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Mini Review

Bronchiectasis as a Disorder of Microbial Ecosystem Imbalance: From Dysbiosis to Precision Therapeutics

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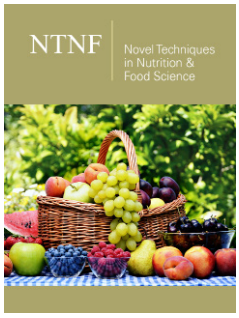

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Abstract

Bronchiectasis has traditionally been viewed as a chronic airway disease driven primarily by persistent bacterial infection and neutrophilic inflammation. Advances in microbiome research suggest that airway dysbiosis, characterized by reduced microbial diversity and depletion of commensal organisms, is associated with disease progression. Culture-independent analyses reveal that bronchiectatic airways harbor polymicrobial communities, in which ecological imbalance often correlates more strongly with clinical outcomes than the presence of individual pathogens. Emerging evidence indicates that depletion of commensal taxa such as Prevotella, Veillonella, and Rothia mucilaginosa is associated with reduced anti-inflammatory signaling, potentially contributing to an environment permissive of neutrophilic activation and protease-mediated tissue injury, although causal relationships remain unestablished. Disruption of the gut-lung axis may further amplify pulmonary inflammation by reducing circulating short-chain fatty acids and increasing systemic exposure to lipopolysaccharides. This review discusses the evolving concept of bronchiectasis as a potential disorder of microbial ecosystem imbalance. It examines therapeutic implications, including evidence from the phase 3 ASPEN trial supporting brensocatib as a neutrophil-targeted therapy, the role of long-term macrolide therapy, and emerging microbiome-directed strategies. The potential role of nutritional modulation through the gut-lung axis is discussed as a hypothesis-generating area. Integrating microbial endotyping into clinical practice may support precision medicine strategies focused on restoring microbial homeostasis.

Keywords: Bronchiectasis; Dysbiosis; Lung microbiome; Gut-lung axis; Lipopolysaccharides; Dietary fiber; Short-chain fatty acids; Neutrophil extracellular traps; Brensocatib; Macrolides; Precision medicine

Introduction

Bronchiectasis is a chronic inflammatory airway disease characterized by irreversible bronchial dilation, impaired mucociliary clearance, chronic infection, and recurrent exacerbations. Historically, the disease has been interpreted through a pathogen-centric framework as driven by persistent bacterial infections, particularly Pseudomonas aeruginosa and Haemophilus influenzae, which trigger airway inflammation and progressive structural damage [1]. This model has informed therapeutic strategies centered on prolonged antibiotics and inhaled antimicrobials. While these interventions provide symptomatic improvement in selected patients, they have not consistently prevented long-term disease progression, and considerable heterogeneity in treatment response remains unresolved [1]. Culture-independent sequencing technologies have altered our understanding of airway microbiology, revealing that bronchiectatic airways harbor polymicrobial communities rather than being dominated by a single pathogen [1,2]. Disease severity often correlates more strongly with ecological imbalance than with the identification of individual organisms. Longitudinal studies demonstrate that patients with microbiomes dominated by P. aeruginosa experience more severe clinical trajectories compared with those maintaining greater microbial diversity [3].

Systemic inflammatory pathways linked to the gut-lung axis are also increasingly recognized in chronic respiratory disease [1]. Recent conceptual advances have reframed the classical "vicious cycle" of bronchiectasis pathogenesis as a "vicious vortex," reflecting the multidirectional and self-amplifying interactions among mucociliary dysfunction, infection, inflammation, and structural damage [4,5]. This review discusses the role of dysbiosis in the pathogenesis of bronchiectasis and explores how microbiome-informed strategies, including established and emerging therapeutics, may reshape treatment approaches.

The Dysbiosis Hypothesis: Associations and Bidirectional Relationships

The classical vicious cycle hypothesis proposed by Cole describes bronchiectasis as a self-perpetuating interaction among impaired mucociliary clearance, infection, inflammation, and tissue destruction. This model largely assumed that persistent infection was the primary initiating factor. The updated "vicious vortex" concept better captures the multidirectional nature of these interactions, in which each component can independently drive disease progression [4,5]. Modern microbiome studies further challenge the infection-centric view by demonstrating that disease severity often correlates more strongly with ecological imbalance than with the identification of individual pathogens [1]. Culture-independent analyses consistently reveal reduced alpha diversity and depletion of commensal taxa, including Prevotella, Veillonella, Gemella, Neisseria, and Rothia in bronchiectatic airways [2,3]. Rogers and colleagues identified microbiota stratification systems capable of predicting future exacerbations, suggesting that ecological structure possesses prognostic significance beyond simple pathogen detection [3]. The relationship between dysbiosis and bronchiectasis progression is likely bidirectional. Structural airway abnormalities impair mucus clearance and create microenvironments favorable to microbial imbalance. In turn, dysbiosis may amplify immune dysfunction and tissue injury. Longitudinal studies demonstrate relative instability in microbial communities over time, particularly in patients with severe disease or repeated antibiotic exposure [3]. The entry of oral commensals into the lower airway represents a complex process: while certain organisms may attenuate inflammation under some conditions, recent evidence demonstrates that in the context of Non-Tuberculous Mycobacterial (NTM) infection, oral commensals, including Veillonella, Prevotella, and Streptococcus, can co-occur with Mycobacterium and contribute to sustained proinflammatory responses marked by elevated Neutrophil Extracellular Traps (NETs) and Th17 cell expansion [6]. This context-dependent behavior underscores the complexity of host-microbiome interactions and cautions against simplistic categorization of taxa as uniformly protective or harmful. An important observation that challenges the infection-centric model is that microbiome studies consistently show no significant differences between stable-state and exacerbation microbiota profiles in bronchiectasis, suggesting that acute bacterial infection may not be the primary driver of exacerbations [7]. The major taxonomic changes during exacerbations appear to reflect a reduction in commensal taxa rather than an excess of pathogenic organisms, and markers of neutrophilic inflammation, such as sputum neutrophil elastase and NETs, are independently associated with bronchiectasis severity even in the absence of chronic Pseudomonas or other infections [7].

Protective Commensals and Immunomodulation

An emerging concept is that certain commensal organisms may exert protective immunomodulatory effects under specific conditions. Organisms such as Veillonella, Prevotella, and Rothia mucilaginosa have been associated with the maintenance of mucosal immune homeostasis. Experimental work demonstrates that R. mucilaginosa possesses anti-inflammatory properties in vitro, inhibiting pathogen- and LPS-induced pro-inflammatory cytokine production, including IL-8, IL-6, IL-1β, and TNF-α [2]. Mechanistic studies indicate that R. mucilaginosa inhibits activation of the NF-κB pathway by reducing phosphorylation of IκBα. In adults with bronchiectasis, the relative abundance of Rothia species was negatively correlated with sputum concentrations of IL-8, IL-1β, and matrix metalloproteinases [2]. However, the immunomodulatory role of these commensals is context-dependent. As noted above, in NTM-positive bronchiectasis, oral commensals that are typically considered protective were associated with enhanced neutrophilic inflammation and severe disease phenotypes in both human observational data and murine models [6]. These observations challenge the conventional distinction between beneficial and harmful bacteria and support viewing the airway as a dynamic ecological system in which the functional role of individual taxa depends on the broader microbial and immunological context. The depletion of protective taxa in bronchiectasis may represent not merely a consequence of disease but a potential contributor to neutrophilic activation and airway destruction. However, causal relationships remain unproven and further interventional studies are needed.

Lipopolysaccharides, Neutrophil Extracellular Traps, and Airway Inflammation

Gram-negative bacteria, including P. aeruginosa and H. influenzae, release Lipo Poly Saccharides (LPS), endotoxins that trigger inflammatory cascades through Toll-Like Receptor 4 (TLR4) activation and downstream NF-κB signaling [2]. LPS may contribute to persistent airway inflammation by stimulating epithelial cells and macrophages to produce pro-inflammatory cytokines, recruiting and activating neutrophils, and promoting NET (Neutrophil Extracellular Traps) formation. NETs are web-like structures composed of DNA and antimicrobial proteins that bind pathogens but can also cause tissue damage, impair mucociliary clearance, and increase inflammation when present in excess [2]. In bronchiectasis, sputum NET levels have been associated with exacerbation frequency, reduced lung function, and increased mortality. High NET levels correlate with microbial dysbiosis and dominance of Proteobacteria genera, including Pseudomonas and Haemophilus [2]. LPS from P. aeruginosa has been demonstrated to stimulate NET formation *in vitro*, providing a mechanistic link

between gram-negative colonization and neutrophilic inflammation [2]. Macrolide therapy reduces sputum NET levels and improves clinical outcomes, suggesting that NETs are not merely markers of disease severity but active contributors to lung inflammation. Elevated circulating LPS levels may additionally contribute to systemic inflammatory burden, though the clinical utility of plasma LPS measurement as a biomarker in bronchiectasis remains to be established.

Non-Tuberculous Mycobacteria (NTM) and the Microbiome

NTM infection represents a critical intersection between microbiome science and bronchiectasis management. In the U.S. Bronchiectasis Research Registry, 50% of enrolled patients had NTM organisms grow in culture [8]. NTM can be both a cause and consequence of bronchiectatic disease, and the relationship between NTM and airway dysbiosis is an active area of investigation [9]. A recent study of 200 bronchiectasis subjects (108 NTM-negative, 92 NTM-positive) demonstrated that NTM-positive airways were enriched for Mycobacterium and oral commensals, and had higher NET levels in bronchoalveolar lavage fluid [6]. Distinct oral commensal taxa were associated with severe disease phenotypes including cavitary disease and frequent exacerbations [6]. The clinical relevance of NTM to the dysbiosis framework extends to therapeutic decision-making. NTM infection must be excluded before initiating long-term macrolide therapy, as macrolide monotherapy carries a risk of inducing azithromycin-resistant NTM [1,2]. This requirement underscores the importance of comprehensive microbiological assessment, including mycobacterial cultures, before implementing microbiome-modulating strategies.

Macrolide Therapy: Dual Antimicrobial and Immunomodulatory Effects

Long-term macrolide therapy occupies a central position in bronchiectasis management and is directly relevant to the dysbiosis framework, given its dual antimicrobial and immunomodulatory properties. The 2025 European Respiratory Society (ERS) guidelines issue a strong recommendation for long-term macrolide treatment in patients at high risk of exacerbations, upgrading from the conditional recommendation in the 2017 guidelines [2]. Meta-analysis of nine randomized controlled trials demonstrates a 52% reduction in exacerbation frequency (hazard ratio-HR 0.48, 95% CI 0.37–0.62) and a clinically meaningful improvement in quality of life (St George's Respiratory Questionnaire (SGRQ) mean difference -7.26 points) with long-term macrolides [2]. Individual patient data meta-analysis confirms that macrolides effectively reduce exacerbations across all subgroups, including patients with P. aeruginosa infection and those with fewer than three exacerbations per year [10]. Beyond their antimicrobial activity, macrolides downregulate proinflammatory cytokines, reduce inflammatory mediators and biofilms, and decrease sputum NET levels [1,2]. These immunomodulatory properties are particularly relevant to the dysbiosis hypothesis: Macrolides may attenuate the inflammatory component of the vicious vortex while simultaneously modifying the microbial environment. However, the impact of long-term macrolide exposure on airway microbial diversity and commensal populations remains incompletely characterized. While the 2025 ERS meta-analysis found no significant increase in antimicrobial-resistant organisms or new pathogen isolation in studies of 6-12 months duration, the long-term ecological consequences of sustained macrolide exposure on the airway microbiome warrant further investigation [2]. The preferred regimen is azithromycin 250mg daily or 500mg three times weekly [1,2]. Before initiation, patients should undergo baseline electrocardiography; Audiometry should be considered; and sputum cultures should be sent to exclude active NTM infection [1,2]. Adverse effects include nausea, diarrhea, QT-interval prolongation, and decreased hearing [1].

Brensocatib: Mechanism-Based Neutrophil-Targeted Therapy

The phase 3 ASPEN trial of brensocatib, a first-in-class dipeptidyl peptidase-1 (DPP-1) inhibitor that prevents activation of neutrophil serine proteases during neutrophil maturation, represents a paradigm shift toward mechanism-based therapies in bronchiectasis [5]. In this international trial of 1721 patients across 35 countries, the annualized rate of pulmonary exacerbations was 1.02 in the 10-mg brensocatib group, 1.04 in the 25-mg group, and 1.29 in the placebo group (rate ratio vs. placebo: 0.79 [95% CI 0.68-0.92, adjusted P = 0.004] with 10 mg; 0.81 [95% CI 0.69-0.94, adjusted P = 0.005] with 25mg) [5]. The HR for time to first exacerbation was 0.81 (95% CI 0.70-0.95) with 10mg and 0.83 (95% CI 0.70-0.97) with 25 mg [5]. At week 52, 48.5% of patients in each brensocatib group remained exacerbation-free compared with 40.3% in the placebo group [5]. Notably, the 25-mg dose demonstrated a significantly slower decline in FEV1 (24 mL vs. 62 mL with placebo; least-squares mean difference 38 mL, 95% CI 11–65, P = 0.04), representing a potential disease-modifying effect [5]. Brensocatib received FDA approval in August 2025 for the treatment of non-cystic fibrosis bronchiectasis in adult and pediatric patients 12 years of age and older [3,11]. The approximate 20% reduction in exacerbation frequency is comparable to estimates from systematic reviews of inhaled antibiotics (22%) but less than the reduction observed with oral macrolides (42%). However, differences in trial design and patient populations preclude direct comparison [4]. A network meta-analysis of 31 trials (N = 4,092) confirmed that both macrolides (rate ratio 0.44, 95% CI 0.35-0.56) and DPP-1 inhibitors (rate ratio 0.73, 95% CI 0.60-0.88) significantly reduce exacerbation frequency, with DPP-1 inhibitors remaining effective irrespective of baseline macrolide use [12]. The incidence of adverse events was similar across groups, except for a higher incidence of hyperkeratosis with brensocatib (3.0% with 25 mg, 1.4% with 10 mg, and 0.7% with placebo), a feature consistent with the phenotype of Papillon-Lefèvre syndrome, a rare genetic disorder due to DPP-1 deficiency [5]. No increase in bacterial infections was observed despite the antineutrophil mechanism [5]. From the perspective of the dysbiosis framework, anti-inflammatory therapies such as brensocatib attenuate protease-mediated tissue damage without

directly restoring microbial equilibrium, underscoring the potential value of combining immunomodulation with microbiome-directed approaches.

The Gut-Lung Axis and Nutritional Modulation of the Microbiome

The gut-lung axis represents an additional dimension of bronchiectasis pathophysiology. Alterations in intestinal microbiota composition may influence pulmonary inflammation through reduced production of Short-Chain Fatty Acids (SCFAs), dysregulation of immune signaling, and compromise of epithelial barrier integrity [1]. SCFAs, produced through bacterial fermentation of dietary fiber in the colon, exert immunomodulatory effects both locally and systemically. Butyrate promotes regulatory T cell differentiation, enhances intestinal barrier function, and inhibits NF-κB-mediated pro-inflammatory signaling [1,13]. A bronchiectasis-specific study by Narayana and colleagues demonstrated that concurrent gut and lung microbiome profiling in 57 patients with bronchiectasis revealed two distinct patient clusters differing by gut-lung microbial interactions, with a high gut-lung interaction cluster (characterized by lung Pseudomonas, gut Enterobacteriaceae, and gut Fusobacterium) associated with increased exacerbations and greater disease severity [14]. This provides direct evidence that the gut-lung axis is clinically relevant in bronchiectasis. Epidemiological evidence from non-bronchiectasis populations supports an association between dietary fiber intake and respiratory health. In the Atherosclerosis Risk in Communities (ARIC) study of 11,897 adults, participants in the highest quintile of total fiber intake demonstrated 60.2 mL higher FEV1 compared with those in the lowest quintile [2]. A meta-analysis of eight epidemiological studies demonstrated that each 10 g/day increase in fiber intake was associated with a 26% reduction in COPD risk [2]. Adherence to the Mediterranean diet has been associated with better lung function in cross-sectional studies [1]. These data are hypothesis-generating for bronchiectasis but derive from general population or COPD cohorts, and disease-specific clinical trials are needed before dietary interventions can be recommended as adjunctive therapy. In bronchiectasis specifically, malnutrition is prevalent and associated with adverse outcomes. Data from the U.S. Bronchiectasis Research Registry indicate that 12.3% of patients are underweight (Body mass index- BMI 18.5 kg/m^2), and underweight status is associated with lower FEV1, greater radiologic severity, and increased mortality [10]. The Bronchiectasis Severity Index incorporates BMI as a prognostic component [10]. Pulmonary rehabilitation programs that include nutritional counseling have been shown to improve BMI, serum albumin, and handgrip strength while reducing exacerbation frequency [8].

The Multibiome Perspective

Bronchiectasis dysbiosis extends beyond bacterial communities. The largest bronchiectasis mycobiome study to date, analyzing the multinational CAMEB cohort, demonstrated a distinct pulmonary mycobiome characterized by enrichment of Aspergillus, Cryptococcus, Clavispora, Botrytis, and Alternaria [12]. Geographic variation in mycobiome composition has been observed, with differing fungal profiles between Asian and European cohorts. Aspergillus fumigatus plays an important role not only in allergic bronchopulmonary aspergillosis but also through sensitization pathways that drive Th2-dominant inflammatory responses. Fungal-associated inflammatory endotypes have been proposed as treatable traits, with highly sensitized individuals demonstrating Th2-dominant profiles that may benefit from antifungal therapy or biologics targeting IgE or IL-5 [12]. Viral infections may additionally disrupt microbial homeostasis and predispose patients to secondary bacterial overgrowth. Integrative multi-kingdom analysis reveals that combining bacterial, fungal, and viral community data improves predictive models for clinical outcomes compared to bacterial profiling alone [12].

Inflammatory Endotypes and Precision Medicine

The heterogeneity of bronchiectasis has prompted efforts to classify patients into biologically distinct endotypes. Choi and colleagues delineated four inflammatory endotypes through cluster analysis of 199 patients across three European centers:

a) Milder neutrophilic inflammation with preserved microbial diversity

b) Mixed neutrophilic and type 2 inflammation

c) Severe neutrophilic inflammation with elevated IL-8, neutrophil elastase, and NETs, associated with P. aeruginosa-enriched microbiome and highest exacerbation risk.

d) Mixed epithelial and type 2 inflammation associated with Streptococcus-dominated microbiome [14,15]. Each endotype exhibited unique microbiome-level characteristics, and these clusters were consistent across patients regardless of country of origin [4].

These endotypes carry potential therapeutic implications. Endotype III, characterized by severe neutrophilic inflammation and Pseudomonas dominance, represents the most logical target population for brensocatib and long-term macrolide therapy. Endotypes II and IV, with type 2 inflammatory features, may benefit from biologic therapies targeting eosinophilic pathways, including anti-IgE or anti-IL-5 agents, particularly when fungal sensitization is present [12,16]. Up to 30% of patients with bronchiectasis may exhibit an eosinophilic endotype, characterized by elevated levels of type 2 cytokines and fractional exhaled nitric oxide [16]. Validation of these endotypes in larger, prospective cohorts and integration with treatment response data will be essential for translating endotyping into clinical practice.

Emerging Microbiome-Directed Therapies

If dysbiosis contributes substantially to the pathogenesis of bronchiectasis, prolonged broad-spectrum antibiotic therapy may paradoxically worsen microbial imbalance. Although antibiotics remain essential for the management of acute exacerbations, repeated exposure may further deplete protective commensal

organisms and reinforce pathobiont dominance [1]. Several microbiome-directed therapeutic strategies are under investigation.

Fecal microbiota transplantation has demonstrated preliminary benefits in modulating systemic inflammation and increasing SCFA production in chronic respiratory disease models, supporting the relevance of intestinal microbial composition in pulmonary immune regulation [1]. Bacteriophage therapy selectively targets pathogenic bacteria while preserving commensal diversity; case reports have described phage use in patients with bronchiectasis and cystic fibrosis, with variable outcomes, including clinical improvement and microbiological clearance, though publication bias inherent to case reports limits interpretation [5]. Five randomized controlled trials of bacteriophage therapy are currently ongoing [5]. Microbiome-based therapies, including prebiotics, probiotics, and postbiotics, represent additional avenues for modulating the gut-lung axis [17]. Genetically modified probiotics engineered to secrete beneficial molecules are being explored as potential adjunctive treatments for chronic respiratory diseases [17]. Direct modulation of the lung microbiome through inhaled probiotics is a plausible but early-stage concept requiring careful strain selection, formulation to preserve viability, and rigorous safety testing [18].

Methodological Considerations

Most existing microbiome studies in bronchiectasis remain observational and cross-sectional, limiting causal inference. Several methodological limitations warrant consideration. The majority of cited studies employ 16S rRNA amplicon sequencing, which provides genus-level (or at best species-level) taxonomic resolution but cannot characterize functional metabolic potential. Shotgun metagenomics offers species-level resolution and functional pathway analysis but is confounded by high proportions of human DNA in respiratory samples, reducing sequencing depth for microbial reads. Methodological heterogeneity across studies, including differences in sample collection (sputum vs. bronchoalveolar lavage), DNA extraction protocols, sequencing platforms, and bioinformatic pipelines, limits cross-study comparisons. Sequencing techniques cannot reliably distinguish viable organisms from residual genetic material, nor do they fully characterize microbial metabolic function. Contamination from sequencing reagents is a recognized concern in low-biomass respiratory samples and may introduce spurious taxa. Longitudinal multi-omics studies integrating metagenomics, metabolomics, and immune profiling will be essential for identifying clinically actionable targets. Integration of microbiome profiling into clinical practice may eventually enable precision medicine strategies tailored to individual microbial and inflammatory phenotypes. Future research should validate dysbiosis scores that incorporate alpha diversity metrics, commensal depletion indices, and pathobiont burden as tools to guide individualized therapeutic decisions.

Conclusion

Bronchiectasis is increasingly recognized as a condition in which an imbalance in the microbial ecosystem, rather than isolated chronic infection alone, may contribute to persistent inflammation and structural lung injury. The updated vicious vortex model, inflammatory endotyping, and gut-lung axis research collectively support a more nuanced understanding of disease pathogenesis. The approval of brensocatib as the first mechanism-based therapy for non-cystic fibrosis bronchiectasis, alongside the strong evidence base for long-term macrolide therapy, provides clinicians with complementary tools targeting the inflammatory component of the vicious vortex. Future studies should evaluate whether dietary and microbiome-modulating interventions can complement pharmacologic therapies by restoring microbial homeostasis through the gut-lung axis.

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