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 2004 Jul 01. PMID: 15231717 [PubMed - indexed for MEDLINE]. Courtesy of Sharon Lawrence. Used with permission.

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7.342 - Cancer Biology
Project 1
Due Date: October 19, 2004

The tumor-suppressing *Rb* gene regulates cell processes, such as S-phase entry, apoptosis and terminal differentiation. When found in humans, a mutation in this gene greatly increases the chance of developing retinoblastoma. Therefore, it is our belief that determining the role of *Rb* in retinal development will lead to a better understanding of the cause of retinoblastoma.

We studied *NesCre1* transgenic mice with *Rb* mutation alone, and also with compound mutations involving *Rb* and *p53*, *p107*, or *p130*, in order to determine the effects of these mutations on retinal development. We found that *Rb* deficiency disrupts exit from the cell-cycle, which leads to *p53*-independent apoptosis in many retinal cells, such as photoreceptors, bipolar cells, and retinal ganglion cells, and abnormalities and nuclear disorganization in amacrine cells and other types of cells. Mutations in both the *Rb* and *p107* genes together led to massive retinal dysplasia and high levels of apoptosis. Mutants lacking both *Rb* and *p130*, however, did not exhibit high levels of dysplasia, and apoptosis levels were similar to levels observed in *Rb* mutants. These results helped to further our understanding of the roles of the *Rb* gene and *Rb* family members in the development of both specific retinal cells and, consequently, retinoblastoma.