If you don't use it you'll likely lose it

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SHORT COMMUNICATION

If you don’t use it you’ll likely lose it
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Summary

This article is a commentary on the recently published manuscript by Drey et al. (2014). The age-related loss of motor units (MU) is an immutable process and understanding the possible role of physical activity in maintaining functional MUs is an important topic. Dysfunctional remodelling of a MU is associated with denervation of the muscle and ultimate death of the spinal motoneurone. Conversely, in cross-sectional studies, high levels of physical activity in humans report a maintenance in the number of functional MUs in older master runners. However, it seems that only those MUs directly associated with the elevated long-term physical activity appear to benefit from any exercise-induced neuroprotective effect.

The authors (Drey et al., 2014) of the paper which is the basis of this commentary, with a large sample size, investigating the age-related loss of muscle mass and motor unit (MU) numbers. The cross-sectional study was designed to characterize the influence of high levels of physical activity in later life (i.e. master athletes) on the typical age-related loss of muscle mass and compared this with MU loss as estimated by a MU number index (MUNIX). A commendable aspect of the experimental design was examining muscle mass and MU number in both males and females and those with distinct training regimes (i.e. power versus endurance master athletes). The authors (Drey et al., 2014) reported that whole-body muscle mass was maintained with a concomitant maintenance in the number of functional MUs of an intrinsic hand muscle group, as indicated by the MUNIX technique, in a group of master athletes compared with older adults. Unfortunately, major methodological limitations blunted the interpretation of the influence of lifelong activity on maintaining MUs in the master athletes relative to the older control group. Critically, the master athletes were an average age of ~58 years; whereas the ‘old’ control group were almost 20 years their senior (~77 years). It has been well established in the literature that there is a gradual decline in the number of MUs up to roughly the seventh decade of life, which is followed by an exacerbated decline thereafter (McComas, 1991; McNeil et al., 2005). Therefore, it is impossible to make any claim of physical activity on maintenance of MU number in this study owing to the age range disparity between the groups tested. Thus, based on the current understanding of MU loss and ageing, the study cannot confirm whether the ‘younger’ master athletes’ MU number estimates were greater due to age or activity levels. An appropriate age-matched control group is required.

Lifelong physical activity has been shown to mitigate age-related loss of MUs (Kanda & Hashizume, 1998; Power et al., 2010; Allen et al., 2013). An emerging factor in the maintenance of functioning MUs appears to be the type of lifelong physical activity. In two separate studies using an animal model, Kanda et al. (1996) investigated the role of increased physical activity on motoneurone (MN) survival using two distinct exercise modalities: one involving muscle synergist ablation that effectively overloaded the remaining musculature, analogous to strength training, and a second focused on whole-body moderate intensity aerobic exercise (Kanda &
Hashizume, 1998) such as exercise typically performed by an endurance athlete. The results of these two investigations were rather divergent, indicating that the type of enhanced activity resulted in distinctly different outcomes. Overloading the muscle (Kanda et al., 1996) resulted in significant compensatory muscle hypertrophy, but MN numbers within the spinal cord were similar to the control limb. Thus, the increased activity did not provide a neuroprotective effect. Conversely, those rats subjected to lifelong swimming (Kanda & Hashizume, 1998) maintained MN numbers well into old age. These neuroprotective findings in the rat were later corroborated in the tibialis anterior of older master distance runners (~65 years) (Power et al., 2012) and world class master athletes (~80 years) (Allen et al., 2013) in which both groups showed a maintenance of MUs relative to age-matched controls.

In the Drey et al. (2014) paper, the authors make specific reference to an investigation our group performed in the biceps brachii as evidence against an overall neuroprotective reference to an investigation our group performed in the same master runners (Power & Hashizume, 1998) which was not loaded directly during running, was investigated in these same master runners (Power et al., 2012). Unlike the tibialis anterior, estimates of the number of MUs in the biceps brachii of the master runners were decreased in old age, similar to older adult controls as compared to the young. Together, these findings in the tibialis anterior and biceps brachii indicate that chronic activation of the MN pool specific to the muscle action is required for delaying the ‘typical’ age-related loss of the quantity of MUs during healthy adult ageing (Power et al., 2012). Drey et al. (2014) found a preservation of hypothenar MUs and argued that owing to the small sample size, Power et al. may have masked any overall net neuroprotective effect. Because Power et al. observed an age-related loss of MUs when comparing young to old with nine subjects in each group and did not observe a difference for the athletic group, this seems to strengthen the concept of a limited overall net neuroprotective effect. Further evidence of muscle-specific MU survival may come from a study by Valdez et al. (2010) who found that caloric restriction had whole-body effects on preservation of MNs while the effects of exercise were localized to preserving the functional integrity of only those neuromuscular junctions involved in the specific exercise. Thus, it is difficult to interpret the underlying mechanism of a maintenance in MU numbers in the present manuscript as a whole-body (hand muscle) neuroprotective effect in master athletes. Despite the large sample size, the findings of an ‘overall neuroprotective effect’ reported by Drey et al. (2014) are limited and may simply be due to an age effect whereby the authors compared a young group of athletes (58 year) with an older (77 year) control group. Thus, the overreaching conclusions of this study should be taken with caution.

**Conflict of interest**

The authors declare no conflict of interests.

**References**


