7.342 Fall 2004 - Project 1 Writing Assignment Proposed abstract for: MacPherson, D., J. Sage, T. Kim, D. Ho, M. E. McLaughlin, and T. Jacks. "Cell Type-specific Effects of Rb Deletion in the Murine Retina." *Genes Dev.* 18, no. 14 (Jul 15, 2004): 1681-94. Epub Jul 01, 2004. PMID: 15231717 [PubMed - indexed for MEDLINE]. Courtesy of Emily Havens. Used with permission.



The *RB* gene is a known tumor suppressor. In humans, inheritance of a mutant allele of *RB* leads to retinoblastoma at about a 90% frequency. Thus, it is important to understand the role that the *RB* gene plays in tumor development; and understanding its role in normal retinal development will aid in understanding this pathology. We have successfully created a model mouse system by utilizing *Rb/NesCre1* with paternal inheritance to completely delete *Rb* in the retina. We found that *Rb* deletion in the retina leads to failure in cell-cycle exit and p53-independent apoptosis. This *Rb/p53* double mutant does not result in retinoblastoma development. However, combining the *Rb* mutation with mutations in *p107* or *p130* causes retinal dysplasia or retinoblastomas with amacrine cell characteristics. Thus, we have successfully created a mouse model that develops retinoblastomas with high histological similarities to their human counterparts which may be useful for future retinoblastoma studies.