Loss of Fascin Enhances Nuclear Actin Filament Formation

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Nuclear actin, while still poorly understood, plays a role in DNA replication, chromatin organization, and gene expression. This research uses Drosophila oogenesis as a model to study in vivo nuclear actin filament biology. Drosophila oogenesis is divided into 14 morphologically defined stages and the follicle consists of somatic cells, nurse cells, and the oocyte. Nuclear actin is observed in the nurse cells between Stages 5-9. My previous research established that nuclear actin filament formation is enhanced by the cellular stress of aging. This research focuses on the impact of loss of Fascin on nuclear actin filaments. Fascin is an actin bundling protein that plays a large role in development and disease. It has recently been discovered in the nucleus; thus, this study attempts to understand how it may regulate nuclear actin. The control sample consisted of Drosophila with wild-type expression of Fascin and the experimental groups are heterozygous and the homozygous loss of Fascin. Follicles were scored for the percentage of nurse cells containing nuclear actin filaments and filament length. The results reveal that loss of Fascin increases the prevalence and length of nuclear actin filaments, with the homozygous experimental group having the greatest impact. The developed analysis parameters provide an unbiased screening tool to quantify and further understand the function and regulation of nuclear actin filaments. As nuclear actin filaments are likely to be similarly formed and regulated in many in vivo contexts, these studies will help provide a better understanding of the role of nuclear actin filaments in cancer and neurodegenerative diseases like Alzheimer's.

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