Aging and Athletic Performance – Don Burris, PhD, CSCS

The aging process produces cardiovascular endurance and muscular strength degradation, even in elite athletes, with the onset of such degradation becoming more prevalent and exacerbated after age 30. In the case of strength athletes, ability may decline, on average, 1-1.5% per year until the age of 70, and more dramatically thereafter depending upon the level of resistance training engaged in by the individual over their lifetime (Haff & Triplett, 2016). Between the ages of 20 and 50, individuals' strength experiences an ~10% decline and, after the age of 50, more dramatically. Maximal aerobic power decreases 1% per year from its peak value which occurs between the ages of ~20 and ~40 (Powers & Howley, 2018). As we age, we experience a decline in VO^2 max over time that perpetuates a degeneration in our ability to engage in activities with the same level of power, endurance, and strength. Aging involves degenerative, physiological changes in maximal heart rate, cardiac output, muscle mass, balance, coordination (neuromuscular efficiency) as well as the potential for the onset or development of pathological limitations, such as arteriosclerosis, atherosclerosis, peripheral vascular disease, osteoporosis, and osteoarthritis. These, degenerative processes lead to decrements in functional capacity and produce significant reductions in endurance, cardiorespiratory fitness, muscular strength, mobility, functional capacity, and proprioceptive neural responses (Clark et al., 2018).

As we age, we experience significant changes in body composition in the form of the loss of bone (osteopenia) and muscle mass (sarcopenia); a decrease in muscle mass produces a correlational loss of muscle strength. After the age of ~30, there is a reduction in the cross-sectional areas of individual muscles, a decrease in muscle density, a reduction in tendon compliance, a denervation of muscle fibers, and an increase in intramuscular fat (Haff & Triplett, 2016). In particular, the gradual denervation of muscle fibers produces a decrease in the ability

of a muscle to generate power; power is lost at a faster rate than muscle strength. However, aging does not serve to enhance or reduce the ability of the musculoskeletal system to experience adaptations to resistance training exercise. As such, marked improvements in muscular strength, muscular power, muscle mass, bone mineral density, and functional capabilities have been found in older individuals who participated in progressive resistance training programs (Haff & Triplett, 2016). Resistance training has been found to increase strength in older individuals by as much as 30% (Powers & Howley, 2018).

The primary contributor to the loss of power and strength as we age is sarcopenia, which results in both a decrease in the size of muscle fibers and, most predominantly, a reduction in the magnitude of fibers. The potential causes of sarcopenia are related to inactivity, free-radical mediated damage (oxidative stress) to muscle fibers, inflammation, and a decrease in anabolic hormone (testosterone) production. The loss of muscle mass occurs most dramatically after the age of ~50; individuals can lose 40-60% of their muscle mass by the age of ~80 (Powers & Howley, 2018). Individuals that are engaged in lifelong resistance training programs experience a much slower decline in the loss of muscle mass as compared to untrained and sedentary individuals.

Degradations in endurance performance and VO^2 max (maximal aerobic capacity) are also related to aging. VO^2 max declines at ~1% per year after the age of ~40. The declination can be slowed by individuals who are engaged in chronic endurance training over there life span (Powers & Howley, 2018). As we age, we experience a decline in maximal cardiac output due to a reduction in maximal heart rate and maximal stroke volume. From age 20 to 60, maximal heart rate can decline by ~60 beats per minute. Additionally, over the course of time, the mechanisms of maximal oxygen are negatively effected as a result of reduced capillary density and

mitochondrial volume (Powers & Howley, 2018). It is noteworthy that exercise economy, the steady-state oxygen consumption during exercising at a submaximal exercise intensity below the lactate threshold, is largely unaffected by the aging process; exercise economy supports the efficient utilization of energy during exercise. Therefore, although aging does not have as deleterious of an effect on exercise economy or lactate threshold, VO^2 max does experience an aging related decline based on diminished maximal heart rate and maximal stroke volume (Powers & Howley, 2018).

References

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