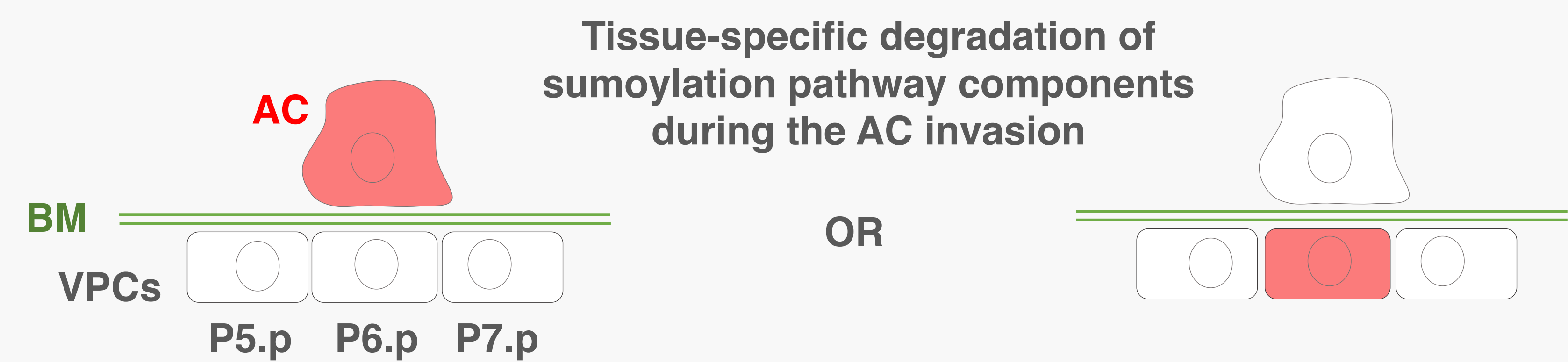


THE ROLE OF PROTEIN SUMOYLATION DURING VULVAL MORPHOGENESIS AND ANCHOR CELL INVASION

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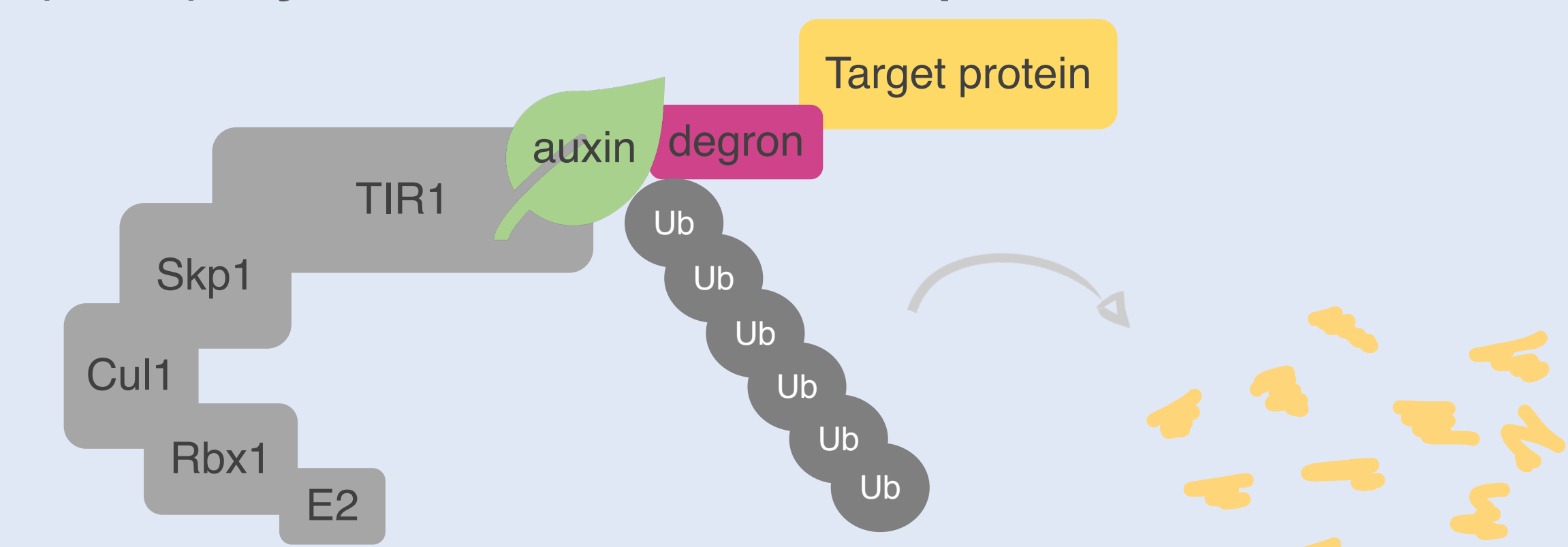
INTRODUCTION

To study how the loss of protein sumoylation affects vulval development and AC invasion, we developed tools to block sumoylation in a tissue-specific and temporally controlled manner. For this purpose, we have been using the auxin-inducible tissue-specific protein degradation system to down-regulate a degron-tagged SUMO E3 ligase (GEI-17) or a degron-tagged SUMO (SMO-1) in different tissues of the worm. We generated transgenic animals expressing a modified *Arabidopsis thaliana* TIR1, an E3 ubiquitin ligase specific for the degron tag, under the VPC and the AC specific promoters.

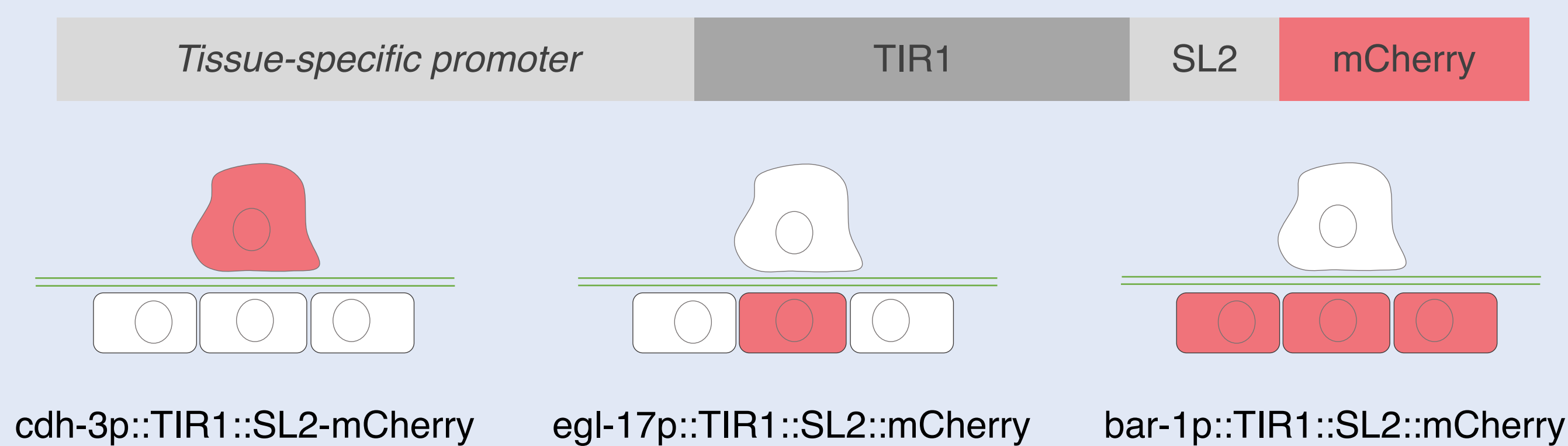


METHODS

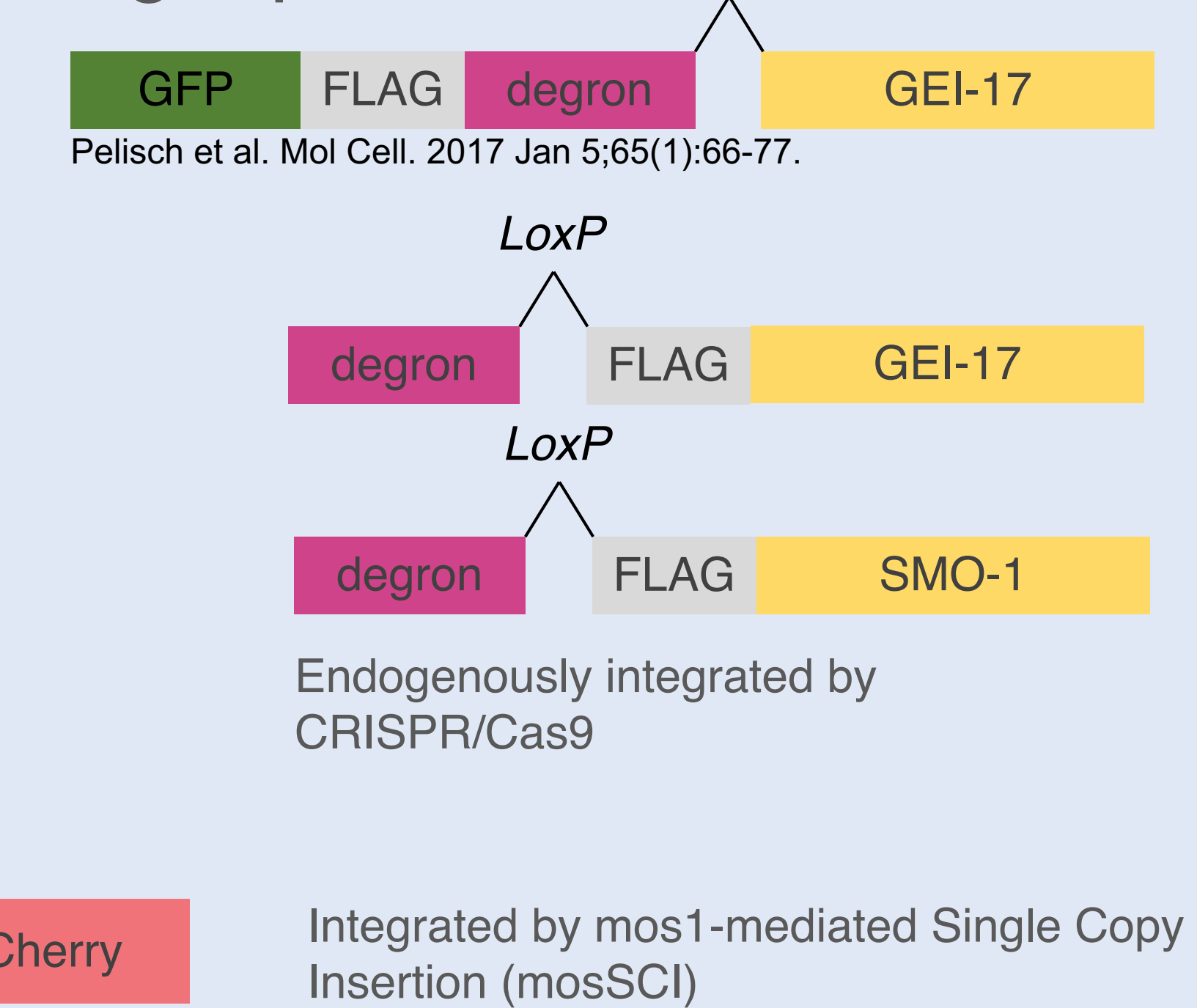
Application of auxin-inducible degradation (AID) system in a tissue-specific manner



Generation of tissue-specific degradation drivers

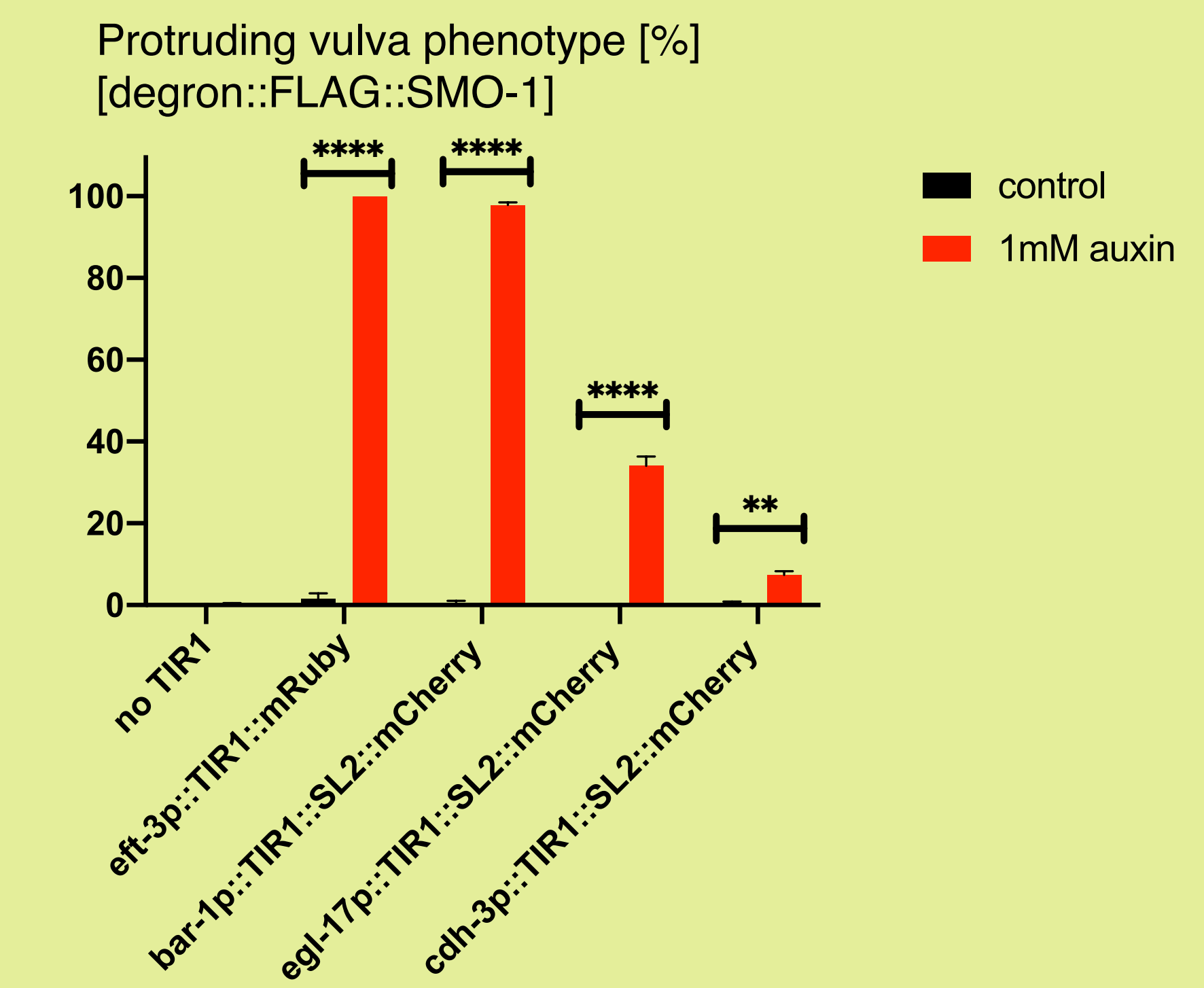


Generation of degron-tagged target proteins



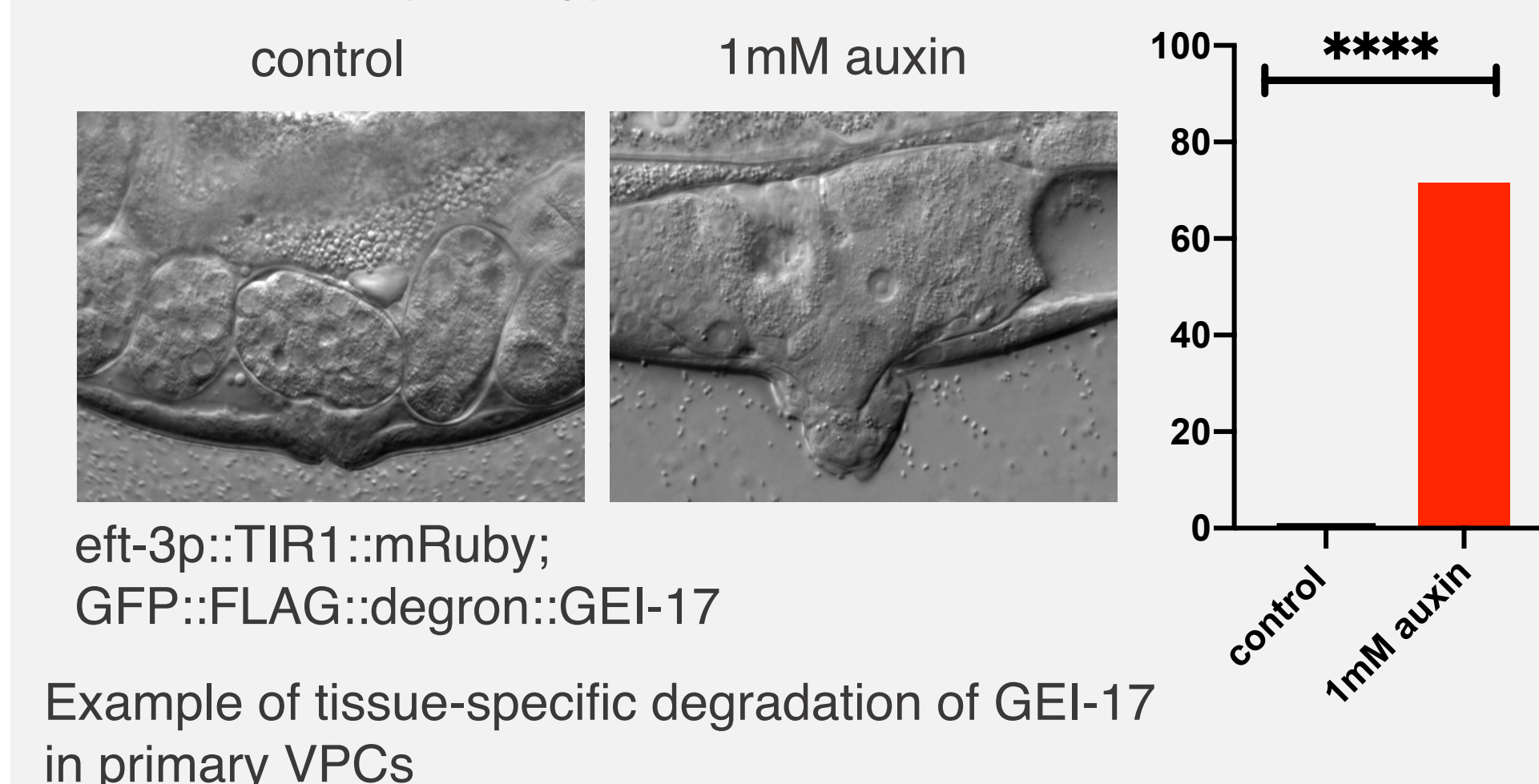
FINDINGS

SUMOYLATION plays crucial role in the VPCs in securing proper vulva formation.

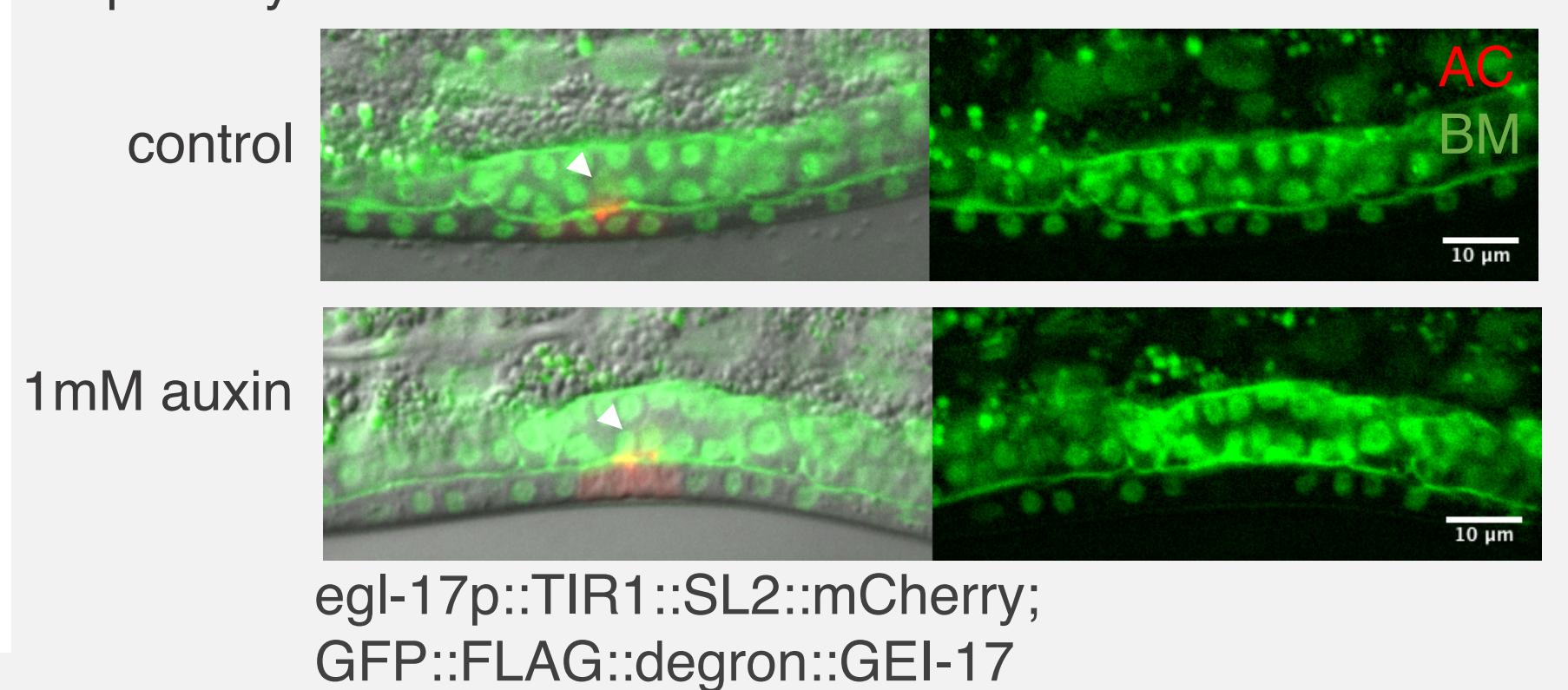


Validation of auxin-inducible degradation system

Degradation of GEI-17 in somatic tissues leads to abnormal vulva phenotype [%]

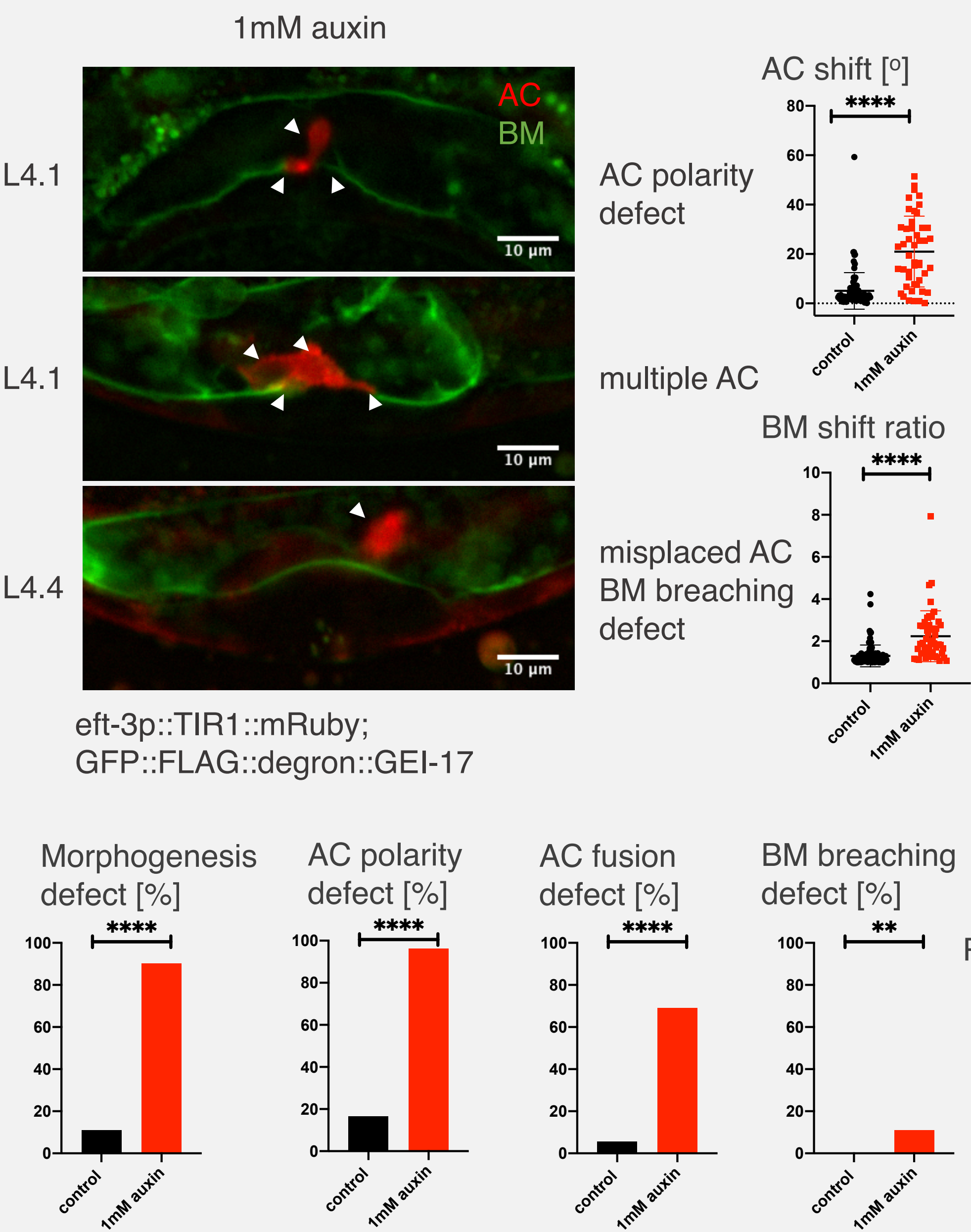


Example of tissue-specific degradation of GEI-17 in primary VPCs

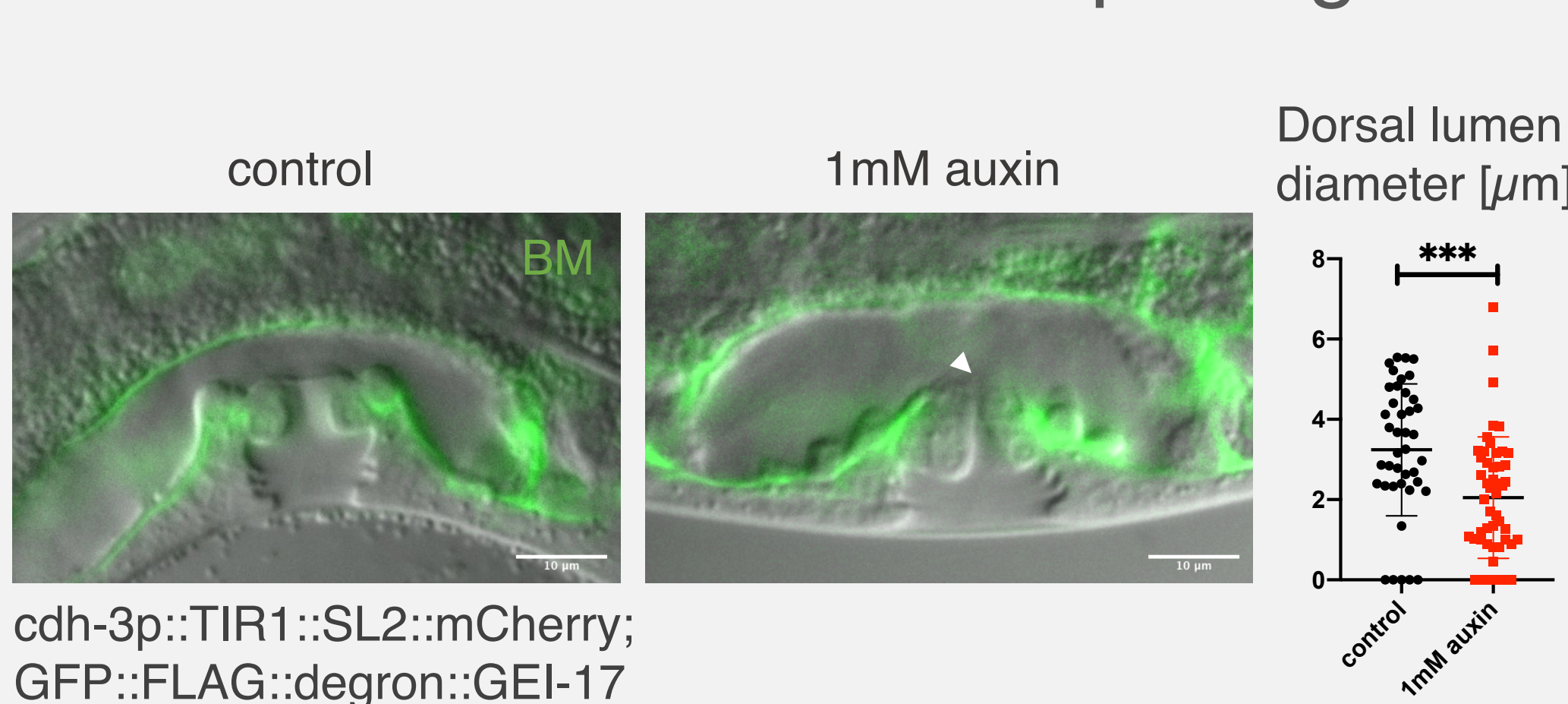


RESULTS

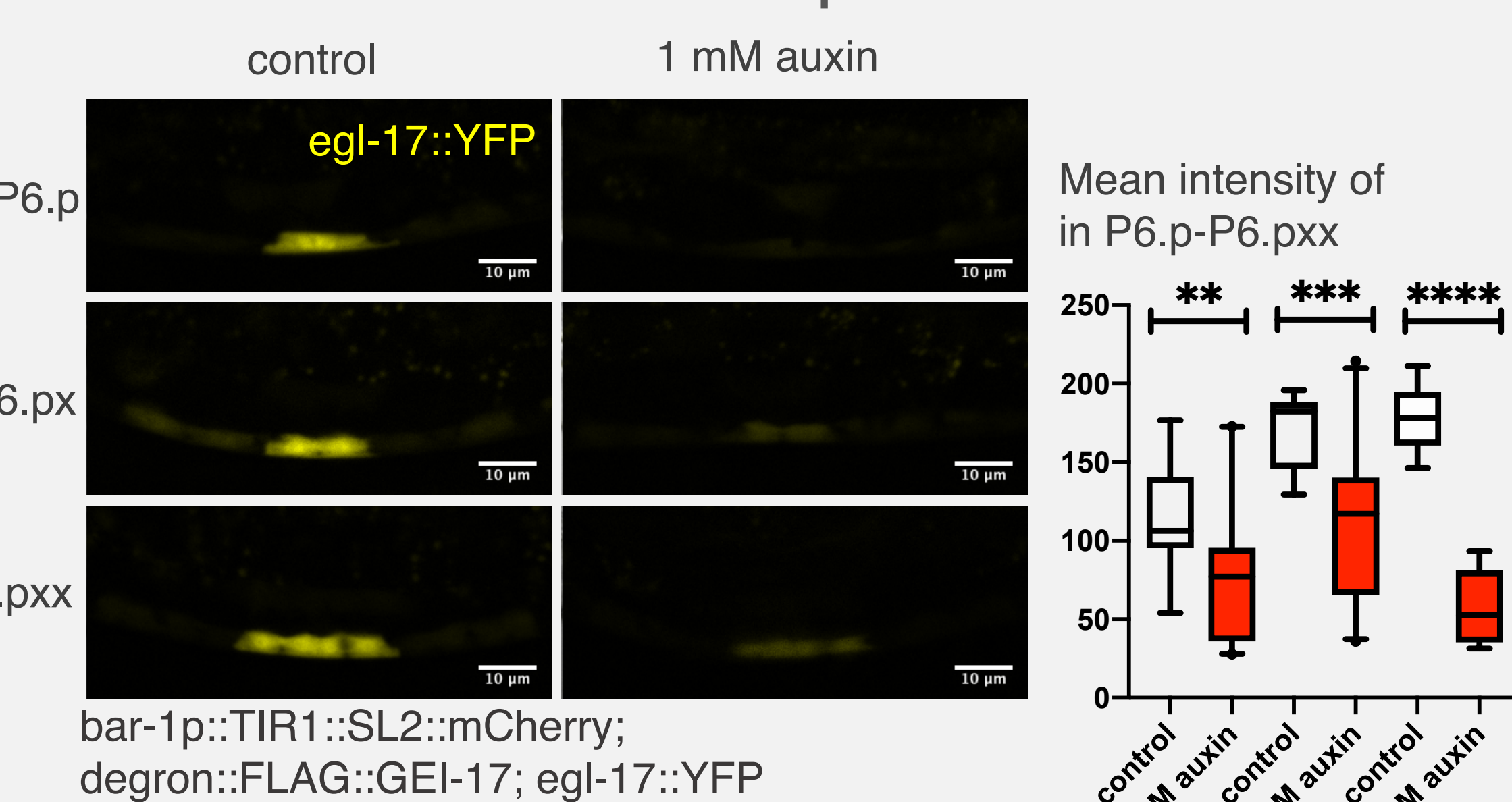
Degradation of GEI-17 in somatic tissues leads to various phenotypes



Degradation of GEI-17 in the AC results in defective dorsal lumen opening



Degradation of GEI-17 in the VPCs leads to defects in cell-fate specification



Degradation of GEI-17 results in toroid formation defects during vulval morphogenesis

