Motor unit loss in aging skeletal muscle is not accompanied by increased heterogeneity of the T2 increase after exercise.

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Introduction:

Previous studies show that sarcopenia in the elderly is preceded by loss of lower motor neurons and decreased motor unit number (1-3). In young subjects loss of motor neurons, for example, due to motor neuron disease or peripheral nerve injury, can be well-compensated for by reinnervation of the denervated muscle fibers from nearby, intact motor axons. However, it is not clear that this reinnervation process is as robust in muscles of elderly subjects (3). Interestingly, a computer model of the muscle denervation-reinnervation process suggested that the resulting increased density of innervated cells within motor unit territories should result in increased heterogeneity of the MRI-measured muscle T2 increase after moderate repetitive exercise (4,5). Therefore, the purpose of this study was to test the hypothesis that motor unit loss in peripheral muscle of elderly vs. younger subjects results in increased heterogeneity of the post-exercise T2 increase.

Methods:

Ten elderly (73.6±4.2 [SD] yrs, 2 female) and seven young adult subjects (26.5±6.6 yrs, 3 female) completed the study after giving informed, written consent. During one experimental session, motor unit number estimation (MUNE) in the right anterior tibial (AT) muscle of each subject was performed using the multipoint stimulation electromyographic method (6). During a second session, subjects lay supine in a GE 1.5T Horizon system (GE Medical, Milwaukee, WI, USA) with the right foot secured to a custom-made device for measuring the force of ankle dorsiflexion. Maximum voluntary force (MVC) of ankle dorsiflexion contractions was measured as the average of the 2 strongest of 4-6 test isometric contractions. Spin-echo images (TR/TE=1000/30, 14 cm FOV, 1 cm slices, 256x160 acquisition matrix) were acquired via a standard quadrature T/R extremity coil at rest and immediately after 3 minutes of dynamic ankle dorsiflexion exercise (1 s duration contractions to peak target force of 33% MVC, performed against an elastic resistance at 0.5 Hz). T2 was computed on a voxel-by-voxel basis, and mean AT muscle T2 and T2 histograms were computed from ROI’s manually drawn in 4 slices encompassing the muscle’s maximum cross-sectional area (CSA), with care to exclude blood vessels and other non-muscle tissue. Comparisons between groups were made by Student’s t-test with Bonferroni correction for multiple comparisons.

Results:

As also reported by others (2), the estimated motor unit number in AT muscle of elderly subjects was half of that in younger subjects (Table). Despite this dramatic decrease in motor unit number, there was no significant difference in maximum AT CSA between groups. As expected from previous studies (e.g., 7, 8), MVC was lower in elderly subjects, but there was a trend toward less fatigue (i.e., percent decline in force) in the elderly subjects at the end of the 3 minute exercise. Mean muscle T2 was significantly higher at rest in the elderly subjects. However, the increase in mean T2 after exercise (illustrated in Figure 1) was similar in the two groups (Figure 2). Furthermore, although the distribution of muscle pixel T2 values (Figure 3) was significantly broader in the elderly compared to younger subjects at rest (mean SD 4.07 ± 0.26 [SE] vs. 2.91 ± 0.14), this difference was abolished after exercise.

Discussion:

Contrary to our hypothesis, there was no significant increase in T2 heterogeneity in muscle of the elderly subjects after repetitive exercise, despite the dramatic decrease in AT motor unit number in these compared to the younger subjects. This result suggests that muscle fiber denervation in the elderly is not primarily compensated for by reinnervation of fibers and increased motor unit fiber density, but instead by hypertrophy of fibers in the surviving motor units, e.g., by hypertrophy of Type IIA fibers (9).

References:


![FIGURE 1](image1.png)

![FIGURE 2](image2.png)

![FIGURE 3](image3.png)