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Commentary

Chronic Inflammation as a Central Driver of Human Disease Mechanisms and Clinical Implications

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DESCRIPTION

Chronic inflammation represents one of the most significant and complex biological responses underlying a wide spectrum of human diseases. Inflammation is a fundamental biological response that protects the body against infections, injuries, and harmful environmental stimuli. Under normal circumstances, acute inflammation is a tightly regulated process that eliminates pathogens, removes damaged tissues, and promotes healing. Once the harmful stimulus has been eliminated, the inflammatory response resolves, allowing tissues to return to their normal physiological state. However, when inflammation fails to resolve and persists over an extended period, it develops into chronic inflammation. Chronic inflammation is characterized by the continuous activation of immune cells and sustained production of inflammatory mediators, leading to progressive tissue damage and impaired organ function. Although chronic inflammation contributes to the development of numerous systemic diseases, it has become increasingly recognized as a major factor in the pathogenesis of several ocular disorders. The eye is an immune-privileged organ with specialized mechanisms that normally maintain a balanced immune environment to preserve vision. Disruption of this balance by persistent inflammatory processes can damage delicate ocular tissues, ultimately resulting in visual impairment or blindness. Therefore, chronic inflammation is now considered a central driver of many eye diseases, influencing both disease progression and clinical outcomes.

The eye contains highly specialized structures, including the cornea, conjunctiva, lens, retina, optic nerve, and uveal tract, all of which require a carefully controlled immune response to maintain transparency and visual function. Unlike many other organs, the eye limits excessive immune activity through mechanisms collectively known as immune privilege. This protective system reduces unnecessary inflammatory reactions that could interfere with light transmission and neural signaling. However, various internal and external factors such as aging, diabetes, autoimmune disorders, infections, environmental pollutants, ultraviolet radiation, oxidative stress, and genetic susceptibility can overwhelm these protective mechanisms. Once chronic inflammation becomes established, immune cells continuously infiltrate ocular tissues and release pro-inflammatory cytokines, chemokines, growth factors, and reactive oxygen species. These inflammatory mediators disrupt normal cellular function, damage blood vessels, impair neuronal survival, and stimulate tissue remodeling, ultimately contributing to the development of chronic eye diseases.

Oxidative stress further accelerates chronic inflammation within the eye. During normal metabolism, cells produce reactive oxygen species in controlled amounts that participate in cellular signaling. However, chronic inflammation significantly increases reactive oxygen species production while reducing antioxidant defenses. Excessive oxidative stress damages proteins, lipids, DNA, and cellular organelles within retinal neurons, corneal epithelial cells, and vascular endothelial cells. This cellular damage activates additional inflammatory pathways, creating a vicious cycle in which inflammation and oxidative stress continuously reinforce one another. Because ocular tissues consume large amounts of oxygen and are constantly exposed to light, they are particularly susceptible to oxidative injury, making chronic inflammation an especially important contributor to age-related ocular diseases. One of the most common inflammatory eye disorders associated with chronic inflammation is dry eye disease. Traditionally regarded as a disorder of insufficient tear production, dry eye disease is now recognized as a chronic inflammatory condition affecting the ocular surface. Tear film instability causes increased evaporation, exposing epithelial cells to environmental stress. Damaged epithelial cells release inflammatory mediators that attract immune cells to the cornea and conjunctiva. These inflammatory cells further damage the lacrimal glands and ocular surface, reducing tear production and worsening tear film instability. Patients experience persistent symptoms including ocular discomfort, burning sensation, foreign body sensation, redness, excessive tearing, fluctuating vision, and sensitivity to light. Without appropriate treatment, chronic inflammation may result in corneal ulceration, epithelial defects, and permanent visual impairment. Age-related macular degeneration is another major cause of irreversible blindness in older adults in which chronic inflammation plays a critical pathogenic role. The macula is responsible for central vision, reading, and detailed visual tasks.

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These deposits activate the complement system, initiating a persistent inflammatory response within the retina. Activated microglial cells and macrophages infiltrate the macular region, releasing cytokines and inflammatory enzymes that progressively damage photoreceptors and retinal pigment epithelial cells.

These activated glial cells release inflammatory cytokines and reactive oxygen species that damage retinal ganglion cells. Even in patients with normal intraocular pressure, inflammatory mechanisms may continue to drive optic nerve degeneration, explaining disease progression despite adequate pressure control.