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REGULATION AND EFFECTS OF FERRITIN ON OVARIAN CELL MIGRATION IN *DROSOPHILA MELANOGASTER*

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Drosophila melanogaster, commonly known as the fruit fly, is an excellent model organism to study development because of its rapid life cycle and it is easy to manipulate genetically. Besides the availability of experimental tools, homology between Drosophila and mammalian genes makes Drosophila an excellent system to characterize developmental processes. Collective cell migration is important in the study of disease progression, wound healing, and animal development. In *Drosophila melanogaste*r there are clusters of cells in the female ovaries, known as border cell clusters, that migrate collectively at a certain time in development. Border cell clusters are useful because we can use genetic and imaging tools to investigate cell migration at a molecular level

Our lab and others have shown that the Janus Kinase/Signal Transducer and Activator of Transcription (Jak-STAT) and steroid hormone signaling pathways are important for the regulation of border cell migration. Flies have a single steroid hormone called ecdysone which binds to its receptor - ecdysone receptor (EcR) that is important for the timing and regulation of border cell migration. We hypothesize that the ferritin complex plays an important role in this pathway. Ferritin is an iron storage molecular complex made of heavy and light chains that are encoded by 3 different genes in *Drosophila*: *fer 1hch*, *fer 2lch*, and *fer 3hch*. Our preliminary results indicate that reducing the expression of ferritin genes affects border cell migration. In this project, we are working to characterize the normal spatiotemporal expression of the ferritin genes and predict how the ferritin gene cluster in border cells is genetically regulated. To do this, we are mining genomic data for transcriptional factor binding site information and regulatory sequence information. Our findings suggest that ferritin is likely regulated by the EcR and STAT signaling pathways during border cell migration

Moving forward, we will take advantage of fly genetics to overexpress or knock down ferritin subunits specifically in the border cells and assay the effects on cell migration. I would also like to characterize the roles of each ferritin subunit and how they affect cell migration. We will also search genome-wide for transcriptional targets of the key transcriptional factors that

regulate border cell migration. This work may suggest an important role for ferritin in other cell types that migrate even in other species.

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