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Commentary

The Impact of Microbiota on Tear Film Stability and Dry Eye Disease

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DESCRIPTION

Dry Eye Disease (DED) is a common condition affecting millions of people worldwide. Characterized by a lack of sufficient lubrication and moisture on the surface of the eye, DED leads to discomfort, visual disturbances, and potential damage to the ocular surface. The pathophysiology of DED is multifaceted, involving tear film instability, increased osmolarity, inflammation, and damage to the ocular surface. Recently, emerging research has begun to explore the relationship between DED and the human microbiota, particularly the ocular and gut microbiomes. This article delves into the interesting connections between DED and microbiota, highlighting recent findings and their implications for understanding and managing this prevalent condition.

Dry eye disease can result from various factors, including aging, hormonal changes, autoimmune disorders, environmental conditions, and prolonged use of contact lenses. The condition is broadly classified into two types: aqueous-deficient dry eye (ADDE) and Evaporative Dry Eye (EDE). ADDE is associated with decreased tear production, whereas EDE is linked to increased tear evaporation. Both types can coexist, leading to a complex interplay of symptoms and underlying causes. The human microbiota comprises trillions of microorganisms, including bacteria, viruses, fungi, and archaea, residing in various parts of the body. These microorganisms play vital roles in maintaining health by aiding digestion, synthesizing vitamins, and modulating the immune system. The microbiota is primarily studied in the gut, but it also exists on the skin, in the oral cavity, and in the ocular environment. The ocular surface, although less densely populated than the gut, hosts a unique microbiome. The composition of the ocular microbiome is influenced by various factors, including the external environment, age, and overall health. Studies have identified several bacterial genera commonly present on the ocular surface, such as *Staphylococcus*, *Corynebacterium*, and *Propionibacterium*. Research suggests that a balanced ocular microbiome contributes to maintaining ocular surface health by preventing colonization by pathogenic microorganisms, modulating the local immune response, and supporting tear film stability. Disruptions in this balance, known as dysbiosis, have been implicated in various ocular conditions, including DED. One of the primary mechanisms by which the microbiota influences DED is through inflammation. Dysbiosis of the ocular microbiome can lead to an overgrowth of pathogenic bacteria, triggering an inflammatory response. Inflammation plays a critical role in the pathogenesis of DED by causing damage to the lacrimal glands and ocular surface, further exacerbating tear film instability and dryness. For instance, *Staphylococcus aureus* and *Propionibacterium acnes* are commonly associated with Meibomian Gland Dysfunction (MGD), a leading cause of EDE. These bacteria can invade and block the meibomian glands, leading to decreased lipid secretion and increased tear evaporation. The microbiota interacts closely with the immune system, and dysbiosis can lead to altered immune responses. In DED, an overactive immune response can cause chronic inflammation and tissue damage. Studies have shown that patients with DED often have altered ocular microbiota profiles, with a decrease in beneficial commensal bacteria and an increase in pathogenic species. This imbalance can exacerbate immune dysregulation, perpetuating the cycle of inflammation and dryness. The stability of the tear film is vital for maintaining ocular surface health and comfort. The microbiota can influence tear film composition and stability through the production of metabolites and enzymes. Certain bacteria produce lipases that break down tear film lipids, leading to increased tear evaporation and instability. Conversely, a healthy ocular microbiome may produce substances that support tear film integrity and reduce evaporation. Beyond the ocular microbiome, the gut microbiota also appears to play a role in ocular health through the so-called gut-eye axis. The gut microbiota interacts with the immune system and can influence systemic inflammation, which in turn affects the eyes. Dysbiosis in the gut can lead to increased intestinal permeability (often referred to as "leaky gut"), allowing bacteria and their metabolites to enter the bloodstream. This can trigger systemic inflammation, which may impact distant organs, including the eyes. Patients with autoimmune diseases such as Sjögren's

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syndrome, which is associated with both gut dysbiosis and DED, exemplify this connection. The gut microbiota produces various metabolites, such as Short-Chain Fatty Acids (SCFAs) that have systemic effects. SCFAs can modulate immune responses and inflammation, potentially influencing ocular surface health. Alterations in gut microbiota composition may lead to changes in these metabolites, contributing to the development or exacerbation of DED. Probiotics, live beneficial bacteria, and prebiotics, substances that promote the growth of beneficial bacteria, have shown potential in modulating the microbiota and reducing inflammation. Clinical studies are exploring the use of oral probiotics to improve gut health and, by extension, ocular health. Additionally, topical probiotics could be developed to restore the balance of the ocular microbiome.