Motor Unit Number Estimates in Masters Runners: Use It or Lose It?

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ABSTRACT

POWER, G. A., B. H. DALTON, D. G. BEHM, A. A. VANDERVOORT, T. J. DOHERTY, and C. L. RICE. Motor Unit Number Estimates in Masters Runners: Use It or Lose It? *Med. Sci. Sports Exerc.*, Vol. 42, No. 9, pp. 1644–1650, 2010. **Introduction**: A contributing factor to the loss of muscle mass and strength during aging is the reduction in the number of functioning motor units (MU). It has been shown that lifelong physically active older rats have greater numbers of MU compared with age-matched sedentary controls, suggesting that chronic exercise may preserve MU function with advancing age. This has not previously been examined in humans. **Purpose**: Thus, the purpose of this study was to estimate the number of functioning MU in the tibialis anterior of masters runners (~65 yr) and to compare the values with recreationally active young (~25 yr) and healthy age-matched controls (~65 yr). **Methods**: Decomposition-enhanced spike-triggered averaging was used to collect surface and intramuscular EMG signals during dorsiflexion at 25% of maximum voluntary isometric contraction. **Results**: The estimated number of MU did not differ between masters runners and young, but MU number estimates were lower in the old (91 ± 22 MU) compared with the masters runners (140 ± 53 MU) and young (150 ± 43 MU). **Conclusion**: These results demonstrate that lifelong high-intensity physical activity could potentially mitigate the loss of MU associated with aging well into the seventh decade of life. **Key Words**: AGING, PHYSICAL ACTIVITY, MUSCLE FUNCTION, MASTER ATHLETES, EMG

Sarcopenia is a gradual deterioration of the neuromuscular system as a result of natural aging, which is characterized by a loss of muscle mass. This age-related deterioration is accentuated in the sixth decade of life, leading to a significant reduction in strength and power and ultimately frailty (39). The mechanisms leading to sarcopenia are multifactorial and include endocrine, inflammatory, nutritional, genetic, and activity level, just to name a few (for a review, see Doherty [12]). Thus, exploring models that may slow or negate this age-related decrement in neuromuscular performance is of considerable importance.

One contributing factor to sarcopenia is a reduction in the number of functioning motor units (MU) (35), which is marked by a gradual reduction during the first seven decades of life followed by a precipitous decline into very old age (7,9,30). The early loss of MU is often undetected because of the preservation of muscle mass and strength (29). This occurs through the process of collateral reinnervation whereby healthy motor neurons (often Type I MU) sprout new axons that reinnervate those muscle fibers orphaned (often Type II) after the death of their parent motor neuron, leading to larger surviving Type I MU (20). The number of functioning MU in a human muscle group can be estimated electrophysiologically by dividing the mean size (amplitude or area) of a representative sample of surface-detected MU potentials (MUP; average size of the MU within a given muscle group) into the corresponding size parameter of the compound muscle action potential in response to maximal stimulation of its motor nerve (M-wave; maximal size of the entire MU pool within the muscle group; for a review, see Bromberg [5]).

In the upper limb, age-related reductions in MU number estimates (MUNE) have been reported for the biceps brachii (7,15) and small intrinsic hand muscles (6,13,19). For the lower limb, age-related declines in MUNE have been reported for the extensor digitorum brevis (8,30), tibialis anterior (29), and soleus (40) muscles. Dalton et al. (11) reported nonsignificant reductions in the number of MU of the soleus by the eighth decade, whereas Vandervoort and

TABLE 1. Anthropometric profile of participants.

Group (n = 10)	Age (yr)	Mass (kg)	Height (cm)
Young	27 ± 3	80.7 ± 10.0	177.6 ± 7.9
Old	66 ± 3	78.8 ± 10.4	171.7 ± 7.0
Masters runners	64 ± 3	72.3 ± 7.7	177.3 ± 6.7

Values are presented as mean ± SD.

McComas (40) reported a 70% age-related loss of MU in the soleus of men older than 90 yr. These two studies suggest MU remodeling may be delayed in a habitually active postural muscle, up to a critical age, but this concept of potential preservation with physical activity has not been tested further.

The most comprehensive study of MUNE with aging was reported by McNeil et al. (29) for the tibialis anterior in which two older groups were compared with a young group. Compared with a group of recreationally active young (~25 yr) men, they found a 40% loss in functioning MU in a recreationally active group of old men (~66 yr) but a further greater loss to 60% in a very old (~82 yr) group of healthy men. Thus, age-related alterations in the numbers of MU may be dependent upon the specific muscle and amount of lifelong physical activity.

Indeed, the observed loss of muscle mass and strength in the lower limbs of typical older adults can be attributed in part to a reduction in intensive physical loading (9). Interventions (i.e., resistance training) designed to maintain function in old age can negate or even reverse the age-related loss of muscle mass and strength (2,31), highlighting the considerable plasticity of skeletal muscle into old age. Furthermore, as demonstrated in rats, although age-related MU loss is immutable, chronic physical activity has been shown to mitigate some of the loss of MU and therefore function (25). For humans, studying individuals who have maintained high levels of physical activity throughout their life span may provide further insight into the neuroprotective effects of higher volumes of lifelong physical activity at the segmental level. Masters athletes, particularly masters runners (>60 yr), engage in high-intensity lifelong physical activity and remain essentially free from confounding factors (i.e., sedentarism) that may provoke age-related declines (34). Because running places the tibialis anterior under chronic stress due to the eccentric overload after the heel strike, this stimulus may promote the preservation of the number of functional MU.

Therefore, the purpose of this study was to derive MUNE for the tibialis anterior of masters runners and to compare the values with healthy recreationally active young and age-matched controls. We hypothesized that because of the chronic physical loading of the tibialis anterior through lifelong running, the MUNE would be greater in the tibialis anterior of old masters runners compared with less active age-matched controls. The significance of the anticipated findings centers on providing an improved understanding of the neuromuscular system through "elite aging" and provides support into the favorable value of long-term physical activity and exercise for protecting neural function.

METHODS

Subjects. Ten young men, 10 old men, and 10 old masters runners (one woman) took part in this study (Table 1). The data from the young men and old men were previously published by McNeil et al. (29) but obtained using the same procedures and analysis programs as described below. Young participants were recruited from the university population, and the old participants were recruited from a local exercise program designed to maintain cardiorespiratory endurance and flexibility. Young and old participants were recreationally active with no known neurological or cardiovascular diseases. Masters runners were all actively training and competing in races around the time of testing. Recruitment was based on finishing times in a 10-mile (16 km) road race held 1 month before the experiment, and a detailed interview was conducted to identify highly trained lifelong runners. Masters runners were included if they had a finishing time under 1.5 h in the 10-mile road race, were actively training for 30+ yr, ran more than 45 km·wk^{-1}, and presented a verifiable race history with personal bests. Runner history included two runners who were former members of their universities' track or cross-country team, one was an international marathon runner, one completed over 25 marathons, two held provincial records in the 10-mile road race, and several runners had finishing times consistent with midlife personal bests. Self-reported training and performance data are presented in Table 2. Participants were asked to refrain from strenuous exercise 1 d before testing and not to consume caffeine on the day of testing. The study protocol was approved by the local university's ethics board and conformed to the Declaration of Helsinki. Informed, oral, and written consent was obtained before testing.

Experimental arrangement. Participants were seated in a custom isometric dynamometer with the hip and knee angles positioned at 90° and the ankle at 30° of plantar-flexion. To minimize confounding hip and knee joint movements during dorsiflexion, an adjustable C-shaped brace was secured to the distal portion of the thigh. Velcro strapping across the toes and the dorsum secured the foot to the dynamometer. All testing was performed on the dominant (right) leg.

Surface EMG was collected from the tibialis anterior using self-adhering Ag-AgCl electrodes (1.5 × 1 cm; Marquette Medical Systems, Jupiter, FL). The skin was cleansed with alcohol before application of the electrodes. An active electrode was positioned on the proximal portion of the tibialis

TABLE 2. Training profile of masters runners.

	Years of Training	Weekly Kilometers	Days Trained per Week	10-Mile (16 km) Finishing Time (min)
Mean	38.2 ± 6.7	59.6 ± 10.1	6 ± 1	79.7 ± 6.8
Range	30–51	45–80	5–7	70–90

Values are presented as mean ± SD.

anterior over the motor point (~7 cm distal to the tibial tuberosity and ~2 cm lateral to the tibial anterior border), and a reference was placed over the distal tendon at the malleolli. Intramuscular EMG signals were recorded via a disposable concentric needle electrode with a recording surface of 0.03 mm^2 (Model N53153; Teca, Hawthorne, NY) inserted into the tibialis anterior, 5–10 mm proximal to the active surface electrode.

Experimental procedures. EMG data were acquired using decomposition-enhanced spike-triggered averaging software on a Neuroscan Comperio system (Neurosoft, El Paso, TX). The surface and intramuscular EMG signals were band-pass filtered at 5 Hz to 5 kHz and 10 Hz to 10 kHz, respectively. Data collection started with determining the maximum twitch torque and M-wave responses. The twitch was evoked using a bar electrode held distal to the fibular head over the common peroneal nerve. A computer-triggered stimulator (model DS7A; Digitimer, Welwyn Garden City, Hertfordshire, UK) provided the electrical stimulation at a pulse width of 100 μs and 400 V. The current was increased until a plateau in M-wave amplitude was reached. The active surface electrode was repositioned to minimize the rise time of the M-Wave, ensuring the electrode was over the motor point. At this point, the stimulation intensity was increased 15% to ensure activation of all motor axons. Next, three maximum voluntary isometric contractions (MVC) were performed of 3- to 5-s duration with at least 2 min rest between attempts. Participants were provided visual feedback of the torque via near real-time display and verbally exhorted. Voluntary activation was assessed using the interpolated twitch technique (3). The amplitude of the interpolated torque evoked during the plateau of the MVC was compared with a resting twitch torque evoked ~1 s after the MVC. Percent voluntary activation was calculated as voluntary activation (%) = [1 − (interpolated twitch/resting twitch)] × 100. During the third MVC, peak root mean square (RMS) value of the raw surface EMG was calculated and is called MVC-RMS.

Upon completion of the MVC, participants were given 5 min of rest to ensure no residual fatigue. Participants were then asked to match a target line of 25% MVC for all subsequent isometric contractions at which time the intramuscular electrode was inserted into the muscle. Previous reports from this laboratory demonstrated this contraction intensity to be the most effective intensity for obtaining a representative MUNE in the tibialis anterior (28). The

investigator manipulated the concentric needle to minimize rise times of the negative-peak amplitudes of the first two to three detected MUP. Repositioning of the needle was completed by either adjusting the depth of insertion or sampling from a new area. Participants were then asked to slowly ramp-up their dorsiflexion torque to the target line within 1–2 s and hold the contraction steady for ~30 s, during which both the intramuscular and the surface EMG were obtained and stored for future analysis. Participants were given at least 1 min of rest between these contractions. These procedures were repeated until at least 20 suitable trains of MUP and their respective surface MUP (S-MUP) were collected (4).

Data reduction and statistics. Decomposed EMG signals were reviewed offline to determine the acceptability of the needle-detected MUP trains and their corresponding S-MUP. First, an acceptable MUP train required greater than 50 detected discharges, which acted as triggers for spike-triggered averaging. Then, the MU discharge pattern was inspected visually for a constant rate (i.e., coefficient of variation ≤ 30%) and a physiological mean discharge rate. Lastly, the interdischarge interval histogram was examined to confirm that it followed a Gaussian distribution. MUP trains that did not meet these criteria were excluded from further analysis. S-MUP were inspected visually to identify a distinct waveform that was temporally linked to the needle potential. The computer-generated negative-peak onset and the negative-peak amplitude markers of the acceptable S-MUP were inspected to ensure they were accurate. Any markers not correctly set were repositioned manually. A computer algorithm then aligned the negative onset markers for all accepted S-MUP and created a mean S-MUP template on the basis of their data point-by-data point average (14). Finally, a MUNE was derived by dividing the negative-peak amplitude of the M-wave by the negative-peak amplitude of the mean S-MUP.

Torque data were sampled online at 1000 Hz using the AcqKnowledge software (AcqKnowledge III; Biopac Systems Inc., Holliston, MA), amplified (DA 100 amplifier; Biopac Systems Inc., Santa Barbara, CA), monitored, and directed through an analog–digital converter (Biopac MP100WSW; Biopac Systems Inc., Holliston, MA) to be stored on the computer. Spike2 software (Cambridge Electronic Design, Cambridge, UK) was used during offline analysis to determine voluntary and evoked isometric torques and contraction duration (time to peak twitch + half relaxation time) of the evoked twitch.

TABLE 3. Neuromuscular properties of the tibialis anterior.

Group (n = 10)	MVC (N·m)	Pt (N·m)	CD (ms)	Target RMS (% MVC-RMS)	Mean MUFR (Hz)
Young	42.6 ± 6.0	6.4 ± 1.5	184.1 ± 15.9	16.5 ± 2.0	13.1 ± 1.3
Old	42.6 ± 7.7	5.9 ± 1.5	201.0 ± 21.8	22.6 ± 8.0	12.6 ± 1.7
Masters runners	31.8 ± 6.3*†	4.9 ± 0.7*	213.4 ± 31.4*	18.3 ± 5.6	12.3 ± 1.2

Masters runners had weaker evoked peak twitch torque (Pt) and maximal voluntary isometric contraction (MVC), with longer contraction duration (CD) than young and old men, whereas target RMS (%MVC-RMS) and mean MU firing rate (MUFR) were similar among groups.
* Masters runners different from young ($P < 0.05$).
† Masters runners different from old ($P < 0.05$). Values are presented as mean ± SD.

All data were analyzed using the Statistical Package for the Social Sciences for Windows (Version 16; SPSS Inc., Chicago, IL). A univariate ANOVA was performed to identify differences between groups for all EMG and torque parameters. When a significant main effect was present, Tukey's HSD *post hoc* test was performed to identify where significant differences exist. Because voluntary activation values are not normally distributed, a Mann–Whitney *U* test was used to test for the statistical significance of this variable. To determine the importance of observed differences, effect sizes were reported. The level of significance was set at $P \leq 0.05$. All data are presented as means ± SD.

RESULTS

Strength, voluntary activation, and twitch properties. Data from the masters runners were combined with data from a previously published study (30) from this laboratory using the same techniques and procedures. Although different investigators analyzed the data, this is not a concern because our laboratory recently reported that the present technique is highly reliable between raters in the tibialis anterior (4). The three groups were of similar mass and stature. The masters runners produced 25% less voluntary isometric dorsiflexion torque than the old and the young ($P = 0.003$; ES = 0.482), whereas the old and the young did not differ ($P = 1.000$; ES = 0.000) (Table 3). Voluntary activation, as assessed using the interpolated twitch technique, was >99% for all groups ($P = 0.265$; ES = 0.046). Electrically evoked peak twitch torque of the masters runners showed a strong trend ($P = 0.070$; ES = 0.200) to be 17% lower than the old and 23% lower ($P = 0.010$; ES = 0.348) than the young, whereas the young and the old did not differ ($P = 0.068$; ES = 0.030). Contraction duration of the twitch was 16% longer in masters runners than the young ($P = 0.011$; ES = 0.277), with no difference between the masters runners and the old ($P = 0.256$; ES = 0.055) and between the young and the old ($P = 0.126$; ES = 0.178).

MU properties. The three groups did not differ ($P = 0.080$; ES = 0.002) in the RMS value of the surface EMG during the targeting contractions (25% MVC) expressed as a percentage of MVC-RMS, and the mean MU discharge rate did not differ ($P = 0.563$; ES = 0.042) among the groups (Table 3). The negative-peak amplitude of the M-wave did not differ ($P = 0.243$; ES = 0.007) between the masters runners (7.8 ± 2.2 mV), the old (6.9 ± 1.0 mV), and the young (6.9 ± 1.1 mV). The negative-peak amplitude of the mean S-MUP for the masters runners (70.1 ± 37.1 μV) did not differ compared with the old (78.2 ± 19.3 μV) and the young (49.1 ± 15.8 μV) groups ($P = 0.100$; ES = 0.020), although it was significantly ($P = 0.021$; ES = 0.539) larger in the old compared with the young. MUNE did not differ between the masters runners and the young, but because of the nonsignificant larger mean S-MUP negative-peak amplitude in the old, MUNE were greater in the masters runners ($P = 0.050$; ES = 0.256) versus the old and smaller in the old

compared with the young ($P = 0.009$; ES = 0.467), whereas there was no difference ($P = 0.737$; ES = 0.022) between the masters runners and the young (Fig. 1). The MUNE derived
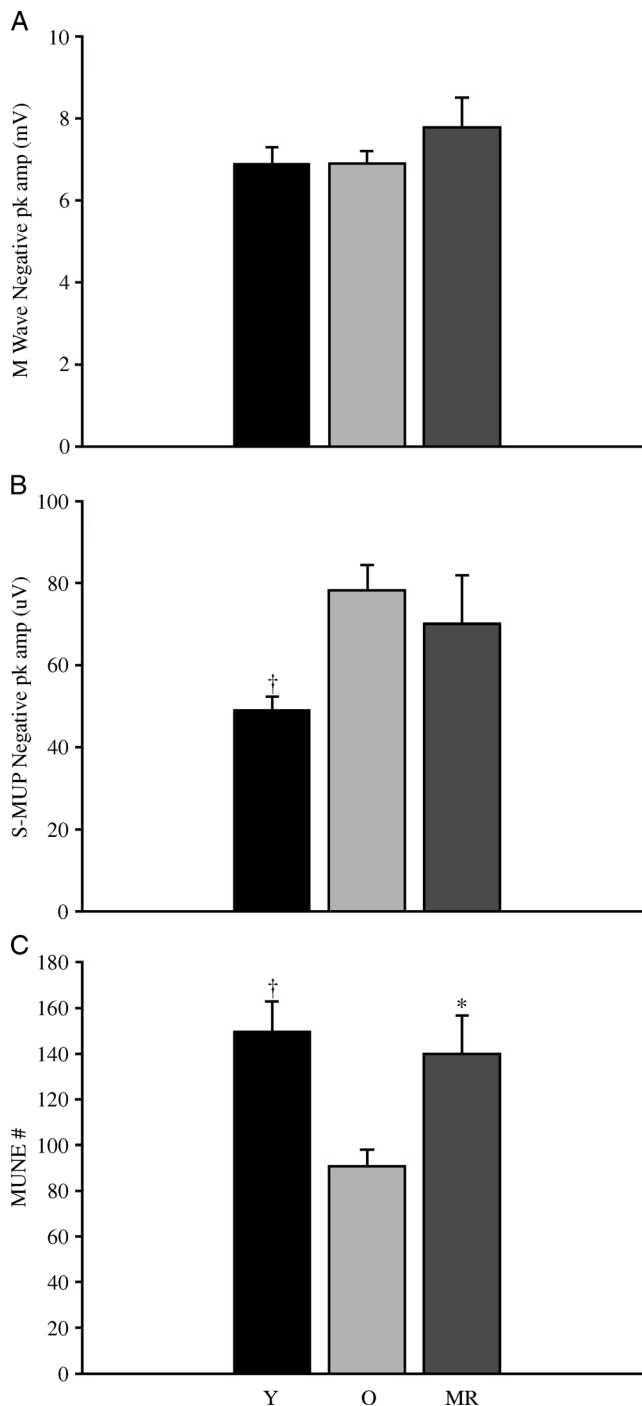
FIGURE 1—Derived MUNE (M-Wave/S-MUP). A, The negative-peak amplitude of the M-wave did not differ between young men (Y), old men (O), or masters runners (MR). B, The negative-peak amplitude of the mean surface MUP (S-MUP) was larger in O compared with Y. C, MU number estimates (MUNE) did not differ between MR and Y, were larger in MR than O, and less in O compared with Y. Values are presented as mean ± SE. *Significant difference between masters runners and old men. †Significant difference between young men and old men.

for the tibialis anterior were 140 ± 53 MU for the masters runners, 91 ± 22 MU for the old men, and 150 ± 43 MU for the young men.

DISCUSSION

We tested the hypotheses that chronic physical loading of the tibialis anterior through lifelong running would preserve MUNE in the tibialis anterior of old masters runners compared with recreationally active age-matched controls. Our hypothesis was confirmed. Thus, despite the lower voluntary and evoked strength in the masters runners, this group had greater MUNE compared with that of the old and similar to the young. These novel results suggest that lifelong high-intensity physical activity has the potential to limit the loss of the number of functional MU associated with natural aging well into the seventh decade of life.

Strength, voluntary activation, and twitch properties. Maximal isometric strength and electrically evoked twitch torque of the dorsiflexors was lower in masters runners compared with old and young men, despite near maximal activation in all groups (>99%). It is common to observe lower strength scores in masters athletes, particularly those training for endurance events (21,26,36). For example, one study (26) in masters endurance athletes (cyclists and long-distance runners) showed a 29% lower MVC in the quadriceps muscle group compared with young men as well as no difference in peak twitch torque and a slower contraction time. Unfortunately, the authors did not include an age-matched control, which would allow for analysis of neuromuscular performance on the basis of age and physical activity level. Thus, the discrepancy in strength in our study can be attributed to physiological acclimations to lifelong endurance training (10,23,36). Compared with sedentary controls, distance runners have smaller (~7%) diameters of both Type I and Type II muscle fibers, which generate 15%–18% less peak single fiber isometric force (36). In addition, masters runners have a smaller total muscle cross-sectional area (21), and longitudinal studies have shown that these measures are consistently lower in masters endurance athletes than age-matched counterparts (37,38). In support of these other indices, the masters runners in this study had a longer contraction duration than the old and the young men, whereas the old and the young men did not differ. Indeed, high-intensity endurance training results in a greater percentage of Type I muscle fiber area that have slower twitch times compared with that of faster Type II muscle fibers (1,33). Hence, the lower strength and slower contractility of the masters runners compared with age-matched controls should not be viewed as an adverse effect of aging but rather as an acclimation to lifelong endurance training.

MU properties. McNeil et al. (29) investigated MUNE in the tibialis anterior of three age groups: young, old, and very old ranging from 23 to 89 yr. They found that although there was no difference in strength between the young and the old groups, there was still a reduction in the estimated number of MU. However, in the very old group, there was a significant loss of strength, and MU numbers were reduced further, suggesting that functional significance of MU loss may not occur until after the seventh decade of life, at which point the extent of MU loss cannot be adequately compensated for by collateral reinnervation. Thus, this process may have masked the initial loss of MU through the preservation of strength in the old men, which only reached functional significance in the very old men when strength was reduced 31% and MUNE was reduced by an additional 33%. Data (17,18) suggest that in conjunction with collateral reinnervation, strength is maintained after the loss of MU by maintaining size, tension, and quality of single muscle fibers. Collateral reinnervation results in maintained muscle mass but subsequently larger MU before precipitous declines in strength and MU numbers occur (29). Indirect evidence of collateral reinnervation can be found from the larger size of the negative-peak amplitude of the mean S-MUP (indirect measure of average MU size) in older compared with younger men. In contrast to previous findings (30) using similar methods in old men, the masters runners had a similar negative-peak amplitude of the mean S-MUP compared with the young men. Despite similar negative-peak amplitude of the evoked M-wave between the old men and masters runners, suggesting similar muscle mass, the negative-peak amplitude of the mean S-MUP was somewhat larger in the old men compared with the masters runners, leading to a greater MUNE in the masters runners. This strongly suggests the masters runners have not undergone substantial MU remodeling as did their age-matched controls. By the nature of cross-sectional designs, selection bias can be a limitation. It is possible that those who excel in long-distance running may have a relatively higher proportion of slow twitch MU in their tibialis anterior muscles. The degree to which this is an important factor in our cross-sectional research design remains unknown; hence, further research is needed to establish longitudinal changes and whether physical activity can prevent or slow the age-related loss of the number of functional MU after the seventh decade.

A recent investigation from our laboratory Dalton et al. (11) reported a nonsignificant decline in functional MU numbers in the soleus of old men, which may result from the frequent activity associated with this postural muscle. The greater activity of the tibialis anterior in the masters runners compared with the recreationally active old men may have also maintained motoneuron numbers. Furthermore, an investigation (25) of the effects of chronic physical activity on the number of MU found that lifelong physically active old rats had greater MU numbers than sedentary old rats. Indeed, the role of physical activity and the effects on the preservation of MUNE may be due to exercise-induced biochemical alterations that may help the motoneuron maintain life (25), such as improved axonal transport and uptake of neurotropic factors (24), which when limited are known to cause cell death (27).

This study is not in accord with the free radical theory of aging. Instead, it parallels a current theory (32) that suggests reactive oxygen species may not be as detrimental to the aging human neuromuscular system as believed previously. Masters runners undergo tremendous oxidative stress through lifelong training and competition, and in keeping with the free radical theory of aging (22), the masters runners in this study should have been much closer to frailty than their age-matched counterparts and would not have MU numbers similar to the young men. However, it appears in this study that chronic high-intensity exercise actually has beneficial effects on the motoneuron and the muscle fibers it innervates (16). Although masters runners place tremendous oxidative stress on their bodies, the possible abundance of antioxidants produced may negate this adverse effect and even partially alleviate natural biological cellular degradation.

CONCLUSIONS

These data are presented as a cross-sectional comparison of three populations distinguished by physical activity level and age. MUNE were greater in masters runners compared with age-matched controls and similar to young men. This novel finding supports our hypothesis and suggests that lifelong running preserves MUNE in the tibialis anterior well into the seventh decade of life and that chronic activity has beneficial effects not only on the muscle fibers but also on the motoneuron. However, it is yet to be elucidated if physical activity can preserve MUNE beyond the seventh decade. Further research is needed to establish if physical activity can indeed prevent the age-related loss of MU after the seventh decade and whether MU loss follows a dose–response relationship with physical activity.

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