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# Synergy of Protein Intake and Strength Exercise in Overcoming Anabolic Resistance in Sarcopenia Management: A Narrative Review

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**ABSTRACT**

**Background.** Sarcopenia is characterized by accelerated muscle loss, driven by chronic inflammation and anabolic resistance, which blunts mTORC1 pathway activation in aging skeletal muscle.

**Aim.** This narrative review analyzes the efficacy and synergistic effects of targeted protein supplementation and resistance exercise in countering sarcopenia.

**Material and methods.** A literature search of PubMed and Google Scholar was conducted to identify recent clinical guidelines and studies on protein supplementation, leucine thresholds, and mechanotransduction in older adults.

**Results.** Overcoming anabolic resistance requires a high intracellular leucine threshold of 2.5–3.0 g per meal. Whey protein effectively provides this rapid spike. Furthermore, progressive resistance training independently upregulates mTORC1 via mechanotransduction. Combining these interventions with specific adjuvants like creatine robustly sustains an anabolic environment.

**Conclusions.** Effective sarcopenia management requires shifting from passive treatments to active interventions combining mechanical loading with targeted nutritional triggers.

**Keywords:** Sarcopenia; Anabolic resistance; Resistance training; Protein supplementation; mTORC1

## 1. Introduction and Purpose

Sarcopenia is a progressive and generalized skeletal muscle disorder characterized by the accelerated loss of muscle mass, muscle strength, and physical performance [1, 2]. As the global population rapidly ages, sarcopenia has emerged as a major public health challenge, with an estimated prevalence ranging from 6% to 22% in adults aged 65 years and older [1, 3]. The clinical consequences are severe; this condition substantially contributes to physical frailty, functional decline, and an increased risk of falls and fractures [2, 3]. Consequently, sarcopenia profoundly diminishes the autonomy and overall quality of life of older adults while simultaneously increasing the risk of hospitalization and all-cause mortality, thereby placing a considerable economic burden on healthcare systems worldwide [1, 2, 3].

A key physiological mechanism driving the progression of sarcopenia is "anabolic resistance" defined as a blunted muscle protein synthesis (MPS) response to anabolic stimuli such as dietary amino acids and physical exertion [4, 5]. Because of this age-related metabolic shift, the current Recommended Dietary Allowance (RDA) for protein is widely recognized as insufficient to maintain skeletal muscle mass and counter sarcopenia in the elderly [4, 5]. Recent clinical evidence strongly suggests that older adults require a significantly higher daily protein intake to effectively overcome anabolic resistance and maximally stimulate MPS [3, 4, 6]. The aim of this study is to analyze the efficacy of protein supplementation in preventing and treating sarcopenia among older adults, specifically exploring the physiological impact of different protein sources and assessing their synergistic effects when combined with resistance exercise programs [1, 5].

To achieve this aim, a comprehensive literature search was conducted using the PubMed and Google Scholar databases. The search strategy focused on peer-reviewed articles, clinical guidelines, and meta-analyses published predominantly in the last decade. The primary keywords and MeSH terms included "sarcopenia", "anabolic resistance", "protein supplementation", "leucine threshold", and "resistance training".

## 2. Description of the State of Knowledge

### 2.1. Clinical Diagnosis and Screening of Sarcopenia

The clinical identification and diagnosis of sarcopenia have been refined by the revised European consensus (EWGSOP2). This consensus establishes a diagnostic pathway encapsulated in the F-A-C-S algorithm: Find cases, Assess muscle strength, Confirm muscle quantity or quality, and determine Severity [1]. The initial "Find" phase relies on opportunistic screening utilizing the SARC-F questionnaire, which evaluates perceived muscle strength,

assistance required for walking, difficulty rising from a chair, ability to climb stairs, and a recent history of falls [3, 7]. A cumulative score greater than or equal to 4 is indicative of sarcopenia risk, signaling the clinical necessity for formal physiological assessment [7]. To quickly identify individuals at risk of muscle decline in clinical practice, the SARC-F questionnaire is recommended [Table 1].

Following a positive screening, the 'Assess' phase quantifies muscle strength. The established clinical cut-off points for low handgrip strength are less than 27 kg for men and less than 16 kg for women [1]. Additionally, the Chair Stand Test serves as a robust proxy for evaluating lower extremity strength. A completion time for 5 rises exceeding 15 seconds is indicative of significant functional impairment [1]. If probable sarcopenia is detected, the "Confirm" phase necessitates the objective evaluation of muscle quantity via DXA or BIA to calculate the Skeletal Muscle Index (SMI = Appendicular Skeletal Muscle Mass / height²) [1, 8, 9]. Finally, severe sarcopenia is diagnosed when poor physical performance is confirmed, such as a gait speed of 0.8 m/s or less or a Timed Up and Go (TUG) test result of 20 seconds or more [1]. Furthermore, poor performance on these functional mobility assessments, particularly the Chair Stand and TUG tests, serves as a strong independent predictor of future physical disability and loss of independence in community-dwelling older adults [10]. "The practical application of this diagnostic pathway is based on the F-A-C-S algorithm [Figure 1].

**Table 1. SARC-F screening questionnaire for sarcopenia risk assessment.**

| Component | Question | Scoring (Points) |
|---|---|---|
| **Strength** | How much difficulty do you have in lifting and carrying 10 pounds? | None = 0<br>Some = 1<br>A lot or unable = 2 |
| **Assistance in walking** | How much difficulty do you have walking across a room? | None = 0<br>Some = 1<br>A lot, use aids, or unable = 2 |

| | | |
|---|---|---|
| **Rise from a chair** | How much difficulty do you have transferring from a chair or bed? | None = 0<br>Some = 1<br>A lot or unable without help = 2 |
| **Climb stairs** | How much difficulty do you have climbing a flight of 10 stairs? | None = 0<br>Some = 1<br>A lot or unable = 2 |
| **Falls** | How many times have you fallen in the past year? | None = 0<br>1-3 falls = 1<br>4 or more falls = 2 |

***Interpretation:*** *A total score of ≥ 4 points indicates a risk of sarcopenia and necessity for further clinical assessment.* ***Source:*** *[7].*

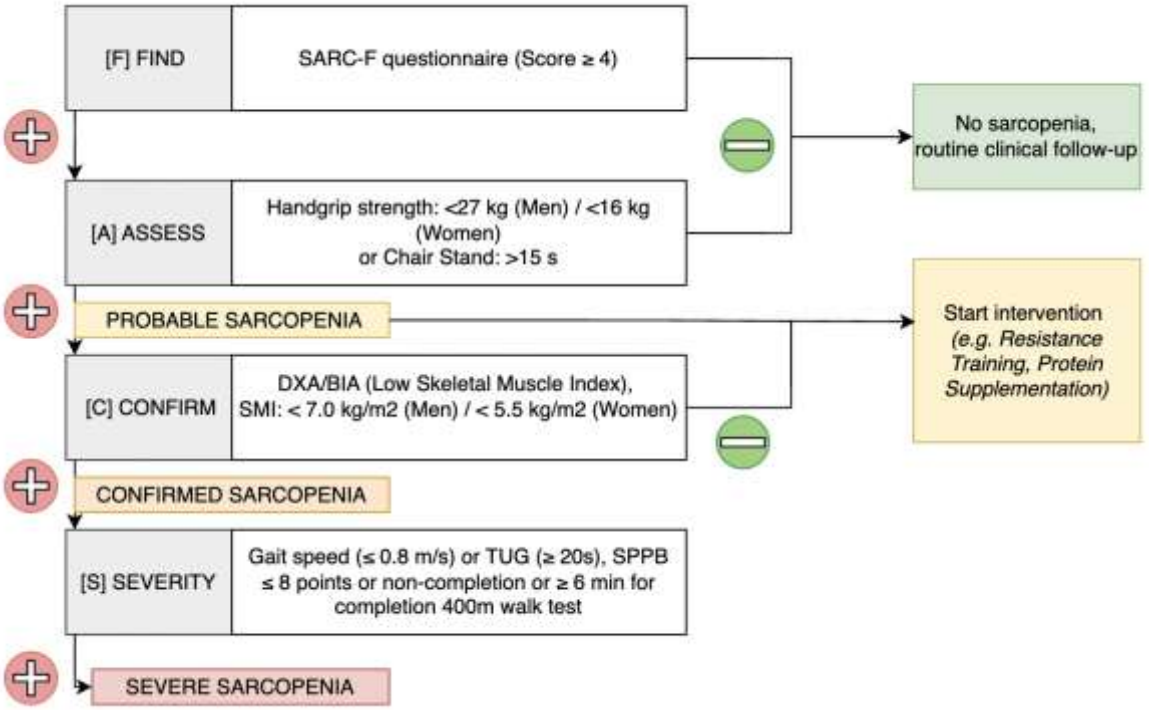


**Figure 1.** Clinical pathway for the identification and diagnosis of sarcopenia using the F-A-C-S algorithm (Find, Assess, Confirm, Severity).

***Abbreviations:*** ***BIA*** *– Bioelectrical Impedance Analysis;* ***DXA*** *– Dual-energy X-ray Absorptiometry;* ***SARC-F*** *– Strength, Assistance in walking, Rise from a chair, Climb stairs, and Falls questionnaire;* ***SMI*** *– Skeletal Muscle Index;* ***SPPB*** *– Short Physical Performance Battery;* ***TUG*** *– Timed Up and Go test.*

***Source****: Own elaboration based on [1].*

### 2.2. Pathophysiological Basis: Anabolic Resistance and Inflammaging

Sarcopenia is driven by a chronic imbalance between muscle protein synthesis (MPS) and muscle protein breakdown (MPB) [11]. In older adults, skeletal muscle develops "anabolic resistance," defined as a blunted mTORC1 activation in response to anabolic stimuli [11,12]. This impairment is heavily exacerbated by "inflammaging", a state of chronic, low-grade systemic inflammation characterized by elevated pro-inflammatory cytokines, notably TNF-alpha and IL-6 [11,13]. At the molecular level, TNF-alpha upregulates the ubiquitin-proteasome proteolytic system via E3 ligases (MuRF1 and MAFbx), directly accelerating muscle protein degradation [14]. Morphologically, this toxic environment induces selective atrophy of type II (fast-twitch) muscle fibers, leading to a profound loss of neuromuscular strength and mobility [11,12]. Clinically, this cascade translates into a significantly higher risk

of falls among older adults, which is often reflected by poor performance in functional assessments such as the Timed Up and Go (TUG) test [15].

### 2.3. Molecular Mechanisms of Muscle Protein Synthesis

#### The mTORC1 Signaling Cascade

The mechanistic target of rapamycin complex 1 (mTORC1) serves as the master regulatory kinase dictating skeletal muscle protein synthesis (MPS) [11, 13]. Upon stimulation by extracellular growth factors (e.g., IGF-1) or mechanical loading, the PI3K-Akt signaling axis acts upstream to fully activate this complex [11, 14]. Once activated, mTORC1 directly phosphorylates two critical downstream translation targets: the 70-kDa ribosomal protein S6 kinase 1 (p70S6K1) and the eukaryotic translation initiation factor 4E-binding protein 1 (4E-BP1) [11,14]. The phosphorylation of 4E-BP1 induces its dissociation from eIF4E, enabling the assembly of the translation pre-initiation complex. Simultaneously, the activation of p70S6K1 drives ribosomal biogenesis and peptide elongation [14]. Ultimately, this molecular cascade shifts the dynamic equilibrium of protein turnover toward net myofibrillar accretion, a fundamental requirement for preserving muscle mass and countering age-related atrophy [5,11]. Thus, the combined activation of the mTORC1 pathway by mechanical loading and amino acid influx remains the primary driver of muscle protein synthesis in older adults [Figure 2].

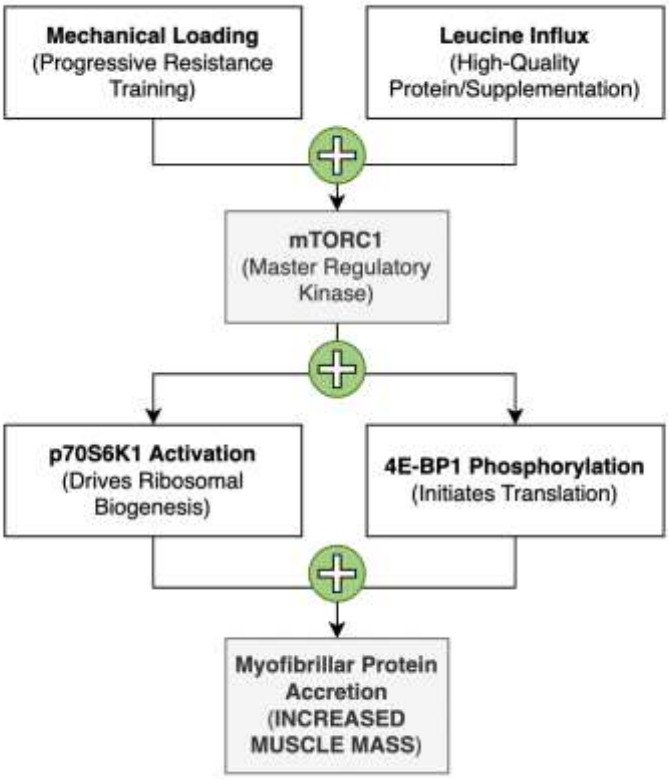


***Figure 2.*** *Synergistic activation of the mTORC1 signaling pathway by mechanical loading and leucine influx.* ***Source:*** *Own elaboration based on [11,14,15,16].*

**The Leucine Sensing Mechanism**

Leucine acts not merely as a structural building block for tissue synthesis but as a potent, independent metabolic signaling molecule that directly triggers the mTORC1 pathway [5,11]. Its intracellular concentration is actively monitored by Sestrin2, a highly specific leucine sensor protein [15]. Under leucine-deprived conditions, Sestrin2 binds to and inhibits GATOR2 [15]. However, upon a significant intracellular leucine influx, the amino acid directly binds to Sestrin2, disrupting the Sestrin2-GATOR2 interaction [15]. This release allows GATOR2 to suppress GATOR1, a known negative regulator of the pathway. The inhibition of GATOR1 ultimately allows the activation of Rag GTPases, which recruit mTORC1 to the lysosomal surface for full activation [15]. This intricate sensing mechanism explains why a sharp peak in intracellular leucine levels is the primary prerequisite for switching on postprandial MPS [5, 11, 15].

**The mTOR-AMPK Crosstalk and Anabolic Resistance**

In addition to targeted nutritional triggers, mechanical loading from resistance exercise induces mechanotransduction, the physiological process by which muscle cells convert mechanical

force and tension into intracellular biochemical signals, which independently stimulates the mTORC1 pathway [16]. However, this anabolic signaling is constantly modulated by the 5'-AMP-activated protein kinase (AMPK), an intracellular energy sensor [15]. When cellular energy is depleted, characterized by an elevated AMP/ATP ratio, AMPK is phosphorylated and acts to suppress mTORC1, shifting the cell toward catabolism to conserve energy [15]. In older adults, the sensitivity of the entire PI3K-Akt-mTOR cascade is severely blunted, creating a physiological state termed "anabolic resistance" [5, 16]. Consequently, the aging muscle exhibits a profound insensitivity to typical anabolic stimuli, requiring significantly higher doses of leucine to breach the elevated anabolic threshold, overcome baseline AMPK-mediated inhibition, and trigger a robust molecular response [4, 16].

### 2.4. Protein Quality, Dietary Patterns, and Individual Considerations

The anabolic efficacy of dietary protein is dictated by its digestion kinetics and Essential Amino Acid (EAA) profile [16]. Animal proteins exert a superior effect on preserving muscle mass compared to plant-based proteins due to higher Digestible Indispensable Amino Acid Scores (DIAAS) [17]. Animal proteins typically exhibit digestibility rates exceeding 90%, whereas plant proteins often reach only 50% due to secondary structural differences and antinutritional factors [12, 17]. Whey protein, a fast-digesting animal protein, induces swift hyperaminoacidemia and a rapid peak in circulating leucine, ranking as the most effective supplement for stimulating postprandial MPS [11, 16, 18]. However, focusing solely on individual protein sources is insufficient if the overall dietary pattern is poor. For instance, high consumption of ultra-processed foods (UPF), which often displace high-quality nutrients, has been directly linked to lower muscle strength, particularly in older women [19]. Furthermore, holistic sarcopenia management must account for individual demographic variables. Ethnic differences, for example, must be considered in clinical practice, as they significantly influence the interpretation of normal Vitamin D levels [3] and dictate the selection of appropriate prediction equations for accurately assessing baseline skeletal muscle mass [8, 9].

### 2.5. The Molecular "Leucine Trigger" and Threshold in Aging

Building upon the importance of protein quality, the specific mechanism driving muscle synthesis in older adults relies heavily on the molecular "leucine trigger." Clinical evidence suggests that to successfully overcome the anabolic threshold, older adults require a dietary intake of 2.5 to 3.0 g of leucine per meal to induce the necessary intracellular signaling spike [4, 6 , 20]. Given that leucine constitutes only about 8–10% of the amino acids in most dietary

proteins, achieving this critical per-meal threshold typically requires the consumption of 25 to 30 g of high-quality protein in a single sitting [4, 6, 16]. Furthermore, to maximize 24-hour anabolism, implementing a pre-sleep feeding strategy with 40 g of protein has been suggested to effectively stimulate overnight MPS in aging populations [6]. Intracellular leucine directly activates the mTORC1 pathway to initiate translation and drive MPS [15,16]. Because of this specific physiological requirement, the choice of protein source becomes crucial. Specifically, whey protein due to its rapid absorption kinetics and naturally high leucine content has proven to be exceptionally effective. This is supported by a recent network meta-analysis by Liao et al. (2024), which demonstrated that whey protein supplementation yields the highest probability of success in improving both muscle mass and handgrip strength compared to other protein sources [18].

### 2.6. Clinical Safety and Renal Considerations

While overcoming anabolic resistance relies on absolute per-meal protein doses to trigger muscle synthesis, total daily requirements are traditionally scaled to body weight to evaluate overall nitrogen balance and organ safety. Increasing dietary protein to 1.2–1.5 g/kg/day does not intrinsically damage renal function in older adults with normal baseline health [4, 6]. A randomized trial demonstrated that supplementing leucine-enriched protein did not impair kidney function, maintaining the estimated glomerular filtration rate (eGFR) within healthy, normal ranges [20]. However, for older adults with advanced Chronic Kidney Disease (CKD) who are not on dialysis and do not have diabetes, dietary protein must be restricted to 0.55–0.60 g/kg/day [6, 11]. For CKD patients who concurrently manage diabetes, the recommendation slightly increases to 0.6–0.8 g/kg/day [6]. Due to these severe restrictions, it is crucial that the majority of consumed protein comes from high-value sources to ensure an adequate supply of essential amino acids and counteract malnutrition [11]. Conversely, once maintenance dialysis begins, protein needs paradoxically increase to 1.0–1.2 g/kg/day or higher to compensate for severe amino acid losses during treatment [11]. Accurate assessment remains a challenge, as self-reported protein intake often diverges from objective biomarkers like 24-hour urinary nitrogen excretion, consistently overestimating actual protein consumption [21].

### 2.7. The Limits of Passive Management: Pharmacotherapy, Diet, and the Absolute Necessity of Exercise

Beyond targeted dietary interventions, the pharmacological management of prevalent metabolic comorbidities must also prioritize muscle preservation in older adults. For instance, in patients

with type 2 diabetes, modern metabolic treatments such as empagliflozin successfully facilitate significant fat loss and glycemic control without exerting detrimental effects on skeletal muscle mass or strength, thereby safely preserving baseline muscle function **[22]**. However, it is crucial to recognize the limits of such passive management. Ultimately, regardless of optimized pharmacological control and high-quality protein intake, isolated interventions remain inadequate for reversing sarcopenia. A recent systematic review confirmed this stark clinical reality, demonstrating that in physically inactive older adults, protein supplementation fails to exert any significant anabolic effect on lean body mass **[23]**. This reinforces the absolute necessity of a concurrent, active exercise stimulus to prime the muscle and fully utilize nutritional and pharmacological support.

### 2.8. Synergistic Power: Combining Resistance Training (RT) with Protein

The synergistic combination of progressive resistance training (RT) and targeted protein supplementation represents the most potent clinical countermeasure against sarcopenia [24, 25]. This synergy is driven by mechanotransduction, where mechanical loading translates into biochemical signals that upregulate the mTORC1 pathway, profoundly sensitizing aging skeletal muscle to circulating amino acids and shifting the metabolic balance toward a highly anabolic state [16]. While traditional guidelines often advocate for higher RT intensities (e.g., 65–80% of One-Repetition Maximum, 1RM = the maximum weight a person can lift for one complete repetition) performed 2 to 3 times per week [4], recent evidence emphasizes that such heavy loads may be unfeasible or unsafe for older adults with osteoarticular comorbidities [16, 24]. Importantly, low-load, high-volume RT (e.g., 20–30% 1RM) performed to volitional fatigue has been shown to stimulate muscle protein synthesis and promote hypertrophy comparably to high-load training, offering a much safer alternative for the geriatric population [16]. Furthermore, recent systematic reviews highlight that to maximize functional adaptations, such as handgrip strength and gait speed, RT should focus on multi-joint, compound movements. By successfully countering this age-related muscle decline, the combined intervention is not only vital for functional independence but also serves as a fundamental strategy to preserve postural balance and significantly reduce the risk of falls in older adults [26]. To safely achieve these crucial clinical outcomes and optimize neuromuscular adaptations, older adults should aim to complete 2 to 3 sets of 8 to 12 repetitions per major muscle group; however, frail individuals or absolute beginners should safely initiate training with a single set before progressively increasing the training volume [25, 27]. Additionally, session durations should be kept relatively short (≤40 minutes) to prevent excessive fatigue and ensure adherence

[25]. Ultimately, a systematic progression of this mechanical load, when consistently paired with adequate protein provision, is essential to prevent physiological plateaus and successfully override chronic anabolic resistance [16, 25]. To further amplify this protection against falls and maximize functional independence, these traditional resistance protocols can be optimized through high-velocity power training. While standard progressive loading effectively triggers mechanotransduction, performing resistance exercises at higher concentric velocities has been shown to induce superior improvements in maximal strength, muscular power output, and functional capacity in older adults compared to traditional slow-speed training [27]. Because explosive force generation is critical for rapid balance recovery during a postural perturbation, incorporating this power-focused stimulus provides a highly specific strategy to mitigate fall risk [27].

### 2.9. Emerging Nutritional Adjuvants: Creatine, Omega-3, and HMB

Beyond mechanical optimization, specific nutritional adjuvants can be layered upon basic protein provision to further override chronic anabolic resistance. Creatine monohydrate supplementation, when combined with resistance training, significantly augments lean tissue mass and upper- and lower-body strength in older adults [28]. At the cellular level, creatine acts as a crucial phosphate buffer to rapidly resynthesize ATP; by preventing an excessive rise in the intracellular AMP/ATP ratio during muscular exertion, creatine mitigates the AMPK-mediated suppression of the mTORC1 pathway, thereby sustaining a highly anabolic environment [28]. Notably, this enhanced energy buffering directly translates to improved sit-to-stand performance, a critical clinical predictor of fall risk, synergizing with the functional goals of power training [28]. Furthermore, overcoming age-related anabolic resistance requires addressing underlying systemic factors such as low-grade inflammation. Supplementation with Omega-3 polyunsaturated fatty acids has been demonstrated to sensitize aging skeletal muscle to amino acids, effectively augmenting the mTORC1-mediated muscle protein synthetic response and facilitating greater adaptations to resistance exercise [29]. Finally, targeted metabolites such as β-hydroxy-β-methylbutyrate (HMB), a downstream derivative of leucine, offer dual therapeutic action. HMB not only stimulates muscle protein synthesis via the mTORC1 pathway but also attenuates muscle protein breakdown by inhibiting the ubiquitin-proteasome system, yielding significant improvements in body composition and strength when paired with mechanical loading [30]. Together, these advanced nutritional modalities represent the next frontier in comprehensively countering sarcopenia.

## 3. Conclusions

In summary, the clinical management of sarcopenia requires a paradigm shift away from passive, isolated treatments toward active and synergistic interventions. As highlighted throughout this review, age-related muscle decline is driven by complex physiological barriers, most notably chronic anabolic resistance and low-grade systemic inflammation. Consequently, passive strategies, such as isolated dietary adjustments or the pharmacological management of metabolic comorbidities, are fundamentally inadequate to restore muscle mass and function on their own. Instead, the most potent countermeasure against sarcopenia lies in the continuous and concurrent application of progressive mechanical loading and targeted nutritional triggers. Resistance training, particularly when incorporating power movements, provides the essential mechanotransduction signals required to sensitize aging muscle. When this mechanical stimulus is consistently paired with optimized protein provision and specific anabolic adjuvants like creatine, Omega-3 fatty acids, and HMB, the mTORC1 pathway is robustly upregulated, shifting the skeletal muscle milieu from a catabolic state to a highly anabolic one. Ultimately, the success of these combined interventions hinges on clinical adherence and the implementation of highly personalized approaches tailored to the geriatric population. Exercise parameters and nutritional prescriptions must be carefully calibrated to account for osteoarticular comorbidities, renal safety, and individual functional limitations. By prioritizing this comprehensive and synergistic methodology, clinicians can effectively override anabolic resistance, preserve postural balance, and sustain long-term functional independence in older adults.

## 4. Disclosure

**Authors' Contributions:**

**Conceptualization:** Ł.W.

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**Investigation:** Ł.W., J.C., H.T.B., Z.M., M.S., F.J., N.G., A.D., K.Z., M.J.

**Writing – original draft:** Ł.W., J.C., H.T.B., F.J., N.G.

**Writing – review and editing:** Ł.W., J.C., H.T.B., Z.M., M.S., F.J., N.G., A.D., K.Z., M.J.

**Visualization:** Ł.W., J.C., H.T.B., Z.M., M.S., M.J., A.D.

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