Effects of Age, Gender, and Myostatin Genotype on the Hypertrophic Response to Heavy Resistance Strength Training

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Background. Because of the scarcity of data available from direct comparisons of age and gender groups using the same training stimulus, it is unknown whether older individuals can increase their muscle mass as much as young individuals and whether women can increase as much as men in response to strength training (ST). In addition, little is known about whether the hypertrophic response to ST is affected by myostatin genotype, a candidate gene for muscle hypertrophy.

Methods. Eleven young men (25 ± 3 years, range 21–29 years), 11 young women (26 ± 2 years, range 23–28 years), 12 older men (69 ± 3 years, range 65–75 years), and 11 older women (68 ± 2 years, range 65–73 years) had bilateral quadriceps muscle volume measurements performed using magnetic resonance imaging (MRI) before and after ST and detraining. Training consisted of knee extension exercises of the dominant leg three times per week for 9 weeks. The contralateral limb was left untrained throughout the ST program. Following the unilateral training period, the subjects underwent 31 weeks of detraining during which no regular exercise was performed. Myostatin genotype was determined in a subgroup of 32 subjects, of which five female subjects were carriers of a myostatin gene variant.

Results. A significantly greater absolute increase in muscle volume was observed in men than in women (204 ± 20 vs 101 ± 13 cm³, p < .01), but there was no significant difference in muscle volume response to ST between young and older individuals. The gender effect remained after adjusting for baseline muscle volume. In addition, there was a significantly greater loss of absolute muscle volume after 31 weeks of detraining in men than in women (151 ± 13 vs 88 ± 7 cm³, p < .05), but no significant difference between young and older individuals. Myostatin genotype did not explain the hypertrophic response to ST when all 32 subjects were assessed. However, when only women were analyzed, those with the less common myostatin allele exhibited a 68% larger increase in muscle volume in response to ST (p = .056).

Conclusions. Aging does not affect the muscle mass response to either ST or detraining, whereas gender does, as men increased their muscle volume about twice as much in response to ST as did women and experienced larger losses in response to detraining than women. Young men were the only group that maintained muscle volume adaptation after 31 weeks of detraining. Although myostatin genotype may not explain the observed gender difference in the hypertrophic response to ST, a role for myostatin genotype may be indicated in this regard for women, but future studies are needed with larger subject numbers in each genotype group to confirm this observation.

The changes in body composition that occur with aging (1) can adversely affect activities of daily living (2) and health status in elderly persons (3). Loss of strength and skeletal muscle mass have been identified as among the primary risk factors for falls and impaired mobility in the very elderly population (2,3). Strength training (ST) has been shown to be a safe (4) and effective (4–18) intervention for counteracting these detrimental changes. Fiatarone and colleagues (8), for example, demonstrated that even in very elderly persons, significant gains in muscle size, strength, and functional mobility could be achieved through ST. Other investigators have reported the effects of moderate to heavy resistance ST on the lean, fat, and mineral components in varying population subgroups, including young (10,19,20) and older (4–6,9,10,13,15,17,20–23) women and men. However, because of the scarcity of data available from direct comparisons of age and gender groups, information is lacking on how the capacity of older individuals to alter their muscle mass with ST compares with that of young people, and whether women adapt differently than men at a given age.
Evaluating the relative usefulness of ST as an intervention for elderly persons of both genders has important implications because of the age-associated losses in strength and muscle mass (18) and because of the relationship between these losses and the health and well-being of older individuals (24). In addition, women may be more susceptible to the adverse consequences and disabilities associated with decreases in strength and muscle mass (25) because they typically live a longer time in infirmity than men (26).

Welle and colleagues (7) compared young and older men and women after 3 months of ST using magnetic resonance imaging (MRI) cross-sectional areas (CSA) of the elbow flexors and the knee flexors and extensors, and concluded that aging reduced the hypertrophic response of muscle. However, the low number of subjects per group (n = 4 or 5) and relatively mild training stimulus may have prevented this study from providing definitive conclusions concerning age or gender responsiveness to ST. The effect of ST on neuromuscular parameters in middle-aged and older men and women was recently studied by Hakkinen and colleagues (27), but no between-group comparisons of muscle mass changes were made. To our knowledge, no direct age or gender comparisons have been reported for muscle volume response to the cessation of an ST stimulus (detraining).

Thus, given the need to better understand whether muscle mass adaptations to ST and detraining are age- or gender-dependent, the primary purpose of this study was to compare muscle mass responses of young and older men and women to the same relative ST and detraining protocols. We hypothesized that there would be no age or gender group differences in hypertrophic response to ST.

Mutations in the myostatin gene have been shown to have a significant impact on muscle phenotype in mice (28) and cattle (29,30), and myostatin protein levels have recently been related to muscle mass in humans (31). We have previously noted sequence variations in the human myostatin (growth and differentiation factor 8) gene (32). Although a role for myostatin genotype in affecting muscle response to ST was not indicated in that preliminary, cross-sectional investigation (32), a significant gender difference in the frequency of a common myostatin variant was detected (unpublished observations). Thus, a second aim of our investigation was to assess the possible role of myostatin genotype on muscle mass response to ST. Based on our preliminary work (32), we hypothesized that myostatin genotype might explain any observed gender differences in hypertrophic responses to ST.

**Methods**

**Subjects**

Eleven young men (20–30 years), 11 young women (20–30 years), 12 older men (65–75 years), and 11 older women (65–75 years) volunteered to participate in this 9-week unilateral ST program. Subjects were screened by a physician who performed a medical history, physical examination, and maximal graded exercise test. All subjects were nonsmokers, free of significant cardiovascular, metabolic, or musculoskeletal disorders. Only sedentary persons who had not exercised regularly (more than once every 2 weeks) during the 6 months prior to the study were allowed to participate. Prior to participation, the purpose and procedures of the study were explained in detail, and the subjects gave their written informed consent to participate. The procedures used in this study were approved by the human subjects institutional review boards of the University of Maryland, College Park, the Baltimore Veterans Affairs Medical Center, the Johns Hopkins Bayview Medical Center in Baltimore, and the University of Pittsburgh.

**Muscle Volume Measurement**

The thighs of both legs were scanned via MRI before and at least 48 hours after the last training session and after 31 weeks of detraining. A Picker Edge 1.5 Tesla MRI scanner (Picker International, Cleveland, OH) was used to obtain a series of axial slices, extending from the superior border of the patella to the anterior superior iliac spine and encompassing the entire quadriceps femoris muscle group. The images were produced using 9-mm thick (1-mm gap) T1-weighted axial scans, with an echo time of 14 ms and a relaxation time of 700 ms. Subjects were instructed not to eat or drink anything after midnight on the night before the scans or perform any vigorous activity prior to the scans, which were consistently performed between 8 and 10 AM. The scan files were stored on magnetic disk for subsequent analysis on a personal computer. The MRI scanner calibration was checked daily and adjusted if needed. MRI accuracy and precision of volume determination were assessed by repeat scanning and analysis of a lean beef phantom with dimensions approximating the knee extensor group. Repeat MRI volume measurements on the beef phantom yielded a 0.12% difference between measurements.

The scan files were imported into National Institute of Health (NIH) Image version 1.61 (NIH, Bethesda, MD) for analysis. For each axial slice, the CSA in centimeters squared (cm²) of the quadriceps muscle group was manually outlined as a region of interest. The quadriceps CSA was outlined in every axial image from the superior border of the patella to a point where the quadriceps muscle group is no longer reliably distinguishable from the adductor and hip flexor groups. The same number of slices proximal from the patella was measured for a particular subject, before and after training, to ensure within-subject measurement replication. The sartorius muscle was not included in the CSA because it does not contribute to knee extension. The same investigator, blinded to both subject identification and training condition, performed baseline and after training analysis. Repeat measurement by the same investigator of 300 different cross sections from different areas of the muscle yielded an average coefficient of variation of 0.78%. Intrarreader variability of total quadriceps volume assessed by repeat determination of the same set of axial scans on different days by the same investigator was 3.5%.

**Muscle Volume Calculation**

The CSA of each axial slice was multiplied by the distance between slices (1 cm) and summed across slices. This value represents quadriceps muscle volume, expressed in cubic centimeters (cm³).

**ST Program**

The training program consisted of unilateral training of the knee extensors of the dominant leg, three times per week, for
approximately 9 weeks. Training was performed on a Keiser K-300 air-powered knee extension machine (Keiser Sport/Health Equipment, Fresno, CA) that allows the subject to change the resistance easily, without interrupting the cadence of the exercise. The untrained control leg was kept in a relaxed position throughout the training program. This was accomplished by having the subject rest their leg in front of the pad on the exercise machine and verified by constant investigator observation during every training session for all subjects.

Prior to the regular training sessions, subjects underwent three familiarization sessions during which they completed a typical training sequence with little or no resistance. Subjects performed a 3-minute warm-up on a bicycle ergometer, followed by supervised stretching of the knee extensor and flexor muscle groups. The training consisted of five sets of knee extension exercise designed to include a combination of heavy resistance and high volume exercise. The first set was considered a warm-up and consisted of 5 repetitions at 50% of the 1-repetition maximum (1-RM) strength value. The second set consisted of 5 repetitions at the current 5-RM value. The 5-RM value was increased continually throughout the training program to reflect increases in strength levels. The third set consisted of 10 repetitions, with the first 4 or 5 repetitions at the current 5-RM value, in which the resistance was lowered just enough to complete 1 or 2 more repetitions before reaching muscular fatigue. This process was repeated within the same exercise set and without changing the cadence until a total of 10 repetitions were completed. This same procedure was used in the fourth and fifth sets, but the total number of repetitions was increased. The fourth set consisted of 5 repetitions at the 5-RM resistance, followed by 10 more repetitions for a total of 15 repetitions carried out in the same manner as described previously for the 10-repetition set. The fifth set consisted of 4 or 5 repetitions at the 5-RM resistance, followed by 15 more repetitions for a total of 20 repetitions performed in the same manner as the other sets. This procedure allowed subjects to use near maximal effort on every repetition, while maintaining a relatively high training volume. The second, third, fourth, and fifth sets were preceded by rest periods lasting 30, 90, 150, and 180 s, respectively.

**Detraining**

Following completion of the unilateral ST program, subjects were instructed to resume their normal lifestyle, but to avoid any form of regular exercise for 31 weeks. During this period, subjects were contacted monthly to ensure that they did not participate in any form of regular exercise or make any other lifestyle changes. Strength tests were performed at the 15-week midpoint and again after 31 weeks of detraining. MRI scans were also taken at the end of the detraining period. These results were compared with those obtained before and after the unilateral training program. The compliance rate for all aspects of the study was 85%.

**Laboratory Methods**

Genomic DNA was prepared from ethylenediamine-tetraacetic acid anticoagulated whole blood or from cheek swabs using standard methods (33). Subjects were genotyped for a human myostatin exon 2 variant that is predicted to result in a lysine to arginine amino acid substitution (Lys 153 Arg) in the myostatin protein, as we reported previously (32).

**Statistical Treatment**

Using the difference between the muscle volume change in the trained leg and the volume change in the untrained leg as the dependent variable, a two-factor (age and gender) ANOVA (ANOVA) was performed to determine the presence or absence of age and gender effects. A separate ANOVA was done using the change values from before and after detraining to determine whether age or gender influenced the response to cessation of training. The extent to which baseline levels of muscle mass influenced the outcomes of these comparisons was assessed by using the volume of the trained leg before training as a covariate. Tukey’s analysis was used to identify specific differences between the mean values shown in Table 2. Within-cell simple effects tests were used when appropriate. Following this primary analysis, 32 subjects were grouped by genotype (with and without the arginine variant) for subsequent analysis of the role of myostatin genotype in muscle mass response to ST, with baseline muscle volume used as a covariate. Because all subjects with the arginine allele were women, a subanalysis of myostatin genotype group effects was performed for female subjects. Values are presented as mean ± standard error. A two-tailed p value < .05 was required for statistical significance.

**RESULTS**

**Subject Characteristics**

At baseline, the men in both age groups were significantly taller and heavier with lower percent body fat and greater nonosseous fat-free mass (FFM) than the women (all p < .05). There were no significant differences in age between men and women within the young or older age groups. Body mass, percent body fat, and total body nonosseous FFM did not change significantly as a result of training, except in the young and older men, who displayed a small but significant increase in body mass after training (p < .05; Table 1).

**Muscle Volume**

Muscle volume increased significantly in the trained leg of all four groups (p < .01; Table 2). Although there was not a significant change in muscle volume in the untrained leg in either the young or older women, the small change seen in the untrained limbs of both the young and older men was significant (p < .05).

Using the difference in change values between the trained and untrained limbs as the dependent variable, age and gender comparisons were made on the muscle volume responses to training. The presence of a significant age by gender interaction with respect to muscle volume gain was indicative of a greater disparity in the response between young gender groups than between the older gender groups (p = .05; Figure 1). Tests of simple effects comparing age groups within genders, and gender groups within ages, are
illustrated in Figure 1. There was not a significant difference in the muscle volume response between ages in either gender group. However, there was a significant difference in the muscle volume response to training between genders in the young age group (p < .01), and a difference that approached significance (p = .057) between genders in the older age group (Figure 1). When values were pooled across genders, there was a nearly identical gain in muscle volume between young and older subject groups (Figure 2). However, there was a significant difference of 104 cm$^3$ between the pooled men’s and women’s groups observed in the muscle volume response to ST, with men achieving greater gains than the women in absolute terms (p < .01, Figure 2). The gender effect remained after covarying for baseline muscle volume (p < .01), indicating that the effect was not a function of smaller baseline volumes in women.

Figure 3 shows the effects of age and gender on the muscle volume response to 31 weeks of detraining. Again, the difference in muscle volume loss between pooled age groups (7 cm$^3$) was not significant, whereas the mean difference in muscle volume loss between men and women (63 cm$^3$) was significant (p < .01; Figure 3). Young men were the only group to have retained a significant portion of their ST-induced gain in muscle volume after 31 weeks of detraining (p < .01; Figure 1). There was no significant interaction in the muscle volume response to detraining.

**Myostatin Genotype**

Based on these results, the role of myostatin genotype was assessed in several of these subjects (n = 32; 18 men and 14 women). The less common myostatin allele was observed in only five female subjects. When all subjects (men and women) were grouped according to myostatin genotype, muscle volume response to ST or detraining was not significantly different between genotype groups. When only women were compared, no significant differences in muscle volume characteristics were noted between genotype groups (Table 3). However, a trend was noted for a genotype effect on the muscle volume response to ST, such that women heterozygous for the rare allele exhibited a 68% higher increase in trained leg muscle volume with ST than women without the variant (p = .056; Table 3). This trend was maintained when baseline muscle volume was covaried in the analysis (p = .067). Although limited, these data indicate a possible role for myostatin genotype in muscle volume response to ST.

**DISCUSSION**

To our knowledge, this study is the first to show that age does not attenuate the hypertrophic response to ST, when comparing young and older individuals in the same study using the same relative training stimulus. ST-induced muscle volume changes in the older subjects were not significantly different than in the young subjects, thus supporting our original hypothesis that aging skeletal muscle retains the capacity to fully adapt to an ST stimulus. However, the finding that young men experience significantly greater increases in muscle volume than young women in response to ST did not support our hypothesis and conflicts with findings of previous investigations (10,14,19). Our findings that men lose significantly greater muscle volume during detraining than women and that the detraining response is not significantly different between age groups are also unique. Finally, although myostatin genotype did not explain the significant gender difference noted in muscle volume response to ST, a possible genotype effect on muscle volume response to ST was indicated in the female subjects.

The finding in the present study that age does not attenuate the hypertrophic response to ST conflicts with the con-
Conclusion of Welle and colleagues (7). Using single MRI cross sections, they observed a relatively attenuated hypertrophic response in elderly persons, based upon a diminished response of the elbow flexors and knee flexors in older compared with young subjects after 3 months of ST. However, Welle and colleagues (7) reported that the knee flexor muscle CSA increased only 1% and the knee extensor CSA only 6% after 3 months of ST in the older group. In comparison, older subjects in the current study had mean relative increases of between 11% and 12% in the volume of the knee extensor group after only 9 weeks of ST. This difference suggests that the training stimulus provided by Welle and colleagues (7) was less intense than that in the present study. The training protocol in the present study was designed to optimize both strength and muscle mass gains in all groups, by requiring subjects to exert a near maximal effort on every repetition, while maintaining a high volume protocol (50 repetitions per session) for the quadriceps muscle group. In addition, the differences between Welle and colleagues (7) and the current study may have been enhanced by using volume measurements of the entire trained musculature, which, to date, have not been utilized in the context of evaluating age and gender responsiveness to ST or detraining. The use of volume measurements is supported by unpublished data from our lab, which show that the change in a single mid-thigh slice across a training period explains only 50% of the variance in volume change. However, the measurement of the change in every other slice from the knee to the hip explained over 98% of the variance in muscle volume change in the same subjects.

In contrast to our finding of a gender difference in the muscle mass change with ST, Cureton and colleagues (19) observed no differences between young men and women for absolute or relative increases in elbow flexor CSA (measured by computed tomography [CT]) with ST. However, training volume was again described by the authors to be lower than in the present study. O’Hagan and colleagues (10), also used CT-measured elbow flexor CSA to observe no differences between young men and women for the relative or absolute hypertrophic response to heavy resistance ST. Staron and colleagues (34) found no significant hypertrophy of muscle fibers in either young men or women after 8 weeks of quadriceps ST, but there was no direct measurement of whole muscle hypertrophy. Moreover, McCartney and colleagues (14) observed no gender differences in muscle hypertrophy after the first 10 months of moderate ST in older men and women, but when an additional training period was introduced, there was a trend toward a greater absolute change in CSA for men compared with women. The important methodological distinction between these previous studies and the present investigation is that these studies did not assess the volume of the entire trained musculature as a means of making the gender comparisons. We reported previously from our investigation on the muscle quality changes in the older men
and women in this study that absolute changes in muscle volume were greater in men compared with women, when the volume of the entire trained musculature is assessed (35). Another limitation related to comparing the results of these studies with the present investigation is the focus on different muscle groups, which may vary in their response to ST. Furthermore, as in most training studies, it was impossible, based on a reading of the papers alone, to evaluate the level of supervision present during each of the training sessions, thus leaving unanswered the issue of whether the ST stimulus was consistently applied between groups.

To our knowledge, no direct comparisons of age and gender groups have been reported during the period after ST has been discontinued (detraining), but some information is available on how skeletal muscle responds to detraining. Narici and colleagues (36) studied four male subjects (23–34 years) during 40 days of detraining following a 60-day unilateral ST program. Upper-leg muscle CSA increased by 9% in the trained leg during the 60 days of training. During detraining, muscle CSA was observed to be lost at the same rate at which it was gained, based upon MRI images taken at 20-day intervals. In contrast, Staron and colleagues (20) studied six college-aged women who had participated in a 20-week lower-limb ST program during 30–32 weeks of detraining and found that some adaptations, specifically fiber area and maximal dynamic strength, were retained for up to 32 weeks of detraining.

Our detraining results show the presence of a gender effect, but no age effect, in the quadriceps muscle volume lost in the trained leg during 31 weeks of detraining. This may have been predictable based upon the nature of the findings during the training phase. An unexpected observation, however, was that except for the young males, who maintained some of their adaptation even 31 weeks after the cessation of the training stimulus (Figure 2), all of the age and gender groups showed no significant differences with baseline levels of muscle volume after the 31-week detraining period. Although Staron and colleagues (20) reported that fiber diameter changes were retained 30 weeks after the onset of detraining in young women, our study is the first to show that it is possible for young men to maintain some of the whole muscle volume changes 31 weeks after the ST stimulus has ended. This conclusion seems to conflict with Narici and colleagues (36), who suggested that young men lose all of their adaptation after 60 days of detraining. However, Narici and colleagues (36) studied their subjects for only 40 days of detraining and thus had to speculate based on the rate of muscle mass lost.

The young men in the present study may have been the only group to retain any of their muscle mass increase for 31 weeks of detraining because they were also the highest responders to ST in absolute terms (Figure 2). Therefore, one possible interpretation of these results is that some threshold of adaptation must be achieved in order for muscle mass gains to be preserved for this long. However, the possibility that the young men may have stayed relatively more active during the detraining period than the other groups cannot be ruled out.

Myostatin is a muscle growth inhibitor (28), high levels of which have been associated with muscle wasting in HIV-infected humans (31). Further, inactivating mutations in the myostatin gene in mice (28) and cattle (29,30) result in a hy-
permuscular phenotype. Despite this background, and consistent with our group’s earlier work (32), a common Lys 153 Arg polymorphism in the human myostatin gene did not appear to explain the gender differences noted in muscle volume response to ST in these subjects. However, when the myostatin Lys 153 Arg polymorphism’s role in muscle volume response to ST was explored in women only, a trend was noted, indicating a possible role for the myostatin Arg allele in explaining muscle volume response to ST. This interesting, yet limited, finding suggests the need for further work in this area. The results of the present investigation and other data from our laboratory may also indicate a gender difference in the frequency of the myostatin Lys 153 Arg variation. In the present study, the Arg allele was observed only in female subjects, and we have also noted a significant difference in the frequency of the Arg allele in a larger, yet still unbalanced, sample (9 of 26 women vs 15 of 127 men carried this allele; chi-squared, \( p < .01 \); unpublished observations).

Table 3. Muscle Volume Characteristics Before and After ST and After Detraining for Female Subjects Grouped by the Lys 153 Arg Polymorphism in the Human Myostatin Gene

<table>
<thead>
<tr>
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<th>A/A Genotype ((n = 9))</th>
<th>A/G Genotype ((n = 5))</th>
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<tbody>
<tr>
<td>Trained leg muscle volume before ST ((\text{cm}^3))</td>
<td>1224.7 ± 107.6</td>
<td>1320.4 ± 125.9</td>
</tr>
<tr>
<td>Muscle volume increase with ST ((\text{cm}^3))</td>
<td>109.0 ± 15.4</td>
<td>183.3 ± 39.2*</td>
</tr>
<tr>
<td>Change in trained leg volume minus change in control leg volume ((\text{cm}^3))</td>
<td>98.23 ± 13.0</td>
<td>133.4 ± 41.5</td>
</tr>
<tr>
<td>Detraining-induced loss of muscle volume ((\text{cm}^3))</td>
<td>89.8 ± 16.5</td>
<td>91.9 ± 39.5</td>
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*Notes: Data are means ± SE. ST = strength training.

\(^*p = .056\) versus A/A myostatin gene variant; \( p = .067\) when baseline muscle volume was covaried in the analysis.
In summary, aging does not appear to attenuate the hypertrophic response to ST as was reported previously. However, gender does influence responsiveness to ST, as the men in the present study had muscle mass gains that were approximately twice as great as women when expressed in absolute terms. There is no information available from this or other studies to determine potential mechanisms for these findings, although differences in hormonal responses to training should be addressed in future studies. The observation of a gender effect and the lack of an age effect on the muscle mass response to detraining also represent new findings. The role of the Lys 153 Arg polymorphism in the human myostatin gene on muscle volume response to ST remains unclear and needs further investigation, particularly in a larger sample size of women.

Acknowledgments

The authors thank Dr. Moriel NessAiver for his technical assistance with MRI scans, Elizabeth Lawrence for her technical assistance with myostatin genotyping, Dorothy O’Donnell for her help with the recruitment of subjects, Dan Barlow for his assistance in data analysis, Mary Lott for her overall coordination, genotyping, Dorothy O’Donnell for her help with the recruitment of subjects, Elizabeth Lawrence for her technical assistance with myostatin genotyping, and Mary Lott for her overall coordination. The authors thank Dr. Moriel NessAiver for his technical assistance with MRI scans, Elizabeth Lawrence for her technical assistance with myostatin genotyping, Dorothy O’Donnell for her help with the recruitment of subjects, Dan Barlow for his assistance in data analysis, Mary Lott for her overall coordination, genotyping, Dorothy O’Donnell for her help with the recruitment of subjects, Elizabeth Lawrence for her technical assistance with myostatin genotyping, and Mary Lott for her overall coordination.

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