AB005. Perspectives on complement injury and choriocapillaris endothelial cell loss in aging and age-related macular degeneration

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Abstract: Genetic studies have revealed that variants in genes that encode regulators of the complement system are major risk factors for the development of age-related macular degeneration (AMD). The biochemical consequences of the common polymorphism in complement factor H (Tyr402His) include increased formation of the membrane attack complex (MAC), which is deposited at the level of the inner choroid and choriocapillaris. Whereas the MAC is normally protective against foreign pathogens, it can also damage resident bystander cells when it is insufficiently regulated. Indeed, human maculas with early AMD show loss of endothelial cells in the choriocapillaris, the principal site of MAC activation. Modeling of MAC injury of choroidal endothelial cells *in vitro* reveals that these cells are susceptible to cell lysis by the MAC, and that unlysed cells alter their gene expression profile to undergo a pro-angiogenic phenotype that includes increased expression of matrix metalloproteinase-9. Strategies for protecting choriocapillaris endothelial cells from MAC-mediated lysis and for replacing lysed endothelial cells will be discussed.

Keywords: Choroid; membrane attack complex (MAC); age-related macular degeneration (AMD)

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