

REVIEW PAPER

A Minimalistic Approach to Promote Health-Span via Bouts of Daily Physical Activity in Older Adults: A Review

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Abstract

While increasing human life expectancy is a significant societal achievement, it often contrasts with a shorter health-span, leading to a higher incidence of chronic diseases, particularly in older adults. Physical activity is crucial for mitigating age-related musculoskeletal decline, yet participation in structured and vigorous exercise among older adults is low. This review aims to provide an overview of the impact of biological aging on the musculoskeletal system and to highlight countermeasures, including viable exercise-related strategies. The review found that biological aging contributes to sarcopenia (age-related loss of muscle mass and quality), sarcopenic obesity (reduced muscle mass with elevated fat mass), dynapenia (age-related loss of muscle strength), and declines in aerobic capacity, alongside changes in appetite regulation and energy expenditure. Importantly, moderate-to-vigorous physical activity (MVPA) is crucial for maintaining muscle mass and preventing sarcopenia. Achieving daily step counts of approximately 6,000-8,000 for adults over 60, or specifically around 8,000 steps for men and 6,900 for women, and/or 15-20 minutes of MVPA daily, appears adequate for preventing sarcopenia. Replacing sedentary time with even small amounts of MVPA, such as 15 minutes daily, can reduce sarcopenia risk by about 15%, with greater benefits seen with longer durations like 60 minutes of MVPA. In conclusion, while structured resistance and endurance training are highly recommended, older adults who find adherence challenging can still enhance their health-span by aiming for daily step targets or incorporating approximately 30 minutes of daily MVPA. This minimalistic approach can help preserve muscle mass, limit fat accumulation, and counteract the detrimental effects of biological aging on the musculoskeletal system.

Keywords: *health span, sarcopenia, sarcopenic obesity, physical activity*

Introduction

Recent history has seen a notable rise in human life expectancy, a metric representing the average lifespan within a population. This trend has been consistent in developed nations for the last two centuries, yet it surpassed projected global limits of 71 years for males and 74 years for females, thereby maintaining its trajectory (Ismail et al., 2021; Oeppen et al., 2002). Although this sustained increase in

life expectancy within developed countries was not anticipated, understanding the factors driving these aging demographic shifts is crucial. Indeed, enhancements in living standards, readily available resources like accessible clean drinking water, improved sanitation, medical progress (such as antibiotic use), and education correlate with reduced mortality rates in early and mid-life stages, thus contributing to the extension of life expectancy (Mitchell



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et al., 2020). Furthermore, advancements in socioeconomic conditions, healthcare accessibility and quality, public health initiatives, and social support systems have collectively improved the quality of life across the entire lifespan (Marzo et al., 2023). Consequently, projections indicate that by 2050, the populations aged over 65, 85, and 100 years will grow by 188%, 551%, and 1004%, respectively (Oksuzyan et al., 2020). In fact, the cohort aged 60 and above has already doubled over the past four decades, now numbering 810 million globally. This demographic trend is anticipated to continue, with the aging population expected to approach approximately 2 billion by the year 2050 (Oksuzyan et al., 2020). Global disparities in life expectancy, along with associated sex-based differences in mortality and morbidity risks, represent significant areas of focus for academia and policy, aiming to promote more equitable and healthy lives for everyone. While this significant extension of life expectancy among aging populations signifies societal progress, this increased longevity does not invariably translate to a proportionately extended health-span (Olshansky, 2018). The former denotes the total duration of life lived, whereas the latter refers to the period experienced free from significant disease. In reality, lifespan and health-span seldom align perfectly. For instance, Garmany and colleagues (2021), comparing 2020 median probabilistic projections for life expectancy and health-adjusted life expectancy, identified a 9.2-year gap between health-span and lifespan. This chronological discrepancy between health-span and lifespan often manifests as non-communicable diseases affecting various biological systems, including the cardiovascular, pulmonary, endocrine, and musculoskeletal systems, and ultimately arises from unhealthy biological aging processes (DiLoreto et al., 2015). For these reasons, the World Health Organization (WHO) has designated healthy biological aging as a research priority for the period between 2016 and 2030 (Keating, 2022). To enhance health-span and promote optimal body mass, the WHO advises a minimum of 300 minutes of physical activity (PA) per week for adults aged 18 to 64 (Bull et al., 2020). This recommendation stems from the fact that PA—any bodily movement generated by skeletal muscles—is the most significant regulator of skeletal muscle plasticity in both younger and older adult populations (Escriche-Escuder et al., 2021). However, for older adults (65+ years), the WHO places increased emphasis on more structured and vigorous forms of PA to prevent sarcopenia and sarcopenic obesity (Bull et al., 2020). Vigorous PA modalities can induce distinct physiological adaptations depending on the type of exercise (Escriche-Escuder et al., 2021). For example, resistance exercise training is considered the most effective non-pharmacological intervention for promoting skeletal muscle hypertrophy, a condition where muscle protein synthesis consistently surpasses muscle protein breakdown, leading to an increased cross-sectional area of the muscle (Damas et al., 2015). This form of training is associated with improvements in muscle pennation angle, neural rate coding, coordination, and increased bone mineral density (Kraemer et al., 2002). Conversely, endurance exercise training enhances muscle capillarization, mitochondrial density, and consequently, maximal oxygen uptake (VO₂max) (Hughes et al., 2018). It is thus understandable that the WHO recommends

combining resistance and endurance training modalities for older adults. Nevertheless, despite these clear health advantages, population-level participation in these recommended exercise forms among older adults (65+ years) worldwide remains low, at only 16% (Elgaddal et al., 2022). Therefore, the primary objective of this review is to provide an overview of the impact biological aging can have on the musculoskeletal system and to highlight countermeasures as well as viable exercise-related strategies

Hallmarks of Biological Ageing

From a biological perspective, aging can be described as senescence, signifying a progressive, cumulative decline in an organism's regenerative capacity and bioprotective functions (Hernandez-Segura et al., 2018). Key initiators of cellular damage, and consequently biological aging, include genomic instability, the shortening of telomeres, epigenetic modifications, and impaired proteostasis. These fundamental processes subsequently trigger various cellular responses, including disruptions in intracellular communication, depletion of stem cell populations, induction of cellular senescence, mitochondrial impairment, and aberrant nutrient sensing pathways (Aubert et al., 2008; Fafián-Labora et al., 2020; Roger et al., 2021). However, inflammation represents a standard physiological reaction to cellular damage, facilitating tissue repair following endogenous or exogenous insults (Weavers et al., 2020). While this acute inflammatory response is typically beneficial and aids healing, persistent low-grade inflammation frequently remains undetected yet can detrimentally affect specific tissues and organs, thereby contributing to the continuation of chronic diseases and debilitating conditions (Minihane et al., 2015). Both aging and obesity are linked to this state of chronic low-grade inflammation, a condition also recognized in various non-communicable diseases like arthritis, type 2 diabetes, and cancer (Adler, 2011). Notably, the elevation in pro-inflammatory markers (cytokines) driven by obesity originates from enlarged adipocytes and immune cells residing within adipose tissue, predominantly lymphocytes and macrophages (Moss et al., 2023). Nevertheless, even a modest body mass reduction of just 5% has demonstrated efficacy in mitigating obesity-associated chronic low-grade inflammation in both human and murine models (Ellulu et al., 2017). In this context, reducing fat mass (FM) is achievable through establishing a negative energy balance, a condition where energy expenditure surpasses energy intake over extended durations, leading to intentional weight reduction (Kim, 2021).

Although inflammation linked to obesity can be lessened via weight reduction, biological aging itself is an unavoidable process and is associated with a persistent age-related chronic low-grade inflammation, often termed 'inflamm-ageing' (Franceschi et al., 2000). Sustained and unresolved inflamm-ageing can subsequently diminish brain plasticity, degrade skeletal muscle quality and function, and promote the accumulation of visceral and ectopic fat deposits, which themselves are pro-inflammatory (Cianciulli et al., 2022). Collectively, these changes result in an elevated probability of physical disability, frailty, and metabolic disorders, ultimately impairing the quality of life for older adults (Figure 1) (Netuveli et al., 2008; Vanleerberghe et al., 2017).

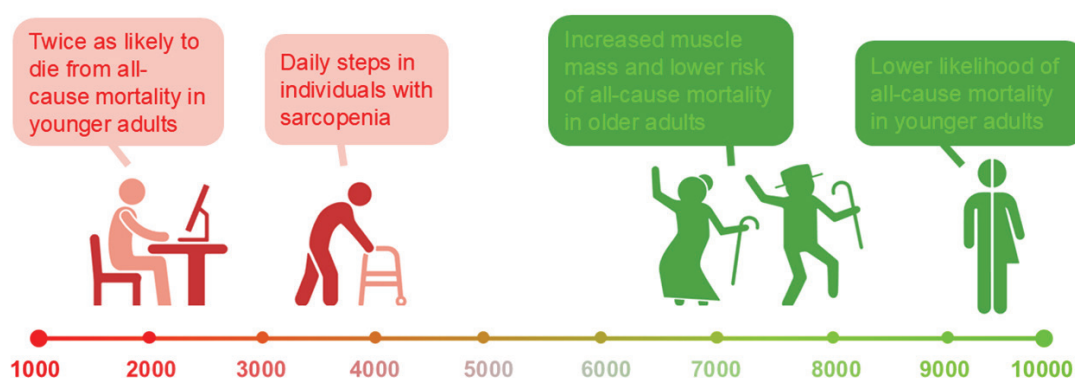


FIGURE 1. Markers of biological aging resulting in reduced health-span

Sarcopenia and Changes in Lean Body Mass

The adverse consequences of biological aging can manifest surprisingly early, potentially beginning in the second decade of life, and impacting various physiological systems such as the cardiovascular and neuromuscular systems, along with the brain (Netuveli et al., 2008; Vanleerberghe et al., 2017). A prime example is the age-associated decline in both the amount and functionality of skeletal muscle mass, a condition known as sarcopenia (Larsson et al., 2019). Sarcopenia's onset can also occur early, leading to a reduction in skeletal muscle mass averaging 0.47% annually in men and 0.37% in women, rates which subsequently increase to 0.98% for men and 0.70% for women beyond the age of 75 (Berger et al., 2010; Marzetti et al., 2017). Additionally, owing to the significant correlation between skeletal muscle mass and bone mineral density, sarcopenia frequently coexists with osteopenia, defined as a decrease in bone mineral density that typically precedes the development of osteoporosis (Lee et al., 2016). As a result of sarcopenia, individuals may lose close to 30% of their peak skeletal muscle mass by their 80th year (Marzetti et al., 2017). This loss is especially significant given that skeletal muscle comprises approximately 40% of total body mass and plays vital roles in movement, postural support, and respiration, besides serving as a primary location for nutrient metabolism and storage (McLeod et al., 2016).

Lean body mass (LBM), encompassing organs like the kidneys, heart, liver, brain, and skeletal muscle, represents a highly metabolically active tissue compartment (Wang et al., 2010). While all these LBM constituents are susceptible to biological aging, skeletal muscle constitutes 70-90% of total LBM and is particularly affected by sarcopenia (Larson et al., 2019). Consequently, alterations in LBM may act as an indicator, reflecting the progression of sarcopenia throughout the lifespan. For example, research by Westertep and colleagues (2021) indicated that LBM reaches its peak between 20-24 years in males and 19-20 years in females, subsequently remaining stable until about age 30. Likewise, Jackson and colleagues (2012) observed a stable LBM period from age 20 to 47, followed by reported annual decreases escalating from 0.42 kg in the fifth decade to 1.96 kg in the eighth decade. Findings from Guo and colleagues (1999) also suggest that LBM decreases become notable in both sexes between the ages of 40 and 50. Investigating LBM variations across healthy individuals aged 18 to 94, Kyle and colleagues (2001) identified the onset of decline around the age of 60.

During aging, women typically experience smaller absolute and relative decreases in skeletal muscle mass compared to men, which largely accounts for the observed sex differences in LBM trajectories (Churchward-Venne et al., 2015). It is noteworthy that women possess approximately 40% less muscle mass in the upper body and 30% less in the lower body compared to men (Janssen et al., 2000). Research by Visser and colleagues (1997) documented that over a 50-year span (ages 20-70), total appendicular skeletal muscle mass loss amounted to 4 kg (~15%) in men and 2 kg (~11%) in women. Another longitudinal investigation by Gallagher and colleagues (1997) reported absolute appendicular skeletal muscle mass reductions of approximately 1 kg in men and 0.6 kg in women across a 7-year timeframe. Furthermore, research examining muscle atrophy rates in older populations indicated slower muscle mass reduction in women relative to men over a 2-year observation period (Zamboni et al., 2003).

Sarcopenic Obesity and Changes in Fat Mass

Declines in skeletal muscle quantity and functionality can lead to improper nutrient partitioning, manifesting as increased fat infiltration within skeletal muscle and the buildup of visceral and ectopic adipose tissue, conditions associated with insulin resistance, dyslipidemia, and specific types of cancer (Larsson et al., 2019). The coexistence of reduced skeletal muscle mass and elevated fat mass (FM) in later life defines sarcopenic obesity, a condition estimated to impact 4.1% of males and 2.65% of females aged 18 to 93 years (Frontera et al., 2015).

Regarding sarcopenic obesity, cross-sectional data suggest that FM expands annually by 1% in both sexes starting from the fourth decade of life (Churchward-Venne et al., 2015; Janssen et al., 2000; Kuk et al., 2009; Kyle et al., 2001). Conversely, one longitudinal investigation found that FM begins to increase from age 40, reaching a peak around age 60 in both males and females, with average yearly gains of 0.37 kg and 0.41 kg, respectively (Guo et al., 1999). A longitudinal study by Visser and colleagues (2003) identified peak FM occurring between the ages of 68 and 80 in both sexes. Comparable results emerged from a longitudinal study by Zamboni et al. (2003), where researchers also observed peak FM levels between 68 and 80 years for both men and women. These observations align with several cross-sectional investigations indicating that peak fat mass occurs between ages 60 and 76 in both male and female participants

(Churchward-Venne et al., 2015; Janssen et al., 2000; Kyle et al., 2001). Further research examining fat distribution patterns in older adults indicates that aging can foster altered fat deposition, resulting in excessive fat accumulation, particularly as visceral fat in the abdominal region (Hirose et al., 2016; Huffman et al., 2009). Indeed, a diagnosis of sarcopenic obesity may be considered when waist circumference surpasses 90 cm for men and 85 cm for women (Barazzoni et al., 2018). Notably, aging correlates with an average yearly increase in waist circumference of about 0.7 cm (Noppa et al., 1980). For instance, longitudinal data from Hughes and colleagues (2019) show that older women experienced an average waist circumference gain of 4 cm over nine years, whereas older men showed a non-significant increase of 1 cm during the same timeframe. Comparable results were reported in another study, with older women exhibiting larger increases in waist circumference over five years than men (+2.8 cm versus -0.2 cm, respectively) (Hairston et al., 2009). These longitudinal findings are corroborated by cross-sectional studies where older women demonstrated greater age-related waist circumference increases compared to their male counterparts (Sakai et al., 2005; Singh et al., 2019). Collectively, these studies suggest that aging women exhibit a greater tendency towards fat mass accretion, particularly the deposition of visceral fat in the abdominal area.

Indirect Markers of Ageing

Muscle strength

Independent of sarcopenia or sarcopenic obesity, the age-associated reduction in muscle strength can further diminish quality of life by negatively impacting personal mobility and overall neuromuscular function. This decline in muscle strength, occurring independently of overt disease, is specifically referred to as dynapenia (Clark et al., 2012). Indeed, the diminution of muscle strength can commence as early as the third decade, exhibiting an average annual decrease of 1.5% between ages 50 and 60, which accelerates to 3–5% per year between ages 60 and 80 (Keller et al., 2014). Generally, muscle strength is commonly evaluated via handgrip dynamometry, with values below 27 kg often considered indicative of sarcopenia and dynapenia (Cruz-Jenoff et al., 2019). The reduction in strength is primarily attributed to the atrophy and denervation of type II muscle fibres at the neuromuscular junction (NMJ), fibers characterized by high glycolytic capacity and greater force-generating potential compared to type I fibres (Callahan et al., 2014). Consequently, the preferential loss of type II fibres in older adults often results in challenges performing routine daily activities, such as ascending stairs, lifting moderately heavy objects, or rising from a seated position (Goodpaster et al., 2006). Furthermore, dynapenia is thought to interfere with the excitation-contraction coupling mechanism, potentially by hindering calcium release from the sarcoplasmic reticulum (Clark et al., 2012). As a consequence, functional limitations are evident, with 17% of women and 9% of men over 65 reportedly unable to lift a 5 kg weight or kneel. Significantly, dynapenia is estimated to affect approximately one-quarter of individuals over the age of 50 (Siparsky et al., 2014).

Aerobic Capacity

Alterations in body composition can lead to modifica-

tions in aerobic fitness. For example, maximal oxygen consumption (VO₂max) represents the capacity of the body to transport and utilize oxygen within skeletal muscle. At the population level, VO₂max serves as a key indicator of overall cardiopulmonary fitness, a parameter strongly associated with health span and risk of all-cause mortality (Hawkins et al., 2007). Consequent to sarcopenia and sarcopenic obesity, VO₂max typically decreases, primarily owing to impaired mitochondrial dynamics (fusion and fission), leading to reduced numbers of functional mitochondria, and a diminished mitochondrial biogenesis response to physical activity (PA) (Liu et al., 2020). Consequently, VO₂max generally begins to decrease after the age of 25, declining by approximately 1% annually, with this rate potentially increasing to 1.5% per year between the ages of 50 and 75 (Betik et al., 2008), mirroring age-related shifts in body composition and metabolic function. Furthermore, VO₂max is recognized as a significant indicator of brain health. Indeed, recent research demonstrated that VO₂max strongly predicts brain health metrics, such as brain volume and mass (Neves et al., 2023).

Appetite

Indeed, there is extensive evidence indicating the presence of age-related changes in brain plasticity which predisposes older individuals to neurological diseases such as dementia and Alzheimer's disease (Hou et al., 2019). Biological ageing, however, tends to also affect the appetite-signalling cascade in the brain, which is the master orchestrator of energy balance (Matafome et al., 2017). For instance, age-related dysregulation in energy intake is characterised by altered appetite due to dampened sense of taste and smell and impaired gastric motility, which contribute to the reduced desire to eat (Clarkston et al., 1997; Crogan et al., 2014; Schiffman, 1997). This phenomenon is now known as, anorexia of ageing and it is estimated to affect 15% to 30% of older adults, with higher rates in older age groups (Malafarina et al., 2013). More specifically, anorexia of ageing has been reported to manifest in 25% of home-dwellers, 60% in hospital inpatients and 80% of nursing home residents (Roy et al., 2016). In turn, this can translate in smaller meals eaten and poor preference for less-palatable nutrients such as dietary proteins, which are key in promoting muscle protein synthesis. This is particularly concerning as older individuals require more dietary protein per meal to elicit a muscle protein synthetic response (Moore 2014). Furthermore, ageing is also related with a reduction in the orexigenic drive that promotes hunger. Ghrelin, a major orexigenic hormone, is known to decrease with advancing of age (Serra-Prat et al., 2009). Similarly, leptin, an anorectic hormone, increases with age pre-prandially and post-prandially (Adamska-Patrano et al., 2019). Ultimately, these adverse effects of anorexia of ageing are mainly the result of age-related changes in homeostatic and non-homeostatic brain circuitry that regulates energy balance and body weight.

Energy Expenditure

Energy intake, including appetite, track the energy demands imposed by LBM and PA. On this note, LBM and PA have been shown to modulate food intake in older adults. For example, in a recent systematic review and meta-anal-

ysis Hubner and colleagues (2021) reported that physically active older individuals (60+yrs) have a better regulation of energy intake to match their energy needs. On this note, a higher appetite is a behavioural response to an elevated total daily energy expenditure (TDEE). A major static component, resting metabolic rate (RMR), comprises of 60-70% of TDEE and can be defined as the energy yield required to maintain biological processes within the body, particularly in metabolically demanding tissue of LBM such as the kidneys, heart, liver, brain and skeletal muscle (Blundell et al., 2012; Villablanca et al., 2015). The latter yields only 13 kcal per kilogram, however, because skeletal muscle accounts for 70 to 90% of all LBM, this highly metabolically active organ becomes a major determinant of RMR in healthy adults (Heymsfield et al., 2018). On this note, Pontzer and colleagues (2021) recently examined differences in TDEE across the life course of 6400 individuals using doubly labelled water and indirect calorimetry, which are gold standard techniques to assess daily energy expenditure and basal metabolic rate in free living individuals. Their findings suggest that there are four metabolic life stages. First, new-borns have similar RMR to an adult, however, at one year of age, infants display a 50% higher RMR when compared to the RMR of an adult, which is due to the increased energy requirements for organ growth. From approximately one year of age, when relative RMR is at its highest, RMR tends to slowly decrease until reaching a plateau at ~20 years of age and remaining stable thereafter until ~60 years of age. Menopause for women as well as puberty in males and females were not factors contributing to substantial changes in RMR (Pontzer et al., 2021). However, the age-related decline in RMR adjusted for body size and body composition becomes significant in older individuals (60+ years). This age-related reduction in RMR is multifactorial, and mainly driven by blunted muscle protein synthesis, organ specific atrophy, as well as decreased enzymatic activity (Manini 2010). In addition, mass of the brain, kidney, liver, skeletal muscle, as well as bone mass, tend to decline by 10-20% from 20 to 60 years of age resulting in substantial reductions in RMR (Zampino et al., 2020). To summarize, promoting maintenance of LBM is a desirable outcome, which may promote metabolic health and locomotion, but also aid appetite regulation in older individuals.

Daily Steps Towards Optimal Health-span

Typically, older individuals cover shorter walking distances compared to younger adults, even when their daily time spent walking is comparable (Zhao et al., 2015). This observation is especially relevant considering reported declines in lifetime peak physical activity (PA) ranging from 40% to 80% (Westerterp, 2018). Indeed, current evidence indicates that a higher walking pace (such as brisk walking) positively correlates with overall fitness levels among older adults (Wu et al., 2021). Consequently, it aligns with the World Health Organization's (WHO) recommendation for older adults to engage in 300 minutes of PA or 150 minutes of moderate-to-vigorous physical activity (MVPA) weekly (Rivera-Torres et al., 2019). At the population level, MVPA is often defined as intentional PA performed at an intensity between 3 and 6 metabolic equivalents (METs), with 1 MET representing the energy expenditure during

quiet sitting. Alternative methods for quantifying MVPA include assessing intensity relative to maximal heart rate or using ratings of perceived exertion. A more recent proposal by MacIntosh and colleagues (2021) suggests categorizing MVPA based on ventilatory and/or lactate thresholds, identifying the first threshold with moderate intensity and the second with vigorous intensity PA. In simpler terms, the 'talk test'—where physical exertion makes carrying on a conversation slightly, but not overly, difficult—can serve as a subjective indicator of MVPA intensity (Reed et al., 2014). Thus, MVPA encompasses activities like brisk walking, ascending stairs, dancing, household cleaning, and gardening. Ultimately, MVPA involves an effort level capable of eliciting physiological adaptations akin to those from formal resistance and endurance training, albeit typically to a lesser degree (Loprinzi, 2015). These everyday, unstructured PA bouts of varying intensities can be measured using triaxial accelerometers (e.g., wrist-worn activity trackers), which also facilitate the quantification of PA through metrics like daily step counts (Henriksen et al., 2018).

While a common public health guideline promotes aiming for 10,000 steps daily (Choi et al., 2007), this specific target lacks robust support from current scientific findings (Hall et al., 2020; Jayedi et al., 2022; Paluch et al., 2022). For instance, a systematic review by Hall et al. (2020) found that higher daily step counts correlated with reduced risks of cardiovascular disease and all-cause mortality. However, this review was not designed to determine a specific dose-response relationship. This aspect was explored in a later systematic review by Jayedi et al. (2022), which identified a dose-response association between daily steps and all-cause mortality risk. Compared to individuals achieving 16,000 daily steps, those walking only 2,700 steps per day exhibited nearly double the risk of all-cause mortality, according to their findings. A more recent meta-analysis encompassing 15 international cohorts and 47,471 adults by Paluch et al. (2022) aimed to estimate the optimal daily step count for maximizing health benefits. Their analysis revealed that increased daily steps correlated with progressively lower mortality risk, with benefits appearing to level off around 6,000-8,000 steps/day for adults over 60 and 8,000-10,000 steps/day for those under 60. However, these large-scale analyses did not specifically examine how age-related variations in body composition might influence these relationships or contribute to suboptimal metabolic health and reduced mobility (McLeod et al., 2016). A cross-sectional study by Westerterp et al. (2021), including 2,000 participants aged 3-96, demonstrated that PA significantly influences body composition throughout life. Nevertheless, these authors emphasized that the specific type and intensity of PA appear particularly crucial for mitigating unfavourable body composition changes (Westerterp et al., 2021). Illustrating this, Scott et al. (2011), studying 697 older adults, observed a robust link between greater daily step counts and higher lower-limb LBM. Likewise, Aoyagi et al. (2010) reported beneficial effects of higher daily step counts on lower-limb strength in older adults, noting apparent thresholds around 8,000 steps/day for men and 6,900 for women. These results align with other research suggesting that achieving 7,000-8,000 steps and/or 15-20 minutes of MVPA daily was adequate for preventing sarcopenia in older men and women (Figure 2) (Park et al., 2010).

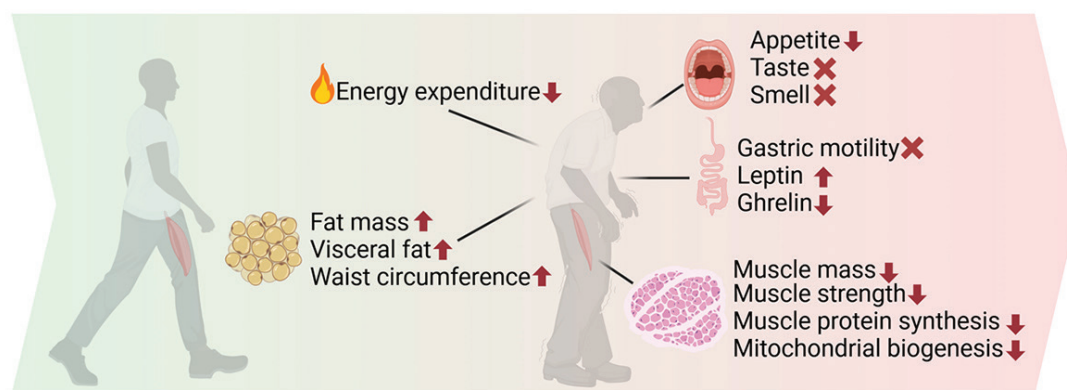


FIGURE 2. Daily step count and association with disease in younger and older adults

Several investigations suggest that substituting sedentary behaviour and low-intensity physical activity (PA), such as walking, with moderate-to-vigorous physical activity (MVPA) could represent a more effective strategy for preventing the development of sarcopenia and sarcopenic obesity (Hamer et al., 2014; Stamatakis et al., 2019; Yasunaga et al., 2018). Specifically, displacing sedentary time with 15 or 60 minutes of daily MVPA has been shown to potentially reduce sarcopenia risk by approximately 15% and 50%, respectively (Sánchez-Sánchez et al., 2019). Similarly, Ogawa and colleagues (2011) determined in a study of 48 older women that MVPA, unlike total daily steps, positively correlated with leg lean body mass (LBM). An observational study by Scott et al. (2021) involving 3,334 Swedish 70-year-olds reported a strong association between greater volumes of MVPA and a lower probability of sarcopenia, independent of MVPA bout duration or time spent sedentary. In a follow-up study with 3,653 community-dwelling individuals, the same research group demonstrated that high levels of MVPA and sedentary time can coexist without increasing the likelihood of sarcopenia (Johansson et al., 2021). Notably, this study also found that low sedentary time combined with low MVPA was insufficient to prevent sarcopenia onset. Conversely, Lai et al. (2022), studying 199 overweight community residents, found that replacing 30 minutes of sedentary time with MVPA decreased fat mass (FM) and waist circumference while maintaining skeletal muscle mass. However, research by Mijnders et al. (2016) over a 5-year period indicated that while participating in at least 60 minutes of weekly MVPA delayed sarcopenia onset in older individuals, it did not entirely prevent it, highlighting the need for sufficient MVPA duration.

Aggio and colleagues (2016) examined the links between objectively assessed MVPA and indicators of sarcopenia and sarcopenic obesity among 1,286 older men. This research group observed a higher prevalence of sarcopenia among participants engaging in 22-33 minutes of daily MVPA compared to those undertaking 38-45 minutes per day. Furthermore, the study highlighted an inverse relationship between sarcopenia severity, assessed via upper arm circumference, and the duration of MVPA. Indeed, the investigators reported that incorporating an additional 30 minutes of daily MVPA nearly halved the risk of severe sarcopenia for individuals in the lower MVPA category (Aggio et al., 2016). A longitudinal study by Menai et al. (2017) including 3,749 adults aged 60-83 demonstrated that individuals classified as 'successful agers' engaged in 8 minutes more daily MVPA

than 'unhealthy agers'. Their research underscored that, relative to those performing less than 10 minutes of daily MVPA, engaging in 10-20 minutes per day doubled the probability of successful aging. Yet, increasing MVPA further to 20-30 minutes daily resulted in only an additional 38% rise in the likelihood of successful aging, suggesting diminishing returns for additional daily MVPA time.

Conclusion and Future Perspectives

The process of biological aging inherently involves a decline in physiological functions, a trajectory often exacerbated by the absence of timely and targeted behavioural interventions capable of mitigating the onset and progression of sarcopenia and sarcopenic obesity. Sufficient levels of physical activity (PA) have demonstrated efficacy in counteracting the age-associated deterioration of body composition and metabolic health. While combined resistance and endurance training represents the principally recommended exercise approach for older adults, long-term adherence to such structured modalities remains challenging. Consequently, for older individuals less inclined to commit to structured exercise regimens, pursuing daily step targets of approximately 8,000 for men and 6,900 for women may be a suitable recommendation. If achieving these daily step counts proves difficult, incorporating additional minutes of moderate-to-vigorous physical activity (MVPA) could enhance health span, particularly when baseline MVPA levels are low (e.g., under 10 minutes daily). Nonetheless, the potential for diminishing returns with increasing daily MVPA duration exists, which might deter participation due to perceived excessive time and effort demands. Thus, advocating for approximately 30 minutes of daily MVPA could be a pragmatic and achievable guideline for preserving muscle mass and limiting fat mass (FM) accumulation in older adults for whom sustained exercise adherence is problematic. It is well-documented in empirical research that seasonal variations and weather conditions significantly influence PA levels, including MVPA. PA participation typically decreases during snowy periods and adverse weather conditions. Indeed, PA levels among community-dwelling individuals can plummet by as much as 75% during snowy intervals (Hasegawa et al., 2019; Matthews et al., 2019). This reduction in PA may, in turn, contribute to unintentional skeletal muscle loss and FM gain, potentially accumulating year-on-year and fostering the development of sarcopenia and sarcopenic obesity.

An area requiring further investigation involves exploring the effects of varying PA levels and age-related alterations

on the appetite-regulating signalling pathways. Observations indicate that older adults generally exhibit lower energy consumption and reduced intake of protein-dense foods. This dietary pattern could potentially worsen the known age-related blunting of the muscle protein synthetic response to dietary protein ingestion. Hence, future studies should focus on ex-

amining how different durations and intensities of PA and/or MVPA influence energy intake, with particular attention to nutrient selection, especially dietary protein. Moreover, careful consideration of exercise modality, volume, intensity, and duration is warranted, as these variables might precisely modulate centrally regulated appetite control.

Acknowledgements

The Slovenian Research Agency provided author N.Š. in the form of partial salary through the program 'KINSPO – Kinesiology for the effectiveness and prevention of musculoskeletal injuries in sport' [P5-0443]. The funder had no role in any aspect of the writing of the manuscript, or in the decision to publish it.

Conflicts of Interest

The authors declare no conflicts of interest.

Received: 11 Aprile 2025 | **Accepted:** 10 May 2025 | **Published:** 01 June 2025

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