Maintaining motor units into old age: running the final common pathway

Geoffrey A. Power (1), Brian H. Dalton (2), Kevin J. Gilmore (3), Matti D. Allen (4), Timothy J. Doherty (5), Charles L. Rice (3,6)

(1) Department of Human Health and Nutritional Sciences, College of Biological Sciences, University of Guelph, Guelph, Ontario, Canada; (2) School of Health and Exercise Sciences, The University of British Columbia Okanagan, Kelowna, British Columbia, Canada; (3) Canadian Centre for Activity and Aging, School of Kinesiology, Faculty of Health Sciences, The University of Western Ontario, London, Ontario, Canada; (4) Schools of Medicine and Kinesiology and Health Studies, Faculty of Health Sciences, Queen's University, Kingston, Ontario, Canada; (5) Departments of Physical Medicine and Rehabilitation and Clinical Neurological Sciences, Schulich School of Medicine and Dentistry, The University of Western Ontario, London, Ontario, Canada; (6) Department of Anatomy and Cell Biology, Schulich School of Medicine and Dentistry, The University of Western Ontario, London, Ontario, Canada.

This article is distributed under the terms of the Creative Commons Attribution Noncommercial License (CC BY-NC 4.0) which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited.

Abstract

Invited Letter to the Editor. This article is a commentary on the recently published manuscript “Use it or lose it: tonic activity of slow motoneurons promotes their survival and preferentially increases slow fiber-type groupings in muscles of old lifelong recreational sportsmen”. Mosole S, Carraro U, Kern H, Loefler S, Zampieri S. Use it or lose it: tonic activity of slow motoneurons promotes their survival and preferentially increases slow fiber-type groupings in muscles of old lifelong recreational sportsmen. Eur J Transl Myol 2016;26:5972. doi: 10.4081/ejtm.2016.5972. We offer some unique perspectives on masters athletes and the role of physical activity in maintaining the number and function of motor units into old age.

Key Words: Motor unit, masters athlete, EMG, motor neuron.
Maintaining motor units into old age

Eur J Transl Myol 2017; 27 (1): 71-73

hypertrophy, but MN numbers within the spinal cord were similar to the control limb. Conversely, those rodents subjected to life-long swimming, maintained MN numbers well into old age. This neuroprotection of MNs in rodents was later corroborated in the tibialis anterior of masters athletes (average age of 65 and 80 years). Not necessarily surprising, there does not appear to be an overall systemic preservation of MUs in masters runners because MUNE in the biceps brachii (a less active muscle in runners) was similar to age-matched controls. Together, most findings in humans appear to support results shown in animal models that chronic activation of the MN pool specific to the muscle exercised seems to be required for delaying the ‘typical’ age-related loss of MUs. The precise mechanisms by which chronic physical activity protects MUs from age-related loss remains unclear. Future work using animal models should shed some light on appropriate therapies to maintain functional MUs during the aging process. In addition to the number of functional MUs, the integrity of neuromuscular transmission with adult aging can be assessed by measuring MU potential stability through measures of variability in the overall shape and timing of consecutively detected near fiber MU action potentials as measured using needle electromyography. In world class octogenarian masters athletes we found a higher number of functioning MUs as compared with age-matched community dwelling controls. Of special interest was that the level of collateral reinnervation was not different (i.e., the negative peak amplitude of the [surface motor unit potential (SMUP)] representing the electrophysiological size of the remodeled motor unit was not different) between groups. Despite the same MU size, the masters athletes exhibited greater MU stability, as determined by their lower near fiber jiggles and jitter values (i.e. MU parameters used to assess MU transmission stability) compared with age-matched controls. The greater levels of MU potential stability in the masters athletes versus the controls may be reflective of protection against abnormalities in muscle fiber action potential propagation, or neuromuscular transmission instability due to the development of dysfunctional neuromuscular junctions of newly reinnervated fibres. Additionally, we reported increased near-fiber potential count in age-matched controls compared with masters athletes, which may be more sensitive than surface EMG measures (i.e., SMUP) in detecting reinnervation, presumably to compensate for prior denervation (i.e. motor unit remodeling). These electrophysiological measures of fiber grouping are consistent with histological evidence of increased MU homogeneity in advanced age. Masters athletes have provided an interesting model in humans to explore healthy adult aging in a population free from many confounding factors known to accelerate the aging process such as obesity and immobility/disuse. Both masters athletes and healthy older adults seem to experience MU remodeling but the masters athletes exhibit less MU loss and more effective reinnervation and more stable neuromuscular transmission, whereas the age-matched controls presented with more extensive remodeling and less stable neuromuscular transmission. The presumed better maintenance of MUs in masters athletes occurs at a time point in the aging process when MU loss is greatest and the loss of muscle mass and strength becomes clinically relevant, potentially maintaining function and attenuating or delaying sarcopenia in this exceptional cohort of older adults. More studies are needed to relate these measures to functional tasks in humans. However, studies in animal models and humans such as reported by Mosole et al., are critical to provide further understanding and insight into the neurophysiology of aging and human neuromuscular function.

Author’s contributions

All authors contributed to the writing and editing of the article.

Acknowledgments

The studies were supported by the Natural Sciences and Engineering Research Council of Canada (NSERC) and the Canadian Institutes of Health Research (CIHR).

Conflict of Interest

The authors declare no conflicts of interest.

Corresponding Author

Geoffrey A. Power, Ph.D.
Neuromechanical Performance Research Lab
Department of Human Health and Nutritional Sciences
College of Biological Sciences
University of Guelph, Ontario, Canada N1G 2W1
Phone: 519-824-4120 x53752
Email: gapower@uoguelph.ca

E-mails of coauthors

Brian H. Dalton: brian.dalton@ubc.ca
Kevin J. Gilmore: kgilmor2@uwo.ca
Matti D. Allen: mallen@qmed.ca
Timothy J. Doherty: tim.doherty@lhsc.on.ca
Charles L. Rice: crice@uwo.ca

References