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Peak Force and Maximal Shortening Velocity of Soleus Fibers After Non-Weight-Bearing and Resistance Exercise

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Abstract

Widrick, Jeffrey J., and Robert H. Fitts. Peak force and maximal shortening velocity of soleus fibers after non-weight-bearing and resistance exercise. *J. Appl. Physiol.* 82(1): 189–195, 1997.—This study examined the effectiveness of resistance exercise as a countermeasure to non-weight-bearing-induced alterations in the absolute peak force, normalized peak force (force/fiber cross-sectional area), peak stiffness, and maximal shortening velocity (V_o) of single permeabilized type I soleus muscle fibers. Adult rats were subjected to one of the following treatments: normal weight bearing (WB), non-weight bearing (NWB), or NWB with exercise treatments (NWB+Ex). The hindlimbs of the NWB and NWB+Ex rats were suspended for 14 days via tail

harnesses. Four times each day, the NWB+Ex rats were removed from suspension and performed 10 climbs (~15 cm each) up a steep grid with a 500-g mass (~1.5 times body mass) attached to their tail harness. NWB was associated with significant reductions in type I fiber diameter, absolute force, normalized force, and stiffness. Exercise treatments during NWB attenuated the decline in fiber diameter and absolute force by almost 60% while maintaining normalized force and stiffness at WB levels. Type I fiber V_o increased by 33% with NWB and remained at this elevated level despite the exercise treatments. We conclude that in comparison to intermittent weight bearing only (J. J. Widrick, J. J. Bangart, M. Karhanek, and R. H. Fitts. *J. Appl. Physiol.* 80: 981–987, 1996), resistance exercise was more effective in attenuating alterations in type I soleus fiber absolute force, normalized force, and stiffness but was less effective in restoring type I fiber V_o to WB levels.

IN A RECENT REVIEW of the literature, Convertino (2) concludes that astronauts experience substantial atrophy of the antigravity muscles of the lower limbs and back and a decline in the voluntary strength of these muscle groups after spaceflight. Because the construction of an orbiting space station will require astronauts to perform a variety of physically demanding extravehicular tasks, the development of countermeasures to prevent or attenuate these potentially detrimental alterations in muscle mass and function is an important research objective of the National Aeronautics and Space Administration.

The soleus, a predominantly slow-twitch ankle extensor muscle, is particularly susceptible to the effects of non-weight bearing (4, 8, 33). Two to 4 wk of rat hindlimb suspension, a ground-based model that mimics the non-weight-bearing conditions of spaceflight, result in substantial reductions in the mass of the soleus, its peak absolute force, and its peak force per muscle mass or muscle cross-sectional area (4, 8, 33). These changes are attributable, at least in part, to qualitatively similar reductions in the size and Ca^{2+} -activated peak absolute force and peak normalized force (force/fiber cross-sectional area) of single soleus fibers expressing the type I myosin heavy chain (MHC) isoform (10, 21, 22, 26, 31). Hindlimb suspension also increases the maximal shortening velocity (V_o) of the soleus (4, 8), which is consistent with an increase in the percentage of soleus fibers expressing fast MHC isoforms (1, 21). However, many atrophied fibers that continue to express type I MHC also display a V_o that is elevated 30–50% above normal weight-bearing levels (10, 21, 31).

It is thought that these functional alterations are, in part, the result of a reduction in the level of mechanical stress experienced by the non-weight-bearing soleus (8). Therefore, brief periods of normal or high-intensity hindlimb contractile activity have been employed as countermeasures to non-weight-bearing-induced soleus atrophy (3, 16, 20, 25). Our laboratory (31) recently reported that simply removing rats from hindlimb suspension and allowing them to stand for four 10-min periods each day was modestly effective in attenuating alterations in soleus fiber function. This intermittent weight-bearing protocol reduced the loss of soleus mass by ~20% and soleus type I fiber atrophy by ~35%. As a result, this treatment was effective in attenuating the loss in absolute peak force even though it had no effect on the decline of normalized peak force. Intermittent weight bearing also reduced the rise in type I fiber V_o by ~50%.

The purpose of the present study was to extend this work by introducing a more strenuous countermeasure treatment. Adult rats were removed from hindlimb suspension and allowed to stand for four 10-min periods each day, exactly as in the previous study of our laboratory (31). However, once each minute the animals climbed a short distance up a steep grid with a 500-g mass attached to their tail harness, an additional load that represented ~1.5 times their body mass. This experimental design made it possible to differentiate between changes in type I fiber contractile function brought about by the high-intensity climbing exercise per se and those effects produced simply by the periods of weight support that separated the climbs.

METHODS

Experimental design.

Adult male Sprague-Dawley rats were assigned to one of three experimental groups: normal caged weight bearing (WB), non-weight bearing (NWB), and NWB with exercise treatments (NWB+Ex). The NWB and NWB+Ex rats were hindlimb suspended for a 14-day period by using a tail harness assembly as previously described ([8](#), [31](#)). This procedure prevented weight-bearing activity by the hindlimbs yet allowed animals use of their forelimbs to gain access to food and water. All groups were maintained on a 12:12-h light-dark cycle. The study was approved by the institutional animal care committee at Marquette University.

Exercise training protocol.

Exercise training sessions, each 10 min in duration, were conducted four times each day (at 0, 4, 8, and 11.75 h of the light cycle). A NWB+Ex animal was removed from suspension, a 500-g mass was attached to the tail suspension harness, and the animal was placed in a standing position in a small wire enclosure. After standing for ~50 s, the rat was lifted and placed near the top of a steeply inclined grid (8° from vertical). The rat was positioned on the inclined grid so that the front paws were at the intersection of the inclined grid and a horizontal grid surface. In this position, the rat would quickly attempt to climb up the vertical grid and onto the horizontal surface. This climb required one to two contractions of each hindlimb and was usually completed in <10 s. Occasionally it was necessary for the investigator to assist animals during climbs by partially lifting the 500-g mass. This occurred primarily during the initiation of climbing activity, and once rats had started to climb they were usually able to complete the climb without additional assistance. On completion of the climb, the rat was placed back in the wire enclosure. After standing for ~50 s, the animal was again positioned on the inclined grid and completed another climb. In this manner, the animal performed 1 climb every minute, completing 10 climbs each training session. The rat was resuspended immediately after the 10th climb.

Solutions.

The composition of the solutions used in this study was as follows. The skinning solution contained (in mM): 125 K propionate, 20.0 imidazole (pH 7.0), 2.0 ethylene glycol-bis(β -aminoethyl ether)-*N,N,N',N'*-tetraacetic acid (EGTA), 4.0 ATP, 1.0 MgCl₂, and 50% glycerol vol/vol. The composition of the relaxing and activating solutions was calculated by using the computer program of Fabiato and Fabiato ([5](#)) and the apparent stability constants compiled by Godt and Lindley ([12](#)). The relaxing solution had a free Ca²⁺ concentration ([Ca²⁺]) of pCa 9.0 (where pCa = -log [Ca²⁺]) and contained (in mM) 20.0 imidazole, 7.0 EGTA, 10.0 caffeine, 4.74 ATP, 14.5 creatine phosphate, 5.40 MgCl₂, and 0.016 CaCl₂ · 2H₂O. The maximal activating solution, pCa 4.5, contained (in mM) 20.0 imidazole, 7.0 EGTA, 10.0 caffeine, 4.81 ATP, 14.5 creatine phosphate, 5.26 MgCl₂, and 7.0 CaCl₂ · 2H₂O. Both solutions contained sufficient KOH and KCl to achieve a pH of 7.0 and a total ionic strength of 180 mmol/l.

Single-fiber preparation.

Animals were anesthetized with pentobarbital sodium (50 mg/kg body wt ip). Soleus muscles were removed, trimmed of excess fat and connective tissue, weighed, and placed in cold (4°C) relaxing solution where they were dissected into small bundles of fibers that were tied at approximately in situ length to small pieces of glass capillary tubing. Bundles were stored in skinning solution at 4°C for 24 h and thereafter at -20°C for up to 28 days. All single-fiber experiments were completed during this 28-day period.

On the day of an experiment, a muscle bundle was placed in cold relaxing solution, and a single fiber segment was isolated and transferred into one of several small experimental chambers that were milled into a stainless steel plate. While submerged under cold relaxing solution, the fiber segment ends were securely attached to small stainless steel troughs as previously described ([31](#)). One trough was connected by a thin stylus to a force transducer (model 400, Cambridge Technology, Watertown, MA) and the other to a DC position motor (model

300B, Cambridge Technology). The experimental chamber was mounted on the stage of an inverted microscope. Sarcomere length was adjusted to 2.5 μm by using an eyepiece micrometer. A Polaroid photograph was then taken of the fiber while it was briefly suspended in air. Fiber width was determined at three points along the length of this photograph, and mean fiber diameter was calculated by assuming that the fiber forms a circular cross-section when suspended in air ([23](#)). Fiber length (FL) was measured as the length of fiber suspended between the two troughs. To ensure disruption of the sarcoplasmic reticulum and to improve sarcomere resolution, the fiber was briefly bathed in relaxing solution containing 0.5% Brij 58 (polyoxyethylene 20 cetyl ether, Sigma Chemical, St. Louis, MO).

It was possible to rapidly transfer the mounted fiber from chamber to chamber by vertical and horizontal translations of the stainless steel plate. The fiber was activated by moving it into a chamber filled with Ca^{2+} -activating solution. The temperature of the activating solution was maintained at 15°C during all experiments. Outputs from the force transducer and position motor were directed to a digital oscilloscope before being amplified and interfaced to a personal computer via a Lab Master input-output board (Scientific Solutions, Solon, OH). Custom software coordinated data collection, analysis, and storage.

Single-fiber contractile properties.

Absolute peak force was determined as the difference between force measured in relaxing solution and peak force attained during Ca^{2+} activation. Normalized peak force was calculated as absolute peak force divided by the fibers cross-sectional area.

V_0 was determined from the slope of the relationship describing the time required for force redevelopment after the imposition of known slack length steps. Briefly, fibers were activated, allowed to attain peak force, and then rapidly released to a shorter length. Force dropped to zero immediately after the slack step but redeveloped once the fiber had shortened sufficiently to reestablish an isometric contraction. The fiber was returned to relaxing solution and reextended to its original FL. The entire procedure was repeated at a minimum of four other slack distances. The computer plotted the times required for the redevelopment of force against the corresponding slack distances and fit the points with a least squares linear regression line, the slope of which was V_0 . The V_0 values in this paper are expressed as FL per second (FL / s). More detailed descriptions of this slack test procedure can be found in previous papers from this laboratory ([10](#), [21](#), [31](#)).

Fiber stiffness was determined from force transients (ΔP) obtained during oscillation of the fiber at a frequency of 1.5 kHz and an amplitude that produced a 0.05% change in FL (ΔL). ΔP and ΔL measurements were obtained while the fiber was in relaxing solution and during subsequent Ca^{2+} activation. Stiffness per fiber cross-sectional area (elastic modulus) was calculated as $[(\Delta P \text{ in activating solution} - \Delta P \text{ in relaxing solution}) / (\Delta L \text{ in activating solution} - \Delta L \text{ in relaxing solution})] \times (\text{FL} / \text{fiber cross-sectional area})$.

Fiber MHC isoform determination.

After contractile measurements, the fiber segment was removed from the apparatus, solubilized in 10 μl of sodium dodecyl sulfate (SDS) sample buffer [containing 6 mg/ml EDTA, 0.06 M tris(hydroxymethyl)aminomethane, 1% SDS, 2 mg/ml bromophenol blue, 15% glycerol, and 5% β -mercaptoethanol], and stored at -80°C . Later, 0.5 nl of fiber volume was run on a Hoefer SE 600 gel electrophoresis system that consisted of a 3% (wt/vol) acrylamide stacking gel and a 5% (wt/vol) separating gel ([21](#)). Gels were silver stained as described by Giulian et al. ([11](#)). A representative gel illustrating MHC isoform identification in single soleus fiber segments is presented in Fig. [1](#).

Fig. 1. Single-fiber myosin heavy chain isoform identification. Each lane of this 5% sodium dodecyl sulfate-polyacrylamide gel represents a single soleus fiber obtained from a non-weight-bearing with resistance exercise (NWB+Ex) animal. All fibers expressed type I myosin heavy chain isoform. However, note that fiber in *lane 2* also expressed type IIa myosin heavy chain. Maximal shortening velocity (V_o) of fibers in *lanes 1–6* were 1.37, 1.71, 1.30, 1.36, 1.47, and 1.11 fiber lengths/s, respectively.

Statistical analysis.

Results are presented as means $\pm$ SE. Significant differences between the three experimental groups were determined with a one-way analysis of variance and, when appropriate, the Student-Newman-Keuls post-hoc procedure. Statistical significance was accepted at $P < 0.05$.

RESULTS

Soleus mass.

Despite the 7% greater body mass of the NWB animals, their average soleus muscle mass was 40% lower than that of the WB animals (Table 1). Consequently, the soleus-to-body mass ratio for the NWB animals was 45% lower than the same ratio for the WB animals. Resistance exercise attenuated the NWB-induced loss in absolute soleus mass by 40% and the reduction in the soleus-to-body mass ratio by almost 50% but did not restore either variable to WB levels.

Table 1. Body and soleus mass of each experimental group

Group	<i>n</i>	Body Mass, g	Soleus Mass, mg	Soleus Mass/Body Mass, mg/g
WB	9	335 $\pm$ 4	158 $\pm$ 7	0.47 $\pm$ 0.02
NWB	9	358 $\pm$ 8*	95 $\pm$ 5*	0.26 $\pm$ 0.01*
NWB + Ex	8	330 $\pm$ 7†	120 $\pm$ 3*†	0.36 $\pm$ 0.01*†

Values are means $\pm$ SE; *n*, no. of animals. WB, weight bearing; NWB, non-weight bearing; NWB + Ex, non-weight bearing with exercise treatments.

*Significantly different from WB mean, $P < 0.05$.

†Significantly different from NWB mean, $P < 0.05$.

MHC isoform distribution.

Single fibers expressing type I MHC comprised 87% (46 of 53 fibers), 76% (42 of 55 fibers), and 85% (45 of 53 fibers) of the fibers studied from the WB, NWB, and NWB+Ex animals, respectively. The remaining fibers expressed either type II MHC or coexpressed type I and II MHC isoforms. Because >75% of the single fibers studied from each group expressed the type I MHC isoform exclusively, our results focus solely on the contractile properties of these fibers.

Fiber diameter.

Type I fibers from the NWB group were, on average, 30% smaller in diameter than similar fibers obtained from the WB animals (Table 2). Resistance exercise attenuated 57% of this non-weight-bearing induced decline in fiber diameter. Nevertheless, NWB+Ex fibers were still 13% smaller than normal WB type I fibers.

Table 2. Diameter, absolute force, normalized force, and maximal shortening velocity of type I soleus fibers

Group	<i>n</i>	Diameter, μm	Absolute Force, mN	Normalized Force, kN/m^2	V_o , fiber length/s
WB	46	67 ± 1	0.48 ± 0.02	134 ± 3	1.09 ± 0.03
NWB	42	$47 \pm 1^*$	$0.21 \pm 0.01^*$	$118 \pm 3^*$	$1.45 \pm 0.05^*$
NWB + Ex	45	$58 \pm 1^{*\dagger}$	$0.37 \pm 0.01^{*\dagger}$	$139 \pm 3^\dagger$	$1.41 \pm 0.04^*$

Values are means $\pm$ SE; *n*, no. of fibers. V_o , maximal shortening velocity.

*Significantly different from WB mean.

†Significantly different from NWB mean.

Absolute force.

The absolute peak force produced by type I NWB fibers was 56% lower than the absolute force produced by the WB fibers (Table 2). Almost 60% of this decline in force production was prevented by resistance training. Still, absolute force production of the NWB+Ex type I fibers remained 23% lower than that of the WB fibers.

Normalized force.

In addition to producing less absolute force, NWB fibers also produced 12% less normalized force than did the WB fibers (Table 2). Figure 2 illustrates that none of the NWB fibers displayed a normalized force $>160 \text{ kN/m}^2$ in comparison to 11% of the WB fibers. Conversely, only 2% of the WB fibers were observed to have a normalized force $<100 \text{ kN/m}^2$ vs. 19% of the NWB fibers. Thus NWB reduced the percentage of type I fibers with relatively high normalized force and increased the percentage of those with relatively low values. These changes were prevented by the resistance exercise protocol. The mean normalized force of the NWB+Ex fibers was identical to the mean of the WB group. The normalized force frequency distributions for these two groups also appeared quite similar, with 13% of the NWB+Ex fibers displaying a normalized peak force $>160 \text{ kN/m}^2$ and only 4% showing a normalized peak force $<100 \text{ kN/m}^2$.

Fig. 2. Normalized force frequency distributions for type I fibers from weight-bearing (WB; A), non-weight-bearing (NWB; B), and NWB+Ex (C) groups.

Fiber stiffness.

Stiffness was determined in a subset of WB, NWB, and NWB+Ex fibers (Table 3). In this subset of fibers, as in the larger sample, the average normalized force of the NWB fibers was significantly less than that of the other two

groups, while no difference existed between the WB and NWB+Ex means. In comparison to the WB condition, NWB was associated with a 38% reduction in peak stiffness and a 37% increase in the normalized force-to-stiffness ratio. Resistance exercise prevented this drop in peak stiffness and restored force/stiffness to the WB value.

Table 3. Peak stiffness and the force-to-stiffness ratio of type I soleus fibers

Group	<i>n</i>	Normalized Force, kN/m ²	Stiffness, kN/m ² × 10 ⁴	Force-toStiffness Ratio
WB	23	124 ± 2	2.80 ± 0.11	46 ± 2
NWB	20	103 ± 3 ^{F3-150}	1.75 ± 0.11 ^{F3-150}	63 ± 3 ^{F3-150}
NWB + Ex	30	125 ± 3 ^{F3-151}	2.60 ± 0.09 ^{F3-151}	49 ± 2 ^{F3-151}

Values are means ± SE; *n*, no. of fibers.

^{F3-150}Significantly different from WB mean.

^{F3-151}Significantly different from NWB mean.

Fiber V_o .

The average V_o of the type I soleus fibers obtained from the NWB group was 33% greater than the WB mean (Table 2). This NWB-induced rise in fiber V_o was due to both a reduction in the number of fibers with relatively low V_o and an increase in the number of single type I fibers displaying an elevated V_o (Fig. 3). Whereas 35% of the WB fibers had a $V_o < 1.00$ FL/s, only 2% of the NWB fibers fell below this level. Conversely, only 4% of the WB fibers displayed a $V_o > 1.40$ FL/s in comparison to 50% of the NWB type I fibers. The average V_o of the type I NWB+Ex fibers was not significantly different from that of the NWB group. The frequency distributions of these two groups also appeared to be similar, with only 2% of the NWB+Ex fibers falling below a V_o of 1.00 FL/s while 42% were distributed above a velocity of 1.40 FL/s. Consequently, the mean V_o of the NWB+Ex fibers remained elevated above the average WB value.

Fig. 3. V_o frequency distributions for type I fibers from WB (A), NWB (B), and NWB+Ex (C) groups.

DISCUSSION

In agreement with previous studies (10, 21, 22, 26, 31), 14 days of non-weight bearing induced significant alterations in the size and function of single permeabilized type I soleus fibers. These changes included reductions in absolute force, normalized force, and peak stiffness, and increases in V_o and the force-to-stiffness

ratio. Our laboratory (31) previously found that simply allowing rats brief periods of weight bearing during hindlimb suspension attenuated these changes in diameter, absolute force, and V_0 but had no statistically significant effect on normalized force, stiffness, or the ratio of force to stiffness. In the present study, hindlimb-suspended rats were subjected to an identical intermittent weight-bearing protocol with the exception that once every minute they performed a short climb up a steep grid while carrying a mass equivalent to 1.5 times their body mass. As detailed in Table 4, this resistance exercise training protocol maintained type I fiber normalized force, stiffness, and the ratio of force to stiffness at weight-bearing control levels over 14 days of non-weight bearing. Furthermore, this treatment was 1.5–2 times more effective than intermittent weight bearing alone in attenuating alterations in the relative mass of the soleus and in the diameter and absolute force of type I fibers obtained from this muscle. However, resistance exercise was less effective than intermittent weight bearing in preventing alterations in the V_0 of type I fibers because this variable was similar for the NWB+Ex and the NWB groups.

Table 4. Relative effectiveness of intermittent weight bearing and intermittent weight bearing with resistance exercise on soleus mass and type I fiber contractile properties

Variable	Intermittent Weight Bearing, %	Resistance Exercise, %
Relative soleus mass	22	49
Type I soleus fiber		
Diameter, μm	36	57
Absolute force, mN	29	59
Normalized force, kN/m^2	NS	100
Stiffness, $\text{kN}/\text{m}^2 \cdot 10^4$	NS	100
Force-to-stiffness ratio	NS	100
V_0 , fiber lengths/s	48	NS

Values represent relative change prevented by each countermeasure treatment after 14 days of non-weight bearing. NS, treatment mean was not significantly different ($P > 0.05$) from NWB mean; 100%; treatment mean was not significantly different ($P > 0.05$) from WB mean. Relative effectiveness of variables that were significantly different ($P < 0.05$) from both NWB and WB means was calculated as $[(\% \text{ difference between WB mean and NWB mean}) - (\% \text{ difference between WB mean and countermeasure treatment mean})] / (\% \text{ difference between WB mean and NWB mean})$. Intermittent weight bearing values from Ref. 31.

The mechanisms responsible for the altered contractile properties of single type I fibers after non-weight bearing, and how resistance exercise attenuates or prevents these changes, are not entirely clear. In addition to simple fiber atrophy, which is thought to reduce the total number of actomyosin cross bridges and lead to a decline in fiber absolute force, non-weight bearing produces a greater loss of thick vs. thin filaments (27) and a disproportionate reduction in myofibrils vs. soleus mass (29). These observations suggest a decline in the number of cross bridges per fiber cross-sectional area and are consistent with the reduction in normalized fiber force observed in the present study.

Historically, fiber stiffness has been used as a relative measure of actomyosin cross-bridge number (9). However, recent work suggests that stiffness measurements may underestimate the number of cross bridges (17) because considerably more compliance (the inverse of stiffness) resides in the thin filament than previously believed (17, 18, 30). Because we do not know whether non-weight bearing affects thin filament compliance, our stiffness results cannot be taken as conclusive evidence that cross-bridge number is reduced by non-weight bearing. Nevertheless, the decline in stiffness noted in this study plus the evidence from the ultrastructural and metabolic studies cited above suggest that the reduction in normalized force after non-weight bearing is

primarily due to a decline in cross-bridge number, although reductions in the average force produced by individual cross bridges cannot be ruled out (22).

It is interesting that all of the fibers analyzed in the present study appeared to express the adult type I MHC isoform exclusively, yet the average V_o of fibers from the NWB group were 33% greater than those obtained from the WB animals. One possible explanation is that the NWB fibers contained small amounts of fast MHC isoforms that were undetected in our gels. However, these fast isoforms would make up <2.5% of total fiber protein, and this level may be insufficient to account for the functional differences between the WB and NWB groups (21). Along similar lines, non-weight bearing may induce the expression of a unique slow MHC that comigrates with the adult type I MHC under our electrophoretic conditions (6). Presumably, fibers expressing this isoform would have a greater V_o than would fibers expressing the adult type I MHC identified on our gels. Alternatively, the disproportionate loss of myosin with non-weight bearing may result in an increase in the spacing between the remaining thick and thin filaments, as suggested by electron micrographs (27). The alterations in single type I fiber contractile function we observed after non-weight bearing, including decreases in absolute force, normalized force, and stiffness and increases in V_o and the force-to-stiffness ratio, are all consistent with the changes in contractile function that have been observed after an increase in myofilament lattice spacing (13, 19, 24).

Resistance exercise during non-weight bearing reduced fiber atrophy and by doing so attenuated the decline in absolute fiber force while completely restoring normalized force and stiffness to normal weight-bearing levels. The most direct interpretation of these results is that the exercise protocol prevented a disproportionate loss of cross bridges per fiber cross-sectional area so that the loss of fiber force after this treatment was directly related to the degree of fiber atrophy. Because resistance exercise during hindlimb suspension has been demonstrated to reduce soleus noncollagenous protein loss (20), it is possible that this countermeasure may have reduced the selective loss of thick filaments and thereby attenuate changes in muscle ultrastructure that may have been responsible for the altered functional properties of atrophied fibers. Although this explanation is supported by the absolute and normalized force and stiffness results of the present study, it is inconsistent with our finding that the V_o of the NWB+Ex and NWB fibers were similar.

When interpreting the V_o results, we believe that it is important to recall the earlier study from our laboratory (31), in which it was found that four 10-min weight-bearing sessions each day attenuated the increase in type I fiber V_o by 48% (Table 4). The present study was designed so that the time spent weight bearing between climbs was similar in duration to the intermittent weight-bearing periods of the previous work from our laboratory. We reasoned that if the climbing protocol had no effect on V_o , this variable would again be ~50% less than the NWB mean. This was not the case because the NWB+Ex and NWB means were identical. We, therefore, conclude that the climbing exercise worked to increase type I fiber V_o above what one would expect to see solely from the intermittent weight support that occurred between climbs.

This interpretation suggests that resistance exercise, like other forms of chronic physical activity, may affect fiber V_o directly. We have previously reported that exercise training, albeit endurance exercise training, results in a significant increase in the average V_o of type I muscle fibers (7, 28, 32). There is evidence that this functional change may be the result of exercise-induced alterations in fiber myosin light chain (MLC) expression (28, 32). Perhaps resistance training also modifies fiber MLC composition and by doing so shifts the V_o of type I fibers toward greater velocities as illustrated in the NWB+Ex histogram of Fig. 3. Note that under this proposed mechanism, the elevation in fiber V_o could occur independently of mechanisms serving to attenuate the decline in fiber force production.

From a practical standpoint, it will be important to determine whether the elevated V_o after this resistance exercise countermeasure has positive and/or negative impacts on fiber and muscle performance. A

higher V_o could be indicative of a potentially beneficial increase in fiber peak power output. Conversely, the faster cross-bridge cycling rate in the NWB+Ex fibers would be expected to increase their adenosinetriphosphatase activity. This could lead to a reduction in the efficiency of contraction, a greater dependence on glycolysis, and an increase in soleus fatigability.

Finally, it is important to note that the average absolute force produced by the NWB+Ex fibers remained significantly less than the WB mean because this countermeasure attenuated only about one-half of the soleus atrophy that occurred during non-weight bearing. Along similar lines, Herbert et al. (16) found that climbing exercise attenuated 74% of the decline in relative soleus mass during 7 days of hindlimb suspension while Kirby et al. (20) reported that electrically elicited maximal eccentric contractions attenuated 80% of the decline in relative soleus mass over 10 days of non-weight bearing. Our finding of a 49% attenuation in the loss of relative soleus mass compares favorably with these previous findings when one considers that the duration of non-weight bearing was 40–100% greater in the present study. Taken together, these findings suggest that resistance exercise may be unable to completely restore soleus mass to weight-bearing levels during even relatively short-term non-weight bearing. This shortcoming may be due to the characteristics of the training protocols, i.e., the duration, intensity, frequency, and mode of training have not been optimal, and/or to systematic factors associated with non-weight bearing, such as changes in anabolic hormone levels and their impact on physiological responses to exercise training (14, 15).

In summary, resistance exercise reduced type I fiber atrophy by almost 60% and served to maintain the normalized peak force of these fibers at a weight-bearing level over 14 days of non-weight bearing. However, type I fiber V_o , which rose 33% during non-weight bearing, remained at this elevated level despite the exercise treatments. Compared with intermittent weight bearing only (31), resistance exercise was more effective in maintaining type I fiber force production but was less effective in restoring type I V_o to normal weight-bearing values. These results suggest that resistance training during non-weight bearing works through two mechanisms, one that has its greatest effect on force production and another that modulates fiber V_o .

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FOOTNOTES

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AUTHOR NOTES

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