Program/Abstract # 190

Sprinter/Wntless is an escort factor for Wg deployment

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In many developing tissues a gradient of Wingless signaling is established whereby there is a concentration-dependent signaling output in the receiving cells to define their fates. There are a number of proposals as to how Wg achieves this differential target activation, but none has been decisively proven. Recently, we and others have identified Sprinter (Srt)/Wntless (Wls) as a factor required for the maturation and deployment of Wg from the signal producing cells. We propose that Srt/Wls acts as a molecular escort to facilitate Wg processing and movement through thesecretory pathway from the endoplasmic reticulum (ER) to the plasma membrane. In developing wing discs that lack *srt/wls*, Wg accumulates in the ER. Movement of Wg from the ER to the cell surface requires physical interaction between these factors. Co-expression of Wg and Srt/Wls results in the redistribution of Wg in cultured cells. Expression of Srt in cell culture and *in vivo* alters the cellular F-actin structure, with the formation of surface protrusions upon with Wg is bound when both factors are co-expressed. These data further elucidate the function of Srt/Wls as a chaperone that can remodel the actin cytoskeleton for Wg deployment and may provide key insights for dissecting the complex processes that lead to Wg morphogen function.

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Program/Abstract # 191

Examining the role of the *C. elegans* uterine-vulval 1 (uv1) cells in egg-laying function

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Wild type *Caenorhabditis elegans* have four cells from the ventral uterine lineage that express the EGF Receptor LET-23 and are specified to adopt the uterine-vulval 1 (uv1) fate by a LIN-3 EGF signal from the underlying vulval cell vulF. Although the role of uv1 cells in *C. elegans* is unknown, we hypothesize a positive or negative role in the function or development of the egg-laying apparatus. Known mutants lacking uv1 cells exhibit other defects in the development of the egg laying apparatus, such as vulval defects, and cause difficulty in determining the role of uv1 cells in egg laying. We have generated a strain lacking uv1 cells with no other obvious defects in the vulva, uterus, or associated muscles and neurons by expressing *let-23* RNA hairpin in the ventral uterine pi lineage using the *egl-13* promoter, causing reduced uv1 fate specification. The resulting strain shows 42.5% penetrance of the uv1 cell defect, defined as three or fewer uv1 cells per animal. 31.9% of animals have zero uv1 cells. To determine the role of uv1 cells in egg-laying, we scored L1-synchronized animals for uv1 cell defects at the L4 stage, and assayed for egg-laying 24 h later by counting the number of eggs retained in the uterus. We did not find a significant difference between the number of eggs retained in uv1-cell defective worms compared to control worms from the same strain with 4 uv1 cells. We conclude that uv1 cell elimination has no effect on the rate of egg-laying, but may still affect other aspects of egg-laying, such as the stage at which eggs are laid or egg-laying regulation in response to stimuli.

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