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Does Lifelong Sports Participation Protect Against Sarcopenia? Prevalence, Diagnostic Criteria, and Sport-Specific Muscle Outcomes in Masters and Former Elite Athletes: A Scoping Review

Zuzanna Olszowiec, ORCID https://orcid.org/0009-0003-6775-8086

E-mail: zuziaol@interia.pl

M. Sklodowska-Curie Provincial Hospital in Zgierz, Parzeczewska 35, 95-100 Zgierz, Poland

Katarzyna Kowalenko ORCID https://orcid.org/0009-0003-5582-9713

E-mail: kjkkowalenko@gmail.com

M. Sklodowska-Curie Provincial Hospital in Zgierz, Parzeczewska 35, 95-100 Zgierz, Poland

Natalia Drobińska ORCID https://orcid.org/0009-0006-9295-7564

E-mail: kingadrobinska@wp.pl

M. Sklodowska-Curie Provincial Hospital in Zgierz, Parzeczewska 35, 95-100 Zgierz, Poland

Jan Szadkowski ORCID https://orcid.org/0009-0009-4276-8636

E-mail: jan.szadkowski.2601@gmail.com

M. Sklodowska-Curie Provincial Hospital in Zgierz, Parzeczewska 35, 95-100 Zgierz, Poland

Hanna Kasprzak ORCID https://orcid.org/0009-0000-9038-1035

E-mail: hania99kasprzak@gmail.com

Norbert Barlicki Memorial Teaching Hospital No. 1 of the Medical University of Lodz, 22 Dr. Stefana Kopcińskiego Street, 90-153 Łódź, Poland

Yana Lavrenchuk ORCID https://orcid.org/0009-0001-1510-2902

E-mail: yanalavrenchuk10.02@gmail.com

Norbert Barlicki Memorial Teaching Hospital No. 1 of the Medical University of Lodz, 22 Dr. Stefana Kopcińskiego Street, 90-153 Łódź, Poland

Krzysztof Wichman ORCID https://orcid.org/0009-0006-8073-6211

E-mail: krzysztof.wichman1@gmail.com

M. Sklodowska-Curie Provincial Hospital in Zgierz, Parzeczewska 35, 95-100 Zgierz, Poland

Kacper Jaskulski, ORCID https://orcid.org/0009-0006-1631-8058

E-mail kacper.jaskulski@gmail.com

Central Clinical Hospital of the Medical University of Lodz, Poland

Kamil Klimas, ORCID https://orcid.org/0000-0002-0201-7928

E-mail: kamkli17@gmail.com

A. Falkiewicz Specialist Hospital: Wrocław, PL

Corresponding author:

Zuzanna Olszowiec, E-mail: zuziaol@interia.pl

Abstract

Background: Sarcopenia is defined by progressive reductions in skeletal muscle mass and strength, with diagnostic frameworks differing in the weight assigned to strength, muscle quantity, and physical performance. The degree of protection afforded by lifelong sport, and whether protection differs between endurance, strength, power, and mixed sports, remains uncertain.

Aim: To map evidence on the prevalence of sarcopenia, diagnostic criteria applied, and sport-specific muscle outcomes in masters athletes and former elite athletes.

Material and Methods: A scoping review framework informed by JBI guidance and PRISMA-ScR was used. PubMed/MEDLINE, Europe PMC, and citation chaining were searched for English-language studies involving competitive masters athletes, lifelong exercisers, former elite athletes, or former Olympians aged 40 years or older. Eligible studies reported sarcopenia diagnoses or at least one relevant domain.

Results: Athlete-focused studies consistently showed a lower prevalence of confirmed sarcopenia than that reported in community-dwelling older populations. Strength and sprint athletes generally displayed greater appendicular lean mass, maximal strength, rate of force development, and preservation of type II fibres than endurance athletes. Lifelong endurance training was associated with low adiposity, superior aerobic capacity, favourable muscle oxidative characteristics, and in several studies preserved muscle quality, but it did not fully prevent lower muscle mass, motor-unit loss, or age-related reductions in power. Continued training after elite retirement appeared more important than historical athletic status alone.

Conclusions: Lifelong sports participation substantially shifts the muscle-ageing trajectory and lowers the likelihood of clinical sarcopenia, but it does not make athletes biologically immune to age-related neuromuscular decline.

Keywords: sarcopenia; masters athletes; former elite athletes; lifelong exercise; muscle mass; muscle strength; muscle power; ageing; scoping review

1. Introduction

Sarcopenia is a progressive skeletal-muscle disorder characterised by reduced muscle strength and muscle quantity, often accompanied by impaired physical performance and an increased risk of falls, disability, hospitalisation, and mortality. The construct has evolved from a mass-centred description of age-related muscle loss to a clinically oriented disorder in which weakness and poor muscle quality are increasingly recognised as central. The revised European Working Group on Sarcopenia in Older People consensus (EWGSOP2) prioritises low muscle strength as the entry point to probable sarcopenia, uses low muscle quantity or quality to confirm the diagnosis, and considers impaired performance a marker of severity [1]. The Asian Working Group for Sarcopenia (AWGS 2019) retains low muscle mass as an obligatory component and combines it with low strength and/or poor performance [2]. The Sarcopenia Definition and Outcomes Consortium (SDOC) gives particular emphasis to weakness and slowness [3], while the Global Leadership Initiative in Sarcopenia (GLIS) conceptual framework identifies reduced muscle mass, reduced strength, and muscle-specific strength as core components, with physical performance considered principally an outcome [4].

These differences are not merely semantic. In global meta-analysis, sarcopenia prevalence varies materially according to the definition, cut-offs, setting, and measurement technology used [5]. Athletes represent an especially challenging population because standard thresholds were derived primarily from general or clinical populations. A lean endurance athlete may have relatively low appendicular lean mass despite excellent mobility and cardiometabolic health,

while a retired strength athlete may retain substantial muscle mass but have clinically meaningful reductions in power, gait adaptability, or neuromuscular function. Conversely, very high baseline function may delay crossing a fixed diagnostic threshold even when the individual has experienced a large within-person decline.

Masters athletes are usually defined by participation in age-group competition, often from approximately 35 or 40 years onward, and may include lifelong competitors as well as individuals who began intensive training later in life. Former elite athletes are a distinct group: their earlier exposure may have produced a large physiological reserve, but protection in older age may depend on whether training was maintained after retirement. Combining these groups without accounting for current training, sport type, sex, body size, injuries, and career history obscures important biological and clinical differences.

The masters-athlete model is valuable because it reduces, although does not eliminate, physical inactivity as a confounder of ageing. Cross-sectional comparisons suggest that chronic exercise can preserve muscle mass, strength, body composition, and function far beyond levels observed in sedentary peers [10-16]. However, mechanistic studies also show persistent age-related motor-unit loss, type II fibre vulnerability, and reductions in explosive performance even among highly trained older adults [17-24]. Therefore, the relevant question is not whether exercise abolishes ageing, but how strongly it shifts the trajectory, which muscle domains are protected, and which training modalities provide the broadest reserve.

The objective of this scoping review was to map the available evidence on three linked questions: (1) What is the reported prevalence of sarcopenia or its components in masters and former elite athletes? (2) Which diagnostic definitions and measurement tools have been used, and what problems arise when applying general-population cut-offs to athletes? (3) How do muscle mass, strength, power, quality, morphology, and motor-unit outcomes differ by sport phenotype, particularly endurance versus strength/power disciplines?

2. Research materials and methods

2.1 Review design and reporting

The review was structured as a scoping review because the evidence base includes heterogeneous cross-sectional cohorts, former-athlete follow-up studies, physiological experiments, imaging studies, muscle-biopsy investigations, and longitudinal observations. The conduct and reporting approach was informed by the Arksey and O'Malley framework [8], subsequent methodological refinements [9], JBI guidance [7], and the PRISMA extension for scoping reviews [6]. The aim was evidence mapping rather than pooled effect estimation or formal grading of intervention efficacy.

2.2 Review questions and eligibility

The Population-Concept-Context framework was used. The population comprised adults aged 40 years or older described as masters athletes, veteran athletes, lifelong exercisers, former elite

athletes, former professional athletes, or former Olympians. The concept was sarcopenia or a related muscle-health domain: total or appendicular lean mass, regional muscle size, muscle strength, muscle power or rate of force development, gait or chair-rise performance, muscle quality, intramuscular adipose tissue, fibre type and morphology, motor-unit number or remodelling, and muscle protein synthesis. The context included competitive and non-competitive sport, current or former participation, and endurance, strength, sprint/power, mixed, or contact disciplines.

Original human studies were eligible when athlete status was explicit and at least one sarcopenia-relevant outcome was reported. Reviews and consensus statements were retained only to define diagnostic frameworks, contextualise prevalence, or support interpretation. Studies limited to cardiopulmonary outcomes without muscle-related measures were excluded. Case reports were used only when they illustrated a measurement issue and were not treated as prevalence evidence. No restriction was imposed on study design, but English-language full text or a sufficiently detailed peer-reviewed abstract was required.

2.3 Information sources and search strategy

PubMed/MEDLINE and Europe PMC were searched from inception through 15 July 2026, supplemented by backward and forward citation chaining from key reviews and athlete cohorts. The core search combined athlete terms with sarcopenia and muscle terms: (master athlete* OR masters athlete* OR veteran athlete* OR lifelong exercis* OR former elite athlete* OR retired athlete* OR Olympian*) AND (sarcopenia OR muscle mass OR appendicular lean mass OR muscle strength OR muscle power OR muscle quality OR motor unit* OR muscle fibre OR muscle fiber OR DXA OR MRI) AND (ageing OR aging OR older). Additional searches used sport-specific terms including endurance, running, sprint, power, weightlifting, strength, and former professional.

2.4 Study selection and data charting

Titles and abstracts were screened for athlete status and muscle outcomes. Full texts or detailed records were then reviewed for age, sex, sport, current training status, comparator group, diagnostic criteria, measurement methods, and principal findings. Because several cohorts generated multiple mechanistic publications, publications rather than unique cohorts were charted. Data were grouped into prevalence/diagnosis, body composition, strength and power, muscle quality and adiposity, muscle morphology, motor-unit physiology, and sport-specific patterns.

2.5 Synthesis approach

A descriptive synthesis was used. No meta-analysis was attempted because of large differences in age ranges, sport exposure, comparator selection, diagnostic definitions, equipment, normalisation procedures, and outcome reporting. Particular attention was paid to whether an apparent benefit reflected higher absolute reserve, slower age-related decline, maintenance above clinical cut-offs, or sport-specific adaptation. These are related but non-equivalent forms of protection.

3. Results

3.1 Scope and characteristics of the evidence

The mapped evidence was dominated by cross-sectional studies of male endurance runners, sprinters, strength athletes, and mixed-sport masters competitors. Women were included in some body-composition and competition cohorts, but sex-specific mechanistic data remained limited. A smaller group of studies evaluated former Olympians or former elite endurance and power athletes in their seventh to tenth decades. Muscle outcomes were measured with DXA, bioelectrical impedance, MRI, ultrasound, dynamometry, grip testing, gait speed, chair-rise tests, jump or sprint performance, muscle biopsy, intramuscular electromyography, and stable-isotope assessment of muscle protein synthesis. The breadth of methods was a strength for biological interpretation but prevented quantitative pooling.

Twenty-three athlete-focused publications were charted in the core evidence map. Several arose from overlapping cohorts and addressed different biological levels, from whole-body lean mass to single-fibre contractility and motor-unit remodelling. The most consistent comparison was between older athletes and age-matched non-athletes; fewer studies directly compared sport types, and only a small number were longitudinal.

3.2 Prevalence of sarcopenia and low diagnostic components

Direct prevalence studies suggest that confirmed sarcopenia is uncommon among active masters athletes. In 156 competitors at the 2014 Pan Pacific Masters Games, Fien et al. found no participant who met the applied sarcopenia definition, although six had low handgrip strength and one had low physical performance [11]. This distinction is important: isolated low components were present even in a healthy competition sample, but the combination required for diagnosis was absent. Selection bias is likely because individuals able and willing to compete in masters sport are healthier than the source population.

Huschtscha et al. examined active adults aged 50 years and older using EWGSOP2-related outcomes. Approximately 6% had low grip strength and therefore probable sarcopenia, but low muscle quantity did not confirm sarcopenia [12]. The study demonstrates that high activity does not guarantee uniformly high strength, and that age, sex, protein intake, and exercise volume contribute to variation within an active group.

The strongest former-elite prevalence evidence comes from athletes who competed at the 1964 Tokyo Olympic Games. Tanaka et al. compared 101 former Olympians, mean age approximately 75 years, with 1,529 community-dwelling older adults using AWGS 2019 criteria. Former Olympians had lower adjusted odds of sarcopenia (OR 0.49, 95% CI 0.20-0.94), with advantages in appendicular skeletal muscle mass and strength [13]. Protection was more pronounced among those who continued exercising and among athletes from disciplines classified as higher intensity. However, low physical function and musculoskeletal pain were not uniformly lower and were more common in some contact-sport groups. Thus, a favourable muscle phenotype may coexist with pain or mobility limitations caused by accumulated injury.

Compared with global estimates of approximately 10-27% depending on diagnostic criteria [5], these athlete cohorts indicate a markedly lower burden of confirmed sarcopenia. Nevertheless, the data do not justify a universal prevalence estimate for masters athletes. Samples differ in age, health, competition level, sex, and sport, and most are not population-based. The oldest-old, women, athletes with disability, and those who stopped training are underrepresented.

3.3 Muscle mass and body composition

One of the most cited masters-athlete studies evaluated 40 high-level recreational athletes aged 40-81 years who trained four to five times per week. MRI-derived mid-thigh muscle area, quadriceps area, lean mass, and quadriceps peak torque did not show the expected age-related decline across the sample [10]. The study challenged the assumption that substantial muscle loss is an unavoidable consequence of chronological ageing and highlighted chronic disuse as a major contributor. Its cross-sectional design, small sample, and unusually active participants limit causal inference, but the visual and quantitative preservation of thigh musculature provided a compelling proof of concept.

More recent sport-stratified work confirms that preservation is modality-specific. Walker et al. compared young and older male athletes from strength, sprint, and endurance disciplines with age-matched non-athletes. In masters athletes, strength athletes had greater appendicular lean mass index than endurance athletes, and both older strength and sprint athletes had greater appendicular lean mass than older controls [14]. Endurance athletes had favourable fat mass but did not show the same degree of muscle-mass reserve. These results support the principle of specific adaptation: high-force loading provides a stronger hypertrophic signal than repetitive low-force endurance activity.

Masters endurance athletes nevertheless often retain more favourable body composition than inactive peers. McKendry et al. reported lower body fat and a more favourable skeletal-muscle index in chronically trained older endurance athletes than in untrained older adults [16]. Piasecki et al. found that older adults who began intensive endurance running after age 50 achieved lower body fat and higher leg lean mass than non-athletes, with body composition and performance similar to lifelong runners [19]. This is an important public-health message: a lifelong competitive history is not required to obtain a substantial late-life muscle and body-composition benefit.

Former-athlete studies suggest that residual advantages may persist, but current activity modifies them. Former Olympic athletes had higher appendicular muscle mass than community controls [13]. Older former elite power athletes generally retained more muscle and strength than former endurance athletes or controls [25]. Yet retirement can be accompanied by reduced activity, weight gain, injury, and altered diet. Historical elite status should therefore be treated as an exposure, not as a current physiological category.

3.4 Strength, power, and functional performance

Muscle strength declines more rapidly than muscle mass in general ageing, and muscle power often declines faster than maximal strength. Masters athletes maintain a higher functional level,

but absolute protection is incomplete. In mixed-sport competition cohorts, grip strength remains high on average but decreases with age [11]. In former elite athletes aged 66-91 years, mobility and muscle strength were generally better than in controls, particularly in former power athletes, although age remained a strong determinant [25].

Sport-specific testing reveals larger differences than generic screening. Lifelong strength-trained men older than 70 years displayed leg-press maximal strength and rate of force development similar to young active controls in the study by Tøien et al., whereas endurance-trained older athletes and recreationally active older adults were lower [20]. These findings indicate that repeated exposure to high force and rapid force production can preserve neuromuscular performance that is directly relevant to preventing falls and recovering balance.

Performance still declines within athletes. Bagley et al. found age-related reductions in both aerobic and anaerobic power among endurance and power masters athletes, with training phenotype shaping the level but not eliminating the decline [27]. A 10-year follow-up of masters sprinters showed preserved histological characteristics yet worsening sprint times and lower jump power [23]. This divergence emphasises that tissue morphology, maximal strength, and whole-body performance are related but not interchangeable. Tendon properties, neural drive, coordination, reaction time, joint health, and training volume can limit performance even when muscle fibres remain relatively well preserved.

For clinical assessment, power deserves greater attention. The ability to generate force quickly is critical for stair climbing, arresting a fall, and maintaining athletic performance. An older endurance athlete may exceed gait-speed thresholds but still have low relative leg power. Conversely, a strength athlete may produce high maximal force but lose movement velocity. A comprehensive athlete assessment should therefore include at least one high-velocity lower-limb measure in addition to grip and mass.

3.5 Muscle quality, intramuscular adiposity, and anabolic responsiveness

Muscle quality refers broadly to force or power produced per unit of muscle size and is influenced by neural activation, architecture, fibre type, contractile quality, fat infiltration, connective tissue, and mitochondrial function. Lifelong aerobic exercise is associated with lower adiposity and, in some muscles and sexes, less intramuscular fat. Chambers et al. showed that ageing and lifelong aerobic exercise influenced muscle size, function, and adipose infiltration in sex- and muscle-specific ways, with higher lifelong training intensity offering greater protection from fat infiltration [21]. This finding helps explain why endurance athletes may retain good function despite less absolute muscle mass.

The anabolic response to habitual activity is complex. McKendry et al. used deuterated water to compare integrated myofibrillar protein synthesis in endurance-trained masters athletes and untrained older adults. Rates were comparable rather than uniformly higher in the athletes [15]. Lifelong endurance exercise may therefore preserve phenotype through repeated training stimuli, better metabolic health, and lower inactivity exposure rather than through a chronically elevated basal synthesis rate. The absence of a large between-group difference also cautions

against assuming that highly trained older muscle is fully resistant to age-related anabolic resistance.

Single-fibre studies provide evidence that contractile quality can be preserved or even enhanced in selected fibre types. Lifelong endurance exercisers have demonstrated larger and more powerful slow fibres than age-matched untrained adults, and in some analyses slow-fibre properties compared favourably with young exercisers [22]. These adaptations are highly relevant to endurance function but do not necessarily protect the fast-fibre pool needed for explosive tasks.

3.6 Muscle fibre morphology and the protection of type II fibres

Ageing is characterised by loss and atrophy of type II fibres, fibre-type grouping after denervation and reinnervation, and progressive motor-unit remodelling. Training modality strongly modifies these patterns. Tøien et al. reported that lifelong strength-trained older men had type II fibre distribution, low prevalence of atrophic fibres, maximal strength, and rate of force development resembling young controls [20]. Endurance-trained older athletes showed a higher proportion of type I fibres, more type I grouping, and more atrophic fibres than strength athletes. These are not necessarily pathological findings; they partly reflect the specific demands of endurance training. However, they indicate less protection of the fast-twitch reserve.

Sprint training also appears protective. Messa et al. reported no ageing-related increase in fibre-type grouping over a 10-year period in masters sprinters who continued training [24]. The longitudinal follow-up by Hendrickse et al. similarly found that key histological features were maintained over a decade, even though sprint and jump performance declined [23]. Together, these studies suggest that high-velocity loading may stabilise aspects of muscle innervation and fibre organisation but cannot fully preserve integrated performance.

Endurance training produces a different beneficial phenotype. McKendry et al. observed favourable muscle morphology and aerobic capacity in chronically trained masters endurance athletes compared with untrained older adults [16]. Long-term endurance training supports oxidative capacity, capillarisation, mitochondrial function, and slow-fibre performance, but the evidence is less consistent for preservation of type II fibre size and explosive power. Therefore, endurance sport protects against one dimension of muscle ageing while leaving another relatively exposed.

3.7 Motor units and neuromuscular remodelling

Motor-unit loss is a major mechanism of sarcopenia and dynapenia. Surviving motor neurons can reinnervate denervated fibres, producing larger motor units and fibre-type grouping. Masters athletes appear capable of more successful compensatory remodelling, but they are not spared from motor-unit loss.

Drey et al. compared power-trained and endurance-trained masters athletes with community-dwelling older adults. Whole-body muscle mass and estimated motor-unit number declined

with age, but the athletes showed evidence compatible with better motoneuron preservation, and power athletes had greater muscle mass than endurance athletes [26]. Piasecki et al. found that long-term endurance and power training may facilitate motor-unit size expansion to compensate for declining motor-unit numbers, particularly in heavily trained muscles [18].

The location and stability of motor units also matter. Jones et al. observed more homogeneous motor-unit potential features across deep and superficial regions of vastus lateralis in masters athletes than in older non-athletes [17]. Other work in tibialis anterior showed that athletic older adults still underwent age-related motor-unit remodelling, although neuromuscular transmission or compensatory features could be more favourable than in inactive peers [28]. The practical interpretation is that lifelong training improves resilience of the motor-unit pool rather than preventing its biological ageing.

3.8 Former elite athletes: reserve, detraining, and accumulated injury

Former elite athletes differ from current masters competitors in three ways. First, they may have entered older age with a larger peak reserve in muscle mass, strength, skill, and cardiometabolic capacity. Second, training often changes abruptly after retirement. Third, years of high loading or contact exposure may cause osteoarthritis, tendon disease, chronic pain, or previous surgery that impairs performance independently of sarcopenia.

The Tokyo Olympian cohort illustrates both reserve and complexity. Former Olympians had lower sarcopenia prevalence and better appendicular muscle mass and strength, especially when exercise continued [13,33]. At the same time, low physical function and musculoskeletal pain were not uniformly reduced, particularly in contact-sport athletes. In Finnish former elite athletes, power-sport backgrounds were associated with stronger late-life muscle outcomes than endurance backgrounds, but age and current physical characteristics remained important [25]. These findings argue against using the label ‘former elite athlete’ as a proxy for healthy musculoskeletal ageing.

The distinction also has implications for causality. Better late-life outcomes may reflect training, genetic endowment, socioeconomic advantage, healthier behaviour, or selection into elite sport. Conversely, those with severe injury or chronic disease may be less likely to attend follow-up studies. Longitudinal designs beginning during the athletic career, with repeated measures through retirement, are needed to separate these influences.

4. Discussion

4.1 Does lifelong sport protect against sarcopenia?

The evidence supports a qualified yes. Lifelong or long-term sports participation lowers the probability that an older adult will cross clinical thresholds for low muscle mass and weakness. Active masters competitors show very low rates of confirmed sarcopenia, and former Olympians have lower adjusted odds than community controls [11-13,31]. The benefit is biologically plausible and supported across multiple levels: higher appendicular and regional

muscle mass, greater strength, lower adiposity, reduced muscle fat infiltration, more favourable fibre morphology, and enhanced motor-unit compensation [10,14,17-21,26].

The protection is nevertheless relative rather than absolute. Masters athletes continue to lose motor units, explosive power, and performance with age. Endurance athletes may preserve aerobic and oxidative capacity while showing lower muscle-mass reserve and less protection of type II fibres. Sprinters and strength athletes can preserve fast-fibre morphology and high force to an exceptional degree, but sprint speed and jump power still decline. The phrase 'protected against sarcopenia' should therefore mean a delayed or attenuated trajectory and maintenance above diagnostic thresholds, not absence of biological ageing [29].

4.2 Sport specificity is central

The clearest cross-study pattern is the superiority of high-force and high-velocity loading for preserving appendicular muscle mass, type II fibres, maximal strength, and rate of force development. Strength and sprint athletes generally outperform endurance athletes in these domains [14,20,23-26]. Endurance athletes display complementary advantages: lower fat mass, excellent gait and aerobic performance, oxidative adaptations, capillarisation, and favourable slow-fibre characteristics [15,16,21,22].

This creates an important training implication. The muscle phenotype most resistant to clinical sarcopenia is unlikely to result from a single sport performed unchanged across decades. Endurance athletes benefit from adding progressive resistance and power training, while strength and power athletes benefit from maintaining aerobic conditioning, mobility, and movement variety. A combined programme can address muscle mass, maximal strength, rapid force production, metabolic health, balance, and cardiorespiratory reserve.

4.3 Diagnostic criteria may misclassify athletes

Current diagnostic thresholds were not designed to detect early decline in high-function athletes. Ceiling effects in gait and chair-rise tests, limited relevance of grip to lower-limb sports, and body-size effects on height-normalized lean mass can distort classification. A binary diagnosis is useful for epidemiology and clinical pathways but insufficient for sports medicine. Athlete assessment should add longitudinal change, regional muscle imaging where appropriate, knee-extension or leg-press strength, jump or stair power, and muscle-specific strength.

The GLIS emphasis on muscle-specific strength is particularly relevant [4]. Strength relative to muscle size can reveal deterioration in neural or contractile quality when absolute mass remains high. However, ratio measures require careful interpretation because both numerator and denominator are influenced by body size, sport, sex, and measurement error. Future consensus work should consider athlete-specific reference distributions or percentile-based change rather than simply importing clinical cut-offs.

4.4 The role of current training after elite retirement

Evidence from former Olympians indicates that continued exercise strengthens the association between former elite status and lower sarcopenia risk [13,32]. This suggests that earlier training creates reserve, but ongoing loading is necessary to maintain it. Detraining, injury, chronic pain, reduced energy expenditure, and weight gain can erode the advantage. Research should therefore report current frequency, intensity, resistance-training exposure, protein intake, and years since retirement, rather than classifying participants solely by their peak career status.

4.5 Sex and the oldest-old

The evidence base remains disproportionately male. This is a major limitation because menopause, sex differences in baseline muscle mass, hormonal changes, sport opportunity, and lifetime training exposure may modify the trajectory [30]. Some lifelong aerobic-exercise studies show sex- and muscle-specific patterns [21], but female strength and sprint athletes are underrepresented. Similarly, relatively few studies include athletes older than 80 years in sufficient numbers for stable estimates. The oldest-old are the group in whom clinical thresholds, injury burden, and survivor selection become most consequential.

4.6 Clinical and sports-medicine implications

Clinicians should not assume that an athlete cannot have sarcopenia, nor should they diagnose it from low lean mass alone. Screening should begin with symptoms and change: reduced training tolerance, slower recovery, loss of sprint or hill-climbing ability, difficulty rising, falls, unintentional weight loss, and reduced strength. Standard EWGSOP2 or AWGS assessment can be followed by athlete-relevant lower-limb tests. Pain, osteoarthritis, tendon disease, and neurological disorders must be distinguished from primary sarcopenia, especially in former contact-sport and power athletes.

For prevention, the evidence favours maintaining at least two exposures that are often lost with ageing: high muscular force and high movement velocity. Progressive resistance training should target major lower-limb and trunk muscle groups, and power work should be performed with appropriate loads and safe intent to move rapidly. Endurance exercise remains essential for cardiometabolic health and helps reduce muscle fat infiltration, but endurance-only training may not provide sufficient stimulus to preserve type II fibres and appendicular lean mass. Adequate energy and protein intake are necessary, particularly when training volume is high or body mass is intentionally kept low.

4.7 Research gaps

Future research should prioritise prospective cohorts that begin before retirement and follow athletes through later life. Repeated DXA or MRI, dynamometry, power testing, injury surveillance, training records, diet, and disease status would allow investigators to distinguish reserve from rate of decline. Studies should stratify by sport loading pattern rather than broad labels. For example, distance running, cycling, rowing, swimming, sprinting, throwing,

weightlifting, and field sports impose markedly different eccentric, impact, force, and velocity demands.

A harmonised core outcome set is needed. At minimum, studies should report the full diagnostic algorithm used; sex-specific muscle mass and grip values; a lower-limb strength measure; a power or rate-of-force-development measure; physical performance; current and lifetime training; and injuries affecting function. Reporting only whether a participant crosses a sarcopenia threshold discards valuable information about exceptional reserve and clinically relevant decline.

5. Conclusions

Lifelong sports participation provides substantial protection against clinical sarcopenia by increasing muscular reserve and attenuating losses in mass, strength, quality, and neuromuscular function. Confirmed sarcopenia appears uncommon in active masters athletes, and former Olympians show lower risk than community peers. Protection is strongest and most comprehensive in athletes exposed to sustained high-force or high-velocity loading, whereas endurance training primarily preserves aerobic capacity, leanness, oxidative phenotype, and selected aspects of muscle quality.

Athletes are not immune to ageing. Motor-unit loss, reduced power, fast-fibre vulnerability, injury, and performance decline persist, and a normal gait speed or high muscle mass can conceal important deficits. Assessment should therefore combine recognised sarcopenia criteria with sport-relevant lower-limb strength, power, and longitudinal change. From a practical perspective, the most defensible strategy for healthy muscular ageing is lifelong activity that integrates endurance, progressive resistance, and power training, supported by adequate nutrition and adaptation to injury and changing recovery capacity.

Disclosure

Author Contributions:

Conceptualization: Zuzanna Olszowiec

Methodology: Katarzyna Kowalenko, Natalia Drobińska

Formal analysis: Jan Szadkowski, Krzysztof Wichman, Kacper Jaskulski

Investigation: Zuzanna Olszowiec, Natalia Drobińska, Yana Lavrenchuk

Resources: Natalia Drobińska, Yana Lavrenchuk, Krzysztof Wichman

Data curation: Hanna Kasprzak, Yana Lavrenchuk, Kacper Jaskulski

Writing – original draft preparation: Zuzanna Olszowiec, Kamil Klimas

Writing – review and editing: Zuzanna Olszowiec, Jan Szadkowski, Hanna Kasprzak

Visualization: Kamil Klimas, Hanna Kasprzak, Kacper Jaskulski

Supervision: Zuzanna Olszowiec, Katarzyna Kowalenko, Krzysztof Wichman

Project administration: Jan Szadkowski, Kamil Klimas, Katarzyna Kowalenko

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Declaration on the Use of Artificial Intelligence

AI-assisted tools were used exclusively for linguistic refinement. The authors take full responsibility for the scientific content, interpretation of the data, and final version of the manuscript.

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