NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

JOURNAL OF EDUCATION, HEALTH AND SPORT

eISSN 2391-8306 · Open Access · Peer-reviewed

apcz.umk.pl/JEHS · Nicolaus Copernicus University in Toruń

Cite as: GRYGORCZUK, Oliwia, LUBA, Gabriela, IGNARSKA, Justyna, IGNARSKA, Magdalena, CZACH, Zuzanna, KUBICZ MZABI, Sabina and ALEKSANDROWICZ, Katarzyna. Nutrition in COPD: From Malnutrition and Sarcopenia to Patient-Tailored Nutritional Strategies. Journal of Education, Health and Sport. 2026;92:72556. https://doi.org/10.12775/JEHS.2026.92.72556

ARTICLE TIMELINE

Received: 25.05.2026 Revised: 25.05.2026
Accepted: 26.05.2026 Published: 20.06.2026

INDEXING & EVALUATION

MEiN points: 40 Unique ID: 201159
Disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

The journal has been awarded 40 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32318). Unique Journal Identifier: 201159. Scientific disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

Punkty Ministerialne z 2019 – aktualny rok 40 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).

Nutrition in COPD: From Malnutrition and Sarcopenia to Patient-Tailored Nutritional Strategies

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Abstract

Background: Malnutrition and sarcopenia are among the most prevalent and clinically significant extrapulmonary manifestations of chronic obstructive pulmonary disease (COPD), a widespread systemic condition. Nutritional depletion leads to impaired physical performance, worse quality of life, and increased exacerbation and mortality risk. Growing evidence suggests that nutritional therapy may play an important role in comprehensive COPD management.

Aim: To present the current state of knowledge on the nutritional burden of COPD, its underlying mechanisms, and the effects of dietary and supplemental interventions.

Methods: A total of one hundred two articles published between 2010 and 2026 were selected from databases such as PubMed, PMC, Cochrane Library, ScienceDirect and analysed based on their relevance and quality of evidence.

Results: Malnutrition and sarcopenia are frequently observed in COPD and correlate with poorer functional status, increased exacerbation rate, and lower survival. Studies show that dietary patterns, depending on their composition, may exert protective effects or worsen disease progression. In addition to increasing body weight and fat-free mass (FFM), oral nutritional supplementation (ONS), when combined with pulmonary rehabilitation, may enhance muscle function and quality of life. Although long-term data remain limited, emerging evidence suggests potential benefits of antioxidants, polyunsaturated fatty acids (PUFAs), and microbiome-targeted therapies.

Conclusion: Nutrition should be considered a key component of COPD management. Early nutritional assessment and individualized interventions may improve outcomes, particularly in patients with malnutrition or sarcopenia. Additionally, alongside ONS, patients with COPD should receive dietary counselling. Despite the growing body of research in this field, long-term randomized controlled trials are still required to optimize strategies and clarify effects on the gut--lung axis.

Key words: chronic obstructive pulmonary disease; malnutrition; sarcopenia; dietary supplements; pulmonary rehabilitation; quality of life

1. Introduction

Chronic obstructive pulmonary disease (COPD) remains a leading cause of morbidity and mortality worldwide, with projections indicating a rising trend in coming years.[1] Despite its primary origin in the lungs, patients suffering from COPD most often present widespread extrapulmonary manifestations, driven by underlying systemic inflammation and persistent airflow limitations. Examples include musculoskeletal dysfunction, metabolic alterations and mental health disorders, among which nutritional impairment and unintentional weight loss are particularly prevalent and clinically meaningful. [2–5] Unlike structural airway damage and airflow obstruction, nutrition remains a modifiable factor. Thus, targeted support can significantly improve clinical outcomes.[6–8] Recent growth of the evidence indicates that adequate nutritional intervention can modulate disease course, reduce exacerbation rates, enhance quality of life (QoL) and selected functional parameters.[9,10] Apart from routine nutritional screening and individualized medical nutrition therapy, integrating nutrition into pulmonary rehabilitation substantially strengthens patient-centred care.[1,11–13] Emerging multimodal and phenotype-specific strategies — including leucine-enriched formulas, polyunsaturated fatty acid (PUFA) supplementation, and interventions targeting the gut–lung axis - are promising, although require further in-depth research is required.[6,14,15] Therefore, nutrition should be considered a crucial pillar of the multidimensional therapeutic approach to COPD.[12,16,17]

2. Aim

This review summarizes current evidence on nutrition in COPD including prevalence and consequences of malnutrition and sarcopenia, mechanisms underlying nutritional depletion, the role of dietary patterns and oral supplementation, and nutrition during pulmonary rehabilitation. Additionally, it evaluates emerging strategies such as microbiome-targeting therapies. Emphasis was placed on translating findings into practical recommendations.

3. Methods

This literature review integrates evidence from one hundred two articles published between 2010 and 2026 identified and selected using databases such as PubMed, PMC, Cochrane Library and ScienceDirect. Priority was given to clinical trials, randomized controlled trials, meta-analyses and systematic reviews to ensure high-quality evidence and reduce the risk of bias, however a few narrative reviews and an experimental study were also included to provide additional context.

4. Malnutrition in COPD: Prevalence and Clinical Significance

Malnutrition, defined as either deficiency, excess or imbalance in energy and/or nutrients intake, affects up to one third of COPD patients when diagnosed using GLIM criteria (Global Leadership Initiative on Malnutrition), compared to up to one quarter when identified by screening tools like MUST (Malnutrition Universal Screening Tool) and MNA-SF (Mini Nutritional Assessment Short-Form). It is associated with more frequent exacerbations, worse functional outcomes, and increased mortality. [5,18–20] National Institute for Health and Care Excellence (NICE) guidelines recommend routine screening using MUST or MNA-SF in all COPD patients during pulmonary rehabilitation, followed by GLIM confirmation when risk is identified.[21] GLIM diagnosis (requiring ≥1 phenotypic criterion plus ≥1 etiologic criterion) reveals 22.6% malnutrition, 12.9% sarcopenia, and 9.7% malnutrition-sarcopenia syndrome prevalence, with severe obstruction (GOLD 3/4) significantly more common.[20] According to GLIM consensus criteria, malnutrition phenotypes include low BMI, unintentional weight loss, or reduced muscle mass, accompanied by etiologic criteria of inflammation or reduced intake.[22] Apart from evident manifestations such as sarcopenia or cachexia, undernutrition also occurs in high-BMI subjects, therefore obesity does not exclude nutritional risk.[23,24]. Observational data describes an „obesity paradox" which reveals that overweight/obese COPD patients (BMI 25-35 kg/m²) have better survival, functional outcomes, and prognosis than underweight patients, particularly in advanced GOLD stages.[25–27] This effect appears limited to former/current smokers, likely because nonsmoker COPD populations remain understudied and less prevalent, thus, requiring further investigation.[28]

5. Mechanisms of Nutritional Depletion in COPD

Nutritional depletion in COPD is mainly driven by systemic inflammation that occurs due to cytokines release, including TNF-α and IL-6.[29,30] Consequently, it stimulates catabolism and leads to muscle atrophy, which may be further exacerbated by oxidative damage due to ROS.[31–33] Additionally, insufficient energy intake associated with high resting energy expenditure due to increased respiratory demands, contributes to a loss of fat-free mass.[13,34] Such metabolic chain reaction significantly worsens the course of the disease and underlines the need for adequate nutritional interventions.[7,29,35]

6. Sarcopenia and Body Composition in COPD

Sarcopenia, defined as a progressive muscle disorder characterized by low muscle strength often with reduced muscle quantity or quality, alongside altered body composition, represents key prognostic factors in COPD.[36] Muscle mass loss and reduced fat-free mass (FFM) independently predict increased mortality, exacerbations, and functional decline beyond traditional markers like FEV1.[37,38] Recent studies have found that poor nutritional status triples exacerbation risk over 12 months in stable COPD outpatients,[39] while reduced FFM correlates with severe airflow limitation and acute exacerbations due to impaired respiratory and immune function.[37,40] Furthermore, unintentional weight loss, which occurs frequently in COPD population, almost triples the duration of hospitalization during exacerbations and predicts worse QoL at 1-year follow-up, independent of baseline BMI.[41] Besides clinical implications of low muscle strength, the functional decline is commonly observed in this group. Sarcopenic COPD patients presented compromised performance in activities of daily living (ADL), independent of airflow obstruction.[20]

6. Dietary Patterns and COPD Risk

Healthy dietary patterns, such as the Mediterranean or DASH diet, are associated with reduced COPD incidence (OR 0.59-0.73) and slower disease progression.[42,43] High dietary intake of omega-3 fatty acids, polyphenols, and antioxidants exerts protective effects on lung health by attenuating inflammation and improving pulmonary function parameters (e.g., FEV1/FVC). In contrast, Western dietary patterns rich in processed meat, refined grains, sugars, and saturated fats increase disease susceptibility.[44] Processed meat intake elevates COPD risk, with each 50 g/week increase linked to 8% higher risk (HR 1.08, 95% CI 1.03-1.13). This phenomenon is most probably triggered by systemic inflammation and oxidative stress, induced by nitrates/nitrites, saturated fatty acids, and advanced glycation end-products in processed meat. However this association remains undetermined and requires further investigation.[45]

7. Nutritional Supplementation in COPD

Given that optimal dietary patterns can both prevent COPD onset and slow its progression—oral nutritional supplementation (ONS) serves as an effective therapeutic tool for the most at-risk individuals, particularly those who are malnourished or sarcopenic. ONS formulations are adjusted to patient profiles by varying protein, fat, and energy content, as well as consistency, to fit in diverse clinical contexts.[35,46] The impact of ONS on the health and well-being of COPD patients has been thoroughly evaluated in multiple randomized controlled trials, complemented further by meta-analyses, consistently demonstrating weight gain,[35,46–50] while selected studies report preservation [35] or even increase[46] in FFM. In contrast, results concerning exercise capacity after ONS therapy are more heterogeneous with limited consensus. Despite the overall positive trend in physical performance,[46,50] individual studies do not show significant gain in exercise capacity.[35] In addition to somatic benefits, validated QoL measures demonstrate significant positive changes with ONS therapy, ranging from QoL preservation[35] to improvement,[47,51] consistent with meta-analytic findings.[46] Regarding macronutrient composition, high-fat ONS (containing 40-55% of fat) have been ranked superior for respiratory physiology, reducing respiratory quotient (RQ) and CO_2 production (VCO_2), which may make them especially beneficial for hypercapnic patients. Both high-carbohydrate and high-fat ONS achieved comparable weight gain, supporting the argument that all formulations are equally effective for nutritional recovery.[49]

Sarcopenic COPD patients are those who will particularly benefit from amino acid and protein-targeted interventions. Due to muscle protein anabolism alteration, these specific nutritional therapy strategies effectively counteract muscle wasting and restore functional capacities . Essential amino acids (EAAs) delivered with ONS stimulate whole-body protein anabolism more than intact protein mixtures and may help prevent muscle loss.[52] In severe COPD with sarcopenia, EAA supplementation has also been associated with gains in body weight, FFM, and physical performance.[53] Leucine - one of the EAAs - is particularly noteworthy as an ‚anabolic trigger’. It acts as a molecular signal that initiates the mTOR signaling pathway, crucial for protein translation.[54] While some stable COPD patients maintain protein sensitivity,[55] those with progressive cachexia or advanced sarcopenia often exhibit a diminished response to standard protein intake.[56] Therefore, these individuals require higher leucine dosages to overcome this so-called „anabolic resistance”.[57]

Besides protein-specific interventions, there are reports of potentially beneficial effects of other micronutrients, antioxidants, and PUFAs on health parameters and disease course in patients with COPD.[58–62] Antioxidants may slow disease progression by reducing oxidative stress and subsequent inflammation, induced by cigarette smoke and other environmental oxidants that activate inflammatory pathways in COPD.[63] Recent meta-analytic evidence suggests that antioxidant nutrient interventions, including vitamin C, vitamin D, vitamin E, selenium, magnesium-fortified beverages, omega-3 fatty acids, and EPA-enriched ONS, may improve handgrip strength, inspiratory muscle strength, and lean body mass index in patients with COPD.[61] ILDA study demonstrated that L-arginine and liposomal vitamin C supplementation alleviates dyspnea and improves QoL and daily-life activities in patients with COPD, suggesting a potential benefit of targeted nutritional support.[64] Nevertheless, the clinical efficacy of these interventions, still appears less consistent than that of synthetic antioxidants, particularly thiol-based compounds such as erdosteine or N-acetylcysteine (NAC), which are commonly used in COPD management. [63,65] Individual studies show an association between vitamin D supplementation and enhanced pulmonary function,[14] whereas others report a strong relationship between low serum vitamin D levels, increased systemic inflammation, and a higher exacerbation rate in patients with severe COPD.[66] Together, these findings support the rationale for treating vitamin D deficiency and for further investigating vitamin D supplementation in COPD to clarify its potential benefits, given the limitations of the available data.[67] By contrast, vitamin C and E supplementation alone did not significantly improve lung function.[14] However, vitamin E has been inversely associated with COPD in meta-analytic and observational studies, suggesting a potential protective role requiring further confirmation.[68]

Supplementation with PUFAs, particularly omega-3 fatty acids such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), may support COPD management by preserving lean mass and attenuating inflammation.[62,69,70] Consistent with these findings, a recent meta-analysis found that PUFA-related interventions were associated with more favorable COPD outcomes, including higher body weight and reduced interleukin-6, although effects on lung function, exercise capacity, and QoL were not significant overall.[60,69] Individual trials reported that EPA/DHA supplementation improved whole-body protein homeostasis, prevented or attenuated lean mass loss, and increased lean soft tissue in patients with COPD.[62,70] Observational data further indicate that higher plasma EPA+DHA levels are associated with better respiratory-specific QoL and a lower risk of moderate exacerbations[58] and that regular omega-3 supplementation is associated with fewer exacerbations and better exercise tolerance.[71] By contrast, role of omega-6 fatty acids appears more context-dependent. In COPD, higher arachidonic acid intake has been associated with higher IL-6 and CRP, whereas higher intake of the omega-3 precursor α-linolenic acid was linked to lower TNF-α.[72] A broader meta-analysis in COPD reported that lower n-3 and higher n-6 PUFA levels were associated with increased COPD risk, [60] although cohort studies also suggest that higher total n-6 intake (particularly linoleic acid) may be protective for cardiometabolic outcomes.[73,74] Conjugated linoleic acid (CLA), an isomer of linoleic acid, has been associated with lower serum levels of proinflammatory cytokines and increased appetite and energy intake, which may contribute to improved nutritional status in patients with COPD.[59,62,69,75,76]

Despite growing evidence suggesting therapeutic benefits from PUFA supplementation in COPD, current data are limited and sometimes inconsistent, based mainly on short-term RCTs and observational data, therefore further research is required, before firm clinical recommendations for routine PUFA supplementation can be made.[77–79]

9. Nutrition During Pulmonary Rehabilitation

Nutritional interventions combined with rehabilitation show a clear but selective impact on the pulmonary function and exercise outcomes. Implementation of protein-rich oral supplements in malnourished or muscle-wasted patients at the early stage of pulmonary rehabilitation (PR) may help preserve lean body mass and skeletal muscle indices and increase quadriceps strength.[13,80] Individualized dietitian-led therapy during inpatient PR, significantly increased energy and protein intake and improved handgrip strength, again without clear short-term changes in performance tests.[11] Targeted multimodal supplementation including leucine, vitamin D, omega-3 alongside high-intensity exercise improved body mass, inspiratory muscle strength, daily steps, and physical activity during and after PR, but its effect on quadriceps strength, cycle endurance, and long-term exercise capacity is less consistent.[35,81,82] Classic NS+PR trials in muscle-wasted COPD showed extra improvements in lean mass and mid-thigh muscle area, with comparable increases in 6-min walk and shuttle tests to PR-only arms.[80] At the level of specific supplements, agents such as melatonin, nitrate, whey protein and vitamin B12 can meaningfully enhance exercise performance, cycle endurance, dyspnea and health status when added to PR,[81,83–86] whereas addition of antioxidants mainly improve muscle strength and antioxidant status without further gains in endurance.[80,81] Overall, current synthesis of evidence demonstrates that nutritional support during PR consistently increases body weight and lean mass, while additional improvements in exercise capacity and QoL beyond PR alone are variable and depend on baseline depletion, supplement composition and program duration.[46,86–89] Therefore, individualized nutrition should be considered a key component of pulmonary rehabilitation in COPD, especially in malnourished or sarcopenic patients, in whom nutritional deficits may limit the effectiveness of rehabilitation.[1,11,89]

10. Emerging Nutritional Strategies

Growing evidence suggests that the gut-lung axis may represent a new nutritional target in COPD, particularly through effects on intestinal permeability, microbiome dysbiosis, and short-chain fatty acid production. Cross-sectional studies show that patients with COPD have an altered gut microbiota profile compared to healthy controls, with reduced diversity and abundance of probiotic genera alongside a shift toward more pro-inflammatory species.[15,90] Numerous studies link dysbiosis to disruption of intestinal immunity and increased susceptibility to chronic inflammation, both of which may ultimately lead to COPD development, highlighting diet, fiber intake, and probiotics/prebiotics as key modulators. The fermentation of prebiotic fiber to short-chain fatty acids is known to improve integrity of epithelium, maintain energy homeostasis, and reduce both mucosal and systemic inflammation. In contrast, low-fiber diets may contribute to epithelial barrier damage, in turn leading to chronic inflammation.[91,92] One of the early interventional studies suggests that prebiotic fiber-containing multi-nutrient supplementation is biologically promising but may require longer duration of treatment to substantially modify the microbiome. Preclinical data also support this hypothesis, as prebiotic fiber-containing supplementation reduced muscle loss in an experimental model of repetitive COPD exacerbations, suggesting potential benefits for skeletal muscle integrity.[93] A meta-analysis of probiotics in patients with COPD further shows modest improvements in FEV_1 and decrease in levels of inflammatory cytokines, indicating that microbiome modulation may be a promising strategy. Nevertheless, the optimal combinations and long-term clinical benefits of prebiotics remain to be defined.[94]

11. Conclusion: Clinical Implications and Future Directions

Malnutrition and sarcopenia remain highly prevalent in COPD and are strongly associated with worse exercise capacity, QoL, and mortality, independent of airflow limitation, underscoring the need for systematic nutritional assessment and early intervention.[95,96] Across randomized trial and meta-analyses nutritional support—especially macronutrient-rich ONS and multi-nutrient formulas—consistently improve body weight, FFM index, functional performance and patient-reported outcomes. Effects on lung function are modest and inconsistent, and appear more robust when delivered alongside pulmonary rehabilitation or within broader multimodal programs.[1,35,46,95] Network meta-analyses comparing supplement types suggest that specific agents (e.g. nanocurcumin, probiotics, butyrate, vitamin D, whey proteins, coenzyme Q10+creatine, melatonin, nitrate, selected traditional herbal combinations) may influence lung function, dyspnea, exercise endurance, or QoL, but overall certainty of evidence is generally low to moderate and direct head-to-head data are limited.[46,97] Multimodal interventions, such as the NUTRAIN trial, indicate that phenotype-tailored supplementation enriched with leucine, vitamin D and PUFAs, combined with counseling and activity feedback, can improve nutritional status, physical activity, psychological well-being and general health status often at acceptable cost per QALY, even without additional gains in maximal exercise capacity.[35,51,98,99] Taken together, this evidence supports integrating structured, individualized, and phenotype-tailored nutritional therapy as a routine component of comprehensive COPD management and pulmonary rehabilitation programs, in line with recent SEPAR–SEEN consensus recommendations for early, systematic nutritional assessment and management in COPD.[16,51,83,100] In addition to ONS, patients with COPD should receive nutritional counseling aimed at promoting healthier dietary patterns.[51,99] At the same time, there is a clear need for long-term RCTs and comparative effectiveness studies to optimize multimodal strategies (including pre/probiotics and PUFA-enriched formulas) and to clarify their impact on pulmonary outcomes, gut–lung interactions and hard clinical endpoints. [6,50,101,102]

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Writing—review and editing: OG, GL, JI, MI, ZC, SKM, KA

Supervision: OG, GL, JI,

Project administration: OG, GL

All authors have read and agreed to the published version of the manuscript.

Funding Statement: This research received no external funding.

Institutional Review Board Statement: Not applicable

Informed Consent Statement: Not applicable

Data Availability Statement: Not applicable

Conflict of Interest Statement: The authors declare no conflict of interests

In preparing this work, the authors used ChatGPT by OpenAI for the purpose of improving language clarity, enhancing readability, and organizing scientific content. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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