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Aging, inflammation, degeneration, and cell death in age-related macular degeneration

Age-related macular degeneration (AMD), the leading cause of irreversible central blindness in the elderly, is characterized most significantly by atrophy of photoreceptors and retinal pigment epithelium, which is sometimes accompanied by choroidal neovascularization. Development of AMD is contingent on both environmental and genetic risk factors, the strongest being advanced age. With normal aging, photoreceptors are steadily lost, Bruch's membrane thickens, the choroid thins, and hard drusen may form in the periphery. The changes are characterized as kinetics between homeostasis and allostasis. In AMD, many of these changes are exacerbated (chronic allostatic overload leading to pathology and disease); additionally, there is development of disease-specific factors such as macular soft drusen, geographic atrophy, and choroidal neovascularization. Para-inflammation, which can be thought of as an intermediate between basal and robust levels of inflammation, develops within the retina in an attempt to maintain ocular homeostasis and physiological allostasis, reflected by increased expression of the anti-inflammatory cytokine IL-10 coupled with shifts in macrophage plasticity from the pro-inflammatory M1 to the anti-inflammatory M2 polarization. In AMD, imbalances in the M1 and M2 macrophage populations and activation of retinal microglia are observed and potentially contribute to tissue degeneration. In the chronic stage of allostatic overload, including oxidative stress, inflammasomes, and inflammatory cytokines (e.g., IL-1β, IL-17 and IL-18) wax and wane; retinal pigment epithelium and photoreceptors degenerate leading to programed cell death via apoptosis, autophagy, pyrotosis and/or necroptosis. Neovascularization may also develop. Therefore, the underlying mechanism of AMD involves homeostasis, allostasis, and allostatic overload, which all depend primarily on aging, inflammation, degeneration, atrophy, and neovascularization.

Biography

Chi-Chao Chan, M.D. is the chief of Immunopathology Section, Laboratory of Immunology and the Head of Histology Core at the National Eye Institute (NEI), National Institutes of Health (NIH). In 1967, Dr. Chan graduated from Chungzhan Medical College (now known as Sun Yat-sen University) in Guangzhou, China. She earned her A.B. in 1972 and M.D. in 1975 at Johns Hopkins University. Following medical school, she completed her medical internship at Maimonides Medical Center in New York and ophthalmology residency at Stanford University Medical Center. Continuing her educational enrichment, Dr. Chan completed two post-doctoral fellowships in ophthalmic pathology at the Wilmer Ophthalmological Institute in 1982, and clinical ocular immunology/uveitis at the NEI in 1985. She is a board certified ophthalmologist and obtained a license of Clinical Laboratory Improvement Act (CLIA), certifying her in clinical laboratory diagnostics in ophthalmic pathology and molecular pathology. Dr. Chan is a world expert in intraocular lymphoma, immunopathology of uveitis, molecular pathology of age-related macular degeneration (AMD), ocular lymphoma and von Hippel-Lindau disease.

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