

# 2008 Ohio Student Research Forum

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## RESEARCH ABSTRACT FORM

**TITLE:** Deletion of E2F1-3 causes disruption of lens development and upregulation of E2F3 targets

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The E2F family of transcription activators and repressors is critical for the regulation of many genes required for cell proliferation. Tissue complexity and genetic redundancy among mammalian E2Fs has made their study *in vivo* challenging. Here we ablated the entire subclass of activators, *E2f1*, *E2f2* and *E2f3*, in progenitor cells of the lens and show that, in contrast to current dogma, triply-deficient cells proliferate and give rise to differentiated cell types. Surprisingly, loss of *E2f1-3* resulted in elevated expression of E2F targets, DNA damage and massive apoptosis. We suggest that the subclass of E2Fs previously classified as activators can act as transcriptional repressors *in vivo* to regulate cell survival. These findings redefine the current paradigm of how E2F regulates cell proliferation *in vivo*.