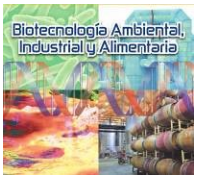
Poster



Using Drosophila to analyse organogenetic gene network evolution

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ABSTRACT

Motivation: Cell migration during development is key to the correct development and morphogenesis of animals. This process normally starts with an Epithelial to Mesenchymal Transition (EMT) allowing the normally cohesive and static epithelial cells to migrate. In Drosophila melanogaster the ring gland is formed by three endocrine organs that are specified in different locations and then migrate together: the corpora cardiaca (CC), the corpora allata (CA) and the prothoracic glands (PG). Previous investigations found that CA, PG and trachea can be homeotically transformed into each other, suggesting they all evolved from a metamerically repeated organ. To investigate the lesser known migration of both CA and PG, a reporter gene named sna-rg-eGFP was made using a specific enhancer that activates the expression of the EMT inducer snail (sna) gene in the CA and the PG.This reporter allows us to track the movement of the CA and the PG during their migration. In this way it was possible to identify deletions affecting the migration process. We will present the defects caused by some of these deletions that were found to have a phenotype of apoptosis. Also, we are studying variants altering the sna-rg enhancer as they can provide information on the up-stream regulators of sna activation and CA/PG specification. These showed that, although STAT sites and a fragment called A1 are required for its expression, in the CA the abscence of STAT sites only delays sna-rg expression. This could mean that after its initial activation, the Sna protein could be self regulating or activating other genes to maintain its expression.

Methods: For the purpose of finding which gene in the deletion causes the apoptosis phenotype we study with fluorescent microscopy different overlapping deletions to narrow down the suspects. After that we check if the mutation of just one gene causes the phenotype and use UAS/SpaltG4 constructs to find if introducing the gene rescues the phenotype. To study the regulation of the sna-rg enhancer and check if sna is maintaining its own expression we are generating a series of progressively shorter versions of the enhancer regulating GFP and performing RNA in situ hybridization to check the expression of the resulting reporters. This way we study directly the expression directed by the enhancer instead of the protein that is more stable and can last longer.

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