Exercise Dosing to Retain Resistance Training Adaptations in Young and Older Adults

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ABSTRACT

BICKEL, C. S., J. M. CROSS, and M. M. BAMMAN. Exercise Dosing to Retain Resistance Training Adaptations in Young and Older Adults. *Med. Sci. Sports Exerc.*, Vol. 43, No. 7, pp. 1177–1187, 2011. Resistance training (RT) is a proven sarcopenia countermeasure with a high degree of potency. However, sustainability remains a major issue that could limit the appeal of RT as a therapeutic approach without well-defined dosing requirements to maintain gains. **Purpose**: To test the efficacy of two maintenance prescriptions on muscle mass, myofiber size and type distribution, and strength. We hypothesized the minimum dose required to maintain RT-induced adaptations would be greater in the old (60–75 yr) versus young (20–35 yr). **Methods**: Seventy adults participated in a two-phase exercise trial that consisted of RT 3 $d \cdot wk^{-1}$ for 16 wk (phase 1) followed by a 32-wk period (phase 2) with random assignment to detraining or one of two maintenance prescriptions (reducing the dose to one-third or one-ninth of that during phase 1). **Results**: Phase 1 resulted in expected gains in strength, myofiber size, and muscle mass along with the typical IIx-to-IIa shift in myofiber-type distribution. Both maintenance prescriptions preserved phase 1 muscle hypertrophy in the young but not the old. In fact, the one-third maintenance dose led to additional myofiber hypertrophy in the young. In both age groups, detraining reversed the phase 1 IIx-to-IIa myofiber-type shift, whereas a dose response was evident during maintenance training with the one-third dose better maintaining the shift. Strength gained during phase 1 was largely retained throughout detraining with only a slight reduction at the final time point. **Conclusions**: We conclude that older adults require a higher dose of weekly loading than the young to maintain myofiber hypertrophy attained during a progressive RT program, yet gains in specific strength among older adults were well preserved and remained at or above levels of the untrained young. **Key Words:** MUSCLE FIBER, SARCOPENIA, EXERCISE DOSE, HYPERTROPHY, AGING, ATROPHY

Viable therapeutic options to counteract age-related loss of skeletal muscle mass (i.e., sarcopenia) and the ensuing physical dysfunction and reduced quality of life remain among the most pressing challenges of biomedicine in our aging society. The efficacy of any sarcopenia countermeasure should be measured by both its potency and sustainability. In our view, potency should be assessed by the therapy's ability to counteract sarcopenia at its roots—by inducing muscle regrowth—as well as restoring or, at least substantially improving, muscle function. Of the countermeasures tested to date including pharmacologic therapies and various modes of exercise training, intense resistance exercise training has consistently shown the highest degree of potency in this context—inducing muscle hypertrophy and markedly increasing strength, power, and mobility (5,8,9,14,17). Although we have shown age differences in resistance training (RT)–mediated muscle hypertrophy (favoring the young) (23,24), clearly the myofibers of older adults are malleable to such training and can regrow in as few as 4 months to reach the size of myofibers in untrained adults 40 yr and younger (24). Further, as we and others have shown, voluntary strength, muscle power, and motor unit activation indices (during submaximal weight bearing tasks) are also largely restored to match the untrained young after relatively brief periods of RT (6,11,24,32). Enhancement of muscle function may in part result from enhanced myofiber functional properties (40) as well as the putative IIx-to-IIa shift in myofiber-type distribution—an adaptation we have found to be induced by RT with equal potency in the old and young (24).

Although potency is established, a major limiting factor of RT as a therapeutic approach to sarcopenia is the second key ingredient that defines efficacy—sustainability. This is not a challenge unique to exercise prescription but one that challenges the utility of most all disease therapies. For example, medications that "control" blood pressure or glucose levels to counteract the untoward consequences of chronic hypertension or diabetes mellitus are effective only during prescribed use—particularly in the absence of any behavioral modification—thus such prescriptions typically continue indefinitely. Exercise prescription is considered a very

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effective form of medical treatment (i.e., American College of Sports Medicine's Exercise Is Medicine initiative), but in a similar fashion, sustainability remains a challenge.

The positive morphological and functional adaptations to RT are reversed when training ceases in any population. In young men, 12 wk of detraining after RT has been shown to result in significant losses in strength and myofiber size (10,16). Staron et al. (35) reported that young women had significant reductions in strength with minimal myofiber atrophy after 30 wk of detraining. A more recent report on young men revealed significant type II fiber atrophy after just 10 d of detraining that followed 3 months of RT (19). Studies that have used magnetic resonance imaging for the assessment of whole-muscle cross-sectional area (CSA) after periods of detraining in older adults have also confirmed significant loss of muscle size in 3 months (1,38). Harris et al. (15) studied the effects of previous training intensity on strength retention in a group of older individuals who had trained 2 $d \cdot wk^{-1}$ for 18 wk. They determined that 20 wk of detraining resulted in a 13.5% decrease in strength regardless of previous training intensity. In one of the few direct comparisons in both young and old individuals, 31 wk of detraining after 9 wk of RT resulted in greater strength decrements in older men and women versus young (28) but no age differences in the degree of muscle atrophy as assessed by thigh muscle CSA (30). On the basis of these combined data, it remains unclear whether aging influences the detraining time course after RT. However, rapid reversal of functional improvements has been noted in octogenarians after short-term training and detraining (20).

Perhaps a more significant question that has yet to be addressed is that of a minimum maintenance exercise dose for the prevention of reversibility after RT in older adults. A comprehensive exercise program that continues indefinitely is ideal; however, many individuals will not continue their intensive program consistently for a prolonged period. Therefore, identifying the minimum dose needed to promote sustainability is crucial if RT is to be embraced as a viable and broadly applicable sarcopenia countermeasure. Trappe et al. (39) demonstrated in five older men that prescribing a maintenance dose equivalent to one-third of the weekly volume used during the RT program was sufficient to maintain muscle strength and whole muscle size for 6 months compared with five older men who underwent strict detraining. However, three important issues regarding maintenance dosing efficacy remain: (i) potential age differences, (ii) minimum dosing required, and (iii) fiber-type–specific adaptations.

The purpose of this study was to determine the age-specific efficacy of two different exercise doses toward the maintenance of gains in muscle mass, myofiber size, and voluntary strength, as well as maintenance of the IIx-to-IIa shift in myofiber phenotype, induced by a 16-wk period of an intense, progressive RT in the young and old. We hypothesized that the minimum dose of resistance exercise required to maintain training adaptations would be greater in older adults versus that in young. To test the hypothesis, we randomly assigned individuals to one of three groups after they completed the 16-wk training program. The detraining/maintenance phase 1 included a detraining control that ceased training and two programs that were equal to one-third or one-ninth of the weekly dose used to induce hypertrophy (24).

METHODS

Subjects. Seventy adults from the Birmingham, Alabama metropolitan area were recruited into two age groups: 60–75 yr (n = 31, 64.1 ± 0.6 yr) and 20–35 yr (n = 39, 27.5 ± 0.6 yr). Subjects were free of any musculoskeletal or other disorders that could potentially affect their ability to complete testing and/or RT. Subjects were not obese (body mass index <30 $kg \cdot m^{-2}$) or had any lower extremity RT experience within the past 5 yr. None of the participants were being treated with exogenous testosterone or other pharmacological interventions thought to influence muscle mass. The study was approved by the institutional review boards of both the University of Alabama at Birmingham and the Birmingham Veterans Affairs Medical Center. Before participation, each subject provided written, informed consent.

Phase 1: progressive RT (n = 70). The RT program that was used has previously been described in detail (24). Subjects trained the knee extensors 3 $d \cdot wk^{-1}$ for 16 wk. Subjects performed a 5-min warm-up on a cycle ergometer or treadmill before each training session. RT consisted of three exercises, including knee extension, leg press, and squats under the direct supervision of a clinical exercise physiologist who held current American College of Sports Medicine–Health Fitness Instructor and National Strength and Conditioning Association Certified Strength and Conditioning Specialist certifications. Each exercise was performed for three sets of 8–12 repetitions using resistance exercise stations or plate-loaded stations (barbell squats and linear 45° leg press). A standardized rest period of 90 s was used between sets and each exercise session lasted approximately 35 min. Initial training loads were based on 75%–80% of baseline one-repetition maximum (1RM) strength. As training progressed, resistance was incremented when a subject completed 12 repetitions for at least two of the three total sets at a given resistance while maintaining proper form (25). The typical increase in resistance was 2.3 kg for knee extension, and it was ~5% for leg press and squat. The goal of this progression was to induce volitional fatigue in the 8- to 12-repetition range for each subject throughout the training program. A minimum adherence rate of 83% was required for participants to remain in the study, and adherence averaged approximately 91%.

Phase 2: detraining/maintenance training (n = 56). After the 16-wk RT program, participants were randomized to one of three groups. Thirteen individuals withdrew before completing phase 2 because of a variety of different reasons (e.g., relocated out of the area, lost interest, family illness),

and one was excluded because of a lack of compliance with phase 2 detraining; thus, phase 2 results are based on $n = 56$. A detraining group (n = 16, 7 young and 9 old) did not perform any further RT but returned for biopsies, dual-energy x-ray absorptiometry (DXA) scans, and strength testing. The second group performed a volume that was equal to one-third of the initial 16-wk program (n = 19, 10 young and 9 old). This was accomplished by maintaining intensity (8RM to 12RM), number of exercises (knee extension, leg press, and squats), and number of sets per exercise (three sets) but by reducing the weekly training frequency by one-third (from 3 to 1 $d \cdot wk^{-1}$). The third group performed at a volume that was equal to one-ninth of the initial program (n = 21, 11 young and 10 old). This was accomplished by maintaining intensity and number of exercises but reducing both the number of sets per exercise (three sets to one set) and weekly training frequency (from 3 to 1 $d \cdot wk^{-1}$). Figure 1 illustrates the group assignment, phases of training, and time course of measures.

Muscle biopsy and tissue preparation. Muscle biopsies were performed in the Pittman General Clinical Research Center at the University of Alabama at Birmingham. Muscle samples were collected from the vastus lateralis muscle by percutaneous needle biopsy using a 5-mm Bergstrom biopsy needle under suction as previously described (7). For the current study, four samples were obtained per subject: baseline, after training (16 wk), and after follow-up periods of 16 and 32 wk during phase 2. At the bedside, visible connective and adipose tissues were removed from the sample with the aid of a dissecting microscope. A portion of the sample to be used for immunohistochemistry was mounted cross-sectionally on cork in optimum cutting temperature mounting medium mixed with tragacanth gum, frozen in liquid nitrogen–cooled isopentane and stored at −80°C.

Immunofluorescence microscopy. Methods used for myofiber typing based on MHC isoform immunoreactivity have previously been described by our group in detail (22,24). We previously confirmed primary antibody (Ab) specificity by immunoblot (24). Briefly, 6-μm sections were fixed in 3% neutral-buffered formalin followed by a sequential series of blocking and primary and secondary Ab incubations: (i) anti-MHC I mouse monoclonal Ab (NovoCastra Laboratories, Newcastle, UK; 1:100) and Alexa 594–conjugated goat antimouse secondary Ab (Pierce Biotechnologies, Rockford, IL; 1:200); (ii) to locate sarcolemmae, anti–laminin mouse MAb (NovoCastra Laboratories, 1:80) and Alexa 488–conjugated goat antimouse secondary Ab (Pierce Biotechnologies, 1:200); and (iii) anti-MHC IIa mouse MAb (University of Iowa Hybridoma Bank, Iowa City, IA; 1:80) and Alexa 488–conjugated goat antimouse secondary Ab (Pierce Biotechnologies, 1:200). Nuclei were revealed by a Hoecsht 33258 DNA counterstain.

High-resolution (48-bit TIFF) fluorescent images were captured at 10×, and image analysis was performed using Image-Pro Plus 5.0 software by a single analyst blinded to age, sex, and time point. Myofiber-type distribution was determined from 980 ± 41 myofibers per sample at baseline, 867 ± 45 after training, 1459 ± 88 after 16 wk of detraining/maintenance, and 1450 ± 103 after 32 wk of detraining/maintenance. Myofibers positive for MHC I and negative for MHC IIa were classified as type I, fibers positive for MHC IIa and negative for MHC I were classified as type IIa, and fibers negative for both MHC I and MHC IIa were classified as type IIx. Myofibers coexpressing more than one MHC isoform were excluded from analyses. Myofiber CSA measurements were performed as detailed elsewhere (21,24).

Voluntary strength. Our 1RM strength assessment methods have been detailed elsewhere (24,33). Subjects attended two familiarization sessions within 5 d before 1RM testing for the three training exercises—squat, leg press, and knee extension. 1RM was defined as the highest load lifted through a full range of motion before two failed attempts at a given load. To accurately compare test performed on weight-stack and free-weight stations, all tests performed on

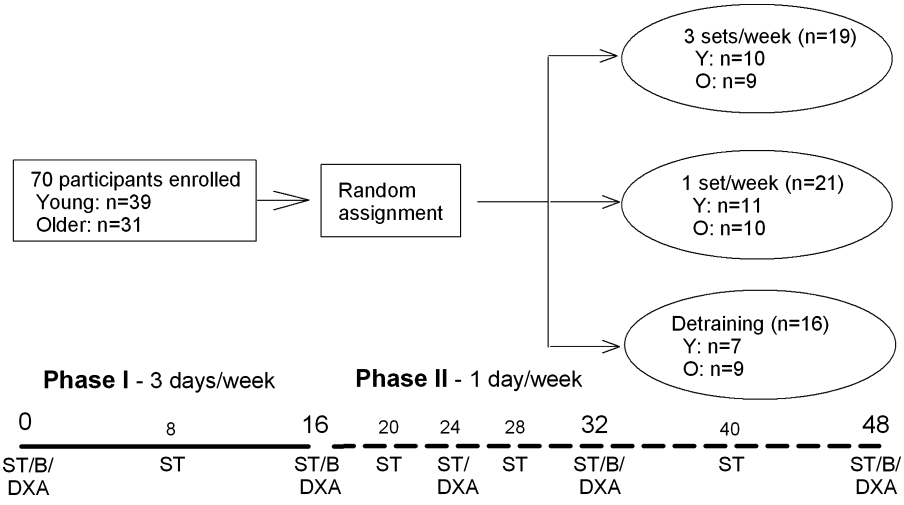

FIGURE 1—Study timeline: Phase 1 = 16 wk of RT. Phase 2 = detraining or maintenance prescription. B, muscle biopsy; ST, strength testing.

resistance exercise stations were converted to actual loads lifted using regression-curve fitting procedures as we have described (33). 1RM testing was repeated at weeks 8, 16, 20, 24, 28, 32, 40, and 48. During phase 2 (weeks 20–48), 1RM was determined for leg press and knee extension only.

Thigh lean mass. Lean mass of each thigh (fat and bone mass excluded) was determined by DXA using a Lunar Prodigy (model 8743; GE Lunar Corporation, Madison, WI) and enCORE 2002 software (version 6.10.029; GE Lunar Corporation, Madison, WI). Quality control was conducted before each session according to manufacturer's instructions; the coefficient of variation for lean tissue in our hands is <2%. Right and left thigh lean masses (TLM) were summed and used to estimate bilateral specific strength of the knee extensors as we have done previously (33).

Statistical analysis. A total of 70 subjects completed phase 1. Fifty-seven subjects went on to complete the randomly assigned, 32-wk phase 2 portion of the study. One of these subjects was excluded because of a lack of compliance with phase 2 detraining, and muscle biopsies were not performed on one subject after the baseline sample because of anticoagulant prescription; thus, $n = 56$ for all phase 2 results except biopsy data ($n = 55$). The combined phase 1 and phase 2 analyses involved 248 scheduled muscle biopsies ((55 subjects × 4 samples) + (14 subjects × first two samples only)). We used mean substitution via established methods as necessary. For example, in 10 (4%) of 248 cases, either the tissue was not collected ($n = 6$) or the tissue yield was not sufficient for histological analysis ($n = 4$). Phase 1 results were analyzed using two factor (time × age group) repeated-measures ANOVA to assess the effects of age and RT on muscle mass, fiber-type–specific CSA, fiber-type distribution, 1RM strength, and specific strength. Phase 2 results were analyzed using a three factor (time × age group × phase 2 group) repeated-measures ANOVA to assess the effects of age and phase 2 group on muscle mass, fiber-type–specific CSA, fiber-type distribution, 1RM strength, and specific strength. Significant main time effects and interaction effects were evaluated *post hoc* using Fisher least squares difference tests. Additional analyses were conducted via ANCOVA (covaried for baseline data) to determine whether pretraining age differences affected phase 1 adaptations (strength, muscle mass, myofiber size). Similar ANCOVA were tested across phase 2 groups for changes in strength, muscle mass, and myofiber size with week 16 data serving as covariate. All statistical analyses were performed using STATISTICA 8.0 (StatSoft, Tulsa, OK). Statistical significance was accepted at $P < 0.05$ for all tests.

RESULTS

Phase 1: Progressive RT (16 wk)

At the conclusion of phase 1, our average adherence was 91%, and there were no study-related injuries reported by the participants. Overall, the 3-d·wk^{-1} phase 1 RT prescription induced the expected age-specific training adaptations, as shown in Table 1. Main effects of age ($P < 0.05$) and time ($P < 0.05$) were found for TLM. The young gained 5.6% during the 16-wk training program, whereas the old gained 4.2%. Type II myofibers were larger among the young (vs old; main age effect, $P < 0.005$), whereas type I CSA did not differ by age. There was a significant group × time interaction for type II myofiber CSA ($P < 0.05$). On average, type II myofiber CSA increased 37% ($P < 0.01$) and 28% ($P < 0.01$) in young and older participants, respectively. There was no significant group × time interaction for type I myofiber CSA, but a significant main time effect for type I fiber CSA was found, and on average, type I myofiber CSA increased 22% and 14% in young and older subjects. Myofiber-type distribution was nearly identical in the two age groups before training, and training adaptations were not different in the old and young (no age group × time interactions). There was no significant alteration in type I fiber distribution for either age group. Type IIa distribution increased in both age groups ($P < 0.01$) with a concomitant decrease in type IIx distribution ($P < 0.01$). Knee extension 1RM strength improved in both age groups during phase 1 ($P < 0.001$). The majority of strength gains were achieved during the first 8 wk. For example, by week 8 1RM improved 27% in both the young and old, with additional increases of 11% and 9%, respectively, from week 8 to 16. There was an expected overall age difference in 1RM strength ($P < 0.001$) because the young were 45% stronger before training. Despite similar relative strength gains in the two age groups, an age × time interaction ($P < 0.001$) was noted because the old achieved strength levels by

TABLE 1. Phase 1 results for myofiber CSA and type distribution, TLM, and knee extension strength (KE 1RM).

	Young ($n = 39$)	Old ($n = 31$)
Myofiber CSA (μm^2)		
Type I		
Baseline	4090 ± 161	4587 ± 241
16 wk	4991 ± 178[a]	5237 ± 241[a]
Type II[b,c]		
Baseline	4333 ± 193	3608 ± 251
16 wk	5917 ± 264[a]	4636 ± 298[a]
Myofiber type distribution (%)		
Type I		
Baseline	36.0 ± 2.2	35.4 ± 1.8
16 wk	34.7 ± 2.1	35.9 ± 2.0
Type IIa		
Baseline	47.5 ± 1.8	50.3 ± 1.4
16 wk	63.1 ± 2.0[a]	62.4 ± 1.9[a]
Type IIx		
Baseline	16.5 ± 1.4	14.2 ± 1.8
16 wk	2.2 ± 0.6[a]	1.7 ± 0.6[a]
TLM[b] (g)		
Baseline	12,429 ± 486	10,717 ± 532
16 wk	13,128 ± 508[a]	11,164 ± 573[a]
Knee extension 1RM[b,c] (kg)		
Baseline	54.2 ± 2.54	36.9 ± 2.1
8 wk	68.8 ± 3.2[a]	47.0 ± 2.7[a]
16 wk	76.2 ± 2.9[a,d]	51.5 ± 2.9[a,d]

Values are means ± SE.
[a] Within-group training effect from baseline ($P < 0.05$).
[b] Main age effect ($P < 0.05$).
[c] Age group × time interaction ($P < 0.05$).
[d] Change from 8 wk ($P < 0.05$).

8 and 16 wk that were no longer significantly different from the young at baseline. Via ANCOVA (covaried across age), adjusted means before training for TLM, type II CSA, and 1RM strength were 11,671 g, 4018 μm^2, and 46.6 kg, respectively. With these serving as covariate, no age differences were noted ($P > 0.05$) after training-induced gains in TLM (week 16 LS-adjusted means ± SE: young = 12,341 ± 102 g; old = 12,154 ± 115 g) or 1RM strength (week 16 LS-adjusted means ± SE: young = 66.2 ± 1.7 kg; old = 64.1 ± 1.9 kg). On the other hand, age groups differed ($P < 0.05$) in type II myofiber CSA after training (week 16 LS-adjusted means ± SE: young = 5621 ± 183 μm^2; old = 5022 ± 210 μm^2), consistent with the age × time interaction via ANOVA.

Phase 2 Detraining or Maintenance Training (32 wk)

TLM. We found a significant time × phase 2 group interaction ($P < 0.01$) for TLM but no significant three-way interaction with age in the model. TLM results (Table 2) focus on phase 2 group assignments irrespective of age; however, data by age group are also displayed for discussion purposes. Subjects assigned to the detraining group gained 4.8% ($P < 0.001$) TLM during phase 1 and, within the first 8 wk of phase 2, lost most all of the gain ($P < 0.01$). Subjects assigned to the one-ninth maintenance prescription gained 6.2% ($P < 0.001$) TLM during phase 1, and at each subsequent phase 2 time point, TLM remained elevated above baseline ($P < 0.001$); however, TLM started to decline after 16 wk of phase 2 ($P < 0.05$). Participants randomized to the higher one-third maintenance dose increased TLM 5.1% during phase 1 ($P < 0.001$) and maintained TLM above baseline throughout phase 2 ($P < 0.05$). When using week 16 TLM data as the covariate, significant ($P < 0.05$) differences persisted at week 48 among phase 2 group assignments within each age group.

Voluntary strength. We found a significant time × phase 2 group interaction ($P < 0.001$) for both knee extensor strength and leg press, indicating that voluntary strength changed differentially among the three phase 2 treatments. There was no age × time × phase 2 group interaction signifying that age had no effect during phase 2 on strength performance. Phase 2 group results were similar for knee extension and leg press strength data; thus, for clarity of data presentation, *post hoc* analyses are presented for knee extension strength and all age groups are shown for discussion purposes (Table 2). *Post hoc* analyses of knee extension strength revealed a small strength loss of 7% after 32 wk of detraining ($P < 0.05$), although strength remained 23% above baseline ($P < 0.001$). On the other hand, both maintenance prescriptions preserved 1RM strength and even improved strength above the phase 1 gains by 7%–8% after phase 2 ($P < 0.01$). When using week 16 knee extensor 1RM data as the covariate, significant ($P < 0.05$) differences persisted at week 48 among phase 2 group assignments within each age group.

Myofiber size. Myofiber size results are presented in Figure 2. We found a significant time × phase 2 group interaction ($P < 0.005$), indicating differential changes in mean fiber area (MFA) among the three randomly assigned treatments during phase 2. The induction of hypertrophy during phase 1 ($P < 0.01$) reversed in the detraining group because MFA was no longer different from baseline at both phase 2 sampling time points. Throughout phase 2, both maintenance prescriptions preserved the larger myofibers attained in phase 1. To further explore the differential effects of phase 2 treatment on myofiber size, we analyzed the sizes of the two primary fiber types (I and II). Type II CSA results generally followed the patterns for MFA. For example, a significant time × phase 2 group interaction ($P < 0.005$) was also noted with the type II population. In

TABLE 2. Phase 2 results for TLM and KE 1RM for detraining (DT), one-ninth, and one-third maintenance prescriptions.

	Baseline	8 wk	16 wk	20 wk	24 wk	28 wk	32 wk	40 wk	48 wk
TLM[a] (g)									
DT	12,145 ± 690		12,725 ± 728[b]		12,268 ± 678[c]		12,164 ± 664[c]		11,880 ± 636[c]
Young	13,049 ± 984		13,719 ± 1055[b]		13,216 ± 1021[c]		12,998 ± 994[c]		12,926 ± 920[c]
Old	11,443 ± 922		11,952 ± 973[b]		11,531 ± 876[c]		11,515 ± 879[c]		11,067 ± 817[c]
One-ninth	11,452 ± 679		12,160 ± 739[b]		11,972 ± 735[b]		11,884 ± 713[b,c]		11,919 ± 730[b,c]
Young	11,592 ± 932		12,531 ± 1005[b]		12,418 ± 1035[b]		12,368 ± 1032[b]		12,345 ± 1000[b]
Old	11,299 ± 1041		11,752 ± 1130[b]		11,481 ± 1078		11,351 ± 1004[c]		11,451 ± 1104
One-third	11,646 ± 744		12,245 ± 845[b]		12,032 ± 833[b]		11,948 ± 805[b,c]		12,089 ± 831[b]
Young	12,882 ± 984		13,611 ± 1167[b]		13,355 ± 1160[b]		13,274 ± 1064[b]		13,580 ± 1101[b]
Old	10,272 ± 987		10,726 ± 1069[b]		10,561 ± 1050		10,474 ± 1072		10,432 ± 1056
KE 1RM[a] (kg)									
DT	47.7 ± 3.8	57.8 ± 4.5[b]	63.1 ± 4.7[b]	62.1 ± 4.0[b]	61.7 ± 4.5[b]	61.3 ± 4.5[b]	63.4 ± 4.6[b]	62.8 ± 5.2[b]	58.8 ± 4.7[b,c]
Young	56.6 ± 6.3	66.2 ± 7.8[b]	72.9 ± 7.6[b]	70.3 ± 5.0[b]	71.7 ± 6.5[b]	72.9 ± 6.1[b]	74.4 ± 7.1[b]	76.4 ± 7.8[b]	70.0 ± 7.2[b]
Old	40.8 ± 3.2	51.3 ± 4.5[b]	55.6 ± 4.8*	55.8 ± 5.3[b]	53.9 ± 5.0[b]	52.3 ± 4.9[b]	54.9 ± 4.7[b]	52.3 ± 4.7[b]	50.0 ± 4.6[b,c]
One-ninth	46.9 ± 3.6	60.1 ± 4.4[b]	66.2 ± 5.8[b]	68.2 ± 6.0[b]	69.6 ± 6.0[b,c]	68.0 ± 5.5[b]	69.0 ± 5.9[b]	69.1 ± 5.7[b]	70.7 ± 6.0[b,c]
Young	51.9 ± 4.8	66.6 ± 6.5[b]	74.8 ± 8.4[b]	77.7 ± 8.8[b]	79.3 ± 8.5[b,c]	77.3 ± 7.7[b]	79.1 ± 8.0[b]	78.2 ± 8.4[b]	80.4 ± 8.5[b,c]
Old	40.8 ± 5.1	52.1 ± 5.0[b]	55.6 ± 6.4[b]	56.6 ± 6.3[b]	57.9 ± 7.0[b]	56.8 ± 6.8[b]	56.6 ± 7.2[b]	58.0 ± 6.0[b]	58.8 ± 6.9[b]
One-third	44.4 ± 3.9	59.6 ± 5.6[b]	65.0 ± 5.9[b]	65.9 ± 5.9[b]	65.0 ± 6.0[b]	65.7 ± 6.2[b]	67.2 ± 6.3[b]	67.8 ± 6.4[b]	70.1 ± 6.4[b,c]
Young	54.8 ± 4.7	74.3 ± 6.7[b]	79.0 ± 7.6[b]	80.6 ± 7.9[b]	80.2 ± 7.8[b]	81.5 ± 7.9[b]	83.8 ± 8.0[b,c]	83.7 ± 8.4[b,c]	86.1 ± 8.4[b,c]
Old	32.8 ± 3.6	43.3 ± 5.4[b]	49.4 ± 5.8[b]	49.7 ± 5.1[b]	48.2 ± 5.1[b]	48.2 ± 5.3[b]	48.8 ± 5.2[b]	50.0 ± 5.3[b]	52.3 ± 5.7[b]

Values are means ± SE.
[a] Time × phase 2 group interaction ($P < 0.05$).
[b] Different from baseline within group ($P < 0.05$).
[c] Different from week 16 within group ($P < 0.05$).

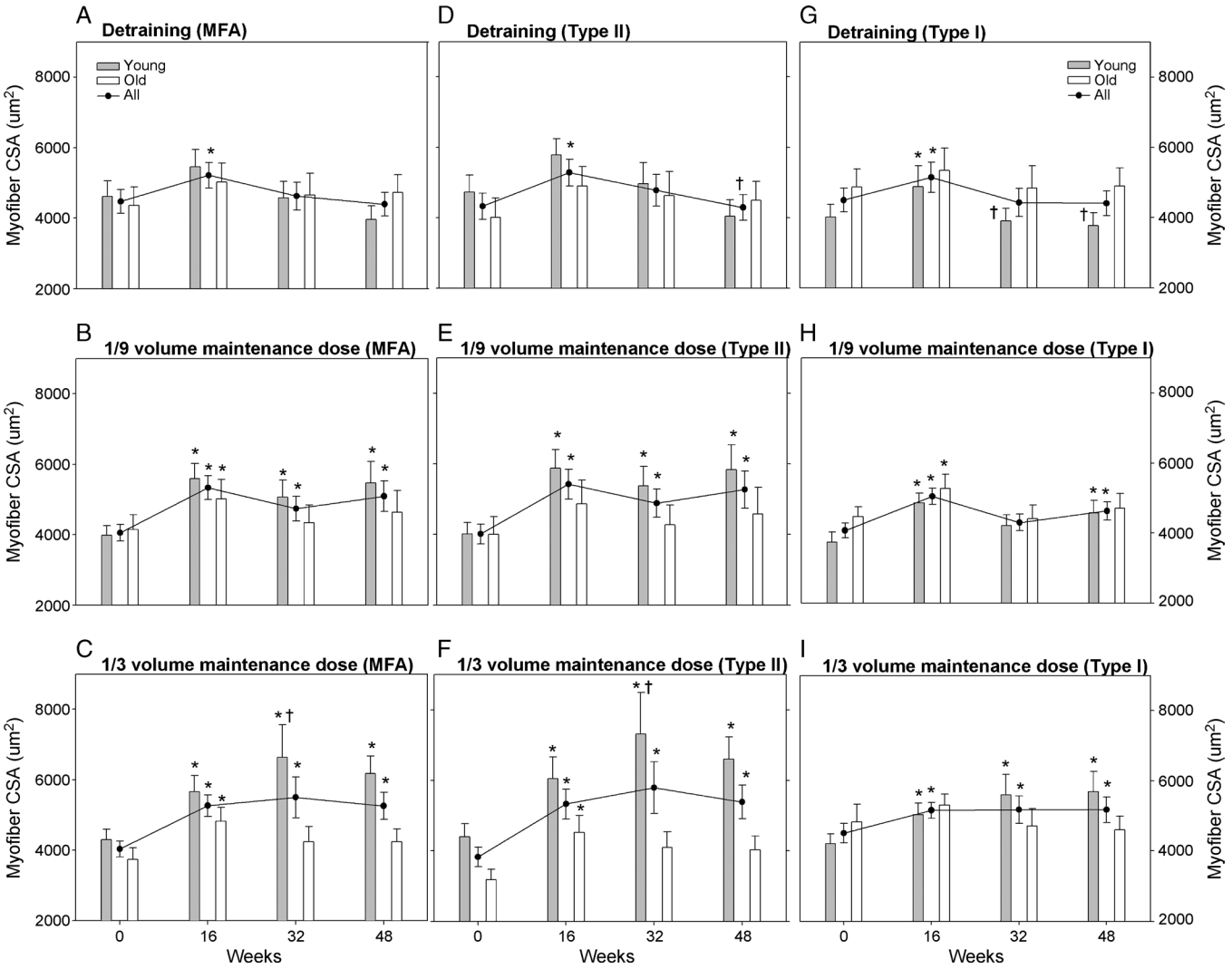

FIGURE 2—Myofiber CSA by phase 2 group. A–C. MFA. D–F. Type II fiber CSA. G–I. Type I fiber CSA. *Different from baseline ($P < 0.05$). †Different from week 16 ($P < 0.05$).

the detraining group, the induction of type II hypertrophy during phase 1 ($P < 0.01$) reversed after only 16 wk of detraining, whereas both maintenance prescriptions proved effective at maintaining the degree of type II hypertrophy found in phase 1. For type I myofibers, we found only a main effect of time ($P < 0.001$); yet much like the findings for type II CSA, detraining resulted in reversal of type I myofiber hypertrophy. Type I CSA was essentially not different from pretraining levels at the two phase 2 sampling time points within the detraining group, whereas both maintenance prescriptions generally preserved the enlarged type I CSA throughout phase 2.

The effect of age on detraining and maintenance training outcomes was tested via three-way interaction (time × age group × phase 2 group). There was a significant three-way interaction for MFA ($P < 0.05$), indicating that the two age groups responded differently to the randomly assigned treatments. This three-way interaction was apparently driven by age differences in the efficacy of the two tested maintenance prescriptions. Irrespective of the tested maintenance dose, reversal or loss of MFA was prevented among the young only—MFA remained above baseline ($P < 0.05$) throughout phase 2 for both maintenance doses (Figs. 2A and B). Unexpectedly, we found a dose response for continued myofiber hypertrophy among the young during phase 2, whereby the one-third maintenance program actually induced further hypertrophy during the first 16 wk of phase 2 ($P < 0.05$). In contrast, among older subjects, the training-induced myofiber hypertrophy was lost by the first sampling time point during phase 2 for both tested maintenance prescriptions—MFA among the old was not different from baseline after 16 or 32 wk of attempted maintenance training. These age-specific changes in MFA during phase 2 seemed to be largely driven by changes in the sizes of type II myofibers (statistical results identical with MFA; Figs. 2A–F). Alterations in type I myofiber size were less robust throughout both phases 1 and 2, but similar to type II CSA, preservation of gains during attempted maintenance training was evident

only among the young (Figs. 2G–I). As mentioned previously, the myofiber CSA of young and old participants responded differently to the RT prescription in phase 1, and there was a significant effect of age on type II myofiber CSA as a result of phase 2. When using type II myofiber CSA at week 16 as the covariate within each age group, significant ($P < 0.05$) differences between phase 2 treatment groups at week 48 were found for the young only.

Myofiber-type distribution. Muscle fiber composition changed similarly in the young and older groups during this 48-wk study (Fig. 3). There were significant time × phase 2 group interactions for type IIa and IIx myofiber distribution ($P < 0.01$), whereas no changes in type I myofiber distribution were noted. The typical RT-induced IIx-to-IIa myofiber-type shift found after phase 1 was fully reversed during detraining (Fig. 3A). The distribution of type IIx myofibers

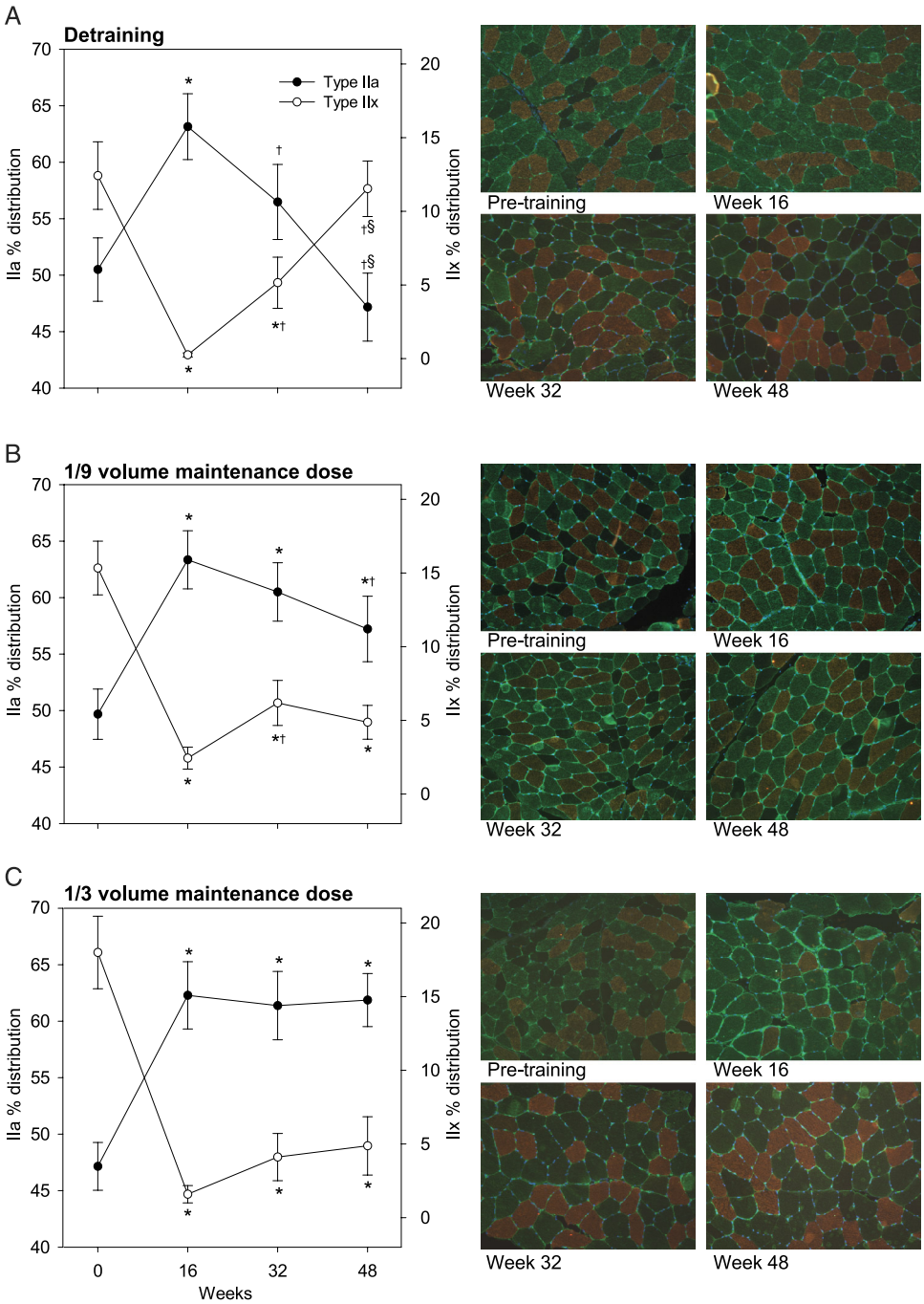

FIGURE 3—Type IIa and IIx myofiber percent distribution by phase 2 group. Representative immunohistological images from each time point are presented on the right (*copper*, type I; *green*, type IIa; *negative or dark*, type IIx). *Different from baseline ($P < 0.05$). †Different from week 16 ($P < 0.05$). §Different from week 32 ($P < 0.05$).

was higher than after training at both phase 2 time points ($P < 0.05$) and had fully returned to baseline by the end of phase 2. Among these detraining subjects, the data for type IIa distribution were essentially the mirror opposite of type IIx across both phases of the project. The one-third volume maintenance prescription generally preserved the IIx-to-IIa shift (Fig. 3C), whereas the lower maintenance dose of one-ninth the volume resulted in a partial shift reversal as statistically detected for type IIx myofibers at the midpoint of phase 2 ($P < 0.05$) and for type IIa myofibers by the end of phase 2 ($P < 0.05$) (Fig. 3B).

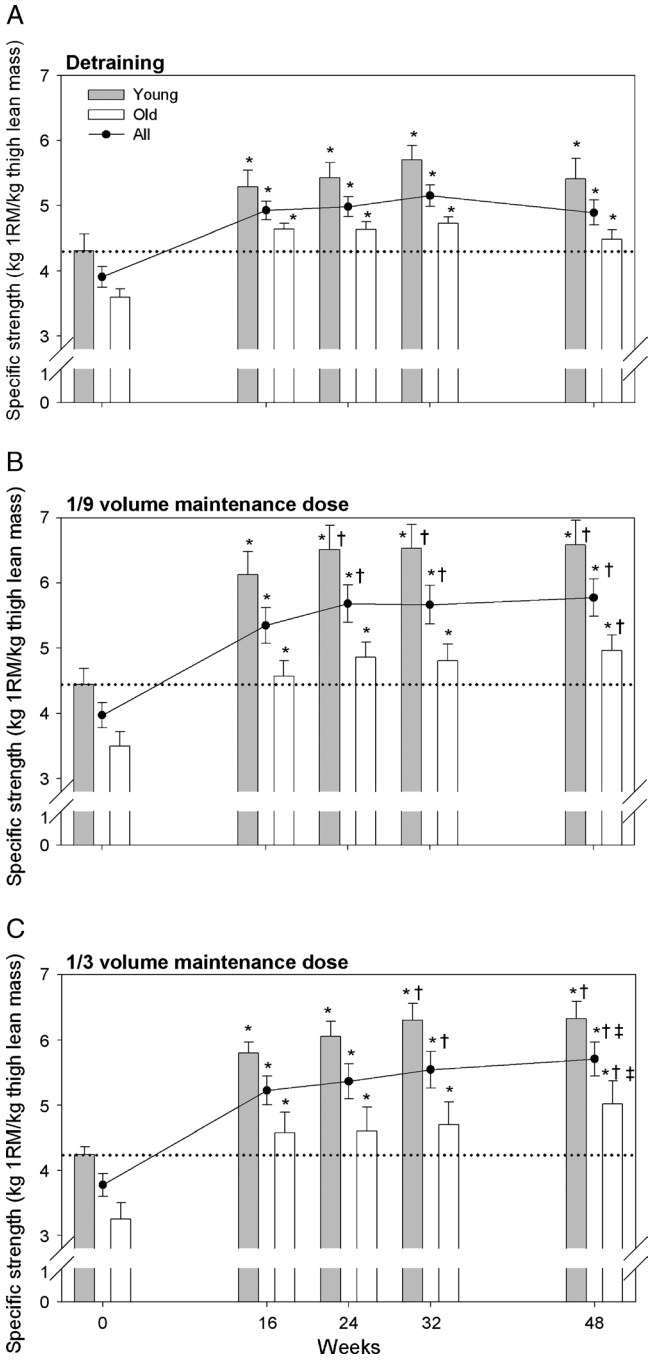

FIGURE 4—Specific strength estimates by phase 2 group determined by ratio of 1RM knee extension (kg) to TLM (kg). *Different from baseline ($P < 0.05$). †Different from week 16 ($P < 0.05$). ‡Different from week 24. *Dotted line* indicates pretraining specific strength of the untrained young.

Specific strength. To estimate specific strength, we analyzed the 1RM knee extension (kg)–to–TLM (kg) ratio and found main effects of age ($P < 0.001$), time ($P < 0.001$), and a significant time × phase 2 group interaction ($P < 0.001$). As shown in Figure 4, specific strength remained elevated above baseline ($P < 0.001$) throughout phase 2, regardless of the phase 2 group assignment. Furthermore, both maintenance prescriptions resulted in additional increments ($P < 0.05$) in specific strength during phase 2 with a noted dose response.

DISCUSSION

To our knowledge, this is the first study to evaluate the dose response efficacy of different prescriptions aimed to maintain progressive RT adaptations, along with potential age differences in maintenance requirements. The primary finding of this study was that a once-per-week exercise dose was generally sufficient to maintain positive neuromuscular adaptations; yet age-specific differences were observed in the required minimum dose for maintenance of muscle size. A clear dose response was evident among the young because one-third volume maintenance dosing led to continued myofiber hypertrophy and strength gains, whereas one-ninth volume effectively maintained improvements in contrast to strict detraining. Among the old, neither prescription was sufficient to maintain the gains in muscle size, but strength improvements were largely retained by both prescriptions. A dose response was also noted (irrespective of age) in the loading dose required to prevent reversal of the IIx-to-IIa myofiber-type shift because only the one-third dose prevented reversal. Despite apparent age differences in maintenance dose requirements for some variables of interest, it is important to emphasize that older adults achieved and generally maintained voluntary maximum strength levels similar to the untrained young.

Our phase 1 progressive RT program had the desired effect on the measurements of interest: increased muscle mass, increased myofiber size with preferential type II hypertrophy IIx-to-IIa myofiber-type shift, and gains in voluntary strength. As expected, the magnitudes of improvement in myofiber size and strength among the young exceeded gains in the old. These findings are consistent with previous studies from our laboratory (3,24) and others (9,13,39,41). Age differences persisted even when using baseline type II myofiber CSA as covariate, further supporting a significant effect of age on the myofiber response to RT. Our primary interest in this study was not to determine whether we could evoke typical changes in skeletal muscle after RT but to begin to assess the optimal training volumes necessary to retain these benefits, particularly among older adults, during periods of reduced exercise frequency. Clearly, it is in the best interest of all adults to adopt progressive RT as a

major component of a regular weekly exercise regimen. The intent of the current study was not to identify a maintenance dose to be implemented indefinitely; rather, during defined periods (e.g., a few weeks) when exercise frequency is difficult to sustain (e.g., extended travel, family illness). Therefore, we felt it important to provide insight as to the dosing necessary to maintain positive adaptations and to assess whether differences exist between young and older individuals.

It is widely recognized that exercise training adaptations are reversed with cessation of training. Depending on the mode of training and the outcome variable(s) of interest, reversibility may be rapid (i.e., days) or delayed (i.e., months). Our detraining strength data are consistent with others who have investigated the effects of detraining on muscle strength and size in older adults. Most studies suggest that declines in strength associated with detraining occur slower than the gains received from RT. For example, Taaffe et al. (36) reported a 68% increase in knee extension strength after 24 wk of training but only a 22% decrease after 24 wk of detraining. Knee extensor strength remained 32% greater than baseline even after a 24-wk detraining phase. A similar study was conducted by Ivey et al. (18), reporting strength gains in older adults of ~30% after a 9-wk RT program, whereas 31 wk of detraining resulted in only a 13% decrease in strength. Again, strength remained significantly higher than baseline measures after the detraining phase. Our older group had a 36% increase in strength after 16 wk of RT, which significantly decreased after 32 wk of detraining, but was still 23% greater than baseline (Table 2). Thus, most studies suggest that strength can be significantly increased with traditional RT programs and that these strength gains are realized for several months even after training ceases. The maintenance prescriptions that were used 1 $d \cdot wk^{-1}$ in this study were sufficient to maintain strength in both age groups, and in fact, the young continued to increase strength above phase 1 levels.

Strength gains during training are due to both skeletal muscle hypertrophy and non–muscle mass adaptations (26). Most of the studies on detraining in older adults have focused on strength gains/losses, whereas data on muscle mass changes are more limited. Ivey et al. (18) reported on muscle volume adaptations owing to 31 wk of detraining in young and older individuals. All of their groups (young and older males and females) increased muscle volume after RT; however, 31 wk of detraining returned all groups, except young men, to baseline levels. Others have reported similar declines in whole muscle size measured by magnetic resonance imaging in younger participants (1). Taaffe and Marcus (37) investigated the specific muscle fiber adaptations in a small group of older male subjects ($n = 7$) after a 24-wk RT program followed by 12 wk of detraining. They reported significant hypertrophy of both type I and II muscle fibers (17% and 26%, respectively), which returned to baseline after the 12-wk detraining phase. To our knowledge, we are the first to report congruent detraining losses of both myofiber size and whole limb muscle mass in the young and old.

The combined results shown in Table 2 and Figure 4 clearly demonstrate that maintenance of strength during detraining is, for the most part, not dependent on maintenance of muscle mass. Both the young and old retained substantial improvements in specific strength throughout detraining. Non–muscle mass adaptations (e.g., improved motor unit activation, better coordination of agonists, and/or inhibition of antagonists [12]) that affect strength performance obviously persist much longer than what seems to be relatively transient muscle hypertrophy. Previous reports have shown that both middle-age and older adults demonstrate large increases in strength (35%–65%) despite being accompanied by only modest hypertrophy (2%–10%) (12), suggesting a disconnect between muscle growth and strength gains. Therefore, we should not be altogether surprised when a similar pattern appears in reverse during detraining. A recent study by Kubo et al. (27) demonstrates that improvements in strength, motor unit activation level, and EMG levels during max contractions last for at least 3 months of detraining in young adults. We are not aware of any studies that have systematically investigated the time course of the loss of non–muscle mass adaptations for longer periods, but as previously mentioned, Ivey et al. (18) report strength gains persisting after 31 wk of detraining in both young and older adults consistent with our findings. We do need to recognize that a possible contribution to the persistent increase in specific strength in our study may have been related to the strength testing that occurred every 4–8 wk during phase 2. From a clinical perspective, it is important to point out that the older adults assigned to detraining maintained a specific strength above pretraining young subjects throughout the 8 months of detraining (see dotted line, Fig. 4). Just 16 wk of heavy RT improves the neuromuscular function of older adults to such a degree that they continue to perform similar to younger untrained subjects even after a period of detraining. This has obvious implications for ambulatory function and may also favorably affect risk of falls.

The primary objective of this trial was to assess the dose response efficacy of two different maintenance prescriptions. Trappe et al. (39) provide some of the only data on the effects of both detraining and exercise maintenance on muscle strength and size in older men. They reported that 24 wk of detraining resulted in an 11% decrease in 1RM with a 5% decrease in whole muscle size as measured by computed tomography. However, training at one-third of the weekly volume for 24 wk was sufficient to maintain both 1RM strength and whole-thigh CSA. Our data further expanded on the work of Trappe et al. (39) by including a young comparison group, a one-ninth weekly volume group, as well as specific myofiber data. Our data are the first to suggest that older adults require more sets per week than younger individuals to maintain hypertrophy. Interestingly, within the young cohort, there was a clear dose response effect to the

phase 2 programs; one-third volume resulted in further hypertrophy, one-ninth volume maintained size, and detraining resulted in myofiber atrophy. The dose response was not evident in the older group because neither phase 2 prescription maintained the myofiber hypertrophy induced by 16 wk of RT. These findings are further supported by using week 16 myofiber CSA as a covariate.

The IIx-to-IIa shift in myofiber type is a well-recognized adaptation to RT (34) that occurs early in a program and is detectable before myofiber hypertrophy. This shift is typically considered favorable for fatigue resistance, and we have found the shift with equal potency in the young and old in the current study and in previous studies (4,24). The time course of shift reversal with detraining, and dose requirements for maintenance, on the other hand, is less clear. During detraining in the current study, approximately half of the shift reversed by 16 wk, and complete reversal was noted by 32 wk, with no age differences. We show a dose response whereby the one-third maintenance dose fully maintained the shift, whereas partial reversal was noted with one-ninth maintenance. This partial shift reversal with the one-ninth dose occurred irrespective of whether type II myofiber hypertrophy was maintained (young) or lost (old). The plasticity of myosin heavy chain isoform expression in response to altered loading is a complex phenomenon with multiple levels of regulation (2). Recent work from the Baldwin laboratory has begun to reveal key promoter elements (29) and epigenetic mechanisms (e.g., histone modifications) (31) that influence transcriptional activities of the MHC genes. In light of the present human findings, it would be of great value in future studies to determine the mechanisms responsible for MHC shift reversal even in the presence of myofiber size preservation during attempted maintenance training.

Conclusions and clinical implications. The consequences of sarcopenia—a major cause of physical frailty—are great and increasing each year as the older population continues to expand at a high rate. Our findings and those of others strongly support RT as a primary intervention strategy to reduce the deleterious effects of sarcopenia. We have demonstrated sustainability for up to 8 months with our 1-d·wk^{-1} maintenance dosing in young adults. Enhanced muscle performance was also sustained among the old; yet they seem to require more frequent dosing to maintain the muscle mass gains realized from RT. The positive health benefits of increased muscle mass among older adults extend well beyond muscle performance (e.g., glucose homeostasis, fatty acid metabolism, aerobic capacity, and bone and joint health). Therefore, we recommend progressive RT continue indefinitely for the health and functional status of all individuals.

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