J Cachexia Sarcopenia Muscle (2011) 2:125–134
DOI 10.1007/s13539-011-0036-4

REVIEW

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

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Received: 22 March 2011 / Accepted: 14 July 2011 / Published online: 28 July 2011

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

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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

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 β-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 α-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 α-tocopherol and γ-tocopherol, and self-reported intakes of vitamin C and β-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 ($1,25OHD_3$; 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 TASOAC, 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 TASOAC, 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.

Acknowledgements This work was supported by the National Health and Medical Research Council of Australia, Arthritis Foundation of Australia, Tasmanian Community Fund and the University of Tasmania Institutional Research Grants Scheme. The authors of this manuscript certify that they comply with the ethical guidelines for authorship and publishing in the *Journal of Cachexia, Sarcopenia and Muscle* [146].

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

References

1. Rosenberg IH. Summary comments. Am J Clin Nutr. 1989;50:1231–3.
2. Rosenberg IH. Sarcopenia: origins and clinical relevance. J Nutr. 1997;127:990–1.
3. Abellan Van Kan G. Epidemiology and consequences of sarcopenia. J Nutr Health Aging. 2009;13:708–12.
4. Cruz-Jentoft AJ, Baeyens JP, Bauer JM, Boirie Y, Cederholm T, Landi F, et al. Sarcopenia: European consensus on definition and diagnosis: report of the European Working Group on Sarcopenia in Older People. Age Ageing. 2010;39:412–23.
5. Fielding RA, Vellas B, Evans WJ, Bhasin S, Morley JE, Newman AB, et al. Sarcopenia: an undiagnosed condition in older adults. Current consensus definition: prevalence, etiology, and consequences. International Working Group on Sarcopenia. J Am Med Dir Assoc. 2011;12:249–56.
6. Muscaritoli M, Anker SD, Argilés J, Aversa Z, Bauer JM, Biolo G, et al. Consensus definition of sarcopenia, cachexia and pre-cachexia: joint document elaborated by Special Interest Groups (SIG) "cachexia-anorexia in chronic wasting diseases" and "nutrition in geriatrics". Clin Nutr. 2010;29:154–9.
7. Marcell TJ. Sarcopenia: causes, consequences, and preventions. J Gerontol A Biol Sci Med Sci. 2003;58:911–6.
8. Waters DL, Baumgartner RN, Garry PJ. Sarcopenia: current perspectives. J Nutr Health Aging. 2000;4:133–9.

9. Aniansson A, Hedberg M, Henning GB, Grimby G. Muscle morphology, enzymatic activity, and muscle strength in elderly men: a follow-up study. Muscle Nerve. 1986;9:585–91.
10. Frontera WR, Hughes VA, Fielding RA, Fiatarone MA, Evans WJ, Roubenoff R. Aging of skeletal muscle: a 12-yr longitudinal study. J Appl Physiol. 2000;88:1321–6.
11. Frontera WR, Reid KF, Phillips EM, Krivickas LS, Hughes VA, Roubenoff R, et al. Muscle fiber size and function in elderly humans: a longitudinal study. J Appl Physiol. 2008;105:637–42.
12. Delmonico MJ, Harris TB, Visser M, Park SW, Conroy MB, Velasquez-Mieyer P, et al. Longitudinal study of muscle strength, quality, and adipose tissue infiltration. Am J Clin Nutr. 2009;90:1579–85.
13. Hughes VA, Frontera WR, Wood M, Evans WJ, Dallal GE, Roubenoff R, et al. Longitudinal muscle strength changes in older adults: influence of muscle mass, physical activity, and health. J Gerontol A Biol Sci Med Sci. 2001;56:209–17.
14. Bassey EJ, Harries UJ. Normal values for handgrip strength in 920 men and women aged over 65 years, and longitudinal changes over 4 years in 620 survivors. Clin Sci. 1993;84:331–7.
15. Rantanen T, Masaki K, Foley D, Izmirlian G, White L, Guralnik JM. Grip strength changes over 27 yr in Japanese-American men. J Appl Physiol. 1998;85:2047–53.
16. Winegard KJ, Hicks AL, Sale DG, Vandervoort AA. A 12-year follow-up study of ankle muscle function in older adults. J Gerontol A Biol Sci Med Sci. 1996;51:202–7.
17. Goodpaster BH, Park SW, Harris TB, Kritchevsky SB, Nevitt M, Schwartz AV, et al. The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. J Gerontol A Biol Sci Med Sci. 2006;61:1059–64.
18. Dutta C, Hadley EC, Lexell J. Sarcopenia and physical performance in old age: overview. Muscle Nerve. 1997;5:5–9.
19. Stenholm S, Harris TB, Rantanen T, Visser M, Kritchevsky SB, Ferrucci L. Sarcopenic obesity—definition, etiology and consequences. Curr Opin Clin Nutr Metab Care. 2008;11:693–700.
20. Doherty TJ. Invited review: aging and sarcopenia. J Appl Physiol. 2003;95:1717–27.
21. Zhai G, Ding C, Stankovich J, Cicuttini F, Jones G. The genetic contribution to longitudinal changes in knee structure and muscle strength: a sibpair study. Arthritis Rheum. 2005;52:2830–4.
22. Arden NK, Spector TD. Genetic influences on muscle strength, lean body mass, and bone mineral density: a twin study. J Bone Miner Res. 1997;12:2076–81.
23. Tiainen K, Sipila S, Alen M, Heikkinen E, Kaprio J, Koskenvuo M, et al. Heritability of maximal isometric muscle strength in older female twins. J Appl Physiol. 2004;96:173–80.
24. Carmelli D, Kelly-Hayes M, Wolf PA, Swan GE, Jack LM, Reed T, et al. The contribution of genetic influences to measures of lower-extremity function in older male twins. J Gerontol A Biol Sci Med Sci. 2000;55:49–53.
25. Reed T, Fabsitz RR, Selby JV, Carmelli D. Genetic influences and grip strength norms in the NHLBI twin study males aged 59–69. Ann Hum Biol. 1991;18:425–32.
26. Huygens W, Thomis MAI, Peeters MW, Aerssens J, Vlietinck R, Beunen GP. Quantitative trait loci for human muscle strength: linkage analysis of myostatin pathway genes. Physiol Genomics. 2005;22:390–7.
27. Sayer AA, Syddall HE, Dennison EM, Gilbody HJ, Duggleby SL, Cooper C, et al. Birth weight, weight at 1 y of age, and body composition in older men: findings from the Hertfordshire Cohort Study. Am J Clin Nutr. 2004;80:199–203.
28. Sayer AA, Dennison EM, Syddall HE, Jameson K, Martin HJ, Cooper C. The developmental origins of sarcopenia: using peripheral quantitative computed tomography to assess muscle size in older people. J Gerontol A Biol Sci Med Sci. 2008;63:835–40.
29. Sayer AA, Syddall HE, Gilbody HJ, Dennison EM, Cooper C. Does sarcopenia originate in early life? Findings from the Hertfordshire Cohort Study. J Gerontol A Biol Sci Med Sci. 2004;59:930–4.
30. Sayer AA, Syddall HE, Martin HJ, Dennison EM, Anderson FH, Cooper C. Falls, sarcopenia, and growth in early life: findings from the Hertfordshire Cohort Study. Am J Epidemiol. 2006;164:665–71.
31. Kuh D, Bassey J, Hardy R, Sayer AA, Wadsworth M, Cooper C. Birth weight, childhood size, and muscle strength in adult life: evidence from a birth cohort study. Am J Epidemiol. 2002;156:627–33.
32. Kuh D, Hardy R, Butterworth S, Okell L, Richards M, Wadsworth M, et al. Developmental origins of midlife physical performance: evidence from a British birth cohort. Am J Epidemiol. 2006;164:110–21.
33. Gale CR, Martyn CN, Kellingray S, Eastell R, Cooper C. Intrauterine programming of adult body composition. J Clin Endocrinol Metab. 2001;86:267–72.
34. Ylihärsilä H, Kajantie E, Osmond C, Forsen T, Barker DJP, Eriksson JG. Birth size, adult body composition and muscle strength in later life. Int J Obes. 2007;31:1392–9.
35. Woo J, Leung JCS, Wong SYS. Impact of childhood experience of famine on late life health. J Nutr Health Aging. 2010;14:91–5.
36. Chodzko-Zajko WJ, Proctor DN, Fiatarone Singh M, Minson CT, Nigg CR, Salem GJ, et al. Exercise and physical activity for older adults. Med Sci Sports Exerc. 2009;41:1510–30.
37. Lenk K, Schuler G, Adams V. Skeletal muscle wasting in cachexia and sarcopenia: molecular pathophysiology and impact of exercise training. J Cachexia Sarcopenia Muscle. 2010;1:9–21.
38. Kostka T, Bonnefoy M, Arsac LM, Berthouze SE, Belli A, Lacour JR. Habitual physical activity and peak anaerobic power in elderly women. Eur J Appl Physiol. 1997;76:81–7.
39. van Heuvelen MJ, Kempen GI, Ormel J, Rispens P. Physical fitness related to age and physical activity in older persons. Med Sci Sports Exerc. 1998;30:434–41.
40. Baumgartner RN, Waters DL, Gallagher D, Morley JE, Garry PJ. Predictors of skeletal muscle mass in elderly men and women. Mech Ageing Dev. 1999;107:123–36.
41. Szulc P, Duboeuf F, Marchand F, Delmas PD. Hormonal and lifestyle determinants of appendicular skeletal muscle mass in men: the MINOS study. Am J Clin Nutr. 2004;80:496–503.
42. Iannuzzi-Sucich M, Prestwood KM, Kenny AM. Prevalence of sarcopenia and predictors of skeletal muscle mass in healthy, older men and women. J Gerontol A Biol Sci Med Sci. 2002;57:772–7.
43. Janssen I, Heymsfield SB, Ross R. Low relative skeletal muscle mass (sarcopenia) in older persons is associated with functional impairment and physical disability. J Am Geriatr Soc. 2002;50:889–96.
44. Baumgartner RN, Romero L, Garry PJ, Heymsfield SB, Koehler KM, Gallagher D, et al. Epidemiology of sarcopenia among the elderly in New Mexico. Am J Epidemiol. 1998;147:755–63.
45. Hughes VA, Frontera WR, Evans WJ, Roubenoff R, Wood M, Fiatarone Singh MA. Anthropometric assessment of 10-y changes in body composition in the elderly. Am J Clin Nutr. 2004;80:475–82.
46. DiPietro L. Physical activity in aging. J Gerontol A Biol Sci Med Sci. 2001;56:13–22.
47. Australian Sports Commission. Participation in Exercise, recreation and sport survey: 2008 annual report. Canberra: Australian Sports Commission; 2009.
48. Bassett DR. Validity and reliability issues in objective monitoring of physical activity. Res Q Exerc Sport. 2000;71:S30–6.
49. Bassey EJ, Bendall MJ, Pearson M. Muscle strength in the triceps surae and objectively measured customary walking

activity in men and women over 65 years of age. Clin Sci. 1988;74:85–9.
50. Tudor-Locke CE, Ainsworth BE, Whitt MC, Thompson RW, Addy CL, Jones DA. The relationship between pedometer-determined ambulatory activity and body composition variables. Int J Obes. 2001;25:1571–8.
51. Chan CB, Spangler E, Valcour J, Tudor-Locke CE. Cross-sectional relationship of pedometer-determined ambulatory activity to indicators of health. Obes Res. 2003;11:1563–70.
52. Thompson DL, Rakow J, Perdue SM. Relationship between accumulated walking and body composition in middle-aged women. Med Sci Sports Exerc. 2004;36:911–4.
53. Roubenoff R. Sarcopenic obesity: does muscle loss cause fat gain? Lessons from rheumatoid arthritis and osteoarthritis. Ann N Y Acad Sci. 2000;904:553–7.
54. Scott D, Blizzard L, Fell J, Jones G. Ambulatory activity, body composition, and lower-limb muscle strength in older adults. Med Sci Sports Exer. 2009;41:383–9.
55. Scott D, Blizzard L, Fell J, Jones G. Prospective associations between ambulatory activity, body composition and muscle function in older adults. Scand J Med Sci Sports. 2010. doi:10.1111/j.600-0838.2010.01229.x.
56. Proctor DN, O'Brien PC, Atkinson EJ, Nair KS. Comparison of techniques to estimate total body skeletal muscle mass in people of different age groups. Am J Physiol Endocrinol Metab. 1999;277:489–95.
57. Wakimoto P, Block G. Dietary intake, dietary patterns, and changes with age: an epidemiological perspective. J Gerontol A Biol Sci Med Sci. 2001;56:65–80.
58. Baumgartner RN, Koehler KM, Romero L, Garry PJ. Serum albumin is associated with skeletal muscle in elderly men and women. Am J Clin Nutr. 1996;64:552–8.
59. Scott D, Blizzard L, Fell J, Giles G, Jones G. Associations between dietary nutrient intake and muscle mass and strength in community-dwelling older adults: the Tasmanian Older Adult Cohort study. J Am Geriatr Soc. 2010;58:2129–34.
60. Mitchell D, Haan MN, Steinberg FM, Visser M. Body composition in the elderly: the influence of nutritional factors and physical activity. J Nutr Health Aging. 2003;7:130–9.
61. Starling RD, Ades PA, Poehlman ET. Physical activity, protein intake, and appendicular skeletal muscle mass in older men. Am J Clin Nutr. 1999;70:91–6.
62. Stookey JD, Adair L, Stevens J, Popkin BM. Patterns of long-term change in body composition are associated with diet, activity, income and urban residence among older adults in China. J Nutr. 2001;131:2433–40.
63. Houston DK, Nicklas BJ, Ding J, Harris TB, Tylavsky FA, Newman AB, et al. Dietary protein intake is associated with lean mass change in older, community-dwelling adults: the Health, Aging, and Body Composition (Health ABC) Study. Am J Clin Nutr. 2008;87:150–5.
64. Meng X, Zhu K, Devine A, Kerr DA, Binns CW, Prince RL. A 5-year cohort study of the effects of high protein intake on lean mass and bone mineral content in elderly postmenopausal women. J Bone Miner Res. 2009;24:1827–34.
65. Semba RD, Lauretani F, Ferrucci L. Carotenoids as protection against sarcopenia in older adults. Arch Biochem Biophys. 2007;458:141–5.
66. Chaput JP, Lord C, Cloutier M, Aubertin Leheudre M, Goulet ED, Rousseau S, et al. Relationship between antioxidant intakes and class I sarcopenia in elderly men and women. J Nutr Health Aging. 2007;11:363–9.
67. Robinson SM, Jameson KA, Batelaan SF, Martin HJ, Syddall HE, Dennison EM, et al. Diet and its relationship with grip strength in community-dwelling older men and women: the Hertfordshire Cohort Study. J Am Geriatr Soc. 2008;56:84–90.
68. Semba RD, Blaum C, Guralnik JM, Moncrief DT, Ricks MO, Fried LP. Carotenoid and vitamin E status are associated with indicators of sarcopenia among older women living in the community. Aging Clin Exp Res. 2003;15:482–7.
69. Michelon E, Blaum C, Semba RD, Xue Q-L, Ricks MO, Fried LP. Vitamin and carotenoid status in older women: associations with the frailty syndrome. J Gerontol A Biol Sci Med Sci. 2006;61:600–7.
70. Semba RD, Bartali B, Zhou J, Blaum C, Ko C-W, Fried LP. Low serum micronutrient concentrations predict frailty among older women living in the community. J Gerontol A Biol Sci Med Sci. 2006;61:594–9.
71. Semba RD, Varadhan R, Bartali B, Ferrucci L, Ricks MO, Blaum C, et al. Low serum carotenoids and development of severe walking disability among older women living in the community: the women's health and aging study I. Age Ageing. 2007;36:62–7.
72. Alipanah N, Varadhan R, Sun K, Ferrucci L, Fried LP, Semba RD. Low serum carotenoids are associated with a decline in walking speed in older women. J Nutr Health Aging. 2009;13:170–5.
73. Bartali B, Semba RD, Frongillo EA, Varadhan R, Ricks MO, Blaum CS, et al. Low micronutrient levels as a predictor of incident disability in older women. Arch Intern Med. 2006;166:2335–40.
74. Cesari M, Pahor M, Bartali B, Cherubini A, Penninx BWJH, Williams GR, et al. Antioxidants and physical performance in elderly persons: the Invecchiare in Chianti (InCHIANTI) study. Am J Clin Nutr. 2004;79:289–94.
75. Lauretani F, Semba RD, Bandinelli S, Ray AL, Guralnik JM, Ferrucci L. Association of low plasma selenium concentrations with poor muscle strength in older community-dwelling adults: the InCHIANTI Study. Am J Clin Nutr. 2007;86:347–52.
76. Lauretani F, Semba RD, Bandinelli S, Dayhoff-Brannigan M, Giacomini V, Corsi AM, et al. Low plasma carotenoids and skeletal muscle strength decline over 6 years. J Gerontol A Biol Sci Med Sci. 2008;63:376–83.
77. Lauretani F, Semba RD, Bandinelli S, Dayhoff-Brannigan M, Corsi AM, Guralnik JM, et al. Carotenoids as protection against disability in older persons. Rejuvenation Res. 2008;11:557–63.
78. Dawson-Hughes B, Harris SS, Ceglia L. Alkaline diets favor lean tissue mass in older adults. Am J Clin Nutr. 2008;87:662–5.
79. Fiatarone MA, Marks EC, Ryan ND, Meredith CN, Lipsitz LA, Evans WJ. High-intensity strength training in nonagenarians. Effects on skeletal muscle. J Am Med Assoc. 1990;263:3029–34.
80. Dominguez LJ, Barbagallo M, Lauretani F, Bandinelli S, Bos A, Corsi AM, et al. Magnesium and muscle performance in older persons: The InCHIANTI study. Am J Clin Nutr. 2006;84:419–26.
81. Adams JS, Hewison M. Update in vitamin D. J Clin Endocrinol Metab. 2010;95:471–8.
82. van der Mei IAF, Ponsonby AL, Engelsen O, Pasco JA, McGrath JJ, Eyles DW, et al. The high prevalence of vitamin D insufficiency across Australian populations is only partly explained by season and latitude. Environ Health Perspect. 2007;115:1132–9.
83. Bischoff-Ferrari HA, Borchers M, Gudat F, Dürmüller U, Stähelin HB, Dick W. Vitamin D receptor expression in human muscle tissue decreases with age. J Bone Miner Res. 2004;19:265–9.
84. Roth SM, Zmuda JM, Cauley JA, Shea PR, Ferrell RE. Vitamin D receptor genotype is asscociated with fat-free mass and sarcopenia in elderly men. J Gerontol A Biol Sci Med Sci. 2004;59:10–5.
85. Geusens P, Vandevyver C, Vanhoof J, Cassiman JJ, Boonen S, Raus J. Quadriceps and grip strength are related to vitamin D

receptor genotype in elderly nonobese women. J Bone Miner Res. 1997;12:2082–8.
86. Visser M, Deeg DJH, Lips P. Low vitamin D and high parathyroid hormone levels as determinants of loss of muscle strength and muscle mass (sarcopenia): the longitudinal aging study Amsterdam. J Clin Endocrinol Metab. 2003;88:5766–72.
87. Scott D, Blizzard L, Fell J, Ding C, Winzenberg T, Jones G. A prospective study of the associations between 25-hydroxyvitamin D, sarcopenia progression, and physical activity in older adults. Clin Endocrinol (Oxf). 2010;73:581–7.
88. Pfeifer M, Begerow B, Minne HW, Schlotthauer T, Pospeschill M, Scholz M, et al. Vitamin D status, trunk muscle strength, body sway, falls, and fractures among 237 postmenopausal women with osteoporosis. Exp Clin Endocrinol Diabetes. 2001;109:87–92.
89. Gerdhem P, Ringsberg KAM, Obrant KJ, Akesson K. Association between 25-hydroxy vitamin D levels, physical activity, muscle strength and fractures in the prospective population-based OPRA Study of Elderly Women. Osteoporos Int. 2005;16:1425–31.
90. Mowe M, Haug E, Bøhmer T. Low serum calcidiol concentration in older adults with reduced muscular function. J Am Geriatr Soc. 1999;47:220–6.
91. Flicker L, Mead K, MacInnis RJ, Nowson C, Scherer S, Stein MS, et al. Serum vitamin D and falls in older women in residential care in Australia. J Am Geriatr Soc. 2003;51:1533–8.
92. Snijder MB, van Schoor NM, Pluijm SMF, van Dam RM, Visser M, Lips P. Vitamin D status in relation to one-year risk of recurrent falling in older men and women. J Clin Endocrinol Metab. 2006;91:2980–5.
93. Shardell M, Hicks GE, Miller RR, Kritchevsky S, Andersen D, Bandinelli S, et al. Association of low vitamin D levels with the frailty syndrome in men and women. J Gerontol A Biol Sci Med Sci. 2009;64:69–75.
94. Visser M, Deeg DJH, Puts MTE, Seidell JC, Lips P. Low serum concentrations of 25-hydroxyvitamin D in older persons and the risk of nursing home admission. Am J Clin Nutr. 2006;84:616–22.
95. Bischoff-Ferrari HA, Dietrich T, Orav EJ, Hu FB, Zhang Y, Karlson EW, et al. Higher 25-hydroxyvitamin D concentrations are associated with better lower-extremity function in both active and inactive persons aged > or =60 y. Am J Clin Nutr. 2004;80:752–8.
96. Houston DK, Cesari M, Ferrucci L, Cherubini A, Maggio D, Bartali B, et al. Association between vitamin D status and physical performance: the InCHIANTI Study. J Gerontol A Biol Sci Med Sci. 2007;62:440–6.
97. Zamboni M, Zoico E, Tosoni P, Zivelonghi A, Bortolani A, Maggi S, et al. Relation between vitamin D, physical performance, and disability in elderly persons. J Gerontol A Biol Sci Med Sci. 2002;57:7–11.
98. Annweiler C, Beauchet O, Berrut G, Fantino B, Bonnefoy M, Herrmann FR, et al. Is there an association between serum 25-hydroxyvitamin D concentration and muscle strength among older women? Results from baseline assessment of the EPIDOS study. J Nutr Health Aging. 2009;13:90–5.
99. Annweiler C, Schott AM, Berrut G, Kressig RW, Herrmann FR, Beauchet O. Vitamin D deficiency-related quadriceps weakness: results of the EPIDOS cohort. J Am Geriatr Soc. 2009;57:368–9.
100. Verreault R, Semba RD, Volpato S, Ferrucci L, Fried LP, Guralnik JM. Low serum vitamin D does not predict new disability or loss of muscle strength in older women. J Am Geriatr Soc. 2002;50:912–7.
101. Wolff JL, Starfield B, Anderson G. Prevalence, expenditures, and complications of multiple chronic conditions in the elderly. Arch Intern Med. 2002;162:2269–76.
102. Fearon K, Evans W, Anker S. Myopenia—a new universal term for muscle wasting. J Cachexia Sarcopenia Muscle. 2011;2:1–3.
103. Lee JSW, Auyeung TW, Kwok T, Lau EMC, Leung PC, Woo J. Associated factors and health impact of sarcopenia in older Chinese men and women: a cross-sectional study. Gerontology. 2007;53:404–10.
104. Scherbakov N, Doehner W. Sarcopenia in stroke—facts and numbers on muscle loss accounting for disability after stroke. J Cachexia Sarcopenia Muscle. 2011;2:5–8.
105. Burns JM, Johnson DK, Watts A, Swerdlow RH, Brooks WM. Reduced lean mass in early Alzheimer disease and its association with brain atrophy. Arch Neurol. 2010;67:428–33.
106. Karasik D, Kiel DP. Evidence for pleiotropic factors in genetics of the musculoskeletal system. Bone. 2010;46:1226–37.
107. Burr DB. Muscle strength, bone mass, and age-related bone loss. J Bone Miner Res. 1997;12:1547–51.
108. Brandt KD, Dieppe P, Radin EL. Etiopathogenesis of osteoarthritis. Rheum Dis Clin North Am. 2008;34:531–59.
109. March LM, Bagga H. Epidemiology of osteoarthritis in Australia. Med J Aust. 2004;180:S6–S10.
110. March LM, Bachmeier CJ. Economics of osteoarthritis: a global perspective. Baillieres Clin Rheumatol. 1997;11:817–34.
111. Arokoski MH, Arokoski JP, Haara M, Kankaanpaa M, Vesterinen M, Niemitukia LH, et al. Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. J Rheumatol. 2002;29:2185–95.
112. Toda Y, Segal N, Toda T, Kato A, Toda F. A decline in lower extremity lean body mass per body weight is characteristic of women with early phase osteoarthritis of the knee. J Rheumatol. 2000;27:2449–54.
113. Zhai G, Blizzard L, Srikanth V, Ding C, Cooley H, Cicuttini F, et al. Correlates of knee pain in older adults: Tasmanian older adult Cohort study. Arthritis Rheum. 2006;55:264–71.
114. O'Reilly SC, Jones A, Muir KR, Doherty M. Quadriceps weakness in knee osteoarthritis: the effect on pain and disability. Ann Rheum Dis. 1998;57:588–94.
115. Nevitt MC, Cummings SR, Kidd S, Black D. Risk factors for recurrent nonsyncopal falls: a prospective study. J Am Med Assoc. 1989;261:2663–8.
116. Slemenda C, Brandt KD, Heilman DK, Mazzuca S, Braunstein EM, Katz BP, et al. Quadriceps weakness and osteoarthritis of the knee. Ann Intern Med. 1997;127:97–104.
117. Guccione AA, Felson DT, Anderson JJ. Defining arthritis and measuring functional status in elders: methodological issues in the study of disease and physical disability. Am J Public Health. 1990;80:945–9.
118. Baker KR, Xu L, Zhang Y, Nevitt M, Niu J, Aliabadi P, et al. Quadriceps weakness and its relationship to tibiofemoral and patellofemoral knee osteoarthritis in Chinese: the Beijing osteoarthritis study. Arthritis Care Res. 2004;50:1815–21.
119. Miller ME, Rejeski WJ, Messier SP, Loeser RF. Modifiers of change in physical functioning in older adults with knee pain: the Observational Arthritis Study in Seniors (OASIS). Arthritis Rheum. 2001;45:331–9.
120. Scott D, Blizzard L, Fell J, Jones G. A prospective study of the associations between self-reported pain, radiographic osteoarthritis and sarcopenia in community-dwelling older adults. Arthritis Care Res 2011; (in press).
121. Kaufman DW, Kelly JP, Rosenberg L, Anderson TE, Mitchell AA. Recent patterns of medication use in the ambulatory adult population of the United States: the slone survey. J Am Med Assoc. 2002;287:337–44.
122. Ashfield TA, Syddall HE, Martin HJ, Dennison EM, Cooper C, Aihie Sayer A. Grip strength and cardiovascular drug use in older people: findings from the Hertfordshire Cohort Study. Age Ageing. 2010;39:185–91.

123. Campbell AJ, Borrie MJ, Spears GF. Risk factors for falls in a community-based prospective study of people 70 years and older. J Gerontol. 1989;44:M112–7.
124. Blake AJ, Morgan K, Bendall MJ, Dallosso H, Ebrahim SBJ, Arie THD, et al. Falls by elderly people at home: prevalence and associated factors. Age Ageing. 1988;17:365–72.
125. Lawlor DA, Patel R, Ebrahim S. Association between falls in elderly women and chronic diseases and drug use: cross sectional study. Br Med J. 2003;327:712–7.
126. Gray SL, LaCroix AZ, Hanlon JT, Penninx B, Blough DK, Leveille SG, et al. Benzodiazepine use and physical disability in community-dwelling older adults. J Am Geriatr Soc. 2006;54:224–30.
127. Lord SR, Anstey KJ, Williams P, Ward JA. Psychoactive medication use, sensori-motor function and falls in older women. Br J Clin Pharmacol. 1995;39:227–34.
128. Vescovo G, Dalla Libera L, Serafini F, Leprotti C, Facchin L, Volterrani M, et al. Improved exercise tolerance after losartan and enalapril in heart failure: correlation with changes in skeletal muscle myosin heavy chain composition. Circulation. 1998;98:1742–9.
129. Onder G, Penninx B, Balkrishnan R, Fried LP, Chaves PHM, Williamson J, et al. Relation between use of angiotensin-converting enzyme inhibitors and muscle strength and physical function in older women: an observational study. Lancet. 2002;359:926–30.
130. Qato DM, Alexander GC, Conti RM, Johnson M, Schumm P, Lindau ST. Use of prescription and over-the-counter medications and dietary supplements among older adults in the United States. J Am Med Assoc. 2008;300:2867–78.
131. ATPIII. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. J Am Med Assoc. 2001;285:2486–97.
132. Bruckert E, Hayem G, Dejager S, Yau C, Begaud B. Mild to moderate muscular symptoms with high-dosage statin therapy in hyperlipidemic patients—the PRIMO study. Cardiovasc Drugs Ther. 2005;19:403–14.
133. Thompson PD, Clarkson PM, Rosenson RS. An assessment of statin safety by muscle experts. Am J Cardiol. 2006;97:S69–76.
134. Graham DJ, Staffa JA, Shatin D, Andrade SE, Schech SD, La Grenade L, et al. Incidence of hospitalized rhabdomyolysis in patients treated with lipid-lowering drugs. J Am Med Assoc. 2004;292:2585–90.
135. Thompson PD, Clarkson P, Karas RH. Statin-associated myopathy. J Am Med Assoc. 2003;289:1681–90.
136. Krishnan GM, Thompson PD. The effects of statins on skeletal muscle strength and exercise performance. Curr Opin Lipidol. 2010;21:324–8.
137. Agostini JV, Tinetti ME, Han L, McAvay G, Foody JM, Concato J. Effects of statin use on muscle strength, cognition, and depressive symptoms in older adults. J Am Geriatr Soc. 2007;55:420–5.
138. McDermott MM, Guralnik JM, Greenland P, Pearce WH, Criqui MH, Liu K, et al. Statin use and leg functioning in patients with and without lower-extremity peripheral arterial disease. Circulation. 2003;107:757–61.
139. Giri J, McDermott MM, Greenland P, Guralnik JM, Criqui MH, Liu K, et al. Statin use and functional decline in patients with and without peripheral arterial disease. J Am Coll Cardiol. 2006;47:998–1004.
140. LaCroix AZ, Gray SL, Aragaki A, Cochrane BB, Newman AB, Kooperberg CL, et al. Statin use and incident frailty in women aged 65 years or older: prospective findings from the women's health initiative observational study. J Gerontol A Biol Sci Med Sci. 2008;63:369–75.
141. Traustadóttir T, Stock AA, Harman SM. High-dose statin use does not impair aerobic capacity or skeletal muscle function in older adults. Age. 2008;30:283–91.
142. Thompson PD, Zmuda JM, Domalik LJ, Zimet RJ, Staggers J, Guyton JR. Lovastatin increases exercise-induced skeletal muscle injury. Metabolism. 1997;46:1206–10.
143. Phillips PS, Haas RH, Bannykh S, Hathaway S, Gray NL, Kimura BJ, et al. Statin-associated myopathy with normal creatine kinase levels. Ann Intern Med. 2002;137:581–5.
144. Scott D, Blizzard L, Fell J, Jones G. Statin therapy, muscle function and falls risk in community-dwelling older adults. QJM. 2009;102:625–33.
145. von Haehling S, Morley J, Anker S. An overview of sarcopenia: facts and numbers on prevalence and clinical impact. J Cachexia Sarcopenia Muscle. 2010;1:129–33.
146. von Haehling S, Morley JE, Coats AJS, Anker SD. Ethical guidelines for authorship and publishing in the Journal of Cachexia, Sarcopenia and Muscle. J Cachexia Sarcopenia Muscle. 2010;1:7–8.