Abstract Title

RETINAL DETACHMENT IN MICE DEFICIENT IN GFAP AND VIMENTIN: REDUCED GLIAL HYPERTROPHY AND INCREASED GANGLION CELL REMODELING

M.R. Verardo 1, G.P. Lewis 1, M. Takeda 3, K.A. Linberg 1, B. Wardak 1, M. Rabena 1, M. Pekny 4, D.F. Chen 3, S.K. Fisher 1,2.
1 Neuroscience Research Institute and 2 Dept. MCD Biology, University of California, Santa Barbara, 3 Schepens Eye Research Institute, Dept. of Ophthalmology, Harvard Medical School, Boston, MA, 4 The Arvid Carlsson Institute for Neuroscience, Institute of Clinical Neuroscience, Sahlgrenska Academy, Göteborg University

Retinal detachment induces Müller glia cell (MC) hypertrophy. Structural and immunolabeling data suggest a prominent role for the intermediate filament cytoskeleton in this process. There also may be a relationship between MC hypertrophy and neuronal remodeling after detachment. Here we studied both of these events after detachment in wild-type C57Bl/6J mice and mice deficient in the intermediate filament proteins GFAP and vimentin (GFAP-/-vim-/-). The retinas were harvested at 7 or 28 days post-detachment. Immunohistochemistry was performed using antibodies labeling both glial and neuronal cell types. Images were collected by confocal microscopy. MC hypertrophy following detachment was greatly inhibited in the GFAP-/-vim-/- mice. Anti-S100 labeling revealed abnormal MC morphology in the attached GFAP-/-vim-/- retina, which was exaggerated after detachment. Extensive neuronal remodeling occurred in both GFAP-/-vim-/- and wild-type animals. However, ganglion cell reactivity, identified by anti-neurofilament labeling, was greatly exaggerated in areas showing damaged MC endfeet. It has already been demonstrated that MC endfeet are more fragile in this strain of mice (Lundkvist, et al. JCS 117, 3481-3488). These results demonstrate the importance of GFAP and vimentin to the hypertrophy of MC after retinal injury and suggests that MC play an important role in regulating ganglion cell remodeling.
Supported by NIH grants EY00888, EY012983, NSF grant 0331697, and the Dept. of Defense.