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Combating post-tuberculosis malnutrition through therapeutic nutrition: A narrative review

Sweta Jaiswal[1], Ankita Sengupta[2], Enakshi Raychowdhury[3], Souvik Tewari[4*]
[1] Department of Physiology, West Bengal State University, Barasat, West Bengal, India
[2] Department of Food and Beverage, KPC Medical College and Hospital, Jadavpur, Kolkata West Bengal, India
[3] Department of Community Nutrition, KPC Medical College and Hospital, Jadavpur, Kolkata West Bengal, India
[4] Assistant Professor, Department of Food and Nutrition, Swami Vivekananda University, Barrackpore, West Bengal, India
Corresponding Author: Souvik Tewari

Abstract
Tuberculosis (TB) remains one of the leading infectious causes of morbidity and mortality worldwide. Although successful antimicrobial therapy cures the infection, many patients continue to experience persistent malnutrition, muscle wasting, impaired immune function, and reduced quality of life after treatment completion. This condition, commonly referred to as post-tuberculosis (post-TB) malnutrition, contributes to delayed functional recovery, increased susceptibility to recurrent infections, and poor long-term health outcomes. Therapeutic nutrition has emerged as a critical component of post-TB care, aiming to restore nutritional status, improve immune competence, enhance muscle mass, and reduce the risk of disease recurrence. This narrative review summarizes the current understanding of post-TB malnutrition, explores the biological mechanisms responsible for persistent nutritional deficits, and evaluates available evidence regarding macronutrient supplementation, micronutrient therapy, oral nutritional supplements, gut microbiome modulation, and lifestyle interventions. The review also discusses current recommendations, identifies existing knowledge gaps, and highlights priorities for future research. Integrating therapeutic nutrition into routine post-TB management has the potential to improve recovery, functional outcomes, and overall quality of life among TB survivors.

Keywords: Tuberculosis, post-tuberculosis, malnutrition, therapeutic nutrition, micronutrients, nutritional rehabilitation, immune recovery

Introduction

Tuberculosis (TB) continues to represent a major global public health challenge despite significant advances in diagnosis, treatment, and prevention. Although effective antimicrobial therapy can successfully eliminate *Mycobacterium tuberculosis* and achieve microbiological cure in most patients, recovery is often incomplete, with many survivors experiencing persistent physical, metabolic, respiratory, and nutritional impairments that extend beyond the completion of treatment (World Health Organization [WHO], 2024; Allwood *et al*., 2021) [1, 11]. Increasing recognition of these long-term sequelae has shifted the focus of TB care from treatment success alone to comprehensive post-treatment rehabilitation that addresses the broader consequences of the disease.

Malnutrition and tuberculosis share a well-established bidirectional relationship. Undernutrition is one of the strongest risk factors for the development of active TB because it compromises both innate and adaptive immune responses, thereby increasing susceptibility to infection. Conversely, active TB induces a hypermetabolic and catabolic state characterized by systemic inflammation, anorexia, increased resting energy expenditure, and accelerated skeletal muscle protein breakdown, leading to substantial weight loss, micronutrient deficiencies, and depletion of lean body mass. Although most patients experience some degree of weight gain during anti-tuberculosis treatment, restoration of normal body composition and nutritional status is often incomplete, particularly among individuals with persistent inflammation, food insecurity, or chronic pulmonary impairment (WHO, 2024; Sinha *et al*., 2020) [7].

The concept of post-tuberculosis disease has gained considerable attention over the past decade as evidence accumulates regarding the long-term health consequences experienced by TB survivors. Persistent malnutrition is now recognized as a major contributor to reduced exercise capacity, chronic fatigue, impaired pulmonary function, immune dysfunction, decreased quality of life, and increased morbidity and mortality following successful TB treatment. Consequently, therapeutic nutrition has emerged as a fundamental component of comprehensive post-TB rehabilitation, with interventions focusing on restoring lean body mass, correcting micronutrient deficiencies, improving immune recovery, and enhancing functional capacity. Integrating individualized nutritional assessment and support into routine post-TB care may therefore improve long-term clinical outcomes and reduce disability among TB survivors (WHO, 2024; Allwood *et al*., 2021) [1].

This narrative review examines current evidence regarding nutritional deficits after TB treatment and discusses nutritional interventions that may enhance recovery.

Burden of Post-Tuberculosis Malnutrition

The prevalence of undernutrition among patients with tuberculosis (TB) remains alarmingly high, particularly in low- and middle-income countries (LMICs), where socioeconomic disparities, food insecurity, and limited access to healthcare exacerbate nutritional deficiencies. A recent systematic review and meta-analysis involving more than 48,000 patients reported a pooled prevalence of

malnutrition of approximately 48%, although individual studies have reported estimates ranging from less than 10% to over 70%, depending on the study population and assessment criteria (Li *et al*., 2023) [2]. Even after successful completion of anti-tuberculosis therapy, many individuals fail to regain their pre-illness body weight or achieve complete nutritional recovery, highlighting the long-term impact of TB on nutritional status (World Health Organization [WHO], 2025) [12].

Persistent malnutrition following successful tuberculosis treatment is multifactorial, arising from the combined effects of prolonged systemic inflammation, persistent loss of appetite, socioeconomic hardship, food insecurity, impaired nutrient absorption, chronic pulmonary impairment, and psychological factors such as depression, which often lead to reduced physical activity and inadequate dietary intake. Even after microbiological cure, residual inflammatory processes may sustain a hypercatabolic state that limits the restoration of body protein stores and delays nutritional recovery. Socioeconomic barriers further exacerbate this problem by restricting access to nutritious foods and healthcare services, while chronic respiratory dysfunction reduces exercise capacity and increases energy expenditure. Consequently, weight gain during recovery does not necessarily reflect complete nutritional rehabilitation. Many individuals regain predominantly adipose tissue rather than lean muscle mass, a phenomenon referred to as sarcopenic recovery. This disproportionate restoration of body composition leaves patients with persistent skeletal muscle depletion, which is associated with reduced muscle strength, impaired respiratory muscle function, diminished exercise tolerance, delayed functional recovery, compromised immune responses, and an increased susceptibility to recurrent infections and other adverse health outcomes. These findings underscore the importance of assessing body composition and functional status, rather than relying solely on body weight or body mass index, when evaluating nutritional recovery in post-tuberculosis patients (Allwood *et al*., 2020; WHO, 2025) [12].

Table 1: Burden and Clinical Consequences of Post-Tuberculosis Malnutrition

Aspect	Description	Clinical Implications
Prevalence	Malnutrition is common among individuals with active tuberculosis and may persist even after successful completion of anti-tuberculosis therapy. Many patients fail to regain their pre-illness nutritional status despite microbiological cure.	Delayed recovery, increased morbidity, and reduced quality of life.
Persistent Inflammation	Ongoing low-grade inflammation after TB treatment promotes protein catabolism, suppresses appetite, and increases resting energy expenditure.	Delayed restoration of lean body mass and prolonged functional impairment.
Reduced Dietary Intake	Loss of appetite, altered taste perception, gastrointestinal symptoms, and psychological distress contribute to inadequate energy and protein intake.	Continued weight loss and inadequate nutritional rehabilitation.
Socioeconomic Factors	Poverty, unemployment, food insecurity, and limited access to healthcare and nutritious foods are common among TB survivors.	Increased risk of persistent undernutrition and poor treatment outcomes.
Muscle Wasting (Sarcopenia)	Chronic inflammation and protein catabolism result in significant loss of skeletal muscle mass, which may persist despite weight gain.	Reduced muscle strength, impaired respiratory function, decreased exercise tolerance, and increased frailty.
Micronutrient Deficiencies	Deficiencies of vitamin D, vitamin A, zinc, iron, selenium, vitamin B12, and folate frequently persist following TB treatment.	Impaired immune recovery, delayed tissue repair, anemia, muscle weakness, and increased susceptibility to recurrent infections.
Impaired Functional Capacity	Persistent fatigue, reduced exercise tolerance, and diminished pulmonary function limit the ability to perform activities of daily living.	Lower productivity, reduced independence, and poorer health-related quality of life.
Altered Body Composition	Weight regain often reflects increased fat mass rather than restoration of lean muscle mass, resulting in sarcopenic recovery.	Persistent physical weakness despite apparent improvement in body weight or BMI.
Psychological and Social Consequences	Depression, anxiety, social stigma, and reduced physical activity may adversely affect appetite, treatment adherence, and nutritional recovery.	Delayed rehabilitation and poorer long-term physical and mental health outcomes.
Long-Term Health Outcomes	Persistent malnutrition increases the risk of recurrent infections, post-tuberculosis lung disease, disability, hospital readmission, and premature mortality.	Highlights the need for comprehensive nutritional assessment and long-term follow-up after TB treatment.

Abbreviations: TB, tuberculosis; BMI, body mass index.

Pathophysiology of Post-TB Malnutrition

Post-TB malnutrition is driven by complex interactions among inflammation, altered metabolism, endocrine dysfunction, and nutritional deficiencies.

1. Persistent Inflammation

Persistent low-grade inflammation is a key mechanism underlying post-tuberculosis (post-TB) malnutrition and delayed nutritional recovery. Although effective anti-tuberculosis therapy successfully eradicates *Mycobacterium tuberculosis*, inflammatory responses may persist for several months or even years after treatment completion because of residual pulmonary damage, immune dysregulation, and ongoing tissue repair (Allwood *et al*., 2020; Ivanova *et al*., 2023) [2]. Elevated circulating pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), interferon-gamma (IFN-γ), and interleukin-1 beta (IL-1β), maintain a chronic catabolic state by stimulating proteolysis and inhibiting skeletal muscle protein synthesis. These cytokines activate the ubiquitin–proteasome and autophagy–lysosome pathways, leading to accelerated degradation of muscle proteins while suppressing anabolic signaling mediated by the insulin-like growth factor-1 (IGF-1)/mammalian target of rapamycin (mTOR) pathway (Sinha *et al*., 2019; Ivanova *et al*., 2023) [2, 8]. Persistent inflammation also contributes to anorexia through cytokine-mediated effects on hypothalamic appetite regulation, reducing dietary intake and exacerbating negative energy

balance. Furthermore, inflammatory mediators increase resting energy expenditure and promote oxidative stress, thereby elevating metabolic demands during recovery. The combined effects of reduced nutrient intake, increased energy expenditure, and sustained muscle catabolism impede the restoration of lean body mass, resulting in prolonged weakness, impaired respiratory muscle function, decreased exercise capacity, and poorer overall functional recovery. Consequently, strategies aimed at controlling chronic inflammation while providing adequate nutritional support are essential components of comprehensive post-TB rehabilitation (Allwood et al., 2020; World Health Organization [WHO], 2025) [12].

2. Increased Energy Expenditure

Increased resting energy expenditure (REE) is a significant metabolic consequence of tuberculosis (TB) that may persist during the post-treatment recovery period, contributing to ongoing malnutrition and delayed restoration of nutritional status. During active TB, systemic inflammation, fever, and immune activation induce a hypermetabolic state characterized by elevated energy demands (Sinha *et al*., 2019) [8]. Although antimicrobial therapy resolves the infection, many patients continue to experience increased REE due to persistent tissue repair, residual immune activation, and chronic pulmonary impairment associated with post-tuberculosis disease (Allwood *et al*., 2020; World Health Organization [WHO], 2025) [12]. The healing of damaged lung parenchyma and other affected tissues requires substantial energy and protein to support cellular regeneration, collagen synthesis, and tissue remodeling. Additionally, individuals with post-tuberculosis lung disease often expend more energy during breathing because of reduced lung compliance, airflow limitation, respiratory muscle weakness, and impaired pulmonary mechanics, thereby increasing the metabolic cost of ventilation and contributing to persistent fatigue and reduced exercise tolerance (Allwood *et al*., 2020). If these elevated energy requirements are not met through adequate dietary intake, patients remain in a negative energy balance, resulting in continued weight loss, depletion of lean body mass, and impaired functional recovery (Sinha *et al*., 2019) [8]. Therefore, nutritional rehabilitation following TB should include individualized assessment of energy requirements and provision of sufficient caloric intake to meet the increased metabolic demands associated with recovery. Early identification and management of persistent hypermetabolism, together with comprehensive nutritional support, are essential to optimize weight regain, preserve skeletal muscle mass, improve physical performance, and enhance long-term clinical outcomes in TB survivors (WHO, 2025).

3. Muscle Protein Catabolism

Skeletal muscle protein catabolism is a hallmark of tuberculosis (TB)-associated malnutrition and remains an important contributor to delayed recovery following successful anti-tuberculosis treatment. Persistent systemic inflammation stimulates the breakdown of skeletal muscle proteins through activation of proteolytic pathways, particularly the ubiquitin–proteasome system (UPS) and the autophagy–lysosome pathway (Shin *et al*., 2021; Sartori *et al*., 2021) [5, 6]. Pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interferon-gamma (IFN-γ), upregulate muscle-specific E3 ubiquitin ligases such as muscle RING finger-1 (MuRF-1) and muscle atrophy F-box (MAFbx/atrogin-1), which target myofibrillar proteins for degradation while suppressing anabolic signaling mediated by the insulin-like growth factor-1 (IGF-1)/mammalian target of rapamycin (mTOR) pathway (Sartori *et al*., 2021) [5]. Inadequate dietary protein intake, prolonged physical inactivity, and persistent hypermetabolism further exacerbate muscle wasting during the recovery phase, resulting in a slower restoration of lean body mass than adipose tissue. Consequently, many TB survivors experience sarcopenic recovery, characterized by disproportionate fat mass gain despite persistent skeletal muscle depletion (Şahin *et al*., 2023). Persistent muscle loss is associated with reduced muscle strength, impaired respiratory muscle performance, diminished exercise capacity, increased fatigue, and poorer health-related quality of life. These observations emphasize the importance of combining adequate dietary protein intake with resistance exercise and comprehensive nutritional rehabilitation to promote muscle protein synthesis, restore functional muscle mass, and improve long-term outcomes among post-tuberculosis patients (Shin *et al*., 2021; Karakousis *et al*., 2023) [1, 3].

4. Micronutrient Deficiencies

Micronutrient deficiencies are common among tuberculosis (TB) survivors and represent a major barrier to complete nutritional and functional recovery following successful antimicrobial treatment. Deficiencies of vitamin D, vitamin A, zinc, iron, selenium, vitamin B12, and folate frequently persist after treatment because of inadequate dietary intake, chronic inflammation, increased metabolic demands, malabsorption, and socioeconomic factors such as poverty and food insecurity (World Health Organization [WHO], 2025; Sinha *et al*., 2019) [8, 12]. These micronutrients play essential roles in maintaining immune competence, antioxidant defense, tissue repair, erythropoiesis, and skeletal muscle metabolism. Vitamin D enhances innate immune responses by promoting macrophage activation and the production of antimicrobial peptides such as cathelicidin, while also contributing to muscle function and bone health (Wagnew *et al*., 2024) [10]. Vitamin A is essential for maintaining epithelial integrity, regulating adaptive immunity, and facilitating tissue repair. Zinc supports lymphocyte development, cytokine regulation, protein synthesis, and wound healing, whereas selenium functions as a key component of antioxidant enzymes that protect cells from oxidative stress induced by chronic inflammation (Wagnew *et al*., 2024 [10]; WHO, 2025). Iron deficiency, often accompanied by anemia of chronic disease, reduces oxygen transport and exercise capacity, although iron supplementation should be guided by laboratory assessment to avoid potential adverse effects associated with iron overload and altered iron homeostasis during infection (WHO, 2025). Vitamin B12 and folate are indispensable for DNA synthesis, erythropoiesis, and neuromuscular function, and their deficiencies may contribute to anemia, fatigue, and impaired cellular regeneration. Collectively, these micronutrient deficiencies compromise immune recovery, delay wound healing, impair muscle regeneration, and prolong functional disability in

post-TB patients. Therefore, comprehensive nutritional assessment and targeted correction of documented deficiencies should be incorporated into post-tuberculosis rehabilitation programs to optimize recovery and improve long-term clinical outcomes (Sinha *et al*., 2019; Wagnew *et al*., 2024) [8, 10].

5. Gut Microbiome Alterations

Alterations in the gut microbiome have emerged as an important factor contributing to persistent malnutrition and impaired immune recovery following tuberculosis (TB) treatment. Standard anti-tuberculosis therapy, particularly its prolonged duration, can significantly disrupt the composition and diversity of the intestinal microbiota by reducing beneficial commensal bacteria and promoting microbial dysbiosis. These changes may persist long after treatment completion and adversely affect several physiological processes essential for nutritional rehabilitation.

A healthy gut microbiome facilitates the digestion and absorption of nutrients, synthesizes essential vitamins such as vitamin K and certain B vitamins, produces short-chain fatty acids that maintain intestinal barrier integrity, and regulates host immune responses. Dysbiosis may impair these functions by increasing intestinal permeability, reducing nutrient absorption, altering energy metabolism, and promoting chronic low-grade inflammation. Furthermore, disruption of the gut microbiota can influence the gut–lung axis, a bidirectional communication network through which intestinal microorganisms modulate pulmonary immunity and systemic inflammatory responses. Persistent microbial imbalance may therefore contribute to delayed immune restoration, increased susceptibility to secondary infections, and prolonged functional recovery in TB survivors. Emerging evidence suggests that dietary interventions, probiotics, prebiotics, and synbiotics may help restore microbial diversity, improve gastrointestinal function, enhance nutrient utilization, and reduce inflammation. However, current clinical evidence remains limited, and well-designed randomized controlled trials are needed to establish the efficacy, optimal formulations, and long-term safety of microbiome-targeted nutritional therapies in post-tuberculosis rehabilitation.

Table 2: Pathophysiological Mechanisms Underlying Post-Tuberculosis Malnutrition

Mechanism	Pathophysiological Process	Nutritional and Clinical Consequences
Persistent inflammation	Sustained production of pro-inflammatory cytokines (TNF-α, IL-6, IFN-γ, IL-1β) following TB treatment maintains a chronic catabolic state, suppresses appetite, and inhibits muscle protein synthesis.	Muscle wasting, anorexia, delayed nutritional recovery, impaired immune function, and prolonged functional disability.
Increased resting energy expenditure (Hypermetabolism)	Ongoing tissue repair, residual immune activation, and increased work of breathing elevate basal energy requirements even after microbiological cure.	Negative energy balance, continued weight loss, depletion of lean body mass, and delayed recovery.
Muscle protein catabolism	Activation of the ubiquitin–proteasome and autophagy–lysosome pathways accelerates skeletal muscle protein degradation, while suppression of the IGF-1/mTOR pathway reduces muscle protein synthesis.	Sarcopenia, reduced muscle strength, impaired respiratory muscle function, decreased exercise tolerance, and physical frailty.
Micronutrient deficiencies	Inadequate dietary intake, chronic inflammation, malabsorption, and increased metabolic demands lead to deficiencies of vitamin D, vitamin A, zinc, iron, selenium, vitamin B12, and folate.	Impaired immune recovery, anemia, delayed wound healing, oxidative stress, reduced muscle regeneration, and increased susceptibility to infections.
Gut microbiome dysbiosis	Prolonged anti-TB antibiotic therapy disrupts intestinal microbial diversity, reducing beneficial bacteria and impairing gut barrier function.	Reduced nutrient absorption, gastrointestinal dysfunction, chronic low-grade inflammation, and delayed immune restoration.
Reduced dietary intake	Persistent anorexia, altered taste perception, gastrointestinal symptoms, depression, and fatigue decrease food consumption during recovery.	Inadequate intake of energy, protein, and micronutrients, contributing to persistent malnutrition.
Impaired nutrient absorption	Gastrointestinal inflammation, dysbiosis, and altered intestinal permeability reduce the absorption of macro- and micronutrients.	Nutrient deficiencies, poor weight gain, impaired tissue repair, and delayed nutritional rehabilitation.
Chronic pulmonary dysfunction	Residual lung damage, airflow limitation, and impaired respiratory mechanics increase the energy cost of breathing and limit physical activity.	Increased energy expenditure, fatigue, reduced exercise capacity, and slower functional recovery.
Hormonal and metabolic alterations	Chronic inflammation suppresses anabolic hormones (e.g., insulin-like growth factor-1) and promotes insulin resistance and endocrine dysfunction.	Reduced muscle protein synthesis, impaired tissue regeneration, and persistent loss of lean body mass.
Socioeconomic and psychosocial factors	Poverty, food insecurity, unemployment, depression, and limited access to healthcare reduce dietary quality and adherence to nutritional recommendations.	Persistent undernutrition, poor rehabilitation outcomes, reduced quality of life, and increased risk of recurrent illness.

Abbreviations: TNF-α, tumor necrosis factor-alpha; IL-6, interleukin-6; IFN-γ, interferon-gamma; IL-1β, interleukin-1 beta; IGF-1, insulin-like growth factor-1; mTOR, mammalian target of rapamycin; TB, tuberculosis.

Therapeutic Nutrition Strategies

Nutritional rehabilitation should be individualized according to nutritional status, age, comorbidities, and socioeconomic circumstances.

1. Energy Supplementation

Patients recovering from TB generally require increased caloric intake to compensate for previous weight loss and support tissue repair. Energy requirements typically exceed those of healthy individuals during early recovery.

Complex carbohydrates, healthy fats, and frequent balanced meals are recommended to maintain adequate caloric intake.

2. High-Quality Protein

Adequate intake of high-quality dietary protein is a cornerstone of nutritional rehabilitation in patients recovering from tuberculosis (TB), as it plays a critical role in restoring skeletal muscle mass, supporting immune function, and promoting tissue repair. During active TB, prolonged systemic inflammation, increased protein catabolism, and reduced dietary intake result in significant depletion of lean body mass, which may persist even after successful antimicrobial therapy. Dietary protein provides essential amino acids required for muscle protein synthesis, collagen formation, enzyme production, and the synthesis of antibodies and immune cells necessary for host defense. High-biological-value protein sources, including eggs, milk and dairy products, fish, poultry, lean meat, soy products, legumes, and pulses, supply adequate quantities of essential amino acids, particularly leucine, which activates the mammalian target of rapamycin (mTOR) signaling pathway and stimulates muscle protein synthesis. Combining sufficient protein intake with resistance or pulmonary rehabilitation exercises can further enhance muscle regeneration, improve physical function, and reduce the risk of sarcopenia during recovery. For individuals who are unable to meet their nutritional requirements because of poor appetite, dysphagia, gastrointestinal intolerance, or socioeconomic constraints, high-protein oral nutritional supplements (ONS) enriched with energy, vitamins, minerals, and, in some formulations, branched-chain amino acids or β-hydroxy β-methylbutyrate (HMB), may provide an effective means of improving protein intake, promoting lean body mass recovery, and accelerating functional rehabilitation. Consequently, individualized assessment of protein requirements and timely nutritional intervention should be considered an integral component of comprehensive post-TB care.

Micronutrient Supplementation

1. Vitamin D

Vitamin D is one of the most extensively studied micronutrients in tuberculosis (TB) because of its critical role in immune regulation and host defense against *Mycobacterium tuberculosis*. It enhances innate immunity by promoting the differentiation and activation of macrophages and stimulating the production of antimicrobial peptides, such as cathelicidin and β-defensins, which possess antimycobacterial activity. Vitamin D also modulates adaptive immune responses by reducing excessive inflammation while maintaining effective pathogen clearance. Deficiency of vitamin D is highly prevalent among patients with active TB and often persists after treatment due to inadequate dietary intake, limited sunlight exposure, chronic illness, and malabsorption. Low serum vitamin D concentrations have been associated with delayed sputum conversion, impaired immune recovery, reduced muscle strength, and poorer clinical outcomes. Although several clinical trials have evaluated vitamin D supplementation as an adjunct to anti-TB therapy, findings have been inconsistent, with benefits appearing greatest in individuals with documented deficiency. Consequently, current evidence supports screening for vitamin D deficiency and providing targeted supplementation where indicated, while routine high-dose vitamin D supplementation for all TB survivors is not currently recommended because of insufficient evidence regarding its universal clinical benefit.

2. Vitamin A

Vitamin A is an essential fat-soluble vitamin that plays a fundamental role in maintaining epithelial integrity, regulating immune function, and facilitating tissue repair during recovery from tuberculosis. It supports both innate and adaptive immunity by promoting the differentiation and function of T and B lymphocytes, enhancing antibody production, and preserving the integrity of mucosal barriers that serve as the first line of defense against pathogens. Vitamin A also contributes to cellular growth, collagen synthesis, and wound healing, making it particularly important for the repair of pulmonary tissue damaged during TB infection. Deficiency is common in individuals with chronic infections and malnutrition and has been associated with impaired immune responses, increased susceptibility to secondary infections, and delayed recovery. Although evidence supporting routine vitamin A supplementation in all TB survivors remains limited, correcting documented deficiency through dietary improvement or supplementation may enhance immune restoration, support tissue regeneration, and contribute to overall nutritional rehabilitation, particularly in populations where vitamin A deficiency is prevalent.

3. Zinc

Zinc is an essential trace element involved in numerous biological processes that are critical for immune competence, protein synthesis, cellular growth, and tissue repair. It functions as a structural and catalytic component of more than 300 enzymes and is indispensable for the development, maturation, and activity of immune cells, including neutrophils, macrophages, natural killer cells, and T lymphocytes. Zinc also plays an important role in antioxidant defense by stabilizing cell membranes and reducing oxidative stress, thereby limiting tissue damage associated with chronic inflammation. Among patients recovering from tuberculosis, zinc deficiency may result from inadequate dietary intake, increased metabolic demands, impaired intestinal absorption, and redistribution of zinc during systemic inflammation. Persistent zinc deficiency has been associated with delayed wound healing, impaired muscle regeneration, weakened immune responses, and increased susceptibility to recurrent infections. Although current evidence regarding routine zinc supplementation in post-TB patients is still evolving, targeted supplementation in individuals with confirmed deficiency may support nutritional recovery, enhance immune function, and improve overall clinical outcomes when combined with adequate dietary protein and comprehensive nutritional care.

4. Iron

Iron deficiency and iron deficiency anemia are frequently observed among individuals with active and post-tuberculosis disease owing to chronic inflammation, inadequate dietary intake, blood loss, and impaired iron metabolism. Iron is essential for hemoglobin synthesis,

oxygen transport, mitochondrial energy production, and normal immune cell function, and deficiency can contribute to fatigue, reduced exercise tolerance, impaired cognitive performance, and diminished quality of life. However, iron metabolism in tuberculosis is complex because inflammatory cytokines increase the production of hepcidin, a hepatic hormone that limits intestinal iron absorption and traps iron within macrophages, resulting in functional iron deficiency despite adequate body iron stores. Furthermore, excess iron may promote oxidative stress and theoretically enhance the growth of intracellular pathogens, emphasizing the need for cautious management. Therefore, iron supplementation should not be administered routinely to all TB survivors but should instead be guided by laboratory evaluation, including measurements of hemoglobin, serum ferritin, transferrin saturation, and inflammatory markers, to distinguish true iron deficiency from anemia of chronic disease. Individualized iron replacement therapy, combined with nutritional counseling and monitoring, can safely improve hematological status and functional recovery in patients with confirmed deficiency.

5. Selenium

Selenium is an essential trace mineral that plays a pivotal role in antioxidant defense, immune regulation, thyroid hormone metabolism, and cellular protection against oxidative damage. It is incorporated into several selenoproteins, including glutathione peroxidases and thioredoxin reductases, which neutralize reactive oxygen species generated during chronic inflammation and infection. Tuberculosis induces substantial oxidative stress through persistent immune activation, leading to increased consumption of antioxidant nutrients and potential depletion of selenium stores. Selenium deficiency may impair T-cell proliferation, macrophage function, cytokine regulation, and overall immune competence, thereby delaying recovery and increasing vulnerability to recurrent infections. In addition, inadequate selenium status has been associated with reduced muscle function and slower tissue repair, potentially contributing to prolonged functional impairment following TB treatment. Although evidence from clinical trials remains limited and somewhat inconsistent, some studies suggest that selenium supplementation may improve antioxidant capacity, reduce oxidative stress, and support immune recovery, particularly in individuals with documented deficiency. Further well-designed randomized controlled trials are required to establish optimal dosing strategies and determine the long-term clinical benefits of selenium supplementation as part of comprehensive nutritional rehabilitation for TB survivors.

Table 3: Therapeutic Nutritional Interventions for Post-Tuberculosis Recovery

Nutritional Intervention	Mechanism of Action	Potential Clinical Benefits	Key Considerations
Energy supplementation	Provides additional calories to meet increased metabolic demands, restore energy balance, and support tissue repair.	Promotes weight gain, reduces energy deficit, improves nutritional status, and accelerates recovery.	Caloric intake should be individualized based on age, body weight, physical activity, and disease severity.
High-quality protein intake	Supplies essential amino acids required for muscle protein synthesis, immune-cell production, and tissue regeneration.	Restores lean body mass, improves muscle strength, enhances immune function, and reduces sarcopenia.	Include high-biological-value protein sources such as eggs, dairy products, fish, poultry, lean meat, soy products, legumes, and pulses.
Micronutrient supplementation	Corrects deficiencies of vitamins and trace elements involved in immune regulation, antioxidant defense, and cellular repair.	Enhances immune recovery, improves wound healing, reduces oxidative stress, and supports muscle regeneration.	Supplementation should preferably be guided by nutritional assessment and laboratory confirmation of deficiency.
Oral nutritional supplements (ONS)	Provides concentrated energy, protein, vitamins, and minerals when dietary intake is insufficient.	Improves body weight, increases lean body mass, enhances physical performance, and supports nutritional rehabilitation.	Particularly beneficial for patients with poor appetite, significant weight loss, or difficulty meeting nutritional requirements through diet alone.
Specialized nutritional formulations	Enriched with anabolic nutrients such as leucine, omega-3 fatty acids, and β-hydroxy β-methylbutyrate (HMB) to stimulate muscle protein synthesis and reduce inflammation.	Preserves skeletal muscle, improves muscle strength, attenuates inflammation, and promotes functional recovery.	Evidence is promising but additional randomized controlled trials are needed to define optimal formulations and duration.
Probiotics and prebiotics	Restore gut microbial diversity, improve intestinal barrier function, and enhance nutrient absorption and immune regulation.	Reduces antibiotic-associated diarrhea, improves gastrointestinal health, supports immune function, and may enhance nutritional recovery.	Current evidence is limited; routine use cannot yet be universally recommended.
Nutrition combined with physical rehabilitation	Combines adequate nutritional intake with resistance and aerobic exercise to stimulate anabolic pathways and improve functional capacity.	Increases muscle mass and strength, improves respiratory muscle performance, enhances exercise tolerance, and promotes independence.	Multidisciplinary rehabilitation programs provide the greatest benefits for long-term recovery.
Individualized nutritional counseling	Tailors dietary recommendations according to nutritional status, comorbidities, socioeconomic factors, and patient preferences.	Improves dietary adherence, optimizes nutrient intake, supports long-term nutritional recovery, and enhances quality of life.	Regular nutritional assessment and follow-up are essential to adjust dietary interventions over time.

Abbreviations: ONS, oral nutritional supplements; HMB, β-hydroxy β-methylbutyrate.

Oral Nutritional Supplements

Oral nutritional supplements (ONS) are an important component of therapeutic nutrition for individuals recovering from tuberculosis (TB), particularly those who are unable to meet their nutritional requirements through a regular diet alone. Commercially available ONS provide concentrated sources of energy, high-quality protein, essential fatty acids, vitamins, and minerals in a convenient and easily consumable form, making them especially beneficial for patients with persistent anorexia, fatigue, dysphagia, or increased metabolic demands during recovery. By supplying additional calories and protein, ONS help counteract the negative energy balance and muscle protein catabolism that often persist after completion of anti-TB therapy. Clinical studies have demonstrated that nutritional supplementation can improve weight gain, increase lean body mass, enhance muscle strength, support immune recovery, and improve overall physical performance and quality of life. Furthermore, adequate nutritional support may enhance treatment adherence during active disease by reducing fatigue and improving patients' overall well-being, while continuing supplementation during the post-treatment period can facilitate functional rehabilitation and reduce the risk of prolonged malnutrition. Individuals with poor appetite, gastrointestinal intolerance, or socioeconomic barriers to accessing nutrient-dense foods are likely to derive the greatest benefit from ONS. Recent advances have also led to the development of specialized formulations enriched with anabolic nutrients such as leucine, omega-3 polyunsaturated fatty acids, and β-hydroxy β-methylbutyrate (HMB), which may further stimulate muscle protein synthesis, attenuate inflammation, preserve skeletal muscle mass, and accelerate recovery. Although current evidence supports the use of ONS as an adjunct to dietary counseling in malnourished TB patients, further large-scale randomized controlled trials are needed to determine the optimal composition, dosage, duration, and cost-effectiveness of supplementation during post-tuberculosis rehabilitation.

Gut Microbiome and Probiotics

The gut microbiome has emerged as an important determinant of nutritional status, immune function, and overall health, and growing evidence suggests that it may play a significant role in recovery following tuberculosis (TB) treatment. Standard anti-tuberculosis therapy, which typically involves prolonged administration of multiple antibiotics, can substantially alter the composition and diversity of intestinal microbial communities, resulting in gut dysbiosis that may persist long after treatment completion. Disruption of the gut microbiota can impair digestion and nutrient absorption, reduce the production of beneficial metabolites such as short-chain fatty acids, compromise intestinal barrier integrity, and promote chronic low-grade inflammation. Furthermore, alterations in the gut microbiome may influence the gut–lung axis, a bidirectional communication network through which intestinal microorganisms regulate pulmonary immune responses and systemic inflammation. Consequently, persistent dysbiosis may contribute to delayed nutritional recovery, impaired immune reconstitution, and an increased risk of recurrent infections among TB survivors. Emerging evidence indicates that probiotics, prebiotics, and synbiotics may help restore microbial diversity, improve gastrointestinal function, enhance nutrient absorption, reduce antibiotic-associated diarrhea, strengthen intestinal barrier function, and modulate systemic inflammatory responses. Common probiotic strains, including *Lactobacillus* and *Bifidobacterium* species, have demonstrated potential benefits in improving gastrointestinal health and immune regulation, while prebiotics such as inulin and fructooligosaccharides selectively stimulate the growth of beneficial gut bacteria. Despite these promising findings, the current evidence is derived primarily from small clinical studies and experimental models, and robust data specifically evaluating post-TB populations remain limited. Therefore, routine use of probiotics or prebiotics in post-tuberculosis rehabilitation cannot yet be universally recommended, and well-designed, adequately powered randomized controlled trials are needed to establish their efficacy, optimal formulations, dosing regimens, and long-term safety.

Nutrition Combined with Physical Rehabilitation

Nutritional rehabilitation alone is often insufficient to restore the functional capacity and physical performance of individuals recovering from tuberculosis (TB), as prolonged illness frequently results in substantial skeletal muscle wasting, reduced cardiorespiratory fitness, and impaired exercise tolerance. Therefore, combining therapeutic nutrition with structured physical rehabilitation has emerged as a comprehensive strategy for optimizing recovery and improving long-term health outcomes. Adequate dietary energy and high-quality protein intake provide the substrates necessary for muscle protein synthesis, while resistance exercise stimulates anabolic pathways, including activation of the mammalian target of rapamycin (mTOR) signaling pathway, thereby promoting muscle hypertrophy, increasing muscle strength, and restoring lean body mass. Regular resistance and endurance training also improve respiratory muscle performance, enhance insulin sensitivity, increase aerobic capacity, reduce fatigue, and promote greater physical independence in daily activities. In addition to musculoskeletal benefits, physical activity contributes to improved cardiovascular fitness, psychological well-being, and overall quality of life by alleviating symptoms of anxiety and depression that commonly persist after TB treatment. Pulmonary rehabilitation programs that integrate supervised exercise training, breathing exercises, nutritional counseling, and patient education have demonstrated significant improvements in exercise capacity, symptom control, respiratory function, and health-related quality of life among individuals with chronic respiratory diseases, including post-tuberculosis lung disease. Although evidence specific to TB survivors is still evolving, current findings suggest that multidisciplinary rehabilitation programs incorporating individualized nutritional support and progressive exercise interventions are likely to provide synergistic benefits, accelerate functional recovery, reduce disability, and enhance long-term clinical outcomes. Future randomized controlled trials are needed to establish standardized rehabilitation protocols and determine the optimal timing, intensity, and duration of combined

nutrition and exercise interventions in post-tuberculosis care.

Special Populations

1. HIV Co-infection

Individuals co-infected with human immunodeficiency virus (HIV) and tuberculosis (TB) represent one of the most nutritionally vulnerable patient populations because both diseases independently contribute to malnutrition, immune dysfunction, and increased metabolic demands. HIV infection accelerates protein-energy wasting through chronic immune activation, increased resting energy expenditure, malabsorption, opportunistic infections, and reduced dietary intake, while TB further exacerbates systemic inflammation and muscle protein catabolism. Consequently, HIV-TB co-infected patients frequently experience severe weight loss, micronutrient deficiencies, anemia, and depletion of lean body mass, all of which are associated with poorer treatment outcomes and increased mortality. Nutritional management should therefore be integrated with antiretroviral therapy (ART) and anti-tuberculosis treatment, emphasizing adequate energy intake, high-quality protein, correction of micronutrient deficiencies, and regular nutritional assessment. Particular attention should be given to potential drug–nutrient interactions, gastrointestinal complications, and metabolic disorders associated with long-term ART. Comprehensive multidisciplinary care that combines medical treatment with individualized nutritional support can improve immune recovery, enhance treatment adherence, reduce complications, and promote long-term health outcomes in this high-risk population.

2. Diabetes Mellitus

Diabetes mellitus is a major comorbidity that adversely affects tuberculosis outcomes and complicates post-treatment nutritional rehabilitation. Chronic hyperglycemia impairs both innate and adaptive immune responses by reducing macrophage function, neutrophil activity, and cytokine regulation, thereby increasing susceptibility to infection and delaying tissue repair. In addition, insulin resistance and poor glycemic control promote protein catabolism, inhibit muscle protein synthesis, and contribute to progressive loss of lean body mass. Individuals with diabetes recovering from TB are therefore at greater risk of persistent malnutrition, delayed wound healing, recurrent infections, and prolonged functional impairment. Nutritional therapy should focus on achieving optimal glycemic control while simultaneously providing adequate energy and high-quality protein to support muscle regeneration and immune recovery. Dietary plans should emphasize low-glycemic-index carbohydrates, adequate dietary fiber, healthy unsaturated fats, and nutrient-dense protein sources while limiting refined sugars and highly processed foods. Regular monitoring of blood glucose levels, body composition, and nutritional status, combined with appropriate pharmacological management and physical activity, is essential for improving metabolic control and optimizing recovery in patients with TB-diabetes comorbidity.

3. Older Adults

Older adults recovering from tuberculosis are particularly susceptible to persistent malnutrition because of age-related physiological changes, multiple comorbidities, reduced appetite, impaired nutrient absorption, and diminished muscle regenerative capacity. Aging is associated with anabolic resistance, whereby skeletal muscle exhibits a reduced response to dietary protein and physical activity, increasing the risk of sarcopenia, frailty, falls, and functional dependence. Tuberculosis further accelerates muscle wasting through chronic inflammation, prolonged immobilization, and increased metabolic demands, often resulting in substantial declines in muscle strength and physical performance. Nutritional rehabilitation in older TB survivors should therefore prioritize adequate caloric intake, higher dietary protein consumption distributed evenly throughout the day, and sufficient intake of key micronutrients such as vitamin D, calcium, vitamin B12, and zinc to support musculoskeletal health. When combined with progressive resistance training, balance exercises, and comprehensive geriatric rehabilitation, these nutritional interventions can enhance muscle mass, improve strength and mobility, reduce frailty, and promote greater independence and quality of life. Individualized assessment and multidisciplinary management are essential to address the complex nutritional and functional needs of older adults during post-tuberculosis recovery.

Conclusion

Post-tuberculosis malnutrition represents an underrecognized but clinically significant consequence of TB disease. Persistent inflammation, muscle wasting, micronutrient deficiencies, altered metabolism, and socioeconomic challenges contribute to delayed recovery despite microbiological cure. Therapeutic nutrition—including adequate energy intake, high-quality protein, correction of micronutrient deficiencies, oral nutritional supplementation where appropriate, and integration with physical rehabilitation—offers a promising strategy for improving long-term health outcomes. Current evidence supports individualized nutritional assessment as an integral component of post-TB care. However, further high-quality clinical trials are needed to establish standardized nutritional protocols and determine the most effective interventions for diverse patient populations. Incorporating therapeutic nutrition into national TB programs has the potential to enhance recovery, reduce disability, and improve quality of life for millions of TB survivors worldwide.

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