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The Dual Burden of Malnutrition and Oral Disease: An Updated Framework for Family Medicine, Dental Care, Nutrition Care, and Community Health Security

Intisar Kalaf Albalawi [(1)] , Mugbil Mohammed Alotaibi [(2)] , Fedha Khamis Nasser Abadi [(3)] , Ali Bader Alsulami [(4)] , Azzah Atitallah Ali Alzahrani [(5)] , Abdullah Ali Mutair Alzahrani [(6)] , Abdulaziz Saeed Alghamdi [(7)] , Mohammed Hebah Badawi [(8)] , Ahmad Abdulsattar Zaghoun [(9)] , Marwan Saud Alharbi [(10)] , Mathail Lafi Alanazi [(11)]

(1) Compliance Department, Ministry Of Health - Riyadh Branch, Saudi Arabia,
(2) Primary Care Center Aldar Alaida, Ministry of Health, Saudi Arabia,
(3) Ministry Of Health, Saudi Arabia,
(4) Erada Mental Health Complex Riyadh, Ministry of Health, Saudi Arabia,
(5) Al-Malaz Phc , Cluster 1 Al-Riyadh, Ministry of Health, Saudi Arabia,
(6) Jeedah - King Abdulaziz Hospital, Ministry of Health, Saudi Arabia,
(7) Ministry Of Health Branch In Jeddah, Saudi Arabia,
(8) Eradah Hospital For Mental Health, Ministry of Health, Saudi Arabia,
(9) Healthcare Strategies - Madinah Cluster, Ministry of Health, Saudi Arabia,
(10) Bed Management - Madinah Cluster, Ministry of Health, Saudi Arabia,
(11) Chest Clinic In Alrawadh, Ministry of Health, Saudi Arabia.

Abstract

Background: The aging population faces a complex, bidirectional relationship between oral health and nutritional status. Age-related physiological decline, compounded by oral diseases like periodontitis, caries, and xerostomia, directly impairs masticatory function, leading to poor nutrition. This, in turn, accelerates systemic conditions like sarcopenia and frailty, creating a vicious cycle of decline.

Aim: This review aims to synthesize an updated framework elucidating the interconnected pathways between oral dysfunction and malnutrition, and to propose integrated, multidisciplinary intervention strategies for family medicine, dental care, and community health.

Methods: The article employs a comprehensive literature review methodology, analyzing the impact of specific oral conditions (edentulism, dry mouth, periodontal disease) on dietary intake and nutritional biomarkers. It further examines the shared pathophysiology, such as chronic inflammation, that links oral disease to systemic muscle loss (sarcopenia).

Results: The analysis confirms that poor oral health is a primary driver of malnutrition in older adults, leading to reduced intake of protein, fiber, and essential micronutrients. Concurrently, oral inflammatory diseases exacerbate systemic inflammation, directly contributing to sarcopenia and frailty, thereby increasing disability and mortality risk.

Conclusion: Breaking the cycle of oral disease and malnutrition requires a collaborative, person-centered approach. Effective management integrates dental rehabilitation, personalized nutrition, and physical therapy within a Comprehensive Geriatric Assessment framework.

Keywords: Oral Health, Malnutrition, Sarcopenia, Aging, Frailty, Interdisciplinary Care, Comprehensive Geriatric Assessment..

Introduction

Advancing age is accompanied by a progressive, multisystem decline in physiological reserve that heightens susceptibility to internal and external stressors and increases the probability of adverse clinical outcomes across care settings [1,2,3]. This biological trajectory intersects with a spectrum of sensory, metabolic, and functional alterations that collectively reshape dietary behaviors and nutritional status in late life. Diminished olfactory and gustatory perception, the emergence of the so-called "anorexia of aging," and age-related reductions in energy expenditure converge to reduce caloric intake and dietary diversity, thereby predisposing older individuals to macro- and micronutrient deficiencies that may accelerate functional decline [4,5,6]. Superimposed illness burden and polypharmacy further perturb appetite regulation and nutrient handling, while oral conditions—loss of teeth, ill-fitting dentures, and mucosal discomfort—amplify aversions to critical food textures, compounding the risk for qualitative and quantitative malnutrition over

*Corresponding author e-mail: Entesarr.K@Gmail.Com (Intisar Kalaf Albalawi).
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time [4,5,6]. Beyond strictly biomedical drivers, late-life nutrition is shaped by social determinants and daily living capacities: living or eating alone, limited financial resources, dismobility, and reduced capacity to shop for or prepare meals erode the stability of food access and the capacity to adhere to therapeutic dietary plans [7,8]. Psychosocial conditions—loneliness, sleep disturbance, cognitive impairment, and depressive syndromes—exert additional, bidirectional effects on appetite, meal timing, and executive function related to food preparation and adherence, thereby deepening vulnerability to inadequate intake and weight loss in community and institutional contexts [9].

Concurrently, aging is characterized by a progressive remodeling of body composition in which skeletal muscle mass and contractile strength decline while total fat mass and intramuscular adipose infiltration increase, a dyad that undermines mobility, metabolic health, and quality of life [10,11]. The construct of sarcopenia, first articulated by Irwin Rosenberg in 1988, captures the pathological reduction of muscle mass and strength culminating in impaired performance and disability, and it has since been refined to encompass structural and neuromuscular dimensions of function [12,13]. Contemporary evidence indicates that sarcopenia is not confined to antigravity or locomotive muscle groups but reflects a whole-body process that includes orofacial, pharyngeal, and respiratory musculature integral to chewing and swallowing [14,15,16]. The atrophy and functional decline of these muscle systems can directly curtail oral intake by lowering bite force, degrading bolus formation, and increasing the effort and perceived risk of deglutition, thereby creating a feed-forward loop in which undernutrition accelerates muscle catabolism and functional compromise [10,17]. Reports of atrophic change in muscles central to ventilatory mechanics and swallowing further underscore the systemic reach of age-related myopathy and its relevance to aspiration risk, hospitalizations, and mortality in geriatric populations [14,18,19,20,21,22].

Oral health status represents a pivotal, yet often underappreciated, determinant of nutritional trajectories in late life. The heterogeneous dental morbidities experienced by older adults—periodontal disease, caries, edentulism, and prosthodontic maladaptation—translate into chewing inefficiency and selective avoidance of fiber-rich fruits, vegetables, and protein-dense foods, thereby narrowing dietary quality and precipitating weight loss and nutrient deficiencies that heighten frailty risk [23]. These pathways intersect mechanistically and clinically with sarcopenia, as reduced protein and energy intake potentiates muscle wasting while masticatory dysfunction limits the practical feasibility of high-quality diets that could otherwise attenuate catabolism [10,23]. In parallel, chronic low-grade systemic inflammation linked to periodontal disease represents an additional axis through which oral pathology can exacerbate frailty and sarcopenic processes, potentially via inflammatory mediators that impair muscle protein synthesis and promote catabolic signaling [24,25]. The prevalence of periodontal disease is elevated among older adults who cannot consistently perform oral hygiene because of functional limitations, cognitive impairment, or lack of caregiver support, which magnifies cumulative inflammatory exposure and further embeds oral health within the causal architecture of frailty [26,27]. Epidemiological and longitudinal data strengthen this connection, associating periodontitis with accelerated declines in handgrip strength and functional metrics relevant to independent living, while implicating chewing difficulties themselves as correlates of frailty phenotypes in late life [28,29,24].

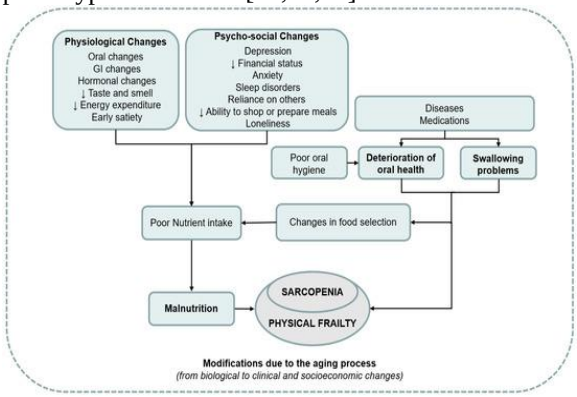

Fig. 1: Interplay between poor oral status, malnutrition, and sarcopenia. GI—gastrointestinal.

Taken together, these observations delineate an interdependent network in which sensory decline, psychosocial stressors, social isolation, and financial constraints converge with oral dysfunction and systemic inflammation to shape diet quality, energy balance, and muscle integrity in older adults [7,8,9]. The resulting malnutrition–sarcopenia–frailty nexus manifests clinically as reduced resilience to acute illness, greater post-hospitalization disability, and heightened care needs, reinforcing the imperative for integrated assessment strategies that bridge dental, nutritional, and geriatric domains [10,11,23]. Strategically, this implies routine screening for taste and smell disturbance, structured evaluation of chewing and swallowing function, targeted periodontal assessment and treatment, and proactive identification of social barriers to meal acquisition and preparation, each aligned with individualized nutrition support to interrupt the downward spiral of anorexia, inflammation, and muscle loss [24,25,26,27]. As populations age, embedding oral health and nutrition as coequal pillars of geriatric care will be essential to mitigate sarcopenia and frailty,

preserve independence, and improve health trajectories in later life [12,13,14,15,16].

Oral Changes with Aging

Poor oral health is not an inevitable consequence of aging; rather, longitudinal prevention, routine professional care, and sustained self-care can preserve functional dentitions well into advanced age, underscoring the life-course nature of oral health equity [24]. Nevertheless, the aging dentition undergoes predictable physiological modifications that shape risk profiles and clinical decision-making. At the crown level, enamel exhibits cumulative microstructural changes—including craze lines, surface staining, and localized translucency—while lifelong attrition and abrasion expose underlying dentin, producing color darkening and altered light transmission; within the tooth, secondary dentin apposition progressively reduces the dimensions of the pulp chamber and root canals, complicating endodontic diagnostics and therapy and modulating nociceptive responses to caries or thermal stimuli [30]. Superimposed occlusal and incisal wear are highly prevalent in later life; epidemiologic evaluations report tooth wear in more than 85% of teeth across both maxillary and mandibular arches in older adults, reflecting the aggregate effects of mastication, parafunction, and erosive challenges [31]. In parallel, age-related diminutions in elastic fibers within the connective tissues lead to oral mucosa that is thinner and less resilient, with delayed recovery from trauma and heightened susceptibility to denture-related lesions and inflammatory conditions [32]. Beyond intrinsic tissue changes, social and structural determinants shape oral outcomes. Older adults—particularly those who are institutionalized or have constrained financial means—encounter substantial barriers to timely dental care, including transportation, caregiver availability, and benefit coverage. Compounding these barriers, many elders struggle to articulate oral complaints or prioritize them amid multimorbidity, leading to deferred presentation until pain or dysfunction becomes intolerable [33]. The clinical sequelae are well documented: high burdens of tooth loss, dental caries, periodontal disease, xerostomia, and an increased incidence of oral potentially malignant disorders and cancers have been reported among aging populations, with functional implications for mastication, speech, and nutrition [34]. Notably, periodontitis and dental caries remain the most common conditions in late life and are the principal drivers of edentulism, with downstream effects on diet quality and systemic health [35]. Around the seventh decade, root and cementum caries peak, a pattern attributed to prolonged tooth retention combined with gingival recession and loss of periodontal attachment that expose root surfaces to biofilm and dietary acids; concomitantly, the cumulative, site-specific nature of periodontal tissue breakdown places multirooted teeth at particular risk for furcation involvement and rapid periodontal deterioration in older adults [36]. Collectively, these age-linked biological changes and access challenges require proactive, tailored strategies—preventive remineralization, root-surface caries control, periodontal maintenance, mucosal surveillance, and pragmatic prosthodontic solutions—to maintain function, comfort, and oral-systemic health across the aging continuum [24,30–36].

Edentulism

Edentulism, whether partial or total, represents a pathological endpoint of cumulative oral disease and health system shortfalls, marked by multiple missing teeth and the attendant loss of occlusal function, aesthetics, and oral–systemic integrity. Its etiology is multifactorial and spans individual predisposition, diet quality, hormonal milieu, comorbid conditions, hygiene practices, and patterns of dental service utilization, while clinical precipitants commonly include refractory periodontitis and advanced carious destruction that prove unamenable to tooth-preserving therapy [37,38]. Importantly, tooth loss is not an intrinsic feature of biological aging; rather, when it occurs, it typically reflects preventable trajectories of neglected self-care, delayed attendance, or inadequate or inaccessible treatment over the life course [39,40]. The consequences of unrehabilitated edentulism are profound. When masticatory function is not restored with appropriately designed and maintained prostheses—whether removable or implant-supported—the biomechanical capacity to comminute food is compromised, prompting avoidance of fibrous fruits and vegetables and protein-dense textures in favor of highly processed, softer alternatives [41,42]. This diet shift is clinically significant: edentulous individuals exhibit a higher risk of macro- and micronutrient deficiencies than their dentate or partially dentate counterparts, with downstream vulnerability to malnutrition, sarcopenia, and clinical frailty phenotypes [43,25]. The ripple effects extend into activities of daily living and social participation; tooth loss becomes a risk factor for disability by undermining self-sufficiency in eating, speech, and social interaction, thereby worsening perceived quality of life and potentially accelerating dependence and institutionalization [42]. From a population health standpoint, reducing edentulism requires integrated prevention that couples upstream caries and periodontal control with equitable prosthodontic rehabilitation that restores chewing efficiency and nutrition to interrupt the malnutrition–frailty cascade [37–43].

Dry Mouth

Saliva is pivotal to oral function, acting as a lubricant, buffer, antimicrobial medium, and vehicle for remineralizing ions; it is integral to bolus formation and the sensory–textural experience that governs swallow safety and dietary choices. Xerostomia denotes the subjective sensation of oral

dryness and can occur with or without measurable salivary gland hypofunction, underscoring the need to distinguish symptom from secretion in assessment and management [30,44]. Prevalence estimates indicate that xerostomia affects roughly 25–50% of older adults, reflecting the convergence of polypharmacy, multimorbidity, and age-related changes in oral sensory perception [45]. Pharmacologic contributors are particularly salient: antihypertensives, antidepressants, antipsychotics, and numerous anticholinergic agents reduce salivary output or alter mucosal wetness perception, while systemic diseases, overall poor health, female sex, and increasing age further elevate risk [46–48]. In addition, therapeutic irradiation for head and neck cancers can permanently damage major salivary glands, leading to persistent, functionally devastating xerostomia that complicates nutrition, speech, and prosthesis wear [49]. Although aging itself is associated with reduced flow in some salivary glands, these changes cannot be wholly attributed to medications, and the literature emphasizes that salivary hypofunction and xerostomia are distinct constructs frequently and improperly conflated in both research and clinical practice [50,33]. Intriguingly, up to one third of elders reporting xerostomia demonstrate normal flow on sialometry, suggesting a psychological or central perceptual component in symptom reporting and highlighting the need for multidimensional evaluation [30]. Nevertheless, true hyposalivation has tangible functional consequences: reduced volume and altered rheology of saliva impair bolus cohesion, lubricity, and oral clearance, compromising chewing efficiency, prolonging oral processing time, and increasing the effort and risk of swallowing. These deficits not only diminish meal enjoyment but also jeopardize the early digestive process and can precipitate nutritional compromise in already vulnerable older adults [51].

Periodontal Disease

Periodontitis is a chronic, biofilm-mediated inflammatory disorder of the tooth-supporting tissues that culminates in progressive attachment loss, alveolar bone resorption, and periodontal pocketing, with clinical sequelae that include tooth mobility, migration, and, ultimately, exfoliation [52]. Masticatory efficiency declines as occlusion destabilizes, intensifying difficulty with tougher textures and contributing to dietary simplification that undermines nutrition. Epidemiologic data in older cohorts reveal wide variability in severe disease prevalence—operationalized, for example, by periodontal index scores with deep pockets (score 4)—ranging between approximately 5% and 70%, reflecting heterogeneity in exposure to risk factors, access to care, and diagnostic thresholds across studies and settings [53]. As a cumulative disease, periodontitis tends to worsen with age when unmitigated, as the burden of historical attachment loss and bone destruction accrues, although aging per se is not deterministic; rather, ongoing plaque control and maintenance can stabilize disease trajectories [30]. At the proximal level, poor oral hygiene drives dysbiotic plaque accumulation and microbial virulence, initiating and perpetuating host inflammatory responses that mediate connective tissue breakdown [54]. Systemic and behavioral cofactors—including tobacco use, poorly controlled diabetes, obesity, psychosocial stress, osteopenia, and suboptimal dietary calcium and vitamin D—modulate susceptibility and progression, demonstrating the multidirectional nexus between oral and systemic health [55]. Given shared pathways of chronic inflammation, mounting evidence links periodontitis with adverse systemic outcomes, including worsened glycemic control in diabetes, elevated cardiovascular risk, adverse pregnancy outcomes, and associations with rheumatoid arthritis, though causal inference and bidirectionality continue to be investigated [56]. Nutrition both shapes and is shaped by periodontal status. Diets low in fruits, vegetables, and micronutrients may amplify periodontal inflammatory responses, while specific nutrient patterns—lower intakes of docosahexaenoic acid, vitamins C and E, β-carotene, milk and fermented dairy, fiber, fruits, and vegetables, and higher omega-6/omega-3 ratios and saturated fats—have been associated with poorer periodontal health in community-dwelling elders [57]. Micronutrient adequacy also influences post-surgical healing, with deficiencies linked to impaired recovery following periodontal procedures [58]. Conversely, tooth loss from periodontitis constrains food choice, often steering patients toward soft, refined diets that perpetuate nutritional deficits and systemic vulnerability, creating a reinforcing cycle of oral disease and malnutrition.

Dental Caries

Dental caries remains one of the most prevalent chronic conditions across the lifespan and retains particular salience in older adults. It is a multifactorial, biofilm-driven disease in which fermentable carbohydrate metabolism yields organic acids that demineralize enamel and dentin; over time, structural compromise progresses from subsurface lesions to cavitation, pulp involvement, and, in advanced cases, tooth fracture and loss if untreated [59]. Among community-dwelling older adults, caries prevalence varies between 20% and 60%, rising to 60–80% in long-term care settings where functional and cognitive impairments, care dependency, and limited access to dental services exacerbate risk [60–64]. Predisposing conditions include frequent consumption of simple sugars, diabetes with its salivary and immune alterations, and socioeconomic disadvantages that limit preventive care, fluoride exposure, and timely restorative treatment [60,65–

68]. Aging-associated physical frailty and cognitive decline complicate daily plaque control and regular attendance, allowing incipient lesions to progress; restorations placed years earlier may fail at margins or require extension, increasing the risk of cusp fracture and, ultimately, endodontic involvement [30]. When pulpal disease ensues, root canal treatment becomes necessary to eradicate infection and preserve the tooth, entailing removal of inflamed or necrotic pulp from coronal and radicular spaces and obturation with gutta-percha to re-establish a bacteria-tight seal. Salivary dysfunction magnifies caries risk. Xerostomia and hyposalivation reduce buffering capacity, diminish calcium and phosphate availability for remineralization, and impair the mechanical clearance of sugars and acids, thereby fostering acidogenic and aciduric bacterial proliferation, accelerating mineral loss from exposed enamel and root surfaces, and compromising mucosal lubrication essential for comfortable prosthesis wear [69]. Clinically, this constellation predisposes older adults to rampant root/cementum caries, secondary caries at restoration margins, and cervical lesions, with a particularly sharp rise around the seventh decade as gingival recession and periodontal attachment loss expose vulnerable root substrates [36]. Effective caries control in late life consequently demands comprehensive strategies that integrate salivary management, fluoride and calcium–phosphate therapies, dietary counseling to reduce fermentable sugar frequency, meticulous plaque control tailored to functional capacity, and timely, minimally invasive restorative approaches to preserve structure and function. Across these domains—edentulism, xerostomia, periodontitis, and caries—the through-line is clear: oral conditions common in older adults are neither inevitable nor inconsequential. They interact bidirectionally with nutrition, systemic health, and functional status, shaping trajectories of independence and quality of life. Preventive, rehabilitative, and interprofessional models that prioritize early detection, risk-based maintenance, prosthetic restoration of function, and management of salivary and inflammatory milieus are essential to break the cycle linking oral disease to malnutrition, sarcopenia, and frailty in aging populations [25,30,33,37–69].

Impact of Oral Health on Nutritional Status

Nutrition is a central determinant of health and functionality in older adults, influencing immune competence, physical resilience, and the capacity to withstand physiological stress. Inadequate nutrient intake is widely recognized as a contributing factor in the onset and progression of numerous chronic diseases, while also playing a crucial role in the complex etiology of sarcopenia and frailty syndromes [70,71,72]. As aging advances, functional decline in several physiological domains—including oral health—compromises dietary adequacy and heightens the risk of malnutrition. Oral disorders, medication effects, and systemic illness can converge to exacerbate anorexia and diminish appetite regulation, leading to reduced consumption of nutrient-dense foods [5,70,73]. Consequently, poor oral health in older individuals often leads to selective food avoidance, limited dietary variety, and ultimately reduced nutrient intake [25]. The link between oral health and systemic disease is increasingly well established. Oral dysfunction not only affects eating ability but also acts as both a mediator and a marker for chronic diseases such as diabetes [74] and cardiovascular disease [75,76,77]. Masticatory performance, one of the most critical factors influencing nutrition, depends on several anatomical and physiological elements: the number of teeth in functional occlusion [78–80], maximal bite force [81,82], proper fit of dentures [83], and adequate salivary flow [84]. Functional occlusion—defined as at least twenty teeth with ten contiguous pairs in each arch—is a benchmark for efficient chewing and bolus formation [85]. When this condition is not met, mechanical efficiency in food processing declines, causing alterations in texture preference and reduced overall nutrient assimilation.

Tooth loss, particularly when unreplaced, markedly diminishes chewing ability and hinders bolus formation, leading to an increased particle size and impaired swallowing function [86,87]. Even among edentulous individuals with high-quality dentures, chewing efficiency remains significantly lower than in dentate peers [88]. This reduction in masticatory performance often precipitates adaptive behaviors, with older individuals opting for softer, more easily chewed foods at the expense of fibrous fruits, vegetables, and protein-rich meats [89,90]. These substitutions have profound nutritional implications, as such foods are principal sources of proteins, vitamins, minerals, and fiber [41,88,91]. Deficiencies in these nutrients are linked to greater risks of malnutrition, sarcopenia, and frailty, conditions that collectively compromise independence and quality of life [24,92]. Micronutrient deficiencies, even when subtle, can initiate oxidative stress and systemic inflammation, both of which accelerate muscle catabolism and exacerbate frailty while simultaneously heightening susceptibility to periodontal disease. Impaired nutrition can also affect enamel remineralization, predisposing individuals to dental caries [93]. Likewise, undernutrition may weaken immune defenses, amplifying the severity of oral infections and their systemic inflammatory impact [94]. Furthermore, older adults frequently gravitate toward soft, processed foods to alleviate chewing fatigue, yet these foods are often high in fats and simple sugars while being poor in essential vitamins and minerals [10,95]. Such dietary profiles promote fat accumulation, oxidative stress, and chronic inflammation—pathophysiologic mechanisms that increase the risk for cardiovascular disease, metabolic

syndrome, and obesity-related oral disorders [88,95,96,97].

Obesity itself is now recognized as a chronic low-grade inflammatory state. The excessive energy stored in adipocytes leads to cellular hypertrophy and hyperplasia, driving mitochondrial stress and endoplasmic reticulum dysfunction, while macrophage infiltration of adipose tissue induces further oxidative damage [98,99]. This inflammatory milieu not only worsens systemic metabolic control but also exacerbates oral conditions such as periodontal disease, dental caries, and eventual tooth loss. Moreover, excessive consumption of simple sugars—common in energy-dense, low-nutrient foods—remains a principal etiologic factor for dental caries, fostering acidogenic bacterial proliferation and enamel demineralization [100,101]. Epidemiological evidence underscores the bidirectional link between oral status and nutrition. Large-scale population surveys, such as the UK National Diet and Nutrition Survey (NDNS) [102] and the US National Health and Nutrition Examination Surveys (NHANES) [103,104], have demonstrated strong associations between poor dental health and inadequate nutrient intake in older adults. Edentulous individuals, regardless of prosthetic rehabilitation, consistently consume fewer fruits and vegetables, leading to deficiencies in dietary fiber, antioxidants, and micronutrients. Similarly, individuals with fewer than twenty-one natural teeth exhibit reduced intake of proteins and vitamins and a corresponding rise in carbohydrate consumption [104]. These findings illustrate how oral dysfunction reshapes dietary patterns and contributes to nutritional decline at the population level. In summary, oral health exerts a profound influence on the nutritional well-being of older adults through multiple interconnected pathways. Tooth loss, masticatory insufficiency, xerostomia, and oral pain limit food variety and nutrient adequacy, while inflammatory oral diseases contribute to systemic metabolic disturbances that reciprocally impair oral integrity. Addressing these challenges requires a multidisciplinary strategy that integrates dental rehabilitation, nutritional counseling, salivary management, and chronic disease prevention. By preserving oral functionality and promoting balanced dietary habits, clinicians can significantly mitigate malnutrition, frailty, and sarcopenia—ultimately enhancing longevity and quality of life in the aging population [70–104].

Sarcopenia and Oral Status

Sarcopenia, defined as the progressive and accelerated loss of muscle mass and function, has emerged as a leading biological substrate for adverse geriatric outcomes, including frailty, incident disability, hospitalization, and mortality [13,105]. Although classically associated with advanced age, sarcopenia may begin earlier in the life course under the influence of disease, lifestyle, and environmental factors, reinforcing its relevance well beyond late life [106]. Since 2016, sarcopenia has been formalized as an independent clinical entity with an ICD-10-CM code, a development that has catalyzed standardized ascertainment, reimbursement pathways, and research comparability across settings [107]. Conceptual advances have been consolidated in the updated European Working Group on Sarcopenia in Older People consensus (EWGSOP2), which re-centers low muscle strength as the primary criterion, reserves low muscle quantity or quality to confirm the diagnosis, and identifies impaired physical performance as a marker of severe sarcopenia, while also operationalizing specific cut-points and a pragmatic algorithm for case finding in clinical practice [106]. This diagnostic pivot toward strength—rather than mass alone—aligns with functional risk, prognostic salience, and therapeutic logic, and it is particularly germane when considering craniofacial and oropharyngeal musculature whose failure manifests as dysphagia, malnutrition, and aspiration. The pathogenesis of sarcopenia is multifactorial, interweaving behavioral, disease-related, and age-dependent biological processes into a self-reinforcing network of decline. Malnutrition and physical inactivity deprive muscle of the anabolic signals required to maintain protein synthesis and neuromuscular integrity; inflammatory diseases, endocrine perturbations, and chronic organ dysfunction compound this deficit; and fundamental age-associated changes—including chronic low-grade inflammation, mitochondrial dysfunction, denervation and motor unit remodeling, satellite cell depletion, and hormonal alterations—advance the catabolic milieu [108,109]. Crucially, sarcopenia is a whole-body process. It does not spare respiratory, masticatory, or deglutitive muscle groups, whose functional erosion undermines ventilation, chewing, and swallowing—competencies that are indispensable to nutrition, safety, and quality of life [14,18,19,20,21,22]. Swallowing itself is a complex, sensorimotor sequence dependent on the precise, temporally coordinated contraction of multiple head and neck muscles; age-related changes in tissue elasticity, craniofacial anatomy, oral and pharyngeal sensation, and dental status can subtly degrade this choreography, producing “presbyphagia,” a subclinical state in which performance is preserved at rest but fragile under stress [110,16,111]. Presbyphagia heightens vulnerability to overt dysphagia and aspiration when acute illness, hospitalization, or other stressors disrupt the compensatory reserve typical of healthy elders [112].

Morphometric and physiologic studies document reductions in the size of the geniohyoid, pterygoid, masseter, tongue, and pharyngeal muscles with aging, alongside measurable declines in the strength and endurance of swallowing muscles in sarcopenic states [20,113,114,115,116]. Maximal

tongue pressure—a surrogate for the force-generating capacity of lingual musculature—declines with age, and converging evidence links tongue strength to global muscle strength, including handgrip, suggesting shared systemic determinants of neuromuscular performance [116,117,118,119,120,121]. Functionally, lower tongue strength associates with impairments in activities of daily living, while reduced tongue thickness is observed in individuals with low body weight, underlining the nutritional–muscular feedback loop that couples oral performance with systemic status [122,20]. The lips contribute a seemingly modest but functionally indispensable role by providing anterior oral competence; poor lip closure permits anterior leakage and undermines bolus control, while decreased lip strength—reported with aging and sarcopenia—correlates with eating and drinking difficulties and exhibits associations with handgrip and clinical markers of aging, such as lip pendency [123,117,124]. These findings substantiate a broader construct, "sarcopenic dysphagia," which recognizes that systemic skeletal muscle loss entails concomitant weakness of the swallowing apparatus, predisposing to impaired intake, aspiration risk, and malnutrition [22,124,125]. The vulnerability of swallowing to sarcopenia reflects, in part, muscle fiber biology: deglutitive muscles contain a high proportion of type II fibers that are selectively sensitive to disuse, malnutrition, and inflammatory catabolism [22]. Yet, heterogeneity exists across craniofacial muscles; jaw-closers such as the masseter display a higher proportion of type I fibers and may be relatively more sensitive to inactivity than to aging per se, offering a pathophysiological explanation for the observed decline in masseter and tongue activity when diets shift toward softer textures that demand less power, a common adaptive pattern in older adults with dental problems [126,127].

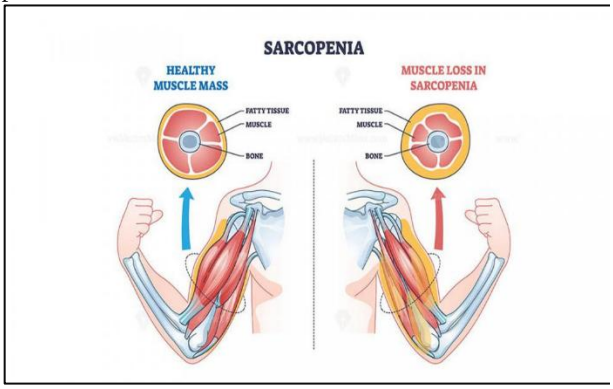

Fig. 2: Sarcopenia.

Oral pathology is not merely a downstream consequence of sarcopenia; it can act upstream as well, amplifying systemic catabolism and weakness. Periodontal disease, a prevalent chronic infection and inflammatory condition, exemplifies how the oral niche can fuel systemic low-grade inflammation, a shared pathway in the causation of frailty and sarcopenia [25,128,129]. The inflammatory burden emanating from periodontitis is not confined to the gingival sulcus; bacteremia, translocation of microbial products, and cytokine spillover provoke systemic metabolic dysregulation, fostering insulin resistance, dysglycemia, and heightened cardiovascular risk, and are associated with rheumatologic disease activity—all conditions that accelerate muscle wasting and functional decline [130–134]. At the cellular level, mitochondrial abnormalities provide a mechanistic bridge between oral inflammation and systemic sarcopenia. Excess reactive oxygen species (ROS) production, a hallmark of mitochondrial dysfunction, is implicated in both periodontal tissue destruction and skeletal muscle catabolism, with cross-talk through autophagic signaling pathways that regulate organelle quality control and proteostasis [108,135,136,137]. Elevated ROS in periodontitis may perturb autophagy, impairing cellular housekeeping and accelerating tissue injury [138]. Consistent with this, patients with periodontitis exhibit increased mitochondrial-derived ROS and evidence of mitochondrial dysfunction in circulating mononuclear cells, linking local oral inflammation to systemic bioenergetic stress [139]. The gingival connective tissue appears highly responsive to bacterial lipopolysaccharides (LPS), particularly from Porphyromonas gingivalis, which evokes robust cytokine production and has been implicated in mitochondrial ROS generation, coenzyme Q10 alterations, and respiratory chain complex perturbation, collectively driving oxidative stress and organelle dysfunction [140,138,139]. Clinically, the systemic reverberations of periodontal disease include measurable decrements in physical performance; accelerated declines in handgrip strength have been reported in association with periodontitis, strengthening the epidemiological connection between oral inflammation and sarcopenic trajectories [29].

The nutritional vector is equally central to the sarcopenia–oral health nexus. As detailed in earlier sections, the spectrum of dental problems experienced by older adults—edentulism, caries, poorly fitting prostheses, xerostomia—reduces masticatory performance, constrains texture tolerance, and fosters selective avoidance of nutrient-dense foods, eroding protein and micronutrient intake [25]. The cumulative effect is a self-perpetuating cycle in which poor oral status precipitates malnutrition, malnutrition accelerates muscle loss, and sarcopenia further degrades swallowing and chewing, thereby worsening oral intake and nutritional status. The clinical footprint of this cycle is conspicuous in high-acuity settings: poor oral status is observed in nearly three quarters of patients in rehabilitation units and in the vast majority of those in acute-care hospitals, with robust associations

to malnutrition, dysphagia, and diminished activities of daily living [141,142,17]. Superimposed inflammation then compounds the problem. Inflammatory mediators suppress appetite, alter hypothalamic signaling, increase resting energy expenditure, and potentiate muscle protein breakdown, tightening the catabolic vise that links chronic disease, oral infection, and systemic wasting [143]. The overlap between sarcopenia and frailty is substantial and clinically important. Both constructs share antecedents in inflammation, endocrine dysregulation, and reduced physical activity, and both converge on the phenotype of diminished strength, endurance, and physiologic reserve [144,145,146]. In practice, distinguishing their contributions to functional decline may be less important than recognizing their co-occurrence and shared modifiability. Oral problems, whether considered individually or collectively, may constitute a tangible and tractable biological substrate within the broader disabling cascade: by precipitating dysphagia, reducing diet quality, and sustaining systemic inflammation, they can tip vulnerable individuals from robustness into frailty, especially when combined with pre-existing sarcopenia. This recognition carries practical implications. Screening for sarcopenia in older adults who present with oral disease or feeding difficulties should be routine, leveraging EWGSOP2 case-finding algorithms tied to simple bedside measures such as handgrip strength and gait speed, followed by confirmatory assessments of muscle quantity and quality where available [106]. Conversely, dental and oral functional assessments should be embedded within sarcopenia evaluations, with particular attention to tongue pressure, masticatory efficiency, denture fit, salivary function, and periodontal status, given the bidirectional influence of these domains on intake and inflammation [116,117,118,119,123].

Intervention strategies necessarily span disciplines. Nutritional support must prioritize adequate high-quality protein, leucine-rich sources, and correction of micronutrient deficits to restore anabolism, while exercise programs incorporating progressive resistance and functional training counter the loss of strength and power that underlies dysphagia risk and mobility decline [108,109]. Dental rehabilitation—ranging from periodontal therapy and caries control to prosthodontic restoration that re-establishes functional occlusion—can directly improve chewing efficiency and diet breadth, thereby amplifying the effectiveness of nutrition and exercise interventions. Management of xerostomia and salivary hypofunction, including medication review, salivary stimulants or substitutes, and meticulous oral hygiene, may mitigate caries risk and enhance bolus formation, while targeted swallowing therapy can rebuild oropharyngeal strength and coordination in sarcopenic dysphagia [30,44,51,124,125]. Finally, reduction of oral inflammatory burden through periodontal care holds promise not only for tooth retention but also for systemic anti-inflammatory gain, potentially easing the catabolic pressure on skeletal muscle and improving functional outcomes [25,128–131,134–139]. In sum, sarcopenia and oral status are entwined through shared biological pathways and reciprocal functional dependencies. The decline of skeletal muscle mass and strength encompasses the craniofacial and deglutitive musculature, rendering eating and swallowing vulnerable to the same catabolic forces that weaken the limbs [14,18–22,116]. In parallel, oral inflammatory disease and mechanical insufficiency propagate malnutrition and systemic inflammation, accelerating sarcopenic change and hastening the transition to frailty [25,128,129]. Recognition of this bidirectional relationship reframes oral health from a peripheral concern to a central determinant of muscular and functional aging. By aligning diagnostic frameworks such as EWGSOP2 with integrated dental, nutritional, and rehabilitative care, clinicians can interrupt the vicious cycle of poor intake, inflammation, and muscle loss, thereby improving resilience, independence, and survival in older adults [106,108,109,141–146].

Interventions

The management of older people should be multimodal and multidisciplinary, especially for those with or at risk of malnutrition [147], in order to improve interdependent conditions such as oral problems and sarcopenia. From a practical standpoint, comprehensive geriatric assessment (CGA) offers a structured, multidimensional diagnostic and therapeutic process to profile medical, psychological, and functional issues, align them with patient priorities, and coordinate a coherent plan. The objective of CGA is to develop an integrated program for treatment and follow-up that maximizes overall health with aging, embeds preventive targets, and anticipates deterioration before crises occur [148]. In this framework, dentistry, nutrition, rehabilitation, nursing, and primary care work in concert rather than sequence. Growing evidence indicates that prosthodontic rehabilitation combined with personalized dietary counselling can improve nutritional status and functional outcomes, particularly when the plan is iteratively adjusted to tolerance and goals [51]. Below is an integrated overview of oral management, nutritional interventions, and exercise-based rehabilitation aimed at interrupting the malnutrition–inflammation–sarcopenia cycle.

Oral Management

The stomatognathic system is vulnerable to cumulative inflammatory and mechanical insults over time, yet with prevention and timely care it can be preserved across the life course [30]. A central

challenge is regular surveillance: older adults frequently struggle to sustain daily oral hygiene and often present late, so clinicians should implement risk-stratified recalls, reinforce teachable self-care routines, and involve caregivers when independence is limited [34]. Person-centered care begins by assessing dependency level, medical complexity, and physical or cognitive impairment, then translating those constraints into a practical oral-health plan that combines professional care with feasible self-care tasks [150]. In many settings, access barriers and waiting lists complicate timely treatment, so private and public providers alike should cultivate geriatric-aware pathways that prioritize symptomatic disease, pain, and nutrition-critical function while planning staged rehabilitation [149]. Disease-directed therapy should prioritize tooth preservation and control of inflammation. Active caries demands prompt, minimally invasive restoration to prevent structural collapse; where pulpal disease exists, endodontic therapy allows retention of occluding units that are essential for mastication and diet breadth [30]. Periodontal care requires both risk modification—smoking cessation, glycemic control, medication review, and plaque control—and professional therapy ranging from nonsurgical instrumentation to surgical procedures when indicated, aiming to reduce pocket depth, stabilize attachment, and lower inflammatory load. Because edentulism exacerbates malnutrition risk, prosthetic rehabilitation—removable, fixed, or implant-supported—should restore functional occlusion and chewing efficiency to enable intake of fiber-rich fruits, vegetables, and protein-dense foods [151]. Management of xerostomia is equally important: saliva substitutes, salivary stimulants, meticulous caries prevention, and rational polypharmacy reduction can mitigate discomfort, reduce caries progression, and improve bolus formation [30]. Throughout, education of patients and caregivers about daily hygiene, denture care, and warning signs (pain, looseness, mucosal ulceration) sustains gains between visits and facilitates early re-entry to care.

Nutritional Interventions

Nutrition is a cornerstone of late-life health. Plans should ensure adequate energy, protein, fluid, and micronutrient intake tailored to individual preferences, comorbidity, and functional status, with explicit goals to prevent or correct deficiencies and improve morbidity and mortality. Assessment precedes prescription: clinicians should document weight trajectory, appetite, oral function, body composition, disease activity, medications, and frailty, using validated tools to stratify risk and to guide monitoring [152]. As a general guide, ESPEN recommends ~30 kcal/kg/day, adapted to clinical context and tolerance [152]. Both ESPEN and the PROT-AGE group advise at least 1.0 g protein/kg/day in older adults, increasing to 1.2–1.5 g/kg/day during acute or chronic illness; because the per-meal anabolic threshold is higher with aging, distributing 25–30 g protein per meal with ~2.5–2.8 g leucine can optimize muscle protein synthesis [147,153]. When appetite or early satiety limits intake, oral nutritional supplements or fortified foods can close energy-protein gaps. Given age-related declines in serum vitamin D, and its associations with reduced muscle mass and strength, supplementation should be considered in those who are deficient, using laboratory guidance to avoid under- or over-replacement [154,155].

Texture adaptation aligns foods with chewing and swallowing capacity to minimize choking and aspiration risk while preserving enjoyment. Hard foods may be modified to bite-sized, minced, or pureed consistencies; liquids can be thickened to slow bolus flow and improve control, with adjustments individualized and revisited as oropharyngeal function improves through therapy [10,156,157]. Caries prevention and metabolic balance both require limiting simple sugars; the World Health Organization recommends restricting free sugars to <10% of total energy intake to reduce caries risk and cardiometabolic strain [101,159]. Emphasizing whole foods—vegetables, fruits, legumes, whole grains, nuts, and dairy or fortified alternatives—improves micronutrient density without excessive glycemic load. Caution is warranted with antioxidant supplements in non-deficient individuals, as excessive doses may blunt exercise adaptations or exert pro-oxidant effects; diet-first strategies are preferred [160]. Dietary intake of fatty fish (e.g., salmon, mackerel, herring, sardines, lake trout, albacore tuna) contributes marine omega-3 fatty acids, which are associated with greater fat-free mass and may improve inflammatory status in periodontal disease and sarcopenia, though optimal dosing and long-term effects require further study [161–164]. Practical adherence tactics—small, frequent protein-rich meals; flavor enhancement; social mealtimes; and denture refitting to reduce soreness—often determine success.

Exercise and Rehabilitative Strategies

Physical inactivity is a principal cause of sarcopenia because it induces resistance to muscle anabolic stimuli and accelerates neuromuscular deconditioning [165,166]. Sedentary behavior may also increase periodontal disease risk, highlighting a shared inflammatory and behavioral pathway between movement and oral health [167]. Progressive resistance training is the most effective countermeasure, improving strength, power, and muscle quality; because sarcopenia is systemic [15,21], programs should be holistic, targeting all major muscle groups at sufficient intensity and volume, with progression, supervision when feasible, and attention to balance and gait to reduce falls [168]. Importantly, masticatory and swallowing muscles respond to training as well. Targeted oral exercises—including lip stretching and strengthening, tongue

protrusion and elevation against resistance, cheek and masseter activation, and sequenced swallowing movements—have improved subjective chewing, swallowing function, salivation, oral dryness, and oral-health quality of life in clinical reports [169–171]. Respiratory/swallowing cross-training such as expiratory muscle resistance training enhances suprahyoid activation and airway protection [172,173]; head-lift exercises (e.g., Shaker) facilitate hyolaryngeal elevation and upper esophageal opening [174,175]; and tongue-strengthening regimens increase maximal tongue pressure and pressure accuracy, improving bolus control and oral intake [176–178]. Notably, tongue exercises have been reported to prevent general sarcopenia, suggesting systemic benefits beyond deglutition [178,179]. The synergistic effect of nutrition with exercise is substantial: protein-timed feeding around training augments anabolism, while improved oral function broadens dietary texture and variety, enabling adequate protein and fiber intake [164,167]. Rehabilitation should be embedded within CGA so that goals, contraindications, and supports (assistive devices, caregiver coaching) are aligned, adherence barriers are anticipated, and progress is tracked with meaningful functional outcomes. In sum, effective intervention for older adults at risk of malnutrition and sarcopenia integrates oral management, personalized nutrition, and structured exercise within a CGA scaffold. Such coordination restores safe, efficient intake; reduces inflammatory and infectious burdens; and rebuilds muscle performance while respecting preferences and capacities. By operationalizing the cited recommendations—prosthodontic restoration with caries and periodontal control, energy-protein targets with texture optimization and sugar restraint, and progressive resistance with targeted oropharyngeal training—clinicians can interrupt the downward spiral linking oral disease, undernutrition, and sarcopenia, thereby improving function, resilience, and quality of life in aging populations [148,151–159,161–179].

Conclusion:

In conclusion, the evidence firmly establishes a powerful, bidirectional relationship between oral health and the dual burden of malnutrition and sarcopenia in older adults. Oral diseases—including tooth loss, periodontal infection, and salivary dysfunction—directly impair chewing and swallowing, leading to impoverished diets deficient in protein and micronutrients. This nutritional decline fuels a cascade of systemic consequences, most notably the acceleration of sarcopenia and frailty. Concurrently, the chronic inflammatory state generated by conditions like periodontitis contributes to a catabolic milieu that exacerbates muscle wasting, creating a self-perpetuating cycle of functional decline. Addressing this complex nexus demands a fundamental shift from siloed healthcare to integrated, multidisciplinary models. Success hinges on embedding oral health screening and rehabilitation as core components of geriatric care. Effective intervention requires a triad of strategies: proactive dental management to restore masticatory function, personalized nutritional support with an emphasis on adequate protein and energy intake, and structured exercise programs, including resistance and oropharyngeal training, to rebuild muscle mass and strength. By implementing these strategies within a Comprehensive Geriatric Assessment framework, clinicians can disrupt the detrimental cycle, preserve independence, and significantly improve the quality of life for the aging population.

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