

HEAT-INDUCED CATARACTS IN THE RAT LENS IN VITRO



Microwaves have been shown to produce cataracts in the eyes of whole animals. Since the main effect of microwaves is believed to be heating, it seemed possible that a simple temperature elevation was causing the lens degeneration as indicated by the experiments of Carpenter and Kramar. We, therefore, thought it important to distinguish between the effect of the electromagnetic radiation and the effect of elevated temperature. We studied the effect of temperature on isolated rat lenses incubated in tissue culture medium. Lenses maintain their clarity in medium 199 (M 199) (containing 10% foetal calf serum) for up to two weeks. When ten times the normal serum glucose levels are included in the incubation, opacities and associated globular degeneration of the lens cells developed within one day. Similar loss of transparency occurred after lenses were warmed to 39° or 41° for one hour and then incubated at 37° for 24 hr. Lenses exposed to higher temperatures (60° and 65°) for 1 hour did not become opaque. They had normal transparency probably because they had been "fixed" by a process similar to histological fixation. Scanning electron microscopy revealed subcapsular cortical globular degeneration in the cataractous areas. The degeneration was deepest in the equatorial region. Glucose-induced and temperature-induced cataracts contained spherical bodies up to 20µ in diameter. Occasionally in temperature-induced cataracts, bodies up to ten times this diameter were found. Lenses were incubated with ³²P_i in Medium 199 to label membrane phospholipids, and while cataract formation was occurring incorporation proceeded into the major phospholipids, including phosphatidylcholine (PC), phosphatidylethanolamine (PE) and sphingomyelin (SM). In cataractous lenses the specific activity of phosphatidylinositol (PI) was higher. Taken together, these observations suggest that membrane changes may be involved in temperature-induced cataract formation.

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