

# The epidemiology of sarcopenia in community living older adults: what role does lifestyle play?

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## Abstract

**Background** Sarcopenia, the age-related decline in skeletal muscle mass and function, is a relatively poorly understood process which may play an important role in the incidence of physical disability and falls in older adults. Evidence demonstrates that both genetic and environmental factors contribute to increased susceptibility for sarcopenia development, yet some of these factors may represent unavoidable consequences of ageing.

**Methods** A review of literature, generally from epidemiological research, was performed to examine the influence that potentially modifiable lifestyle factors (general physical activity, dietary nutrient intake and sun exposure), as well as chronic disease and medication use, may have on sarcopenia progression.

**Results** The review demonstrated that while physical activity, nutrient intake and sun exposure often decline during ageing, each may have important but differing benefits for the prevention of muscle mass and functional declines in older adults. Conversely, age-related increases in the prevalence of chronic diseases and the subsequent prescription of pharmacotherapy may exacerbate sarcopenia progression.

**Conclusions** The prevalence of poor physical activity, diet and sun exposure, as well as chronic disease and medication use, within older adult populations may be modifiable through simple lifestyle and health care interventions. As such, these factors may represent the most effective targets for sarcopenia prevention during the ageing process.

**Keywords** Sarcopenia · Epidemiology · Physical activity · Diet · Vitamin D · Chronic disease

## 1 Defining sarcopenia

Irwin Rosenberg first noted that age-related declines in muscle mass are dramatic and have wide-ranging health outcomes [1], and proposed the descriptor ‘sarcopenia’ in 1988 [2]. In subsequent literature, the term has evolved to additionally describe age-related declines in muscle function [3], and recent recommendations for clinical definitions of sarcopenia promote combined measurement protocols, including measures of low walking speed [4–6]. However, an internationally accepted consensus definition of sarcopenia does not presently exist, in part due to conjecture regarding whether it is a disease state or process of normative ageing [7]. The present review analyses sarcopenia as a continuous and ubiquitous process impacting both skeletal muscle mass and function.

Symptoms of sarcopenia may develop as early as the fourth decade of life [8], but observed rates of progression vary by population and assessment techniques. Most longitudinal studies analyse changes in muscle strength rather than muscle mass, and results indicate that decreases in knee and elbow strength are in the range of 1–4% per year in older adult populations [9–12], but declines may be significantly greater for men than women [13], indicating

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sex-specific differences. Hand grip strength may decline at a faster rate in UK [14] than in US [15] older adults, and decreases in strength may also vary between muscle groups [16].

Prospective changes in thigh cross-sectional area (CSA) assessed by computed tomography reveal decreases of only 0.5–1.0% per year [10, 11]. Indeed, the Health, Aging, and Body Composition (Health ABC) study demonstrated that annual decreases in muscle strength are two- to fivefold greater than decreases in muscle CSA [12], and muscle mass changes explain as little as 5% of the variation in change in muscle strength in older adults [13, 17]. Changes in ‘muscle quality’, the amount of force produced per unit of muscle mass, may therefore explain much of the strength loss during ageing [18] and are possibly attributable to age-related neuromuscular changes, including reductions in muscle fibre size, number and contractility, as well as skeletal muscle fat infiltration [19]. Muscle quality decreased by 5–9% in 3 years in US adults aged 70–79 years [17], suggesting it may be a useful indicator of sarcopenia progression. Neuromuscular changes along with changes in body fat, systemic low-level inflammation, oxidative stress, protein metabolism, and endocrine activity contribute to sarcopenia, but may be a result of normative ageing and are therefore beyond the scope of this review. This review instead focuses on common lifestyle behaviours, chronic disease and medication use, all of which may be modifiable.

## 2 Lifestyle determinants of sarcopenia

The aetiology of sarcopenia is multifactorial, and its progression is generally attributed to age-related changes in skeletal muscle: an increased withdrawal of, or resistance to anabolic factors, and a concurrent increase in catabolic factors [20]. However, the heritability of muscle mass and strength may be as high as 50–60% [21–25], indicating that the development of sarcopenia is under significant genetic control. Indeed, a number of genes have already been identified as potential mediators of sarcopenia [26].

Additionally, early life influences may determine sarcopenia progression in later life. In the Hertfordshire Cohort Study (HCS), including nearly 3,000 UK older adults, birth weight and weight at 1 year positively predicted fat-free mass [27], muscle CSA [28], and hand grip strength [29], and lower infant growth was associated with a history of falls in men [30]. Birth weight was also positively associated with grip strength after adjustment for childhood and adult body size in a cohort of adults born in the UK in 1946 [31], and standing balance and chair rise ability was positively predicted by weight gain before the age of 7 years in men [32]. Low birth weight has also been

associated with low lean mass and hand grip strength in population-based studies of European older adults [33, 34], and early life experience of famine was associated with slower walking speed, shorter stride length, and recurrent falls in older adults in Hong Kong [35].

Life course studies therefore suggest that a modest but significant proportion of the origins of sarcopenia may be found in the prenatal and post-natal environment, and that declines in skeletal muscle mass and function during older age may be related to lifestyle behaviours during early development. It is likely that lifestyle choices during later life are also predictive of sarcopenia progression.

### 2.1 Physical activity

The effectiveness of resistance training interventions in reversing skeletal muscle mass and function declines indicative of sarcopenia progression has been demonstrated unequivocally [36]. However, general physical activity levels may be a more appropriate target from a public health standpoint, and it would appear logical that even general physical activity could hold benefits for skeletal muscle given that muscle contractions are associated with anabolic effects including increases in anti-inflammatory and antioxidative activity [37].

Studies have examined associations between general physical activity, muscle mass and strength in older adults, but its potential impact on sarcopenia prevention is unclear. A cross-sectional study of older French women revealed a positive association between self-reported habitual physical activity, leisure-time physical activity and maximal anaerobic power of the quadriceps [38], but a study of Dutch older adults reported no association between self-reported leisure-time physical activity and hand grip strength [39]. The New Mexico Aging Process study revealed positive associations between self-reported physical activity and muscle mass for older men and women [40] and relative appendicular lean mass (ALM) was greater with increased intensity of work activities, and in those who reported regular leisure-time exercise [41].

However, self-reported physical activity was not associated with ALM assessed by dual-energy X-ray absorptiometry (DXA) in over 300 older adults [42], and the US National Health and Nutrition Examination and the New Mexico Elder Health Surveys have provided conflicting findings regarding whether low self-reported physical activity is associated with a greater likelihood of being sarcopenic [43, 44]. Differences in cross-sectional associations are likely to be explained in some part by the use of different instruments for assessing self-reported physical activity, all of which have a substantial measurement error. Prospective studies of associations in older adults between self-reported physical activity, muscle mass, and function

are uncommon although higher levels of physical activity attenuated decline in thigh girth, but not knee extensor or flexor strength, in older adults over nearly 10 years [13, 45]. Physical disability has been associated with low self-reported physical activity in other prospective population-based studies [46].

Few studies of sarcopenia have objectively analysed physical activity levels. Ambulatory activity (AA) is the most popular form of leisure-time physical activity for older adults [47] and may be assessed by several objective measurement tools [48]. A significant association between strength of the triceps surae and total daily AA was assessed by pedometer in older men, but not women [49]. Pedometer-determined AA has been associated with lower adiposity in cross-sectional studies of older adults [50–52], and AA may indirectly prevent declines in skeletal muscle quality and function by reducing body fat levels [53].

The Tasmanian Older Adult Cohort (TASOAC) study was a prospective, population-based study of 1,100 community-dwelling older adults (mean age, 62 years). Baseline analyses revealed that pedometer-determined AA was negatively associated with total body fat and trunk fat mass in both sexes, and positively associated with leg strength and muscle quality in women only [54]. However, prospective analyses over 2.6 years demonstrated that habitual AA predicted declines in body fat for men, increased leg strength and muscle quality in women, and increased leg lean mass in both sexes [55]. These findings indicate that general physical activity may be of benefit in the prevention of sarcopenia, particularly through maintenance of desirable body composition, and there does not appear to be a threshold for this association.

## 2.2 Diet

In healthy adults, muscle constitutes over half of total body protein, but this decreases with age [56] due to greater rates of protein breakdown than synthesis. Daily energy intake declines by as much as 5,000 kJ in men and 3,500 kJ in women in their 80s, with substantial declines in intakes of nutrients, including protein [57]. Decreased dietary intakes of protein, as well as other nutrients, may contribute to sarcopenia progression.

Serum albumin is positively associated with DXA-assessed skeletal muscle mass in older adults [58], and in an Australian population-based study, participants who failed to meet recommendations for protein intake demonstrated significantly lower mean ALM [59]. However, several cross-sectional studies examining protein intake in older adults have reported no associations with muscle mass or strength [40, 60, 61]. Longitudinal studies have provided more support for the notion of a protective effect of dietary protein for sarcopenia; gains in mid-arm muscle

area were associated with higher self-reported baseline protein intake in a 4-year study of Chinese older adults [62], and those in the highest quintile for protein intake in the Health ABC Study had around 40% less decline in lean mass over 3 years than those in the lowest quintile [63]. Similarly, baseline protein intake was positively correlated with whole body lean mass, ALM and upper arm muscle area 5 years later in a study of Australian older women [64], and was a positive predictor of change in ALM over 3 years in Australian older men and women [59].

Literature examining the influence of non-protein dietary components on sarcopenia is less common. Carotenoids may be protective against oxidative stress, and subsequently, sarcopenia [65]. In Canadian adults aged 60–75 years, odds for sarcopenia were greater in those who reported failing to meet recommended dietary allowances for the antioxidants selenium and vitamins A, C and E [66], and in the HCS a positive association was observed for hand grip strength with  $\beta$ -carotene, selenium and vitamin C [67]. In the Women's Health and Aging Study (WHAS) of nearly 700 community-dwelling women aged 70–79 years, high plasma carotenoid and  $\alpha$ -tocopherol (a form of vitamin E) status were associated with reduced odds for low muscle strength [68] and frailty [69]. Over 3 years in this study, low plasma carotenoid status at baseline predicted development of frailty [70], walking disability [71, 72] and ADL disability [73]. Several examinations of carotenoid status have also been conducted within the InCHIANTI study. Objectively measured plasma  $\alpha$ -tocopherol and  $\gamma$ -tocopherol, and self-reported intakes of vitamin C and  $\beta$ -carotene, were positively associated with knee extension strength and physical performance score [74], and low plasma selenium intake was associated with higher risk of poor muscle strength [75]. A 6-year follow-up revealed that a higher baseline carotenoid status predicted a reduced risk of developing poor hand grip, knee and hip strength [76], and walking disability [77]. The TASOAC study, however, revealed no associations between vitamin C and E and prospective changes in ALM over 3 years in older adults although zinc, which may have antioxidant properties, was a positive predictor of change in ALM even after adjustment for protein intake [59].

Diets high in fruit and vegetables may also be beneficial due to increased potassium intake, which may reduce metabolic acidosis. Urinary potassium and potassium intakes have been positively associated with lean mass and muscle strength in older adults [78, 79] but not prospective changes in lean mass over 3 years [59, 78]. Magnesium may also be preventative of skeletal muscle declines by contributing to muscle adenosine triphosphate and cell structure. Serum magnesium concentrations were positively associated with grip strength, lower limb muscle power and lower limb extension strength in the InCHIANTI

study [80], and also in a small trial involving nonagenarians [79] and was also a positive predictor of change in ALM in a 3-year prospective study of older adults [59]. Evidence therefore suggests that several dietary components may be involved in sarcopenia progression.

### 2.3 Vitamin D

Vitamin D is a secosteroid hormone produced following ultraviolet B light exposure and also obtained in small amounts from some foods [81]. Vitamin D insufficiency and deficiency may be prevalent in older adults and particularly amongst those who live at higher latitudes [82]. Nuclear 1, 25 vitamin D receptors (VDRs) located in skeletal muscle may bind 1,25-dihydroxyvitamin D (1,25OHD<sub>3</sub>; the active form of vitamin D) and promote protein synthesis, and the apparent decrease in VDRs within the muscle during ageing may explain some of the age-related decline in protein synthesis [83]. Indeed, VDR polymorphisms have been associated with low fat-free mass in older men and muscle strength in older women [84, 85].

The majority of sarcopenia research in this area assesses 25-hydroxyvitamin D (25OHD). ALM relative to body height is significantly lower in older men with low 25OHD values [41], and the odds of losing more than 3% of muscle mass over 3 years were around two times greater in Longitudinal Aging Study Amsterdam (LASA) participants with low (<25 nmol/L) serum 25OHD levels, compared to participants with high (>50 nmol/L) levels at baseline [86]. Relative ALM was positively associated with 25OHD in cross-sectional analyses in TASSOAC, although not in longitudinal analyses [87]. Higher 25OHD levels have been also associated with decreased body sway [88] and improved balance [89], and this may partly explain the association of vitamin D with reduced falls, in older adults [90–92]. High levels of 25OHD are also associated with better performance in assessments of frailty [69, 93] and independence [94], gait speed [89], stair climbing ability [90], walking ability and sit-to-stand tests [95], and the Short Physical Performance Battery [96].

Whether vitamin D-associated improvements in physical performance in older adults can be attributed to increased skeletal muscle function is unclear. In a cross-sectional analysis of the Osteoporosis Prospective Risk Assessment study, positive associations were observed between 25OHD and thigh strength in women aged 75 years [89], and 25OHD was positively associated with knee and arm extension strength in Italian women aged 68–75 years [97]. Also, 25OHD was positively associated with hand grip strength in the InCHIANTI study [96], and in an examination of Norwegian older adults [90]. Cross-sectional analyses from an Australian population-based study demonstrated that participants with insufficient

25OHD had lower leg strength and muscle quality compared to those with normal 25OHD [87]. In contrast, a study of women with osteoporosis demonstrated no association between low 25OHD and trunk muscle strength [88], and in randomised samples of French older women no significant associations were observed for 25OHD with quadriceps or hand grip strength [98, 99].

Prospective studies suggest vitamin D may be important for muscle function, however. A 3-year follow-up analysis in the WHAS reported that women classified across varying 25OHD levels had similar changes in lower and upper extremity strength, and physical function [100], but women in the lowest quartile of 25OHD at baseline had around a 30% increased risk of developing frailty [70]. Also, in the LASA, the odds for a loss of grip strength greater than 40% over 3 years were around two times greater for participants with low serum 25OHD levels, than for those with high 25OHD at baseline [86], and in longitudinal analyses of TASSOAC, baseline 25OHD was a positive predictor of change in leg strength and muscle quality after adjustment for physical activity, sun exposure and season of blood sampling [87]. It appears that vitamin D may therefore play an important role in the maintenance of muscle function for older adults.

### 2.4 Chronic disease

Chronic disorders are highly prevalent in older adults [101], and declines in skeletal muscle mass and function may be an additional comorbid outcome of chronic disease. It has recently been suggested that skeletal muscle wasting directly attributable to chronic disease should be termed “myopenia”, with sarcopenia referring only to muscle declines associated with ageing [102]. However, a consensus is yet to be reached on this debate or the appropriate terminology, and so the following section examines chronic disease as a potential predictor of sarcopenia, rather than of myopenia.

Men with low muscle mass are significantly more likely to report lung and cardiovascular disease [40, 44], and chronic lung disease and atherosclerosis are also associated with low height-adjusted ALM [103]. Although stroke-associated disability is usually attributed to brain injury, the disability is also associated with muscle atrophy and neuromuscular changes [104]. Lean mass has been shown to be reduced in older adults with early Alzheimer’s disease [105], and poor psychological health also predicted a steeper decline in hand grip strength over 4 years in a population-based study of UK older adults [14]. Moreover, in older Japanese-American men, a steeper decline in grip strength after around 27 years was associated with prevalent stroke, diabetes, arthritis, coronary heart disease and chronic obstructive pulmonary disease [15].

Components of the musculoskeletal system are highly interrelated and common genetic factors may influence age-related skeletal muscle and bone changes [106]. Also, mechanical influences, particularly skeletal muscle strength, influence the control of bone loss during ageing [107]. In concert with the association between chronic disease and sarcopenia, this raises the potential of a synergistic relationship between muscle and skeletal components in which age-related bone disorders influence the development and progression of sarcopenia in older adults. Osteoarthritis (OA) represents a group of diseases which affect all components of the joints and results in symptoms including joint pain, tenderness and movement restriction [108]. OA prevalence increases markedly with age [109] and is a major cause of disability in older adults [110], and such disability may in part be explained by OA influences on declines in skeletal muscle function.

Older men with hip OA have significantly lower CSA of the thigh and pelvic muscles on the most severely affected hip compared to the opposite hip [111], and older women with knee OA have reduced lower limb lean mass compared to age- and sex-matched controls [112]. Research has demonstrated that reduced knee extension strength is a correlate of self-reported knee pain [113], and isometric quadriceps strength was lower for older adults with knee pain compared to those without, partly due to reduced muscle activation [114]. Also, increased odds of falling have been reported for older adults with self-reported OA [115].

It has been reported that quadriceps weakness may be present in patients demonstrating knee radiographic OA (ROA) but not reporting pain [116], suggesting that the early structural changes associated with OA may also be predictive of sarcopenia progression. Men with hip OA have demonstrated lower strength of the hip muscles compared to controls [111], and knee ROA has also been associated with increased self-reported stair climbing and walking disability [117]. However, in Chinese older adults, the significant association between quadriceps weakness and knee ROA was mediated by pain [118].

This finding is supported by the only prospective analyses we are aware of. In 480 adults over the age of 65, baseline knee ROA predicted a significant decline in performance of a stair climbing task over 30 months [119], although the investigators reported that the association was mediated by knee pain intensity and knee strength. Similarly, baseline knee pain (but not ROA) predicted a greater decline in leg strength and muscle quality, as well as a greater increase in objectively measured falls risk over almost 3 years, although these associations were observed for women only [120]. Thus, it is possible that OA and other age-related disorders exacerbate sarcopenia progression in older adults as a result of avoidance of activity due

to the pain experience and effective management of chronic disease and pain may represent an important target for sarcopenia prevention.

## 2.5 Medication use

Prevalence of prescription medication usage may be as high as 80% in community-dwelling older adults [121]. An increase in medication use over 10 years is associated with greater decline in knee strength in older men [13]. Hand grip strength significantly decreases [122], and self-reported falls increase, with higher self-reported medication use in older adults [123]. Older adults receiving hypnotics and antidepressants may in particular be at an increased risk for falling and disability [124]. Hypnotics, anxiolytics and antidepressants have been associated with increased odds of falls in British older women [125], and benzodiazepines have also been shown to be related to incident mobility and ADL disability [126]. A 1-year prospective study of Australian older women lends weight to the argument that such outcomes may be related to skeletal muscle function decline; in this study, the use of benzodiazepines and antidepressants was significantly associated with multiple falls and impaired lower limb muscle strength [127].

Cardiovascular medications may also influence skeletal muscle changes in older adults. Angiotensin-converting enzyme (ACE) inhibitors may be preventative of functional declines through improvements in the fatigability of the skeletal muscle [128], and in the WHAS, women who reported continued use of ACE inhibitors demonstrated lower declines in knee extensor strength and walking speed over 3 years compared to intermittent and non-users [129]. A cross-sectional analysis of the HCS, however, observed that while cardiovascular drugs, including furosemide, nitrates, calcium channel blockers and fibrates were associated with decreased hand grip strength, no associations were observed for ACE inhibitors [122].

Statins (or HMG-CoA reductase inhibitors) are the most widely used prescription medication [130] due to their protective effects for coronary events and total mortality [131]. Statins have relatively few side effects; the most common being myopathy including muscle pain, although rhabdomyolysis occurs in rare cases [132–134]. The mechanisms by which statin use may cause myopathy are unclear; however, pathways may include reductions in the production of sarcolemmal and sarcoplasmic reticular cholesterol, ubiquinones and coenzyme Q10 or isoprenoids and regulatory proteins [135, 136].

However, in a 1-year study of over 750 older adults, statin users actually demonstrated significantly better performance in a chair stand test than non-users [137]. Similarly, in adults with and without peripheral arterial disease (PAD), statin users demonstrated improved perfor-

mance in walking ability, balance, and chair rise tests [138], and amongst PAD patients statin use was again associated with improved performance in these tests after 3 years [139]. However, the incidence of frailty development was similar between statin users and controls after 3 years in US older women [140], and in the HCS, no associations were observed between statin use and hand grip strength [122]. High dose statin supplementation for older adults has resulted in no declines in aerobic or skeletal muscle performance [141].

Nevertheless, statin users may demonstrate increased muscle damage in response to exercise [142], and in a small clinical trial, patients were able to repeatedly distinguish periods of statin use from placebo use, and hip muscle strength tests demonstrated weakness during statin therapy. Skeletal muscle biopsies also indicated mitochondrial dysfunction which was reversed during subsequent placebo use [143]. Moreover, statin users in a 3-year study of older adults demonstrated greater decreases in leg strength and muscle quality, and a greater increase in falls risk than non-users, and this effect was reversed for those who reported cessation of statin use during the follow-up period [144]. Research to date is yet to clarify whether statin therapy contributes to functional decline; however, it is possible that older adults receiving some forms of pharmacotherapy are more at risk of disability due to increased rates of sarcopenia progression.

### 3 Summary

This review demonstrates that a number of modifiable lifestyle-related factors may be associated with skeletal muscle mass and function changes, but further prospective population-based studies are required to confirm these associations and examine other potential lifestyle behaviours which may contribute to sarcopenia.

Nevertheless, whilst factors that contribute to sarcopenia such as age-related body composition, inflammatory, metabolic and endocrine changes may be inevitable; a focus on improving lifestyle behaviours and overall health of older adults may represent the most effective means of reducing the impact of sarcopenia. Intervention studies will be important in demonstrating the potential for older adults to adopt and maintain lifestyle changes, as well as the magnitude of skeletal muscle mass and functional improvements that may be associated with these behaviours. In order to effectively design and implement interventions though, it is vital that practitioner and public awareness is increased and that consensus is achieved on the clinical definition of sarcopenia [145].

Such studies may include trialling of health promotion techniques to encourage adequate general activity and

nutrient intake in community-dwelling older adults, investigation of vitamin D supplementation as a therapeutic intervention for sarcopenia progression, investigation of effective pain control techniques and strengthening exercises for OA patients, and randomised-controlled trials to clarify the functional impact of statin use in older adults.

Sarcopenia is associated with a reduced ability to complete everyday tasks and an increased risk of falls, both of which may result in loss of independence in older adults. As western populations age, the social and economic burden associated with sarcopenia will increase. It is important that future studies continue to investigate both the aetiology of sarcopenia and its potential treatments, and promote these effectively to other researchers, practitioners and older adults themselves.

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**Conflict of interest statement** The authors declare that they have no conflict of interest.

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