) and resting blood pressure. Furthermore, they were interviewed about their general health and motivation toward the study. A cardiologist went through the subjects’ ECG data before they were given a position as a subject. Overall, 32 subjects went through prescreening, and out of these, 26 healthy subjects (age, 24.6 ± SD 3.8 years; height, 180 ± SD 7.3 cm; mass, 77.0 ± SD 10.0 kg) started the study. Each subject was informed of all potential risks and discomforts of the study and the possibility to dropout from the research project at any time. After that, they signed a written informed consent document. Two subjects dropped out of the study because of health problems unrelated to the study.

The study received ethical approval from the Ethics Committee of the University of Jyväskylä and was conducted according to the Declaration of Helsinki.

**Procedures**

**Training.** The intervention lasted 16 weeks including 10 weeks of strength training. The subjects trained 3 times per week. There was always at least 1 full day of rest between training sessions. The first session of the week was held either on Tuesday or on Wednesday depending on the subject’s availability. The second training session was held on Friday, and the subjects started each their training session from the legs and moved on to the upper body. On the third session of the week, which was on Wednesday or Sunday, it was vice versa to minimize the order effect.

Overall, 30 training sessions were conducted during the intervention. The average participation number in the training sessions was 29.1 ± 0.93 sessions, resulting in the participation rate of 96.9%. All the training sessions were supervised by an expert from the study group.

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### Figure 1. Overview of the experimental design of the study. The control tests lasted 1 week; thereafter, the control period was 1 week, the strength training intervention lasted 10 weeks, the detraining period was 6 weeks, and overall, the timeline covered 18 weeks.
The training program consisted of 3 initial medium weeks so that the volume of training was progressively increased, and one set was added to both bench press and leg press exercises every week, followed by 4 hard weeks, when the volume and intensity were both raised. The intensity was increased in bench press and leg press 5% every week, and the volume was raised so that one set was added to accessory exercises every week. It was followed again by 3 medium weeks with the same volume but with a progressively rising intensity. The intensity was raised again in both bench press and leg press by 5% every week. A medium week was described, when only one variable increased (volume or intensity). A hard week was described when 2 variables were increasing (volume and intensity). Overall, the volume of training (sets) increased over the first 7 weeks, and thereafter, the volume remained approximately the same and the training intensity increased (percentage loads from the 1-repetition maximum [1RM]). The intensity was increased by increasing the percentage load. The subjects trained either unilaterally or bilaterally. The overall training volume was carefully equated between the groups.

The training program consisted of leg press and bench press 3 times per week (Table 1). The training program also included knee extension, knee flexion, dumbbell bench press, seated French press, elbow flexion and extension movements, horizontal row, and core exercises. The eccentric portions of the lift were always done with the 3-second tempo and the concentric portion of the lift as fast as possible. Rest time between the main exercises was 3 minutes, and between the accessory exercises, it was 60 seconds. Rest time between the sets remained the same during the whole intervention. In addition, the subjects did isometric training in the knee extension and in knee flexion machines. The knee angle was 90° in both exercises. The subjects also did isometric bench press with the elbow angle of 90°. The isometric training covered approximately 5% of the whole volume of the intervention. The results of the isometric training were always shown to the subject, and he was then encouraged to go over the previous value.

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. However, implementation of the nutritional plan was not controlled in this study.

Detraining. The subjects were advised to continue their life in the same way as they did before the training intervention. However, they were instructed not to do any strength training or high-intensity physical activity. They were allowed to do normal daily physical activities, for example, short commute biking (<5 km) and physical activity related to household chores. The detraining process was controlled by a subjective questionnaire at week 3 and again at week 6. Every subject replied that they had followed the instructions given for the detraining period and had not performed any strength training or other intensive physical activities.

Data Collection and Analyses: Whole-Body Composition and Lean Body Mass. Dual-energy x-ray absorptiometry (DXA) (LUNAR Prodigy Advance, GE Medical Systems, Madison, WI) was used to measure whole-body composition and lean mass before and after the intervention. Dual-energy x-ray absorptiometry was used only at before and after the 10-week training 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 using Styrofoam and elastic straps and the arms by rice bags to prevent any movement during the scan. The subjects came to the DXA scan overnight fasted, and they had been 24 hours without training. They could have one cup of water in the morning before the scan. Before the measurement, all metal objects were removed from the subject, and they were instructed to be in their

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Training program and exercise selection.*†</th>
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<tbody>
<tr>
<td></td>
<td>Weeks 1–3</td>
</tr>
<tr>
<td><strong>Tuesday</strong></td>
<td>Dyn LP 5 × 10 × RM</td>
</tr>
<tr>
<td></td>
<td>Iso KE 2 × 60 s</td>
</tr>
<tr>
<td></td>
<td>Iso knee flexion 2 × 60 s</td>
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<tr>
<td></td>
<td>Three rounds of core work</td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Friday</strong></td>
<td>Dyn LP 3 × 10 × 70%</td>
</tr>
<tr>
<td></td>
<td>Dyn KE 3 × 12 × 60%</td>
</tr>
<tr>
<td></td>
<td>Dyn prone knee flexion 3 × 12 × 60%</td>
</tr>
<tr>
<td></td>
<td>Dyn DB BP 3 × 10 × 50%</td>
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<tr>
<td></td>
<td>Dyn seated French press with DB 2 × 10 × RM</td>
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<tr>
<td></td>
<td>Dyn narrow grip HR 3 × 12 × 60%</td>
</tr>
<tr>
<td></td>
<td>Plank + iso BE 2 × 45 s + 10 s</td>
</tr>
<tr>
<td><strong>Sunday</strong></td>
<td>Dyn LP 3 × 10 × 70%</td>
</tr>
<tr>
<td></td>
<td>Dyn narrow grip HR 3 × 12 × 60%</td>
</tr>
<tr>
<td></td>
<td>Dyn zottmann curl with DB 3 × 12 × RM</td>
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<tr>
<td></td>
<td>Dyn LP 3 × 10 × 70%</td>
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<tr>
<td></td>
<td>Dyn KE 3 × 12 × 60%</td>
</tr>
<tr>
<td></td>
<td>Dyn prone knee flexion 3 × 12 × 60%</td>
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<tr>
<td></td>
<td>Seated abdominals 2 × 10</td>
</tr>
</tbody>
</table>

*RM = repetition maximum; Dyn = dynamic; Iso = isometric; LP = leg press; KE = knee extension; BP = bench press; DB = dumbbell; HR = horizontal row; BE = back extension.
†Isometric training was done so that 5 seconds was work and 15 seconds was rest. That was repeated 3 times and that was counted as 1 set.
underwear. The same investigator performed all the measurements and analyses.

**Muscle Cross-Sectional Area.** Vastus lateralis 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. Anatomical landmarks for the CSA determination were measured from the middle section between the joint space on the lateral side of the knee and to the greater trochanter. The 40% of femur length was marked, and the line was drawn from the lateral to medial diaphysis of the right thigh. 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 used. Great care was taken not to compress the muscle tissue. Ultrasound images were combined automatically to a panorama view in the device. Three panoramic CSAs were measured, and the mean of those was used in the analyses. The CSA was then determined with Image-J program (version 1.37, National Institute of Health). Within Image-J, the analysis 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 to previous images (2). Great care was used to complete the analyses. The same investigator performed all the measurements and analyses.

**Echo Intensity.** Echo intensity was assayed by mean gray scale analysis using the standard histogram function in Image-J. The mean echo intensity was used in the analyses. It is the number between 0 and 255 (a complete black is 0, and a complete white is 255). In vastus lateralis, the echo intensity was determined from the analyses. The CSA was then determined with Image-J—program (version 1.37, National Institute of Health). Within Image-J, the analysis 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 to previous images (2). Great care was used to complete the analyses. The same investigator performed all the measurements and analyses.

**Electromyography and Isometric Force.** Maximal bilateral leg extensor strength was measured on the custom-built horizontal leg press (Biology of Physical Activity, University of Jyväskylä) at a knee angle of 107°. Muscle activity was recorded during the isometric strength testing from the agonist muscles, VL and vastus medialis (VM), of the right leg. Skin was prepared by shaving, scraping, and disinfecting. Thereafter, the electrodes were placed according to SENIAM guidelines (20). 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 measurement during the study (24). Electrodes were bipolar Ag/AgCl electrodes with 5-mm diameter and 20-mm interelectrode distance.

During the measurements, the raw signals were amplified (500 gain) at a bandwidth of 10–500 Hz, the sampling frequency was 3,000 Hz. Thereafter, the signals went through the transportable pack to the receiving box (Telemyo 2400R; Noraxon, Scottsdale, AZ) and then to an AD converter (Micro1401; Cambridge Electronic Design, United Kingdom) and recorded by Signal 4.04 software (Cambridge Electronic Design). Electromyography (EMG) signals were analyzed by a customized script. Maximum iEMG values were obtained at the contraction period of 500–1,500 milliseconds (ms). The highest values of the VL and VM were combined and expressed as a mean value.

**Quadriceps Muscle Electrical Stimulation.** Constant current stimulator (Digitimer Stimulator Model DS7AH; Digitimer Ltd., United Kingdom) was used to stimulate the quadriceps muscle group of the right leg. Four, galvanically paired electrodes (6.98 cm V-trodes, Mettler Electronics Corp.) were placed on the proximal and middle regions of the quadriceps muscle so that they would cover up muscle CSA as much as possible. Skin under the electrodes was shaved and disinfected.

The resting stimulation was performed first. The subjects sat on the custom-made chair with the knee angle of 107°. Their right leg was strapped into the chair, and the left leg was placed to the platform in front of them so that it could be relaxed. Upper limbs were crossed in the lap. Single 1-ms pulses were given by a constant-current stimulator until a force plateau was found. Thereafter, the maximum voluntary contraction was produced, and an additional 25% of stimulation was added to the identified current. During the maximal voluntary contraction (MVC), hands were instructed to keep on the side of the bench. The stimulation was given during the plateau of peak torque and then one more pulse 2 seconds after a contraction to assess voluntary activation.

**Dynamic Strength Testing.** Maximal bilateral concentric of 1RM was measured in the leg press (David 210; David Health Solutions Ltd, Helsinki, Finland). In the starting position, each subject was seated in the device with a knee angle of 60°. They were required to lift the load to a fully extended position. The weight was progressively increased using 5-kg increments, until the subjects could no longer lift the load. The rest time between efforts was 3 minutes.

**Transcranial Magnetic Stimulation.** Transcranial magnetic stimulation (TMS) was delivered using double-pulse, Magstim Bistim² Stimulator with a 7-cm figure-eight–shaped double cone coil (Magstim, Whitland, United Kingdom). The coil was positioned on the subject in the place that elicited the greatest motor-evoked potential (MEP) at rest; furthermore, subjects’ scalps were marked to keep the coil position constant and ensure corrected repositioning. Resting motor threshold (RMT) was defined as the lowest stimulus intensity to elicit a visible MEP with a peak-to-peak amplitude of 50 µV in 3 of 5 consecutive trials. Motor-evoked potentials of vastus lateralis muscle were elicited at 100, 110, 120, 130, and 140% of RMT. Ten trials for each intensity were recorded. The order and timing of these stimulation were randomized to prevent any anticipatory reactions by the subject. During these measurements, the subjects were asked to perform an attention task, which consisted of silently counting backwards from 200.

Motor-evoked potential responses to TMS were measured with the same EMG electrodes and settings as during the MVC measurements. Peak-to-peak amplitudes of each MEP were analyzed, and finally, an average of all the MEPs of different intensities were calculated.

**Blood Samples.** Blood samples were collected from antecubital vein via sterile techniques. Blood samples were drawn into serum tubes (Venesafe, Terumo Medical Co., Leuven, Belgium) by a qualified laboratory technician. Overall, 6 ml of blood were collected, which included approximately 2.5-ml serum. Resting
serum blood samples were obtained in the morning in the fasted state to determine basal hormone concentrations. The subjects fasted approximately 12 hours. The subjects could drink a glass of water before coming to the blood collection. All food and other liquids were prohibited. The collected blood was held for 15 minutes at room temperature before it was centrifuged for 10 minutes at the speed of 3,500 rpm (Megafuge 1.0R, Heraeus, Germany). Serum samples were then placed into the refrigerator (−20°C) and stored for future analysis. Serum testosterone, cortisol (C), growth hormone, and sex hormone–binding globulin (SHBG) were analyzed from the samples. Analyses were accomplished by chemiluminescent immunometric techniques (Immulite 2000) and hormone-specific immunoassay kits (Immulite, Siemens, IL). 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 Luteinizing hormone (LH). Intra- and interassay reliability (Coefficient of Variation %) were within acceptable limits (total testosterone = 8.3%, cortisol = 6.1%, SHBG = 2.5%, and LH = 3.6%).

Statistical Analyses

Standard statistical analyses were used for descriptive variables of means and SDs. Normal distributions were determined through the Shapiro-Wilk test, and acceptable levels of skewness and kurtosis were also checked. All dependent variables were evaluated using a 2-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 pairwise differences. Differences between the subgroups were analyzed by the 1-way ANOVA. SPSS Statistics version 24 (IBM corp., New York, NY) was used for statistical analyses. For all tests, the alpha level was set at \( p \leq 0.05 \).

Results

Vastus lateralis CSA increased in the total group of subjects statistically significantly by 10.7 ± 12.5% after 10 weeks of strength training (Table 2). Relative changes in VLCSA in each individual are shown in Figure 2. Both total lean mass and legs lean mass increased significantly from pre to post (Table 3). Dynamic bilateral 1RM strength increased significantly by 16.3 ± 11.8% after the 10-week intervention (Table 2).

After the intervention, subjects were split into 3 groups according to the magnitude of the increase of the VLCSA during the 10-week training period (Figure 3) as follows: HR >15% (n = 10), MR 15–4.5% (n = 7) and LR <4.5% (n = 7). High responders showed a significant increase in VLCSA from pre- to midtraining (+14.9 ± 5.1%; \( p = 0.008 \), from pre- to posttraining (+23.0 ± 6.3%; \( p = 0.002 \)), from pre- to detraining 1 (+10.0 ± 7.2%; \( p = 0.021 \)), and from pre- to detraining 2 (+12.7 ± 4.9%; \( p = 0.002 \)). Medium responders reached a statistically significant increase from pre- to posttraining (+7.2 ± 3.1%; \( p = 0.017 \)), whereas LR did not reach a significant change in VLCSA.

Only HR to hypertrophy increased their bilateral leg press 1RM significantly from pre- to posttraining by 21.1 ± 11.7% (\( p = 0.001 \)), pre- to detraining 1 by 23.8 ± 12.3% (\( p = 0.001 \), and pre- to detraining 2 by 19.1 ± 13.2% (\( p = 0.004 \)) (Figure 4). During the detraining phase, HR showed a decrease of −10.5% in their VLCSA, whereas MR and LR maintained their VLCSA (−0.7% and −0.6%, respectively). High responders and MR lost more strength (−20.0% and −25.0%, respectively) than LR (−0.9%), although not significantly (Figure 4).

There was a statistically significant increase in maximal EMG activity (mean of VL and VM muscles) of 21.3 ± 22.9% from pre to posttraining for the whole group in the bilateral leg press (\( p = 0.009 \)) (Figure 5). For the different subgroups, MR increased significantly their pre to posttraining maximum mean iEMG value in bilateral leg press (\( p < 0.001 \)) (Figure 5).

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>(Δ%) pre to post</th>
<th>SD</th>
<th>( p )</th>
<th>DT (±6 wks)</th>
<th>(Δ%) post to DT ±6 wk</th>
<th>SD</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>VLCSA (cm²)</td>
<td>32.1 (±5.9)</td>
<td>35.1 (±5.0)</td>
<td>10.7</td>
<td>12.5</td>
<td>0.025</td>
<td>33.2 (±5.1)</td>
<td>−4.9</td>
<td>7.3</td>
<td>—</td>
</tr>
<tr>
<td>Bilateral leg press 1RM (kg)</td>
<td>155 (±24.9)</td>
<td>179 (±25.0)</td>
<td>16.3</td>
<td>11.8</td>
<td>&lt;0.0001</td>
<td>176 (±24.6)</td>
<td>−1.2</td>
<td>3.7</td>
<td>—</td>
</tr>
</tbody>
</table>

*VLCSA = vastus lateralis cross-sectional area; RM = repetition maximum; DT = detraining.
None of 3 subgroups showed significant changes in the maximal voluntary activation level after the 10 weeks of strength training (Figure 6). In HR, MR, and LR, maximal activation level decreased at week 10 slightly by $-3.25 \pm 3.79\%$, $-0.52 \pm 3.06\%$, and $-1.82 \pm 3.49\%$, respectively. However, all subgroups showed some rebounds in maximal activation back at week 3 of detraining from posttraining, and in MR, this change was significant ($p = 0.038$).

The average MEP values of the different subgroups did not show any significant changes during different time points or differences between the groups (Figure 7). However, a decreasing trend in MEPs toward the end of the strength training intervention could be seen in all the groups. This reduction was significant at week 10, when the groups were calculated as one ($p < 0.01$). In addition, a clear recovery in MEPs could be observed at week 3 of detraining, especially for HR. This was not significant within or between the groups but again a significant joint effect could be found ($p < 0.01$).

In the total group of subjects, no significant changes occurred during the study period in serum basal hormone concentrations and SHBG (Table 4). Serum basal testosterone-to-cortisol ratio increased slightly from the pre value of 0.043 ($\pm 0.02$) to the post value of 0.049 ($\pm 0.025$) during the 10-week training period (Table 4). After 6 weeks of detraining, the testosterone-to-cortisol ratio slightly decreased close to the baseline. After the strength training period, the testosterone-to-SHBG ratio increased from 0.567 ($\pm 0.165$) to 0.664 ($\pm 0.481$) and further slightly increased after the detraining period up to 0.705 ($\pm 0.381$).

No significant changes were observed in the serum testosterone-to-SHBG ratio in 3 different subgroups during the 10-week strength training and 6-week detraining periods (Figure 8). The HR subgroup showed a higher, but not significant, testosterone-to-SHBG ratio throughout the training period until week 3 of detraining. There was a trend of a significant correlation ($r = 0.365; p = 0.079$) during the 10-week strength training period between individual changes in VLCSA and average individual values in the testosterone-to-SHBG ratio for the whole group (Figure 9).

### Discussion

The present 10-week, hypertrophic, strength training intervention increased maximal concentric 1RM strength significantly from pre to post by 16% in the total subject group. In addition, VLCSA increased in the total group by 10.7%, indicating that our intervention was effective. The present subjects could be split into 3 subgroups according to the magnitude of increase in VLCSA: HR $>15\%$ ($n = 10$), MR $15–4.5\%$ ($n = 7$), and LR $<4.5\%$ ($n = 7$).

The HR and MR subgroups increased VLCSA significantly from pre to post by 23% and 7%, whereas the LR did not achieve a statistically significant change. Mobley et al. (32) have found similar results in the responder subgroups, although all their subgroups increased statistically significantly significantly from pre to post. During the 6-week detraining phase, HR lost $-10.5\%$ of their VLCSA, whereas MR and LR lost only $-0.7\%$ and $-0.6\%$ of their VLCSA. In addition, both HR and MR lost more strength during detraining than LR. Thus, the HR group tended to lose muscle mass and strength faster than the other 2 responder groups during the detraining phase. It seems that strength training adaptations in muscle mass and strength take place more quickly for HR and MR in both directions. For LR, these adaptations seem to occur more slowly. Low responders might just be “slow responders” and might need more training time for adaptations to occur. During the detraining phase, the present LR did not lose those minor adaptations gained as much as the other 2 subgroups, indicating a slower adaptation time course. A longer, strength training, intervention period is needed for a better understanding of this phenomenon. To the best of our knowledge, these results are, however, quite unique and probably published for the first time that the different responder groups also demonstrated different degrees of muscle mass loss during the detraining phase.

The present study additionally showed that none of the subgroups displayed decreases in maximal dynamic strength during the first 3 weeks of detraining, and HR showed a slight (by 2.5%) rebound in their strength (Figure 4). The time course of this finding is interesting because Häkkinen et al. (1985a) (25) showed a very large decrease in both strength and maximal EMG after 4 weeks of detraining. A significant training–induced increase took place in maximal EMG (mean of VL and VM muscles) in bilateral leg press for the present whole group (Figure 5) and for MR after 10 weeks of strength training. Minor increases in the maximum voluntary AL in all subgroups occurred after strength training at week 5, whereas at week 10, a slightly decreased value ($-3.3 \pm 5.8\%$, ns) was observed especially in HR (Figure 6). This probably indicates that the neuromuscular system of the HR was most stressed by the end of the present strength training period.

Interestingly, during the first 3 weeks of detraining, no significant changes occurred in maximal EMGs in the bilateral leg press, and maximal voluntary AL turned into slight increases for all subgroups, with MR showing a significant difference. Only slight (ns.) decreases in maximal EMGs occurred during the latter 3 weeks of detraining and no changes in maximal AL. The data during the detraining suggest that the maximal neuromuscular performance in HR and MR may bounce somewhat back and up from strenuous strength training at week 3 of the detraining period compared with LR.

The present results also showed that MEP averages were at the lowest ($-10.8\%$) also for HR right after the strength training period (Figure 7). Interestingly, HR MEP increased very fast after 3 weeks of the detraining ($+19.6\%$ compared with the previous value) but decreased back close to the baseline value. Low responders remained again rather steady and increased by $+3.1\%$ of their MEP averages after the first part of the detraining but remained approximately in the same condition during the second parts of the detraining. Thus, when all the data are taken into account, it seems that HR have more fluctuation in all of these values measured. Muscle hypertrophy, strength gains, and muscle activation adaptations may occur faster in HR and that peak

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### Table 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre Mean (g)</th>
<th>Post Mean (g)</th>
<th>Δ%</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td>Total mass</td>
<td>75.2</td>
<td>78.2</td>
<td>4.2</td>
<td>0.01</td>
</tr>
<tr>
<td>SD</td>
<td>11.0</td>
<td>10.2</td>
<td>3.3</td>
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</table>

### Table 4

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre Mean (kg)</th>
<th>Post Mean (kg)</th>
<th>Δ%</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total mass</td>
<td>58,595.0</td>
<td>61,032.1</td>
<td>4.9</td>
<td>0.0001</td>
</tr>
<tr>
<td>SD</td>
<td>6,440.8</td>
<td>5,947.7</td>
<td>2.6</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

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maximal dynamic strength may be reached, not right after the strenuous strength training period, but after 2–3 weeks of detraining.

The present training program was effective for the needs of HR leading to considerable gains in muscle mass and maximal strength. For LR, the training program used might not have been that suitable. Manipulation of training variables could have induced different outcomes for different individuals. Manipulation of resistance training frequency can alter individual responsiveness to strength training (12). Higher training frequency may also confer a potentially superior hypertrophic adaptation (42). On the other hand, although higher training frequencies can accumulate greater volumes of training, the weekly resistance training frequency may not meaningfully impact muscle hypertrophy, when the volume is equated (40). However, the training volume might have a considerable impact on hypertrophic adaptations (16). Higher training volume is also associated with increased ribosomal biogenesis (18). High responders might have optimal ribosome biogenesis for hypertrophy. Ribosome biogenesis has occurred as an important regulator of muscle hypertrophy and maintenance by altering the translational capacity of muscle cells (17). There is also a difference in the overall signaling pathways as the HR’s cellular responses seem to be more like a growth response and in the nonresponders more like a magnified inflammatory response (41). The effect of training variables to the individual adaptations remains to be elucidated.

Figure 3. Relative changes (mean and SD) in VLCSA in 3 different subgroups after 2, 5, and 10 weeks of strength training and 3 and 6 weeks of detraining. Con refers to the control measurements before premeasurements. *Significantly greater than the corresponding pretraining value, *p < 0.05, **p < 0.01. VLCSA = vastus lateralis cross-sectional area.

Figure 4. Relative changes (mean and SD) in dynamic bilateral 1RM leg press strength in 3 different subgroups after 10 weeks of strength training and 3 and 6 weeks of detraining. Con refers to the control measurements before pre measurements. **Significantly greater than the corresponding pretraining value, **p < 0.01, ***p < 0.001. 1RM = 1-repetition maximum.
Other factors, such as nutritional intake, daily rhythm, sleep habits, genetic environment, stress, and the like, could also affect the ability of subjects to adapt to strength training. Thus, changing the training variables might have had an impact on the present results. The level of responsiveness has been suggested to be strongly affected by the duration of the exercise intervention, with more positive responses, when subjects train more (11, 33). This may indicate that LR would just need more specific training. Furthermore, it must be taken into consideration that the results reflect the adaptive capacity of individuals at a given time. In addition, in the total group of subjects, there was no correlation between individual baseline strength levels and individual gains in strength during the present strength training intervention. Thus, baseline values did not predict, which responder group the individual subject ended. Subjects did or did not respond to our intervention training program, but they might behave differently if the intervention would be repeated (38).

Our results also indicate that HR gained more muscle mass already during the first 2 weeks of strength training. Also, HR gained 9.2% (± 8.3) in their VLCSA, and even MR gained 6.3% (± 9.1). Even though individual variation was rather large, the possibility to gain muscle mass during the hypertrophic type of strength training sooner than usually proposed (34) may thus be possible. However, we measured hypertrophy only from one
muscle (VL), whereas the whole quadriceps hypertrophy was not measured. The results must, therefore, be interpreted with care. Few studies have also detected early adaptations in skeletal muscle size (30,37). Illera-Domínguez et al. (30) found that only after 14 days of strength training, a large change in the quadriceps CSA took place (5.5% ± 1.9%). In the present study, ultrasound scans were performed after ≥ 48 hours from the last strength training session, but we used the echo intensity method to measure whether there would be any muscle swelling in the muscles. These echo intensity scans revealed no statistically significant changes after the first 2 weeks of strength training. This suggests that muscle hypertrophy may have taken place. However, there was the decrease of –3.1% (±5.5) in echo intensity from pre to week 2 (ns.) indicating possibly that some muscle swelling may have taken place. Thus, the results should be interpreted with care.

Gains in strength during the present training period seemed to be as variable as hypertrophic adaptations and highly individual. We found that the gains in dynamic strength in bilateral leg press after the 10-week intervention ranged as much as from –9.7% to +41.7%. Other investigations have also noted that the changes in muscle force and physiological CSA vary substantially between individuals (3,14,22). Thus, it seems that although larger variability exists, nearly everyone will get stronger when they start to train. However, hypertrophic adaptations do not occur so easily, and almost every study has found some “nonresponders” to hypertrophy (3). However, the resistance training–induced muscle hypertrophy can explain notable proportions of interindividual changes in isometric and isoinertial strength (15). Overall, it seems that in previously untrained subjects, hypertrophy can explain to a rather low extent the strength gains in strength during initial weeks of training. However, in strength trained athletes, muscle size and strength have correlated more strongly, and these correlations have varied between $r = 0.59$ and $r = 0.69$ (4,5).

Bickel et al. (8) have observed as the primary finding that a once-per-week exercise dose was generally sufficient to maintain
positive neuromuscular adaptations (both strength and muscle mass) during the 32-week-long maintenance training (with a lowered training frequency) period. We detected similar results, when strength decreased only by $-1.2\% \pm 3.7$ during the detraining period, whereas VLCSA decreased by $-4.9\% \pm 7.3$. In addition, strength can remain elevated during the 3-week detraining period despite some decrease in muscle CSA (26). Thus, strength may be easier to maintain, at least for 2–3 weeks or so, compared with muscle CSA.

In the present study, we also measured serum testosterone concentrations and testosterone-to-SHBG ratios repeatedly throughout the experimental period. The average serum testosterone-to-SHBG ratio was somewhat higher for HR during the entire experimental period compared with other 2 responder groups (Figure 8). Interestingly, the standard deviation in the testosterone-to-SHBG ratio in HR increased throughout the present hypertrophic strength training intervention. There was also a modest correlation ($r = 0.37; p = 0.079$) between individual levels in the testosterone-to-SHBG ratio and individual muscle hypertrophy in the total group of subjects (Figure 9).

Häkkinen et al. (27) have earlier reported that in elite weightlifters, individual changes in the testosterone-to-SHGB ratio have correlated significantly with changes in weightlifting performance during the very stressful training period of some weeks. Ahtiainen et al. (1) also found a significant correlation between averaged individual testosterone concentrations and individual changes in isometric strength in strength-trained athletic men (1).

In the present study, serum testosterone concentrations increased slightly during the present strength training intervention for the whole group. Ahtiainen et al. (1) have reported similar results, with no significant changes in basal serum concentrations, when untrained and strength-trained men trained for 21 weeks. However, basal testosterone and free testosterone increased during the first 14 weeks (with the increase of training volume) and decreased from week 14 to week 21 in strength-trained men (with the decreased volume) (1). In addition, the volume or intensity of strength training has been shown to affect serum testosterone concentrations (27,29). These findings suggest that...
serum testosterone concentrations can differ with regard to the volume of strength training and can be an important factor for strength development in strength-trained men. Häkkinen et al. (28) found earlier that individual changes in maximal strength and individual changes in anabolic hormonal concentrations correlated significantly during the later stressful training weeks of the prolonged, 6-month, strength training intervention, indicating the possible importance of serum testosterone for trainability.

The present study had some limitations that must be considered when attempting to draw evidence-based conclusions. First, the low sample size of 24 subjects was a limitation when divided into 3 subgroups. 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 3 subgroups, it is possible that the results between the groups could have diverged with a longer intervention protocol. The subjects also trained either unilaterally or bilaterally. Even though the training volume was carefully equated between the groups, the different training styles might have influenced the results. Muscle hypertrophy was measured using ultrasound at the middle length of the QF and not using, for example, MRI. It should also be pointed out that the corticospinal excitability was measured on a passive muscle, which is usually the case in similar experiments. However, it is possible that the passive condition is not able to reveal neural adaptation processes. In the future, it would be interesting to measure corticospinal excitability during high muscle contractions in strength training experiments. Additionally, we gave protein and carbohydrate supplementation to the subjects, and the subjects 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 influence the results of the intervention. Moreover, there were only subjective questionnaires during the detraining process, and no actual activity tracking was done during that period.

In summary, the present study included 10 weeks of progressive hypertrophic resistance training followed by 6 weeks of detraining. After the strength training period, we were able to identify 3 different responder groups for the present hypertrophic strength training program. Our results also indicate that a subgroup of HR in the gains in muscle CSA during the present training were the ones who also tend to lose muscle mass somewhat faster than LR during the detraining phase. In addition, strength gains and maximal muscle activation adaptations may take place faster in HR, and a peak in maximal dynamic strength may be reached, not right after the strenuous strength training period, but after 2–3 weeks of detraining or with lowered volume training. It is important to create individualized strength or hypertrophy programs to maximize the effectiveness of each strength training period. Furthermore, the time for tapering, detraining, and off-season could also be individualized so that the long-term development would be optimal for every individual. Future research is required to determine more accurately the optimal training intensity, volume, exercise selection, and programming for different responder groups.

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**References**


**Practical Applications**

Different responders and actually 3 responder subgroups were observed in our strength training program. Some subjects responded well to our intervention, whereas others did not. In addition, differences in maintaining muscle CSA and maximal strength were found during the detraining. A peak in maximal dynamic strength may be reached, not right after the strenuous strength training period, but after 2–3 weeks of detraining or

**In summary, the present study included 10 weeks of progressive hypertrophic resistance training followed by 6 weeks of detraining. After the strength training period, we were able to identify 3 different responder groups for the present hypertrophic strength training program. Our results also indicate that a subgroup of HR in the gains in muscle CSA during the present training were the ones who also tend to lose muscle mass somewhat faster than LR during the detraining phase. In addition, strength gains and maximal muscle activation adaptations may take place faster in HR, and a peak in maximal dynamic strength may be reached, not right after the strenuous strength training period, but after 2–3 weeks of detraining. The present results highlight the different adaptation capabilities of different individuals. In addition, it expresses the need for personal training programming and tapering for maximum development in hypertrophy and maximal strength in the long term.


