The age-related decrease in muscle mass (referred to as “sarcopenia”) and strength has been well characterized. Indeed, sarcopenia has been demonstrated to be associated with decreased functional capacity among very old individuals and decreased energy needs. What is less well understood is the relationship between muscle size and strength and the development of power. Power is the product of force generation and speed of muscle contraction. Bassey and colleagues (1) demonstrated that among frail nursing home residents, leg muscle power is more important than strength for performing daily activities such as stair climbing, rising from a chair, and walking. Older men and women who required the use of assistive aids to perform these tasks had 42–54% less leg extensor power than those who could complete these tasks without assistance. The ability to generate force rapidly is a critical component of ambulation. Preservation of strength and prevention of sarcopenia, while important, may not result in a preservation of the ability to perform mobility-related activities of daily living. Indeed, muscle quality (defined as the amount of force production/unit of muscle) decreases with advancing age (2), and muscle power decreases to a greater extent than does muscle strength (3). Although sarcopenia has been demonstrated to be a predictor of disability in older people (4), the strongest predictor of late-life mobility-related disability appears to be body fatness (5–7). While a number of studies have indicated that low body weight or weight loss is associated with an increase in the risk of hip fracture (8–10), increased body weight and body fat has also recently been shown to increase the risk for a hip fracture in older White women (11). The explanation for this may be in the greatly reduced ability to generate power with increased amounts of body fat. Reduced muscle quality and decreased power production are also risk factors for hip fractures among older people (12). In addition, weight loss without resistance exercise will likely result in an exaggerated loss of muscle mass in older people.

The article by Martin and colleagues in this issue (13) demonstrates that muscle power is significantly reduced with advancing age and may be related to muscle fiber composition. Because the power output of type II muscle fibers is four times that of type I fibers (14), the selective atrophy of type II fibers with advancing age may hasten the decreases in muscle power in late life. Another potential cause for decreased power is an age-related increase in muscle stiffness (15) that may be secondary to increased nonelastic connective tissue. This loss of power with age has recently been demonstrated to have severe functional consequences (16). Among a group of sedentary, community-dwelling elderly women (mean age 74.8 ± 5.0 years), leg power was the strongest predictor of functional status (compared with medical diagnoses, neuropsychological status, or other physiological parameters). It is clear that the capacity of elderly people to increase muscle strength and size with progressive resistance exercise training (17–19) is well preserved into late life, and exercise programs designed for elderly, fall-prone people can reduce the risk and incidence of falls (20). We have demonstrated that increased muscle power results from progressive resistance training (21); however, muscle strengthening exercises may not always produce an optimum increase in power. Strategies designed to provide an increase in muscle power in elderly people must also be tested. Perhaps incorporating more rapid force-generating exercises at lower intensities should be considered in any exercise program for elderly people.

The preservation of muscle power into late life can greatly decrease the risk of disability and enhance functional independence. Research into strategies to increase muscle power in old people and to prevent the age-related loss of muscle power should be seen as a very high priority.

Acknowledgment

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References


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