

Sarcopenic Obesity: The Confluence of Two Epidemics

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The two greatest epidemiological trends of our times are the aging of the population and the obesity epidemic (1). Each of these trends has important effects on body composition, morbidity, and mortality. Aging causes a progressive loss of muscle mass and strength, independent of any disease process, called sarcopenia, from the Greek for “poverty of flesh” (2). Sarcopenia is an important cause of frailty, disability, and loss of independence in the elderly, and recent estimates suggest that it costs the United States over \$18 billion per year, a sum on par with the economic consequences of osteoporosis (3–6). Weight loss is not necessary for the development of sarcopenia, but it accelerates it (4,7).

In general, normal aging in the developed world means that most adults gain weight between the third and sixth decades, and then level off and lose a modest amount of weight through the ninth decade. Although decline in muscle mass can be documented as early as the fourth or fifth decade, especially in sedentary adults, overt sarcopenia is seen in 5–10% of adults in their sixties, rising to over 50% in healthy, ambulatory, community-dwelling adults over age eighty (8). Of course, the onset of acute or chronic illness accelerates muscle loss above and beyond the age-related sarcopenia.

Although the prevalence and time course of sarcopenia is reasonably well worked out, the impact of obesity on it is only now emerging as an important public health problem. Ironically, although weight gain causes an increase in lean mass as well as fat mass, obesity in the elderly acts synergistically with sarcopenia to maximize disability. The “fat frail” have the worst of both worlds as they age—increased weakness due to sarcopenia and a need to carry greater weight due to obesity (9). With the increase in obesity that has occurred in the past two decades, the prevalence of obese elderly is rising, and the impact of sarcopenic obesity is likely to be dramatic in the next decade.

In this issue of the journal, Villareal et al. (10) provide one of the first systematic evaluations of sarcopenic obesity in contrast to both nonsarcopenic obese and lean adults of comparable age. The authors compared 52 obese elderly adults, 52 nonobese frail adults, and 52 nonobese, nonfrail persons, matched by age and sex. Despite a higher absolute amount of fat-free mass in the obese elders, these subjects had lower muscle quality (force per unit of cross-sectional muscle area) than the other two groups, and their functional performance, aerobic capacity, strength, balance, and walking speed were as severely reduced as the frail nonobese elders. Thus, these obese elderly adults had sarcopenia (low relative muscle mass, low muscle strength per muscle area) despite appearing the opposite of the stereotypical frail elderly adult.

What is the biological connection between sarcopenia and obesity? Are we simply seeing the results of decades of little physical activity and positive energy balance? In fact, there is good reason to suspect that sarcopenia and obesity reinforce each other at several levels, both behavioral and biological (11). As people age, their physical activity level falls, reducing the most important trophic effect on muscle while at the same time predisposing people to positive energy balance and weight gain, most of which is fat. Beyond this, however, loss of muscle reduces the mass of available insulin-responsive target tissue, promoting insulin resistance, which, in turn, promotes the metabolic syndrome and obesity (12). Moreover, increasing fat mass promotes production of tumor necrosis factor- α , interleukin-6, and other adipokines that further promote insulin resistance as well as potentially a direct catabolic effect on muscle. Thus, a vicious cycle is created that leads to more gain in fat and more loss of muscle, until a threshold is crossed at which functional consequences such as disability and illnesses such as hypertension and diabetes occur.

The results of the study by Villareal et al. suggest that sarcopenic obesity is a major public health problem in its own right. Until recently, the research communities studying each of these topics have had little contact with each other. Clearly, in the future more collaboration between those who study fat gain and those who study muscle loss

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will be needed if we are to solve the metabolic consequences at the conjunction of two epidemics.

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