



ABSTRACT

*“Challenging the textbook:
the paradigm for protein sorting to the lytic compartment
does not apply to plants”*

Prof David G. Robinson

**Department Cell Biology
Heidelberg Institute for Plant Sciences
University of Heidelberg
GERMANY**

Receptor-mediated sorting processes in the secretory pathway of eukaryotic cells rely on mechanisms to recycle the receptors after completion of transport. The most frequently described example for this in all major cell biology textbooks is the mannosyl 6-phosphate receptor for trafficking of acid hydrolases to the lysosome. Following this principle, plant vacuolar sorting receptors (VSRs) are believed to recycle, after the dissociation of receptor-ligand complexes in a prevacuolar compartment (PVC). This recycling is supposed to be mediated by retromer, a cytosolic coat-complex consisting of sorting nexins (SNX) and a large heterotrimeric subunit. However, as a result of an extensive analysis using immunoelectron and fluorescence microscopy, it turns out that the AtSNX1, AtSNX2a and VPS29p localize to the *trans* Golgi network (TGN), which is considered to represent the early endosome of plants. In those plants where VSRs are found principally at the PVC, the inhibition of retromer function *in vivo* by expression of AtSNX1 or AtSNX2a mutants as well as transient RNAi knock-down of all sorting nexins leads to an accumulation of the VSR BP80 at the TGN. However, quantitative protein transport studies as well as live cell imaging using fluorescent vacuolar cargo molecules has revealed that the arrival of these VSR ligands at the vacuole is not affected under these conditions. This indicates that the TGN is the point of retromer-mediated recycling of VSRs and that transport towards the lytic vacuole downstream of the TGN is receptor-independent. It seems that in plants retromer recycles VSRs to the ER, and we have clear evidence that VSRs also interact with their ligands in the ER. VSRs and their cargo molecules exit the ER in a process which is dependent on adaptins/clathrin rather than COPII since expression of a μ -adaptin mutant leads to ER retention of VSRs and vacuolar cargo.