

**P009** Distinct genetic properties of ESCRT-II components in *Drosophila*

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Genetic studies in yeast have identified class E *vps* genes which form the ESCRT complexes required for protein sorting at the early endosome. We are just beginning to understand the phenotypic consequences of loss of *vps* function in metazoan organisms. In *Drosophila*, mutations of the ESCRT-II component *vps25* cause endosomal defects leading to increased non-autonomous cell proliferation and increased non-autonomous apoptotic resistance due to failure to down-regulate Notch and Jak/STAT signaling. Here, we genetically analyze the remaining ESCRT-II components *vps22* and *vps36*. Like *vps25*, mutants in *vps22* and *vps36* display endosomal defects causing increased Notch and Jak/STAT signaling. However, while *vps22* mutants cause strong non-autonomous overgrowth, they do not affect non-autonomous apoptotic resistance. In contrast, *vps36* mutants display the reciprocal phenotype: they increase apoptotic resistance, but have little effect on non-autonomous proliferation. Therefore, *vps25* mutants combine these phenotypes. In summary, despite their intimate physical relationship, the ESCRT-II components *vps22*, *vps25* and *vps36* display distinct genetic properties.