Aug 9th, 10:15 AM - 12:00 PM

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Repository Citation

King, Gregory and Andres, Andrew, "The Role of an ABC transporter as a steroid antagonist in Drosophila" (2011). *Undergraduate Research Opportunities Program (UROP)*. 22.
https://digitalscholarship.unlv.edu/cs_urop/2011/aug9/22

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The Role of an ABC Transporter as a Steroid Antagonist in Drosophila
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Introduction

Drosophila melanogaster are holometabolous insects that have several distinct life stages including larva and a winged adult. The larval stage is marked by a time of feeding and growth, while the adult stage is optimized for sexual reproduction and dissemination. The larval stage can be divided into three time periods, or instars: 1st (L1), 2nd (L2), and 3rd (L3) (Figure 1). Larval growth — both between instars and beyond — depends on specific signaling pathways controlled by a cholesteroid derived steroid, 20-hydroxyecdysone (20E). Although 20E is a systemic developmental signal, little is known about the molecular details of how different tissues respond to the hormone. We have been studying one gene induced by 20E in target tissues that we believe is the ABC transporter gene (ABC). Gene expression of this gene in the larval gut results in a phenotype similar to that seen in the larva. This gene, E23, encodes a ATP binding cassette (ABC) transporter protein that may function to limit hormone exposure in tissues where it is expressed.

Hypotheses

E23 functions as a 20E antagonist, meaning when it is overproduced in any target cell the cell will not respond to hormone and produce the normal developmental response. Since the late cells respond to 20E by stimulating larval ecdysone, E23 overproduction in these specific cells should result in the larva displaying ecdysone-deficient phenotypes.

Materials and Methods

To control the production of E23 within specific tissues, a powerful genetic system can be used: the UAS-Gal4 system (Figure 5). This system, which has been adopted from yeast, consists of two parts: the Upstream Activator Sequence (UAS), and the GAL4 “driver” protein which binds to UAS. This allows for transcription and production of the UAS controlled E23 only in cells that express the UAS-driven construct. Figure 6 is a diagram of the protocol to assay for defects.

Results and Conclusion

Shown below is a graph comparing the control (UAS-Gal4) and experimental (UAS-E23/UAS-Gal4) larvae (Figure 7). The graph, from data following 15H larvae each, contains mortality by measuring liveness or lethality. On the right is a comparison of the third instar larva (Figure 8). Ecdysone defects are indicated by a second set of mouth hooks, cuticular structures that are created during molting. If the larva undergoes ecdysone, the smooth hooks remain attached rather than falling off with the old cuticle.

Discussion

Along with observing the effects of E23 on the molting and ecdysone process, I will be studying the effect of 20E suppression throughout the larval period. The epidermal cells that secrete the cuticular structures, can be controlled through another recently acquired another stock, A58-Gr-Ca. We have hypothesized that overproduction of E23 in these cells would result in a phenotype similar to the UAS-E23 expression. At the stages of ecdysone, 24 hours to 48 hours (2nd and 3rd instars), the mortality gap increases to only 25% remaining for the experimental versus 87% remaining for the control.

Acknowledgements

Andrea Fly Lab, Elena Paladino, Kathryn Lantz

References


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References