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Age Related Motor-Unit Remodeling and Its Effect on Muscle Performance

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summary

Age-related changes in motor-unit structure are not without functional consequences. Changes in gross strength, rate of force production, and control of muscular force are factors strength professionals need be aware of when working with older adults.

Aging is associated with loss of muscle mass and strength as a result of numerous non-pathological changes within the neuromuscular system. Two commonly reported changes include loss of fiber number and size; however, these factors alone fail to account adequately for the magnitude of strength loss (21). Despite detailed observations and quantification of declining strength and mass, the underlying mechanisms precipitating such changes are not fully understood. Neurologic degeneration of-

fers a plausible explanation to account for such changes.

Progressive destruction of the nervous system as a function of aging has a significant impact on the structure and function of the neuromuscular system and, consequently, performance of aging muscle. Examination of age-related changes in the most fundamental unit of neuromuscular control, the motor unit (MU), can provide insight regarding the functional consequences of aging on muscle performance. Predicated on the role of the MU, this review is presented to provide the strength community with a summary of the concept of age-related MU remodeling and its effect on skeletal muscle performance.

Motor-Unit Function

A MU is a lower motor neuron (alpha motor neuron) and all the muscle fibers it innervates (3, 8, 17). Not to be confused with gamma motor neurons, which provide efferent (motor) innervation to the reflex-mediating muscle spindle, alpha motor neurons provide efferent innervation to skeletal muscle fibers. Termed the “final common pathway,” the MU relays motor messages

from the central to peripheral nervous systems resulting in activation of skeletal muscle (16).

Expression of muscular force is a complex event dependent on three primary factors related to the MU recruitment: (a) the number of MUs recruited, (b) the size of the MUs recruited, and (c) the frequency with which a MU is recruited (3). With increasing demand for force, more MUs may be activated, larger MUs recruited, and already recruited MUs activated more often. Variations in the control of force through these mechanisms is termed “coding” and is responsible for the great variation with which muscular force can be generated.

The recruitment of MUs has long been described by the “size theory of recruitment,” which states that the order of MU recruitment is directly related to the size and electrical threshold at which cell bodies can be activated (11). Likewise, the recruitment of MUs initially is described as additive, indicating the functional specificity in the recruitment of slow or fast MUs. This implies that for a specific task, similar-size MUs are recruited first. Once muscular force has

been generated by additive recruitment, sequential recruitment allows for greater force to be generated. As an example, slower functions such as walking and control of balance are predominated by additive recruitment of slower/smaller MUs; however, with more explosive/faster activities such as jumping and sprinting, the activation of slower MUs is subsequently accompanied by the recruitment of larger/faster MUs (3, 23).

MU size and innervation ratio are determined by the number of fibers per motor neuron, and both influence MU function. MUs with a greater number of fibers are capable of generating greater force than those with fewer fibers. However, larger MUs with greater innervation ratios offer less precision, or ability to grade force, versus MUs with lesser fibers (9). “Coding” signals are used to alter force output with precision, but activation of large MUs would subsequently activate a larger number of muscle fibers than when a smaller MU with fewer fibers is activated.

Motor-Unit Remodeling

MU remodeling has been defined as the natural cycle of turnover of neuromuscular connections between the motor neuron and muscle fibers. Through the process of denervation via the death of motor axons, subsequent sprouting of existing MUs, and re-innervation of previously denervated fibers, MUs are remodeled (4). Initially, the capacity for re-innervation is capable of compensating for the denervation, but in more advanced years, the neurogenic disruption exceeds the capacity to restore innervation, leaving a greater number of muscle fibers without neural supply. This, in turn, precipitates muscle fiber atrophy and death. The rate of MU loss is estimated at 1% per year beginning in the third decade and increasing exponentially in advanced years (24). From the second to tenth decades, data re-

port an average MU loss of 25% (24) with additional studies showing up to a 50% loss in total MU number (6, 24).

However, MU remodeling results in a decrease in the total number of viable MUs while increasing MU size via collateral sprouting and incorporation of abandoned fibers into the existing MU (18). Ansved et al. (1) have suggested, however, a limit to the number of fibers that can be incorporated into an existing MU, and this limit appears to be dependent on the specific muscle studied, the MU type, and species examined. Whether a limit is due to an inherent inability of the motor neuron to meet the metabolic needs during reinnervation is unknown (14).

Age-related neural degeneration, although present in all MU types, appears to preferentially target type II (fast) muscle fibers, which are innervated by larger MUs (1, 4, 24). This is evidenced by an increased innervation ratio in slow MUs paralleled by a decline in the number of type II fibers (14). This occurs, however, at a cost as the abandoned type II fibers are incorporated through collateral sprouting into slower MUs supplying type I (slow) muscle fibers (21). Consequentially, type II fibers incorporated by reinnervation into slower type I MUs become, by physiologic and biochemical properties, type I fibers (13, 18).

Numerous sources within the literature provide evidence for the remodeling of MUs with aging (5, 13–16, 20–22). Biochemical evidence shows changes in myosin heavy-chain (MHC)—the specialized portion of myosin providing the power stroke and molecular basis for speed of muscle contraction—in the fast-fatigable type IIb fibers suggesting a transformation to fast-fatigue resistant type IIa fibers (22). The normal random distribution of small and large MUs within the muscle normally seen is disturbed with MU remodeling

as evidenced by histochemical data showing clustering or grouping of type I fibers with a decline in type II groups (5). These findings are indicative of the structural alterations secondary to MU remodeling and are not without functional consequences.

Functional Consequences of Motor-Unit Remodeling

MU remodeling results in three primary functional changes in muscle performance: (a) decreased strength, (b) decreased rate of force development, and (c) declining control of force. The first change in performance, decreased strength, may be the most apparent consequence of aging. Numerous reviews are available, which detail the more-systemic effects of aging on the neuromuscular system beyond those discussed here, yet they all address declining strength (2, 4, 15, 20). At lower force levels, activation of smaller/slower MUs occurs by additive recruitment. With the demand for higher force, larger MUs are recruited. Because the generation of force at high levels entails the sequential recruitment of larger MUs along with additive recruitment of smaller MUs, loss of the large MUs would decrease force generation as fewer large MUs would be available for recruitment. Additionally, type II fibers formerly innervated by large motor axons but now remodeled into slow MUs exhibit the physiologic characteristics of slow MUs and show a reduced capacity for generating force (13). Collectively, changes at the level of the MU appear to contribute significantly to the magnitude of strength loss observed with aging.

Loss of MUs is also reflected in the declining ability to generate forces quickly. Rate of force development (RFD), or the amount of force generated per unit time, decreases with age, and this decrease has been correlated with a decline in the number of the large/fast MUs, which include type II fibers (25, 26). Vandervoort and Mc-

Comas reported prolonged contraction times in older muscles showing loss of large/fast MUs versus younger muscle without such changes (25). The decrease in RFD in older muscle is asserted to be inherently related to loss of type II fibers, and interestingly, RFD declines more rapidly than gross strength (10). This provides direct support for the contention that atrophy may be more significant in type II fibers resulting in a more obvious decrease in the ability to generate forces quickly but also a decline in maximal force.

Decreased RFD is also evidenced when assessing motor nerve conduction velocity (MNCV). Concomitant with a loss of large MUs and type II fibers, MNCV declines with increasing age in a linear manner (7, 19). Previous investigators have suggested that peak MNCV is attained as early as 10 years, remains relatively consistent until age 50, and thereafter undergoes a near-linear decline ranging from 0.15 to 0.18 m/s per year (27). Considering changes in MNCV, loss of the large, fast MUs remains a plausible explanation for such slowing.

The third alteration in muscle performance is decreased control of force output. Evidence suggests that remodeling of MUs, with concomitant increased MU size and decreased MU number, results in greater force per MU but decreased ability to grade force output (9, 12). Thus, to grade/control force, sequential recruitment of MUs of increasing size leads to recruitment of larger MUs with higher innervation ratios. With fewer fibers per MU, a smaller innervation ratio allows for greater precision in the summated force exerted. With more fibers per MU, the remodeling from lower to higher innervation ratios results in a decreased ability to control force. Consequentially, the ability to modulate force

output is impaired as larger MUs with more fibers are activated, manifesting a decreased ability to control/grade force.

Conclusion

Age-related neurogenic reorganization in the form of motor unit remodeling results in a decline in total number, but increase in size, of existing MUs. The selective denervation of type II fibers and subsequent re-reinnervation by type I fibers are likely influences on functional changes observed in skeletal muscle performance. These changes include a more obvious decline in maximal strength but are also present in less overt parameters of muscle function such as rate of force development and control of force. As many of those in the strength community are involved with older adults, a basic understanding of the effect of aging on skeletal muscle performance is fundamental. The functional consequences of MU remodeling decrease the ability to produce large forces, generate force quickly, and control force. Strength professionals need to consider these changes with program design and progression in keeping with known physiological changes in older adults. ♦

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