SPECTROSCOPY AND THE ETIOLOGY OF CATARACT AND DRY EYE

DOUGLAS BORCHMAN, *Ophthalmology and Visual Sciences, University of Louisville, Louisville, KY, USA*.

NMR and Infrared spectroscopies were instrumental in determining the relationships between lens and tear lipid composition, conformation and function. The major lipid of the human lens is dihydrosphingomylein, discovered by NMR spectroscopy and found in quantity only in the lens. The lens contains a cholesterol to phospholipid molar ratio as high as 10:1. Lens lipids contribute to maintaining lens clarity, and alterations in lens lipid composition due to age are likely to contribute to cataract. Lens lipid composition reflects adaptations to the unique characteristics of the lens: no turnover of lens lipids or proteins and contains almost no intracellular organelles. Long-lived species such as humans and the bowhead whale exhibits lens lipid adaptations that confer resistance to oxidation, and thereby allowing the lens to stay clear for a relatively longer time than is the case in many other species. With cataract, light scattering increases due to the increase in the lipid order of lens membranes measured using infrared spectroscopy. It is plausible that the increase in lipid-lipid interactions may contribute to myopia by causing greater compaction and overall stiffness of the lens. The TFLL is a thin, 100 nm layer of lipid on the surface of tears covering the cornea that contributes to tear film stability. NMR spectroscopy found that the major lipids of the TFLL are wax esters and cholesterol esters. The hydrocarbon chains associated with the esters are longer than those found anywhere in the body, as long as 32 carbons, and many are branched. More ordered lipid with dry eye, measured using FTIR, could inhibit the flow of meibum from the meibomian glands and contribute to the formation of a discontinuous patchy TFLL, which in turn results in deteriorated spreading, and decreased surface elasticity. One may also speculate that more ordered lipid results in the attenuated capability to restore tear film lipid layer structure between blinks.