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## Role of ecdysone signaling in fat body remodeling

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### **ROLE OF ECDYSONE SIGNALING IN FAT BODY REMODELING**

Climate change is fundamentally connected to animal development and survival, and the life history of an organism must be coordinated with predictable seasonal changes of the environment. Climate change affects the life cycle of plants, a major food source for insects. If photoperiod, the primary environmental cue that insects utilize to determine the proper emergence time, and food availability becomes out of sync, many populations of insects and other animals could be threatened. Understanding animal development can provide insight into this issue and could provide clues that may help the scientific community predict how insect populations may respond to climate change.

During *Drosophila* metamorphosis, most of the larval tissues are destroyed, but the fat body is an exception. The larval fat body escapes destruction and is instead remodeled from flat, polygonal and attached sheets of cells to round, spherical and detached free-floating cells (Nelliot, et. al, 2006). It has been hypothesized that Ecdysone signaling is necessary for fat cell detachment. To test the hypothesis that Ecdysone signaling is necessary for fat cell detachment, I am using genetic techniques to create mosaic animals. These techniques will allow me to generate clones of cells that are deficient or hyperactive in certain Ecdysone signaling targets.

Currently, I am trying to establish animals for the first part of a two-step cross. Next, using the FLP/FRT and the Mosaic Analysis with a Repressible Cell Marker system (MARCM), I will generate mitotic clones of cells. These clones will be comprised of small populations of cells mutant for Ecdysone signaling factors and will be surrounded by normal (wild type) cells. I predict that the populations which are deficient in Ecdysone signaling factors will not undergo fat body remodeling while the surrounding pools of wild type cells will complete the remodeling program. These two types of cells can be distinguished from each other because I will also label the Ecdysone signaling-defective cells with green fluorescent protein. The data will be procured on the Confocal Microscope in the Center for Biological Imaging.

# Role of Ecdysone Signaling in Fat Body Remodeling in *Drosophila melanogaster*

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## ABSTRACT

In Dipterans, metamorphosis is depicted by the loss and transformation of larval tissues as the animal gets ready for adult life. Ecdysone signaling mediates several distinct biological responses of the metamorphosing animal including the programmed cell death of most larval tissues. During *Drosophila* metamorphosis, most of the larval tissues are destroyed, but the fat body is an exception. The larval fat body escapes destruction and is instead remodeled from flat, polygonal and attached sheets of cells to round, spherical and detached free-floating cells (Nelliot, et. al, 2006). It has been demonstrated that Ecdysone signaling does play a role in fat body remodeling (Cherbas et al., 2003). Here I present data that demonstrates a cell autonomous role for Ecdysone signaling mediated fat body remodeling.

## INTRODUCTION

Ecdysone signaling triggers many developmental events in *Drosophila melanogaster* such as larval molts, puparium formation, pupal formation, and metamorphosis. The life history of *Drosophila* is characterized in part by three larval stages. After the third larval stage the animal prepares for metamorphosis. During metamorphosis, most of the larval tissues are lost through programmed cell death and new adult tissues are differentiated. All of these changes are initiated by the insect hormone 20-hydroxyecdysone (herein referred to as Ecdysone).

Our research focuses on the larval fat body of *Drosophila melanogaster*. During *Drosophila* metamorphosis, most of the larval tissues are destroyed but the fat body is an exception. The larval fat body escapes destruction and is instead remodeled from flat, polygonal and attached sheets of cells to round, spherical and detached free-floating cells (Nelliot, et. al, 2006). It has been demonstrated that Ecdysone signaling does play a role in fat body remodeling (Cherbas et al., 2003).

During pupariation, Ecdysone levels in *Drosophila* increase in preparation for metamorphosis. Different forms of EcR are then associated with different programs of differentiation during the subsequent adult development (Riddiford, 1993). Aside from triggering developmental events, Ecdysone also initiates transcription of certain genes. One such gene is Ftz-F1, which is a competence factor dependent on the decrease of the Ecdysone titer.

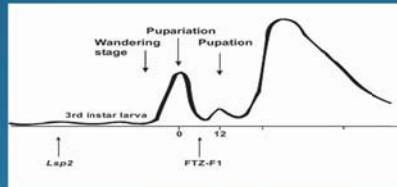


Figure 1. Ecdysone titer and its correlation to LSP2, FTZ-F1, and development. Adapted from Riddiford, L.M. (1993).

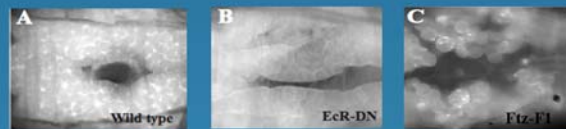


Figure 2. Fat-body cell remodeling in *Drosophila*. (A) Wild-type fat body dissociation: round, spherical, and detached cells. (B) EcR-DN fat body mutant: flat, polygonal, and attached. (C) FTZ-F1 fat body mutant: premature fat body dissociation: round, spherical, and detached.

## METHODS

Stock animals were established for a two-step cross. Using the FLP/FRT and the Mosaic Analysis with a Repressible Cell Marker system (MARCM), mitotic clones of cells were generated. These clones are comprised of small populations of cells mutant for Ecdysone signaling factors and are surrounded by normal (wild type) cells.

### FLP/FRT Recombination

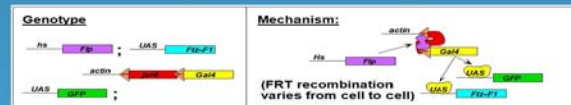


Figure 3. FLP/FRT site specific recombination to vary the levels of Ftz-F1 within the fat body cells. Adapted from Dr. Andres.

### Mosaic Analysis with a Repressible Cell Marker System (MARCM)

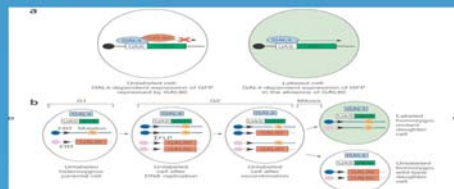


Figure 4. Protocol for MARCM to investigate gene function in fat body cells. Adapted from Wu, J. & Luo, L. (2006)

## RESULTS

We have determined that Ecdysone is necessary for fat body dissociation and remodeling. *Ftz-F1* is a gene which encodes for a transcription factor and allows genes to respond to Ecdysone. When *Ftz-F1* was blocked, the larval fat body populations which are deficient in Ecdysone signaling factors did not undergo fat body remodeling while the surrounding pools of wild type cells completed the remodeling program. These two types of cells were distinguished from each other by labeling the Ecdysone signaling-defective cells with green fluorescent protein. The data was then procured on the Confocal Microscope in the Center for Biological Imaging.

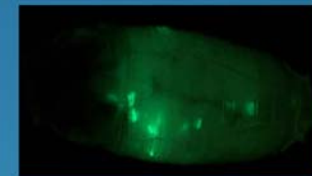


Figure 5. Fat cell clones mutant for Ftz-F1.

## CONCLUSION

Ecdysone signaling is necessary for fat cell detachment during fat body remodeling. Ecdysone signaling is cell autonomous and triggers expression of proteins that cause developmental changes in the individual cell.

## REFERENCES

- Cherbas L, Hu X, Zhimulev I, Belyaeva E, Cherbas P. (2003). EcR isoforms in *Drosophila*: Testing tissue-specific requirements by targeted blockade and rescue. *Development* 130:271-284.
- Nelliot, A., Bond, N., Hoshizaki, D. (2006). Fat-Body Remodeling in *Drosophila melanogaster*. *Genesis*, 44, 396-400.
- Riddiford L.M. (1993). Hormone receptors and the regulation of insect metamorphosis. *Receptor*; 3:203-209.
- Wu, J. & Luo, L. (2006). A protocol for mosaic analysis with a repressible cell marker (MARCM) in *Drosophila*. *Nature Protocols*, 6, 2582-2589.

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