HEALTH RESEARCH ABSTRACT SUBMISSIONS #14

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College *	College of Liberal Arts and Sciences
Department *	Genetics
Title of Research *	Drosophila Myb interacts with NURF to repress cell cycle genes in non-mitotic tissues
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Introduction & Purpose *

c-Myb is encoded by a proto-oncogene associated with leukemias and lymphomas in birds and mammals. Vertebrates have three representatives of the Myb gene family consisting of A-, B- and c-Myb, all of which encode DNA-binding factors that are important for the proper expression of large numbers of genes including those that regulate cell cycle progression and cell differentiation. Drosophila melanogaster contains a single Myb gene (Dm-Myb), mutants of which die before reaching adulthood. Dm-Myb mutant flies display cell cycle defects such as aneuploidy and abnormal centrosome number, both hallmarks of cancer. Additionally, the Dm-Myb protein was identified in a complex containing a large number of proteins including the nucleosome remodeling factor NURF. However, no study has attempted an empirical analysis of transcriptional changes occurring in Myb mutant animals. Moreover, the mechanism by which Dm-Myb regulates gene expression is poorly understood.

Experimental Design *	We performed yeast-two hybrid and genetic screens to identify putative interactors of Dm-Myb. Also, we conducted gene expression arrays experiments in Myb mutant and the newly identified interactor mutant animals to assess gene expression changes in hopes of elucidating a co-regulation mechanism. Finally we performed tiling microarray studies to empirically assess genome-wide transcription in our mutant animals.
Results *	We have now established that Dm-Myb physically and genetically interacts with the major subunit of NURF (Nurf301). Also, there is a strong overlap of the genes regulated by these two proteins and, in contrast to the prevalent view in the field, we have observed a prominent transcriptional repression function for Myb and Nurf301, specifically in non-mitotic tissues. Moreover, there is an upregulation in the transcription of transposable elements in Dm-Myb and Nurf301mutant animals.
Conclusions *	These data suggest that, in addition to activation of cell cycle genes in dividing cells, Myb and NURF work to repress such genes in non-dividing cells. Even more surprising, our tiling microarray data indicate that Dm-Myb and Nurf301 are working in concert to silence transposable elements.

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