

Sarcopenia, Nutritional Risk, and Functional Independence in Older Adults Receiving Palliative Care

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Abstract

Functional independence in older adults receiving palliative care can deteriorate through interacting changes in skeletal muscle, nutritional status, disease burden, frailty, treatment exposure, and the environment in which daily activities are performed. Sarcopenia is therefore clinically important, although its meaning cannot be reduced to low muscle quantity or interpreted independently of nutritional and functional context. This conceptual article applies a functional-reserve perspective to examine how muscle mass, strength, physical performance, nutritional risk, inflammatory and catabolic burden, and frailty jointly shape the capacity to sustain meaningful activity during serious illness. Functional reserve is defined here as the conditional margin between a person's current capabilities and the level of physiological, functional, and contextual stress at which meaningful activities become difficult to maintain or recover. The resulting account predicts nonlinear and potentially self-reinforcing relations between nutritional deterioration and functional decline. Functional independence in palliative care is thus best interpreted as an emergent outcome of interacting reserves and stressors rather than as a direct consequence of muscle mass alone.

Keywords: Sarcopenia, Palliative care, Nutritional risk, Functional independence, Functional reserve, Frailty

Introduction

Sarcopenia has moved from a predominantly muscle-mass-centered concept toward a multidimensional understanding in which strength and physical performance carry distinct clinical meaning. Contemporary international consensus describes sarcopenia through deficits in skeletal-muscle characteristics and function rather than through muscle quantity in isolation [1, 2].

That question becomes especially important in palliative care, where the same impairment can have different implications according to prognosis, symptom burden, prior function, care setting, and goals. In older adults with cancer, malnutrition, sarcopenia, and cachexia can coexist, yet they remain clinically distinguishable conditions and do not necessarily overlap in a simple one-to-one manner [3, 4].

The distinction matters because a person may have reduced muscle reserves without active cachexia, may develop profound catabolic wasting despite nutritional support, or may remain functionally independent despite reduced muscle quantity if strength, performance, environmental support, and compensatory capacities remain sufficient [5].

The central claim is conditional: sarcopenia and nutritional risk contribute to functional decline through their effects on the capacity available to meet physiological and everyday demands, but the resulting level of

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Received: 09 April 2026; **Revised:** 03 June 2026; **Accepted:** 30 June 2026

How to Cite This Article: Novak P, Dubois S, Müller T, Weber A, Johansson E. Sarcopenia, Nutritional Risk, and Functional Independence in Older Adults Receiving Palliative Care. *J Integr Nurs Palliat Care*. 2026;7(1):226-36. <https://doi.org/10.51847/S9qMpwjqN8>

independence also depends on strength, performance, inflammatory and catabolic load, disease severity, frailty, multimorbidity, treatment burden, and the conditions under which activities are performed.

Conceptual approach

The functional-reserve perspective begins by separating measurable impairment from the capacity to withstand additional stress. Global consensus work similarly emphasizes that clinically meaningful sarcopenia must be related to defined health outcomes rather than treated as an isolated body-composition state [6, 7].

For the present analysis, functional reserve denotes the conditional margin between an individual's current capabilities and the level of combined physiological, functional, and contextual stress at which meaningful activities can no longer be sustained or recovered. It is an analytical construct, not a diagnostic test, validated scale, or claim that a hidden biological quantity can currently be measured directly.

This distinction is consistent with evidence that the major muscle domains contribute differently to disability. Meta-analytic evidence indicates that low muscle mass, weakness, and impaired physical performance are all associated with limitations in activities of daily living, although their associations are neither identical nor uniform across studies and measurements [8]. Such variation is theoretically important.

A functional-reserve perspective therefore shifts attention from single abnormalities toward configurations. Reduced strength can contribute to dependence, yet dependence may also arise because pain, dyspnea, cognition, environmental barriers, or caregiving circumstances prevent the use of preserved physical capacity.

Sarcopenia in advanced age and serious illness

Ageing and serious illness can converge on skeletal muscle through partly overlapping processes. Longitudinal evidence in community-dwelling older adults shows that composite sarcopenia is associated with subsequent disability and mortality, while isolated low muscle mass without concomitant weakness or impaired performance did not show the same significant associations in that cohort [9].

The components of muscle capability also change on different trajectories. In a large Danish cohort spanning young adulthood to very old age, lean mass, strength, power, and functional performance differed substantially across age, and their age-related patterns were not synchronized [10].

Serious illness adds further complexity. Neuromuscular impairment can accompany different stages of sarcopenia and may involve neural as well as muscular contributors [11]. Consequently, the muscle phenotype observed during palliative care may represent age-associated vulnerability, disease-related sarcopenia, cachectic processes, treatment effects, or several of these mechanisms simultaneously.

A low-muscle-mass phenotype accompanied by preserved strength, stable intake, and independent mobility signifies a different functional state from an apparently similar level of muscle mass accompanied by profound weakness, declining intake, escalating inflammation, and frailty.

Table 1. Distinguishing muscle failure, nutritional vulnerability, catabolic burden, and functional consequence in older adults with serious illness

Analytical unit	What is being observed	What the observation can indicate	What it cannot establish by itself	Functional-reserve relevance
Muscle quantity	Amount of skeletal muscle or lean tissue	Structural depletion; possible loss of available contractile tissue	Current strength, independence, recovery potential, or cause of loss	One structural component of available capacity
Muscle quality	Composition and functional characteristics of muscle tissue	Reduced efficiency of existing muscle; possible myosteatosis or impaired contractile quality	Degree of disability or reversibility	Helps explain different capability at similar muscle quantity
Muscle strength	Force-producing capability	Clinically important impairment in muscle function	Full mobility, endurance, or independence	Direct contributor to usable functional capacity
Physical performance	Execution of mobility-related tasks	Integrated expression of muscular, neurological, cardiopulmonary, and cognitive capacities	The isolated mechanism producing impairment	Observable manifestation of multiple interacting reserves
Nutritional risk	Increased probability of inadequate intake or nutrition-related deterioration	Vulnerability requiring assessment	Established malnutrition, sarcopenia, or cachexia	Potential reduction in substrates needed to maintain or recover capacity
Malnutrition	Phenotypic nutritional deterioration with an etiologic basis	Clinically relevant nutritional deficit	The existence or severity of sarcopenia or cachexia	May reduce maintenance and recovery potential
Catabolic/inflammatory burden	Disease-related inflammatory and metabolic pressure	Ongoing biological drive toward tissue breakdown or impaired anabolism	Whether nutritional provision will restore function	Can consume reserve despite adequate nutritional support

Disease-related sarcopenia	Muscle impairment occurring in the context of disease-associated mechanisms	Integrated muscle consequence of illness-related exposures	A single causal mechanism or inevitable dependence	Represents loss of one major component of reserve
Frailty	Broader vulnerability to stress across multiple systems	Reduced resilience and increased risk from physiological stressors	A synonym for sarcopenia	Modifies how muscle and nutritional deficits translate into function
ADL independence	Ability to perform meaningful everyday activities	Realized functional outcome in the current environment	Latent recovery capacity or the isolated cause of dependency	Endpoint produced by the interaction of capacity, demand, and context

Note: Categories are intentionally non-equivalent. A patient may meet criteria in several rows simultaneously, and movement in one domain does not imply proportional movement in another.

Muscle mass, strength, and performance

The differentiation among muscle quantity, strength, and performance is central to the functional-reserve account. Age-related patterns demonstrate that these components diverge [10], while physiological investigations indicate that neuromuscular impairment can contribute to reduced force production at different stages of sarcopenia [11]. Muscle quality helps explain why individuals with similar quantities of muscle may function differently. The observation does not establish that modifying muscle quality will restore independence, but it undermines the assumption that quantity alone captures the functional state of skeletal muscle [12].

Trajectory also matters. Among older adults with mobility limitations, adherence to physical activity has been associated with subsequent mobility-disability risk, with baseline functional status influencing the relationship [13]. This supports a dynamic view in which present impairment and future deterioration are not identical states. Accordingly, muscle evaluation in serious illness should be interpreted as a multidimensional profile. Low mass may indicate structural vulnerability; weakness may signal loss of usable force; poor performance may reveal integrated system limitation.

Nutritional risk

Nutritional vulnerability is closely linked with sarcopenia but requires separate conceptual treatment. In older adults attending a frailty clinic, poor nutritional status was associated with several definitions of sarcopenia, although the strength and presence of these associations varied according to the diagnostic framework used [14]. Across settings, malnutrition is common among older adults with sarcopenia, but prevalence varies substantially between hospital, institutional, and community populations [15]. More importantly, it indicates that care environment, disease burden, and selection processes influence how frequently nutritional and muscle impairments coincide.

Malnutrition can also add prognostic information when sarcopenia is present [16]. Yet association does not establish a universal sequence in which nutritional deterioration first causes muscle loss and muscle loss then causes functional dependence. In serious illness, reduced intake may precede muscle decline, follow worsening function, develop in parallel with systemic catabolism, or arise from treatment and symptom burden.

The Global Leadership Initiative on Malnutrition provides a useful distinction by requiring phenotypic and etiologic criteria for malnutrition after initial risk screening [17]. Nutritional risk identifies vulnerability; diagnostic malnutrition characterizes a more specific clinical state.

Inflammatory and catabolic burden

Inflammatory and catabolic processes can alter the relationship between nutritional intake and muscle function. These processes may impair anabolism even when food provision is increased, creating a biological context in which restoration of nutritional intake does not translate predictably into restoration of muscle or independence [18].

This distinction is essential for palliative reasoning. A person whose muscle loss is driven predominantly by reversible undernutrition presents a different intervention problem from a person with intense disease-related catabolism and rapidly declining physiological reserve.

Body composition can also complicate recognition. Sarcopenic obesity demonstrates that clinically relevant muscle impairment may coexist with excess adiposity and metabolic dysfunction [19, 20]. These configurations illustrate why visible wasting is an unreliable proxy for the extent of functional vulnerability.

Within a functional-reserve account, inflammatory and catabolic burden operates as a stressor that can consume or constrain the effective value of available nutritional and muscular resources.

Disease-related sarcopenia

Disease-related sarcopenia emerges when muscle impairment is embedded within active illness and its consequences. This clinical reality cautions against assigning disease-related muscle loss to a single pathway [21].

A disease-related configuration can include inflammatory catabolism, immobility, treatment toxicity, pain, fatigue, reduced oral intake, endocrine changes, hospitalization, and deconditioning. These exposures may reinforce one another. Simultaneously, active disease may reduce anabolic response and limit the benefit attainable from additional nutritional provision.

International cancer-cachexia guidance similarly emphasizes multimodal assessment and management, with disease stage and expected survival affecting the appropriateness of intervention [22]. Together, these sources support an interaction model in which muscle deterioration is embedded in a wider disease system [23].

Catabolism, malnutrition, inactivity, neuromuscular impairment, and treatment burden may all contribute, but their relative importance can differ between patients and across time.

Reduced oral intake and treatment burden

Reduced oral intake is one of the most clinically visible pathways through which serious illness can threaten nutritional and functional resources [24]. At the same time, nutritional intervention itself can create burden through monitoring, supplements, feeding procedures, appointments, adverse effects, or conflict with preferred patterns of eating and care.

Evidence from randomized nutritional interventions in incurable solid cancer illustrates the resulting uncertainty. It indicates that provision of nutrients cannot be assumed to restore function uniformly when disease mechanisms, baseline reserve, adherence, symptoms, and prognosis differ [25].

Implementation conditions further influence what care patients actually receive. A systematic review of strategies for integrating nutrition into cancer care identified knowledge and resource limitations as recurrent barriers and education and audit/feedback among commonly used implementation strategies [26].

Treatment burden must therefore be incorporated alongside anticipated benefit. The same escalation may offer little value when severe catabolism, short prognosis, or high burden sharply constrains the possibility of functional improvement.

Functional reserve

Functional reserve can now be specified more precisely. It should not be interpreted as a hidden quantity that can currently be measured with a single instrument. The construct instead organizes relationships among observable domains.

Functional ability research supports the need for this broader perspective. That validated index is distinct from the construct advanced here, but it demonstrates the limitation of interpreting function as a single muscular endpoint [27].

Exercise evidence further illustrates heterogeneity in recovery potential. Such variation is compatible with differences in reserve: the same intervention challenge may produce improvement, stability, or little observable benefit depending on baseline capacity and competing stressors [28, 29].

Functional reserve therefore occupies the conceptual space between present impairment and realized dependency. It may be partly buffered when strength is preserved, symptoms are controlled, food and assistance are accessible, activity remains possible, and compensatory strategies reduce task demands.

The resulting interaction pathways are summarized in **Table 2**.

Table 2. Mechanistic pathways that can alter functional reserve in disease-related sarcopenia

Functional-reserve pressure	Primary process	Potential interaction with muscle	Potential interaction with nutrition	Expected functional implication	Major interpretation boundary
Disease activity	Ongoing physiological stress and organ dysfunction	Accelerated weakness, deconditioning, impaired recovery	Appetite loss or altered metabolism	Smaller margin for maintaining activities	Disease severity may dominate the trajectory independently of sarcopenia
Inflammatory/catabolic load	Increased tissue breakdown and impaired anabolic response	Progressive loss of muscle quality or quantity	Nutrient provision may yield attenuated tissue response	Recovery may lag behind nutritional correction	High intake does not imply anabolic recovery
Reduced oral intake	Insufficient energy/protein and related nutrients	Less substrate for maintenance or rebuilding	Direct worsening of nutritional status	Reduced endurance and recovery potential	Intake deficit may be symptom-driven or goal-concordant
Inactivity and deconditioning	Reduced mechanical loading and cardiovascular challenge	Declining strength, power, and performance	May reduce appetite or ability to obtain/prepare food	Narrowing capability for everyday tasks	Immobility may reflect disease or treatment rather than muscle failure alone

Treatment toxicity/burden	Symptoms, fatigue, procedures, repeated healthcare demands	Reduced activity and direct or indirect muscle effects	Nausea, taste change, dysphagia, disrupted meals	Functional effort redirected toward treatment demands	More treatment may increase burden despite biological rationale
Frailty/multisystem vulnerability	Limited resilience across physiological systems	Greater effect of a given muscle deficit	Greater impact of nutritional perturbation	Earlier transition from impairment to dependence	Frailty remains broader than sarcopenia
Environmental support	Assistance, equipment, accessible meals, adapted tasks	Can compensate for reduced physical capacity	Can preserve access to food and eating support	Maintains realized independence despite impairment	Support changes observed function without necessarily changing biological capacity
Rehabilitation opportunity	Activity, exercise, task practice, symptom control	Potential preservation or improvement of usable capability	May act synergistically with adequate intake	May expand functional margin in selected patients	Response depends on prognosis, adherence, disease burden, and goals
Progressive multimorbidity	Competing physiological limitations	Muscle impairment becomes one of several constraints	Dietary needs or restrictions may conflict	Multiple simultaneous routes to dependency	Muscle-focused intervention may have limited effect when another system dominates

Note: The pathways are non-sequential. “Functional reserve” denotes the article’s analytical synthesis; it is not presented as a validated clinical score or diagnostic category.

Activities of daily living and independence

Activities of daily living provide an observable endpoint through which many underlying impairments become meaningful to patients and caregivers. Longitudinal evidence likewise associates the composite sarcopenia phenotype with later disability [8, 9].

Yet ADL performance is an integrated outcome. Balance, cognition, vision, cardiopulmonary reserve, pain, fatigue, motivation, assistive devices, housing, caregiving, and task expectations all influence whether a physical deficit becomes dependency.

The distinction between capacity and independence is especially important in palliative care. Intervention can therefore change independence through different routes: increasing capability, reducing task demand, improving symptom control, providing equipment, reorganizing care, or supporting caregivers.

Evidence that exercise interventions produce heterogeneous ADL outcomes in frail older adults reinforces this contextual interpretation [28]. A change in one biological or functional domain does not guarantee a proportional change in independence.

Feedback between functional decline and nutritional risk

The relationship between functional decline and nutritional deterioration can become reciprocal. These findings establish association; they do not by themselves demonstrate a self-sustaining feedback mechanism. The feedback interpretation arises when those associations are considered alongside the practical effects of declining function [14, 16].

Loss of mobility can reduce shopping, cooking, sitting tolerance, utensil use, meal attendance, or the ability to eat without assistance. Declining intake can then reduce the nutritional resources available for muscle maintenance, while continued disease burden may amplify catabolic losses.

The reverse pathway is similarly plausible. This can create a loop in which lower activity further accelerates deconditioning. The intensity of that loop should vary according to disease state, baseline strength, environmental support, symptom control, and the extent to which nutritional deterioration is reversible.

The study demonstrates that nutrition and activity can interact favorably under appropriate conditions; it does not establish that the same response is attainable during advanced catabolic disease. In incurable cancer, nutritional intervention results remain considerably more heterogeneous [25, 30].

The feedback account is therefore conditional. Functional deterioration may worsen nutrition, and nutritional deterioration may further impair function, but each connection can be weakened by assistance, rehabilitation, symptom relief, accessible meals, or stable disease.

Frailty and multimorbidity as modifiers

Frailty broadens the analysis beyond skeletal muscle by representing vulnerability across multiple physiological systems. Frailty therefore modifies the consequences of sarcopenia without being reducible to sarcopenia itself [31].

This distinction explains why similar muscle deficits can have very different effects. A patient with extensive frailty may have little compensatory margin, so the same decrement in muscle capability precipitates loss of mobility or self-care.

Multimorbidity adds another layer. Heart failure, chronic lung disease, neurological impairment, arthritis, renal disease, cognitive disorders, or sensory limitation may become the dominant constraint on independence even when sarcopenia is present. These factors establish competing pathways to disability and make it inappropriate to attribute every decline in an older person with low muscle mass to sarcopenia [19].

Frailty also changes intervention tolerance. Exercise, nutritional supplementation, diagnostic procedures, repeated appointments, and treatment escalation all impose demands. The relevant clinical state therefore reflects both what is biologically modifiable and what the person can reasonably tolerate.

Figure 1 depicts these relationships while retaining a separate route for competing causes of disability.

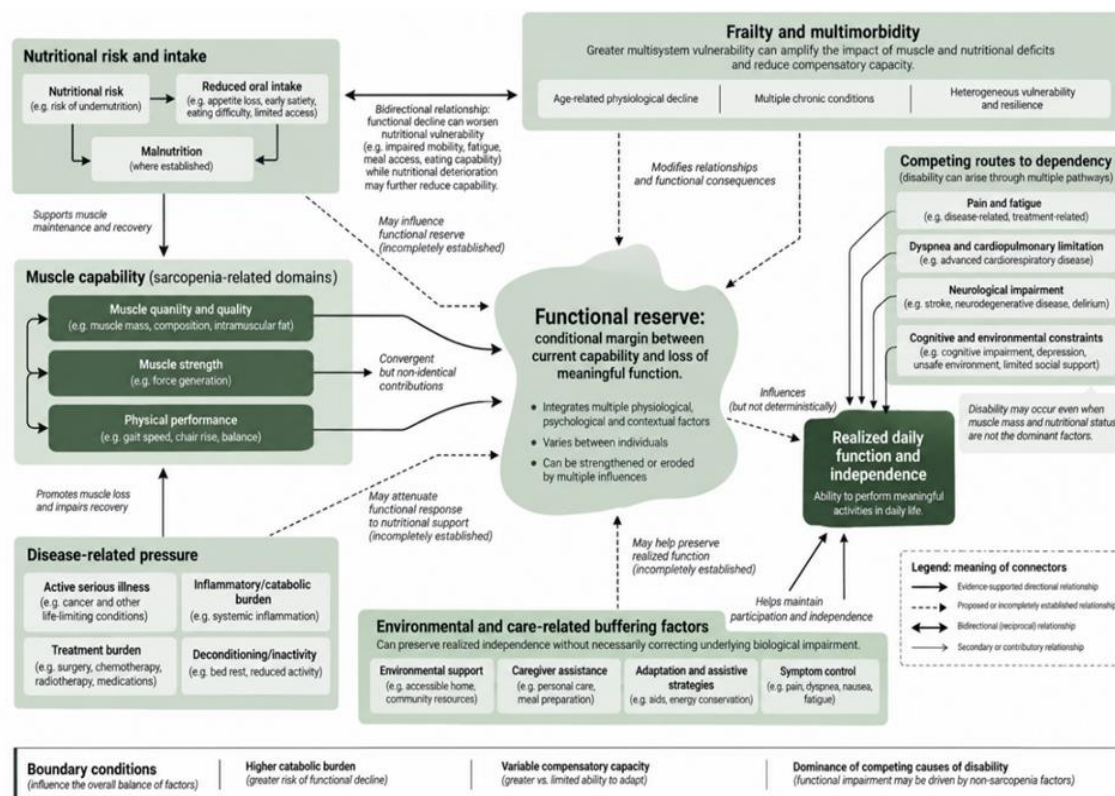


Figure 1. Conditional depletion and buffering of functional reserve in older adults with serious illness

Palliative goals and proportionality of intervention

In palliative care, identification of potentially modifiable muscle or nutritional deficits does not automatically determine intervention intensity. Expected survival and disease trajectory also influence whether nutritional, rehabilitative, pharmacological, or supportive strategies remain feasible and proportionate [21, 22].

Multicomponent cachexia interventions are similarly diverse, and adding more components does not itself establish greater clinical benefit [32]. The appropriate question is therefore not how many correctable abnormalities are detectable, but which interventions are likely to advance outcomes that remain meaningful to the individual [25].

Geriatric nutrition guidance likewise supports individualized decisions based on clinical condition, feasibility, and overall goals [33]. A strategy intended to restore mobility may be reasonable when nutritional depletion and reversible deconditioning dominate. The same strategy may offer little value when refractory catabolism, severe frailty, or another disabling disease process makes meaningful functional recovery improbable.

Palliative goals therefore establish a boundary around the interpretation of reserve. Preserving independence, making transfers safer, maintaining the ability to eat socially, or reducing fatigue may justify intervention even when normalization of muscle measurements is unrealistic [34-39].

Proposed sarcopenia–nutrition–function framework

Muscle quantity and quality contribute structural capacity; strength and physical performance indicate usable capability; nutritional status influences maintenance and recovery resources; inflammatory and catabolic burden can consume those resources; frailty and multimorbidity modify vulnerability; and environmental support changes the demands required to remain independent [40-43].

A patient with reduced muscle mass but preserved strength, stable intake, manageable symptoms, and supportive surroundings may retain substantial ability to compensate. Evidence showing that muscle quality and strength

contribute information beyond muscle quantity supports this differentiation [12], while multidimensional functional-ability research reinforces the importance of context when interpreting realized function [27]. Functional reserve is inferred from the relationship among current capability, stressors, modifiers, and subsequent trajectory; it is not proposed as a new diagnostic score. Stable function despite serious illness may indicate effective compensation, whereas rapid deterioration after a relatively modest stressor may signal a narrow reserve margin.

Palliative proportionality further changes how the framework is used. The decision that follows depends on the expected reversibility of the limiting pathway, competing causes of disability, intervention burden, prognosis, and patient goals.

Table 3. Clinical interpretation matrix for sarcopenia–nutrition–function configurations in palliative care

Clinical configuration	Dominant interpretation question	Assessment emphasis	Potential intervention orientation	Boundary condition preventing automatic escalation
Reduced muscle quantity with preserved strength and independence	Is structural loss currently limiting meaningful activity?	Strength, performance, trajectory, symptoms	Surveillance, activity preservation, nutrition review where relevant	Low muscle quantity alone does not establish dependency or treatment need
Weakness or poor performance with potentially reversible nutritional deficit	Is inadequate intake materially limiting recovery?	Intake, malnutrition criteria, symptoms, muscle capability	Symptom treatment, nutritional support, graded activity where tolerable	Benefit depends on disease burden and ability to translate nutrition into function
Progressive muscle decline with high inflammatory/catabolic burden	Is biological stress overwhelming anabolic recovery?	Disease activity, weight trajectory, strength, intake, functional change	Multimodal supportive management with realistic objectives	Greater nutritional provision may not reverse disease-driven wasting
Functional decline with substantial frailty or multimorbidity	Which system is primarily constraining independence?	Frailty, comorbid disease, ADL, cognition, mobility	Selective treatment of reversible contributors plus adaptation/support	Sarcopenia may be only one contributor to dependency
Preserved biological capability but environmental dependency	Can function be maintained by reducing task demands?	Home/care environment, assistance, equipment, meal access	Environmental modification, caregiver or service support	Dependency may improve without altering muscle biology
High treatment burden with limited recovery potential	Does intervention advance a goal that remains meaningful?	Prognosis, symptoms, treatment load, patient priorities	Comfort-focused or low-burden supportive care	Correctability of an abnormality does not establish proportional benefit
Recoverable deconditioning with acceptable burden	Is there sufficient reserve for rehabilitation to translate into meaningful function?	Baseline performance, activity tolerance, nutrition, goals	Goal-directed rehabilitation with nutritional support when indicated	Response remains individual and should be reassessed longitudinally

Note: These configurations are proposed interpretation zones rather than validated treatment categories, prognostic strata, or numerical thresholds.

Clinical assessment domains

Sarcopenia assessment can separate strength, muscle quantity or quality, and physical performance [6], while nutritional assessment should distinguish initial risk from established malnutrition and identify etiologic factors such as inadequate intake or disease burden [17, 23].

Current ADL performance, recent functional change, frailty, symptoms, treatment burden, cognitive status, mobility aids, caregiving support, and environmental demands help determine whether an observed biological deficit is translating into meaningful dependency [31].

Discordance—such as low muscle quantity with preserved performance, or severe functional loss with relatively preserved muscle—should trigger investigation of alternative pathways rather than forced classification within a muscle-centered explanation [44-49].

Goal-concordant intervention zones

Assessment can be translated into intervention zones without creating a rigid treatment ladder. One zone concerns potential restoration, where a reversible contributor such as inadequate intake, deconditioning, uncontrolled symptoms, or remediable treatment toxicity is limiting function and the patient wishes to pursue recovery. A second concerns maintenance and compensation, where complete recovery is unlikely but preserving transfers, mobility, eating, or self-care remains meaningful. A third concerns burden minimization, where progressive

disease, refractory catabolism, severe frailty, or short prognosis makes intensive intervention unlikely to produce proportional functional benefit.

These zones may overlap and can change over time. Cachexia guidance supports multimodal management but does not establish one intervention sequence appropriate for every patient [22, 26].

The governing principle is reassessment. An intervention should remain aligned with what it is achieving, what it costs the patient, and whether the target outcome continues to matter [50, 51].

Nursing implications

Nursing practice occupies a particularly important position because changes in eating, mobility, fatigue, self-care, symptoms, and treatment tolerance may become visible through repeated everyday contact before they are evident in isolated measurements [26, 33].

A reserve-oriented nursing assessment therefore emphasizes change over time. These observations gain meaning when linked with patient priorities and communicated across the interdisciplinary team.

The nursing role is consequently both observational and coordinative: identifying potentially reversible contributors, supporting low-burden adaptations, monitoring whether an intervention produces meaningful benefit, and recognizing when escalation no longer aligns with the patient's goals.

Research propositions

The framework generates propositions that can be tested rather than accepted as assumptions.

Proposition 1: Strength and physical performance should explain subsequent loss of independence beyond muscle quantity alone, particularly when muscle quality differs between individuals.

Proposition 2: Nutritional deterioration should have stronger functional consequences when baseline muscle capability or frailty indicates limited compensatory capacity.

Proposition 3: High inflammatory or catabolic burden should attenuate the association between increased nutritional provision and recovery of function.

Proposition 4: Repeated within-person assessment should discriminate functional trajectories more effectively than isolated sarcopenia or nutritional measurements.

Proposition 5: Frailty, multimorbidity, and environmental support should modify the translation of muscle impairment into ADL dependency.

Proposition 6: Goal-concordant interventions should produce different clinically meaningful endpoints according to reserve state; preservation of a valued activity may be an appropriate outcome even when muscle measurements do not normalize.

Prospective palliative-care cohorts with repeated muscle, nutritional, symptom, functional, and goal measurements are required to test these propositions.

Results and Discussion

The principal contribution of this article is the separation of impairment, reserve, and realized independence. The functional-reserve synthesis connects these domains without turning them into a new diagnostic syndrome [6] [17].

Low muscle mass can coexist with retained independence; nutritional support can improve intake without restoring function; severe dependency can arise through pathways that are only partly muscular; and meaningful clinical improvement can occur through adaptation or symptom control without normalization of body composition.

Multicomponent cachexia care can be reasonable, but current evidence remains heterogeneous [32]. Functional reserve therefore serves as an explanatory bridge between multidomain assessment and proportional action.

Limitations

The framework is limited by the evidence from which it is constructed. Findings should therefore not be assumed to transport unchanged across diagnoses, stages of illness, or care settings.

Definitions and measurement approaches also vary. Some of the proposed reciprocal relationships, especially the function–nutrition feedback loop, are supported by plausible directional evidence without having been established as complete causal cycles in palliative populations.

Most importantly, functional reserve is a proposed analytical construct in this article. It has not been validated as a clinical instrument, numerical score, diagnostic category, or prognostic classifier.

Conclusion

Sarcopenia, nutritional risk, and functional independence intersect in older adults receiving palliative care, but they should not be interpreted as a single linear sequence. Muscle quantity, muscle quality, strength, performance,

nutritional status, inflammatory and catabolic burden, frailty, disease trajectory, environmental support, and patient goals contribute differently to whether meaningful everyday function can be maintained.

It directs attention to the margin between current capability and dependency, to the conditions that consume or buffer that margin, and to the possibility that similar biological impairments can lead to different functional trajectories. The resulting interpretation is inherently conditional: correcting a measurable deficit is most meaningful when the pathway is sufficiently reversible, the intervention burden is proportionate, and the resulting function matters to the person receiving care.

Acknowledgments: None

Conflict of interest: None

Financial support: None

Ethics statement: None

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