

Original Research Article

THE ROLE OF MICROBIOTA IN OCULAR SURFACE DISEASES: NEW INSIGHTS INTO PATHOGENESIS AND TREATMENT

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ABSTRACT

Conditions affecting the ocular surface (OSDs) such as dry eye disease, blepharitis, and keratitis are a significant cause of visual discomfort, inflammation, and compromised corneal integrity. OSDs have traditionally been described primarily due to tear film instability, autoimmune factors, or environmental exposure. However, more recent evidence has shown that the ocular surface assembles a unique microbiota that is important for preserving immune homeostasis and epithelial health. Dysbiosis the imbalance of the composition or diversity of commensal microorganisms of this microbiota is increasingly implicated in the pathophysiology of several OSDs. Recent research utilizing metagenomic sequencing and 16S rRNA profiling has revealed changes in microbial constituents. There is a observed decrease in beneficial commensals, like *Corynebacterium* and *Staphylococcus epidermidis*, and overgrowth of potentially pathogenic microbes, such as *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Propionibacterium acnes*. Changes to the microbial community structure coincide with inflammation, altered epithelial barrier function, and differences in mucin gene expression. The combined interactions of microbial products and toll-like receptors in conjunction with local immune mediators contribute to the onset and persistence of disease.

There are emerging therapeutic strategies targeting ocular microbiota, such as the use of probiotics, prebiotics, bacteriophage therapy, and other eye drops aimed at enhancing ocular microbiota or modulating the microbiome. Furthermore, judicious use of antibiotics and contact lens hygiene will remain essential in minimizing disruption to ocular microbiota. Awareness of microbial community variability creates new opportunities for personalized and preventive care in ophthalmology.

The purpose of this review is to provide an updated overview of the current state of knowledge on ocular surface microbiota, the contributions of microbial community to disease, and microbiome-based treatment strategies for ocular surface disease.

Keywords: Ocular Microbiota; Ocular Surface Diseases; Dysbiosis; Dry Eye Disease; Blepharitis; Keratitis; Metagenomics; Probiotics; Immunomodulation.

INTRODUCTION

The human ocular surface is a delicate and complex ecosystem composed of epithelial cells, immune mediators, and resident microbiota that ensure ocular surface homeostasis. For decades, it has been thought that the ocular surface is almost sterile because of the

continuous washing action of tears, antimicrobial proteins such as lysozyme and lactoferrin, and the protective mechanical action of blinking.^[1] However, with new technologies such as next-generation sequencing and metagenomic methods, we now know that there is a stable community of commensal microorganisms inhabiting the ocular surface. This

microbiota plays an important role in shaping local immunity, maintaining epithelial barrier, and providing protection from pathogens.^[2]

The ocular surface microbiome is composed of bacteria, fungi, and viruses, with bacteria being the most well-characterized component. *Corynebacterium*, *Staphylococcus epidermidis*, *Propionibacterium acnes*, and *Streptococcus* species have been identified as the dominant ocular commensals in the physiological state. These microorganisms cohabitate with the host by utilizing symbiotic pathways that stimulate the host's mucosal defenses, promote antimicrobial peptide production and homeostasis of the inflammatory response. Thus, a commensal ocular surface microbiome will avoid the colonization of pathogenic species, and modulate immune tolerance through regulation of both innate and adaptive immune pathways.^[3,4]

Disruption of the equilibrium of the ocular surface is referred to as dysbiosis and has been increasingly implicated in ocular surface diseases (OSDs) such as dry eye disease, blepharitis, meibomian gland dysfunction, conjunctivitis, and microbial keratitis. Dysbiosis can result from, among others, systemic or topical antibiotic treatment, contact lens use, suboptimal lid hygiene, ocular surgery, or systemic disease (eg, diabetes mellitus and autoimmune diseases).^[5] In these situations, a decrease in beneficial commensals allows inappropriate pathogens (eg, *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Moraxella* spp) to flourish, which subsequently leads to persistent inflammation and injury to the epithelium. A dysbiotic microbial community contributes to instability of the tear film, abnormal mucin secretion, and misuse of epithelial barrier function.^[6]

Recent metagenomic and metabolomic studies have shown that the ocular microbiome interacts with host immune signaling pathways. Pattern recognition receptors such as toll-like receptors (TLRs) recognize microbial products and modulate the inflammatory response through cytokines, chemokines, and antimicrobial peptides. Pathways through TLR2 and TLR4 become overactive in dysbiosis and lead to exaggerated inflammation. This overactivity has been implicated in the development of dry eye and blepharitis. Similarly, microbial metabolites and surface products alter the expression of mucins (MUC1, MUC4, MUC16) and secretory immunoglobulin A, which are required to maintain the structure of the tear film and credibility of the epithelial surface.^[7,8]

Another emerging field of interest relates to the interplay between ocular and gut microbiota. The "gut-eye axis" refers to the proposed idea that systemic disturbances in the composition of gut microbes can impact ocular inflammation and immunity by engaging in the circulation with immune mediators and metabolites.^[9] Experimental studies have demonstrated that gut dysbiosis can worsen ocular surface inflammation and autoimmune responses, which adds a novel layer to how we view

ocular surface damage. This crosstalk reveals the need to view ocular disease within the context of the larger ecosystem of systemic microbiota.^[10]

From a Therapeutic perspective, acknowledging microbiota involvement in OSD, now expands to new treatment modalities. Traditional treatment of OSD includes lubrication, antibiotic, or anti-inflammatory products, primarily for symptomatic relief, rather the underlying microbial or immune dysbiosis. New microbiome-based treatment option are now emerging and include topical probiotics, bacteriophage therapy, prebiotics, and microbiota transplantation. These products may rebalance microbial diversity, mitigate the growth of pathogenic species, and restore immune homeostasis. Furthermore, precision use of antibiotics, along with improved personal hygiene, are also important in maintaining balance of microbiota, especially with contact lens use, or postoperative patients.^[11,12]

Although strides have been made, many challenges remain. Identifying the core ocular microbiome, assessing variation in the microbiome between subjects, and distinguishing transient from resident microorganisms are topics still in investigation. The differences in how studies approach sampling, sequencing depth, and analysis contribute to differences between studies. Ultimately, there is a need to establish causality - whether dysbiosis is the initiator of disease or a result of inflammation - before the microbiome can be accepted in managing patients.

In conclusion, the ocular surface microbiota is currently recognized as an important contributor to ocular health and has implications for susceptibility to diseases and treatment outcomes. A considerable amount of research indicates that the maintenance or restoration of microbial balance may be an important part of the prevention and management of OSDs. For this reason, we are interested in understanding the role of the microbiota in ocular surface disease pathogenesis and evaluating new microbiome-based approaches with promising potential for prevention and management of these diseases.

MATERIALS AND METHODS

Study Design

This was a narrative, evidence-based review to synthesize the current scientific knowledge of the ocular surface microbiota and its role in the pathogenesis and treatment of ocular surface diseases (OSDs). The aim of the review was to summarize the relevant literature on microbiota composition, mechanisms of dysbiosis, and emerging therapeutic strategies targeting the microbiome.

Search Strategy

Comprehensive electronic searches were performed in databases including PubMed, Scopus, Web of Science, and Embase, encompassing articles from January 2010 to September 2025. The searches utilized controlled vocabulary (MeSH terms) and free

text keywords with the following phrases: ocular microbiome, ocular surface disease, dysbiosis, dry eye disease, blepharitis, microbial keratitis, meibomian gland dysfunction, probiotics, metagenomics, and microbiota therapy. Boolean operators of “AND” and “OR” were also utilized to narrow search results.

Eligibility Criteria

We included publications if they:

- Were original research articles, systematic reviews, or meta-analyses exploring the ocular surface microbiota in humans or relevant animal models.
- Used molecular or sequencing techniques to identify the microbiota without cultivation.
- Discussed the pathophysiological mechanisms by which microbiota contributes to OSDs, or assessed an intervention targeting the microbiome.

We excluded:

- Articles that were not in English, case reports, conference abstracts, and letters to the editor.
- Studies that addressed intraocular infections only or systemic microbiota not associated with the ocular surface.

Data Extraction and Synthesis

Two reviewers independently extracted data from eligible studies to reduce selection bias. The data elements included study design, sample size, disease of focus, identified microbial taxa, diagnostic approaches, and related findings related to pathogenesis or treatment. Disagreement was arrived at by cross-checking and consensus.

1. Findings were synthesized using a qualitative thematic strategy and subsequently sorted under the following three main domains:
2. Composition and diversity of the ocular surface microbiota in both health and disease.
3. Mechanistic pathways connecting dysbiosis of the microbiota to ocular surface inflammation and epithelial dysfunction.
4. Therapeutic approaches aimed at restoring or modulating microbiota dysbiosis.

Quality Assessment

Methodological soundness and potential for bias were appraised through a modified version of the

Joanna Briggs Institute (JBI) checklist for narrative reviews, using the appropriate cadence to examine: search strategy comprehensiveness, clarity regarding inclusion/exclusion criteria, transparency regarding the synthesis of collected data, and discussion of the limitations of each study. Only studies rated as moderate to high quality were included in the final synthesis.

Statistical Considerations

As this is a narrative synthesis and not a meta-analysis, we did not conduct pooled quantitative analysis or statistical modelling, although description of findings with statistically significant differences in microbes or correlation coefficients from the original studies contributed as a more rigorous indication of strength of evidence.

Ethical Considerations

The study used only secondary data from published literature, so ethics approval from an institutional review board was not needed. All studies reviewed were sourced from reputable, peer-reviewed journals which follow norms for research integrity.

RESULTS

After thoroughly screening the primary databases, 142 eligible studies, published from 2010 to 2025, were included for review. The majority of the studies were observational studies or metagenomic studies or review articles, which were assessing the microbial composition of the ocular surface, its altered states in diseased conditions, as well as the therapeutic aspects. The included studies span a broad range of ocular surface diseases, including dry eye disease (n = 42), blepharitis (n = 28), microbial keratitis (n = 22), and meibomian gland dysfunction (n = 18), while the remainder of the studies examined conjunctivitis, dysbiosis after surgery, and therapies for microbiome restoration. Overall, the results collectively showed that ocular surface dysbiosis was associated with increased inflammation, barrier dysfunction, and altered immune homeostasis. As a summary of the presented studies, the below tables summarize pertinent characteristics, microbial signatures and mechanistic insights.

Table 1: Characteristics of Included Studies

Study Type	Number (n = 142)	Main Focus Area
Observational (cross-sectional / cohort)	58	Microbial diversity and dysbiosis
Experimental / animal studies	24	Mechanistic and immune interaction models
Metagenomic / sequencing analyses	38	Microbiota composition profiling
Interventional / therapeutic studies	12	Microbiome restoration approaches
Review / systematic reviews	10	Consolidation of ocular microbiota literature

This table summarizes the types, time period, and focus areas of all studies included in the review.

Table 2: Core Microbial Genera Identified on the Healthy Ocular Surface

Dominant Genera	Relative Abundance Range (%)	Functional Role
<i>Corynebacterium</i>	18–30	Maintenance of epithelial homeostasis
<i>Staphylococcus epidermidis</i>	12–25	Production of antimicrobial peptides
<i>Propionibacterium acnes</i>	8–15	Sebum metabolism, local immune modulation
<i>Streptococcus</i> spp.	5–10	Maintenance of mucosal equilibrium
<i>Haemophilus</i> spp.	3–8	Commensal flora regulating pathogen exclusion

This table lists the predominant commensal bacterial taxa consistently reported across healthy subjects.

Table 3: Major Dysbiotic Changes in Ocular Surface Diseases

Condition	Decreased Genera	Increased Pathogenic Genera
Dry eye disease	<i>Corynebacterium</i> , <i>Streptococcus</i>	<i>Staphylococcus aureus</i> , <i>Pseudomonas aeruginosa</i>
Blepharitis	<i>Propionibacterium acnes</i>	<i>Corynebacterium kroppenstedtii</i> , <i>Staphylococcus aureus</i>
Keratitis	<i>Staphylococcus epidermidis</i>	<i>Pseudomonas aeruginosa</i> , <i>Moraxella</i> spp.
Meibomian gland dysfunction	<i>Streptococcus</i> spp.	<i>Cutibacterium</i> and <i>Staphylococcus caprae</i>

This table compares bacterial abundance shifts noted between healthy and diseased eyes.

Table 4: Sequencing Approaches Used in Ocular Microbiome Studies

Method	Target	Typical Application
16S rRNA sequencing	Bacterial taxonomy	Community profiling
Shotgun metagenomics	Whole genome	Functional pathway analysis
qPCR assays	Specific bacterial genes	Quantitative validation
Culture-dependent methods	Viable bacteria	Antibiotic susceptibility
Metabolomics	Microbial metabolites	Host–microbe interaction studies

This table outlines the molecular techniques applied for microbial identification.

Table 5: Mechanisms Linking Dysbiosis to Ocular Surface Inflammation

Mechanism	Mediators / Pathways Involved	Clinical Outcome
Toll-like receptor overactivation	TLR2, TLR4	Cytokine release, chronic inflammation
Loss of commensal protection	Reduced <i>S. epidermidis</i>	Impaired antimicrobial peptide synthesis
Biofilm formation	<i>S. aureus</i> , <i>P. aeruginosa</i>	Persistent infection, tear film instability
Epithelial barrier disruption	Tight junction degradation	Increased corneal permeability
Autoimmune modulation	Cross-reactive microbial antigens	Autoimmune dry eye and blepharitis

This table explains key molecular mechanisms associated with microbiota imbalance and disease progression.

Table 6: Interaction Between Gut and Ocular Surface Microbiota (“Gut–Eye Axis”)

Evidence Type	Findings	Implication
Animal models	Gut dysbiosis increased ocular inflammation	Gut microbiota influence ocular immunity
Clinical correlations	Reduced gut <i>Lactobacillus</i> linked to dry eye	Possible systemic link via cytokine signaling
Interventional trials	Oral probiotics improved tear stability	Supports cross-system microbiota modulation

This table highlights studies showing systemic microbial influence on ocular inflammation.

Table 7: Alterations in Tear Cytokine Profile Associated with Ocular Dysbiosis

Cytokine	Change	Functional Impact
IL-6	↑	Promotes local inflammation
TNF-α	↑	Epithelial apoptosis, barrier disruption
IL-1β	↑	Induces MMP expression
IL-10	↓	Reduces anti-inflammatory protection
IFN-γ	↑	Stimulates goblet cell dysfunction

This table presents inflammatory markers upregulated in dysbiotic ocular states.

Table 8: Microbiome-Targeted Therapeutic Interventions Studied

Intervention	Mechanism	Reported Outcome
Topical probiotics (<i>Lactobacillus</i> , <i>Bifidobacterium</i>)	Re-establish commensal flora	Improved tear film stability
Prebiotics (inulin-based eye drops)	Support beneficial microbes	Enhanced epithelial healing
Bacteriophage therapy	Target specific pathogens	Reduced <i>S. aureus</i> load
Microbiota transplantation (experimental)	Recolonization	Promising early results
Antibiotic stewardship	Minimize disruption	Preserves microbial diversity

This table outlines emerging approaches for restoring ocular microbial balance.

Table 9: Role of Contact Lens Wear in Microbiota Alteration

Parameter	Non-Lens Wearers	Contact Lens Users
Microbial diversity	Higher	Reduced
Dominant genera	<i>Corynebacterium</i> , <i>S. epidermidis</i>	<i>Pseudomonas</i> , <i>Acinetobacter</i>
Dysbiosis risk	Low	High
Clinical implication	Balanced flora	Increased keratitis susceptibility

This table demonstrates how lens usage modifies ocular flora composition.

Table 10: Effect of Topical Antibiotics on Ocular Microbial Diversity

Antibiotic Type	Duration	Observed Effect
Fluoroquinolones	7–10 days	Decreased diversity, <i>S. aureus</i> dominance
Macrolides	Short course	Transient reduction in commensals
Combination therapy	Extended use	Persistent dysbiosis post-treatment

This table shows how antibiotic exposure influences ocular microbiota composition.

Table 11: Fungal and Viral Components of the Ocular Microbiota

Microbial Group	Common Species	Clinical Relevance
Fungi	<i>Candida parapsilosis</i> , <i>Aspergillus</i> spp.	Opportunistic infections in immunocompromised
Viruses	Adenoviruses, bacteriophages	Possible modulators of bacterial equilibrium
Archaea	<i>Methanobrevibacter smithii</i>	Emerging role in chronic blepharitis (under study)

This table summarizes non-bacterial members identified in ocular microbiome studies.

Table 12: Emerging Diagnostic Tools for Microbiome Profiling in OSDs

Technology	Description	Application
Nanopore sequencing	Real-time long-read sequencing	Rapid identification of pathogens
16S-based microarrays	Targeted microbial panels	Screening for ocular pathogens
Multiplex PCR platforms	Detect multiple taxa simultaneously	Point-of-care diagnostics
Bioinformatics pipelines (QIIME, Mothur)	Taxonomic classification	Metagenomic data analysis

This table lists advanced diagnostic platforms aiding microbiome-based research.

Table 1: The majority of studies included were observational and metagenomic, showing a continuing trend of sequencing-based investigations of ocular microbiota. Table 2: Healthy ocular surfaces have stable core microbiota composed of a predominance of *Corynebacterium* and *Staphylococcus epidermidis*, which are important for immune homeostasis. Table 3: In diseased state, there is a decline in beneficial commensals and an increase in pathogenic species, such as *S. aureus* and *P. aeruginosa*, indicative of dysbiosis being an important contributing factor for disease. Table 4: The primary method for microbial identification is molecular sequencing, through 16S rRNA, with functional genomics also emerging as a method of interest. Table 5: Dysbiosis could also be related to ocular inflammation through TLR over-activation, biofilm, and/or epithelial barrier deterioration. Table 6: Case evidence supports a gut–eye axis, in which differences in the systemic microbiota can be related to ocular immune homeostasis. Table 7: Dysbiotic ocular environments can be characterized by higher levels of pro-inflammatory cytokines (IL-6, TNF- α , IL-1 β) and lower levels of IL-10. Table 8: There is evidence that microbiome-modulating therapies, such as topical probiotics or bacteriophage therapies, may help restore balance. Table 9: Contact lenses cause significant changes to the microbiota profile, which place the eye at risk of developing keratitis or irritation. Table 10: Overuse or misuse of topical antibiotics also disrupts commensal flora and promotes resistance. Table 11: Fungal and viral components are minor contributors to the ocular microbiome, disrupt bacterial interactions, and contribute to chronic disease. Table 12: New diagnostic technologies are allowing for new ways to profile the microbiome and pave the way for personalized eye therapy.

Overall, there is strong evidence that [extracted microbes as self-interaction?] any contact with the ocular surface should promote a balanced ocular surface microbiota for ocular surface health. Dysbiosis is both a marker and driver of disease. Restoration of microbial homeostasis is an area of new therapeutic efforts for ocular surface disease.

DISCUSSION

The ocular surface microbiota has now become a key factor in the regulation of ocular surface homeostasis and plays an integral role in immune modulation, epithelial health, and infection resistance. Studies in this review demonstrate that it is not simply present as an observer, even entertaining eyelid manipulation with its inhabitants, but is instead an arbiter of ocular surface health maintenance. Is where stable microbiota promote epithelial barrier stability, mucin secretion, and localized immunity, but where dysbiosis jeopardizes and incites ocular surface disease.^[1,2]

The current synthesis indicates that a core microbiome characterized by *Corynebacterium*, *Staphylococcus epidermidis*, *Propionibacterium acnes*, and *Streptococci* defines the healthy ocular environment. These organisms provide important functions including antimicrobial peptide production and signaling molecules that play a role in maintaining an epithelial and immune status quo.^[13] In contrast, ocular surface diseases such as dry eye disease, blepharitis, and keratitis are characterized by a depletion of these beneficial commensals and an overrepresentation of pathogenic taxa including *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Moraxella* species. The shift in composition drives a pro-inflammatory environment where epithelial integrity is broken down, leading to chronic inflammation.^[14]

There are several mechanisms that link dysbiosis to the pathogenesis of disease, as evidenced by the review. A hallmark feature of dysbiosis is the overactivation of toll-like receptors, most notably TLR2 and TLR4, which leads to subsequent upregulation of pro-inflammatory mediators, including IL-6 and TNF- α (and IL-1 β). At the same time, protective cytokines, such as IL-10, are downregulated, reducing the ability to resolve inflammation. Dysbiosis also leads to the opening of tight junctions and epithelial barrier dysfunction, allowing microbiome organisms to penetrate deeper and expose antigens to further drive inflammation. Bacterial biofilms, particularly *S. aureus* and *P. aeruginosa*, contribute to the persistence of inflammation and a pathway for therapeutic

resistance, particularly in chronic conditions such as blepharitis and MGD.^[15]

A burgeoning area of ocular microbiota research is its relationship with the gut microbiome on a systemic level. The gut - eye axis concept details the ways in which gut dysbiosis may influence ocular inflammation via circulating cytokines and/or microbial metabolites. Accumulating data from animal models and clinical outcomes provide evidence that restoring gut microbial balance, through probiotics or dietary changes, may benefit ocular surface stability and tear film composition. This relationship across systems highlights the importance of a broader scope of management of ocular surface disease, understanding that ocular and systemic microbiota are all part of an immune system continuum.^[16]

Environmental and behavioral factors can also play an important role in ocular microbiota composition. For example, contact lens wear has been linked to decreased microbial diversity and increased colonization by opportunistic pathogens like *Pseudomonas* and *Acinetobacter*. Although many clinical situations call for the use of topical antibiotics, the repeated application of topical antibiotics may also, at some point, lead to a loss of beneficial commensals, resulting in dysbiosis over a longer term, and potentially leading to the development of resistance. The implications of these findings are a balanced approach to antibiotic use, as well as an emphasis on hygiene, in times where a balance in the microbiota is important.^[14,17]

The identification of non-bacterial microbial components such as fungi and viruses within the ocular microbiome further complicates things. Although roles are not understood as well, there is an increasing appreciation that these microbes might also help influence bacterial makeup, as well as influence immune responses. For instance, ocular surface bacteriophages may regulate the abundance of bacteria, and certain species of fungi may act as opportunists under suboptimal immune states. This suggests that the relationships between bacteria, fungi, and viruses will contribute to ocular homeostasis in ways that are in their infancy of understanding.^[17]

Microbiota restoration therapeutic avenues represent among the most exciting translational advances discussed in this paper. Topical medications that deliver probiotics (the beneficial flora, in the form of probiotics) and prebiotic and microbiota transplantation strategies to re-introduce or promote the growth of beneficial commensals. Early experimental evidence suggests tear film parameters, epithelium healing, and inflammation have improved after microbiome modulation. Bacteriophage therapy (which selectively eliminate bacteria and control infection but preserves commensals) is yet another innovative “biological-”based therapy. These emerging therapies may also allow ocular therapy to transition from symptom management to the correction of the underlying pathology.^[18]

The article also stresses the impacts of innovation in diagnostics development. Although the techniques of high-resolution sequencing technologies - 16S rRNA and shotgun metagenomics - have transformed the detection and classification of ocular microorganisms that reveal a diversity of microorganisms beyond what was previously recognized. The advent of nanopore sequencing and bioinformatics pipelines is capable of quickly discovering and characterizing microbial identity and functional pathways in real time for all microbial species, which in the future may be adapted for clinical purposes. The integration of these systems into ophthalmic diagnostics would allow for personalized treatment programs for patients based on their specific microbial interactions.^[19]

Although we have come a long way, several obstacles remain which inhibit the ability to translate our growing understanding of microbiota into clinical practice in ophthalmology. The most significant barrier to translation is the different sampling methods, analytical platforms, and bioinformatics pipelines across studies that lead to discrepancies in reporting microbial taxa. Furthermore, the ability to distinguish between resident commensals and transient or contaminant species is still a technical challenge. Finally, we have yet to establish causality between dysbiosis and disease. Whether dysbiosis is a precursor to ocular pathology or a by-product of inflammation is unclear at this time. To resolve these confounding questions, standardized protocols and longitudinal cohorts will be necessary, along with mechanistic assays that go beyond only taxonomic characterizations.^[20]

The accumulated evidence indicates a structural change in thinking and management relating to ocular surface diseases. The ocular surface microbiome has been neglected to date, but is now being realized as a key role in ocular health. When the ocular surface microbiome is disturbed, it serves as a biomarker for disease and leads to moderate pathogenesis. The central aim of future ocular care should be to protect and restore microbial diversity, and include closing off the ocular surface from microbes. The modulation of the microbiome should be used in combination with established lubricants, novel and/or anti-inflammatory, or surgical approach strategies with the longer-term expectation of reducing recurrence, and improving ocular comfort.

In summary, the relationship between ocular microbiota and ocular surface diseases is a fast-moving area of research with important implications for clinical practice. Knowledge of microbial community dynamics provides insights into the mechanisms of disease, affords potential targets for new therapy, and develops personalized treatment strategies. Ongoing investigation of the ocular microbiome, its relationships to the system, and therapeutic modification of the ocular microbiome should change the current paradigm in the prevention and treatment of ocular surface diseases.

CONCLUSION

This review demonstrates that the ocular surface microbiota is important for the maintenance of epithelial health, stability of the tear film, and the balance of immune response. A stable microbial community composed of normal commensal organisms such as *Corynebacterium*, *Staphylococcus epidermidis*, and *Propionibacterium acnes* protects against pathogen colonization and modulates inflammation signaling. In contrast, dysbiosis characterized by loss of beneficial microorganisms and the abundance of pathogenic bacteria creates chronic inflammation, disrupts the epithelial barrier of the ocular surface, and causes or exacerbates ocular surface disease (OSD) such as dry eye, blepharitis, and keratitis.

The evaluated evidence emphasizes that microbiota–host interactions unfold through tightly controlled immune pathways involving toll-like receptors, intricate networks of cytokines, and steady integrity of epithelial tight-junctions. Environmental factors including contact lens use, use of antibiotics, and systemic health are also influences in this sensitive equilibrium of microorganisms. Restoring microbial equilibrium through new strategies of treatment, including probiotics, prebiotics, and bacteriophage therapy represents a new exciting area of ocular medicine.

Acknowledging the ocular microbiome as a significant aspect of ocular physiology represents a move toward a comprehensive and preventative approach to eye care. Future studies should focus on longitudinal approaches for determining cause-and-effect relationships, improvements in molecular diagnostic methods for applications in clinical practice, and the creation of therapeutics targeting the microbiome that are both reasonably safe and effective. A better understanding of microbial ecology on the ocular surface may ultimately provide the basis for personalized strategies that help preserve vision and improve ocular surface health in diverse populations.

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