

Mechanisms of Growth Control

Yan Yan

Division of Life Science, The Hong Kong University of Science and Technology, Hong Kong SAR, P. R.

China

Email: yany@ust.hk

It has long been proposed that cell competition functions to remove precancerous clones. A classical model is the removal of polarity-deficient clones such as the scribble (scrib) mutant clones in *Drosophila* imaginal discs. The activation of Ras, Yki or Notch signaling robustly reverses the scrib mutant clonal fate from elimination to tumorous growth. Using single-cell transcriptomics techniques to profile wing imaginal discs harboring the scrib mutant clones in combination with different signals, we found that a critical converging point downstream of Ras, Yki and Notch signals is the upregulation of Upd2, which is necessary to promote tumorous growth. Unexpectedly, while Upd2 is not required for cell survival per se, Upd2-deficient clones are efficiently wiped out from epithelia, indicating that Upd2 is a previously unrecognized cell competition factor. We further found that Upd2 likely serves as an intercellular messenger to communicate fitness level among cells and Upd2 regulates cell competition through Xrp1.