SCAN ME

How flies can help us learn more about cancer

Characterizing expression patterns of LexA and LexAop lines for development of a Drosophila cancer model.

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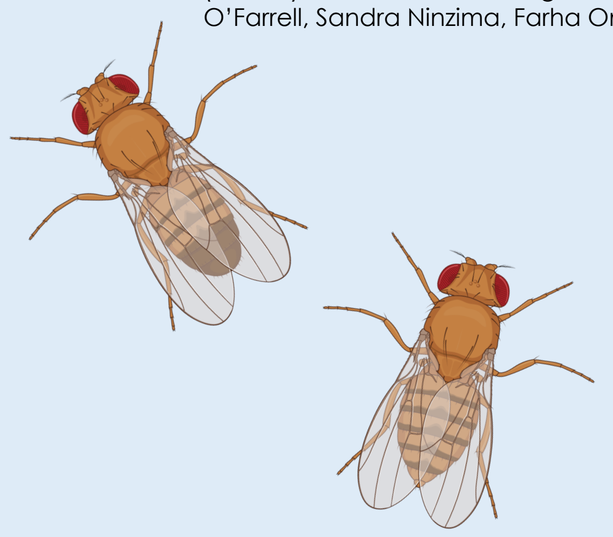
1 The fly and its magical wing disk

The reasons for using *Drosophila* in addition to the fun work are many! It only takes 10 days from a cross until the F_1 generation adults start to come out. The genome of *Drosophila* is closely related to that of humans, 60% of the genes in general and 75% disease related genes are overlapping within the two species. *Drosophila* has a simpler genome, providing better opportunity to control gene expression. The imaginal wing disk, which becomes the wing of the organism, is easily accessible and the genetic expression within this area easily controllable, making it our key to achieve success.



The Fly Lab

Pushing → Flipping → Crossing → Dissecting

Me selecting flies by pushing the flies on the pad with a paint brush

Selecting flies based on phenotype

How we keep the stocks, boxes with tubes containing flies

Maintaining the flies

Phenotypes: Sp (top left), CyO (top right), Tm6c (bottom left) and Dr (bottom right)

Making new flies with known genotype

Shows the dissection in action, the magical wing disk in the red circle

Stealing their magical wing disk

2 Reasons for research

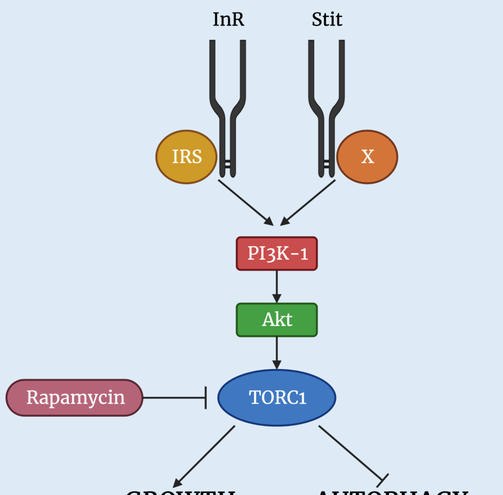


Figure: TORC1 downstream signalling pathway, promoting growth initiated by oncogene Stit [1]

Malignant tumour growth and spread is the cause of mortality in more than 90% of all cancer patients. What triggers this switch to malignancy, and how does the communication between the tumour and the surrounding cells effect this?

We need a system where we can induce a tumour in one cell and control gene expression in the neighbouring cells. The *Drosophila* provides such a system, using RET/Stit which promotes tumour combined with LexA for controlling expression patterns.

3 How to choose flies

I was provided with a selection of different LexA driver lines, and I needed to find the suitable LexA drivers and compare effects of RET/Stit expression to tumour development.

All RNAi (interfering RNA capable of reducing gene expression) lines and the oncogenes RET/Stit rely on binary system GAL4/UAS. As we want to knock down genes surrounding the oncogene cells independent of RET/Stit expression we have to use a second binary system, LexA/Aop.

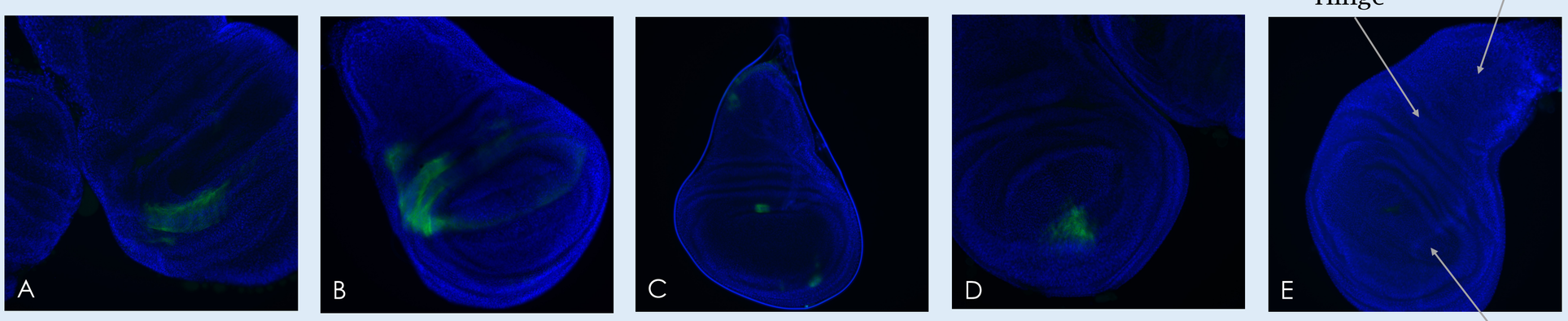
GAL4/UAS → Surrounding cells iRNA

LexA/Aop → Tumour induction RET/stit

4 Creating expressions in the magical wing disk

Using GFP reporter to mark the differences and spreading within cells

We have identified four LexA drivers out of our starting selection which are suitable to be used for comparing effects of RET/Stit expression in tumour development. The positive expression sites within the magical wing disk are indicated in green being GFP positive cells.



The figures A-E show the wing disk of five LexA driver lines, A: aptorous, B: aptourus, C: patched, D: trithorax, E: knot. The blue regions mark cells within the organ, the green area are GFP positive cells and show the expression site of the selection of LexA driver lines. A, B, C, and D show positive signs of expression, E show no expression within the wing disk.

REFERENCES

[1] O'Farrell, F., Wang, S., Katheder, N., Rusten, T. E., and Samakovlis, C. (2013) *Two-Tiered Control of Epithelial Growth and Autophagy by the Insulin Receptor and the Ret-like Receptor, Stitcher*, PLOS Biology. V:17, I:7.

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