

## IN BRIEF

# Crosstown Trafficking: The Retromer Complex Component VPS29 and Recycling of the Vacuolar Sorting Receptor

For plant growth and development to proceed normally, cellular trafficking of proteins must flow more smoothly than the cars on the crosstown expressway (reviewed in De Marcos Lousa et al., 2012). Cargo proteins move from the endoplasmic reticulum, via the Golgi, through the trans-Golgi network, to the plasma membrane or the vacuoles. Cargo can also be moved from the plasma membrane via endocytic transport. Transport proteins must move their cargo accurately and may return from their destinations to facilitate multiple rounds of transport. Although the signals for vacuolar sorting have been known for some time, the dynamics of recycling of transport proteins has emerged as a complex, active area of research. The vacuolar sorting receptors (VSRs) are transport proteins that act in targeting soluble proteins to the vacuole, for example, sorting enzymes to the lytic vacuole or storage proteins to the storage vacuole during seed development. Although many VSRs have been identified, the mechanisms of VSR recycling have remained mysterious.

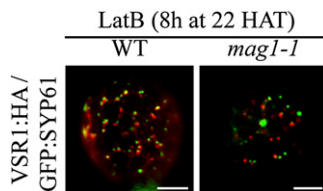
The retromer complex, which acts in targeting to the trans-Golgi network in endocytic transport, may be one component of VSR recycling in plants (reviewed in Bonifacino and Hurley, 2008). In mammalian systems, the retromer complex acts in recycling lysosomal sorting receptors. To probe the involvement of the *Arabidopsis thaliana* retromer complex in VSR recycling, Kang et al. (pages 5058–5073) examine the retromer component vacuolar protein sorting 29 (VPS29) by characterizing the *Arabidopsis* VPS29 mutant *maigo1-1* (*mag1-1*). Using a protoplast system, they find that *mag1-1* mutants do

not effectively traffic soluble proteins, such as *Arabidopsis* aleurain-like protein:green fluorescent protein (GFP) and sporamin:GFP to the lytic vacuole, but can move membrane proteins, such as  $\beta$ Fructosidase 4, via the secretory pathway to the lytic vacuole or plasma membrane. This indicates a specific effect on vacuolar targeting, rather than a general effect on trafficking.

In *mag1-1* mutants, soluble cargo proteins become trapped in the trans-Golgi network, in a traffic jam of sorts. The authors showed that this jam was caused by a failure of transport, in this case because reduced levels of VPS29 inhibit recycling of VSR1 from the prevacuolar compartment to the trans-Golgi network. Treatment with latrunculin B inhibits actin filament assembly and thereby inhibits movement of VSR1 from the trans-Golgi network to the prevacuolar compartment, but permits trafficking in the

reverse direction. In wild-type protoplasts, Latrunculin B treatment caused VSR1 to build up in the trans-Golgi network by inhibiting its movement to the prevacuolar compartment. By contrast, in *mag1-1* protoplasts treated with Latrunculin B, VSR did not accumulate in the trans-Golgi network, showing that its retrograde trafficking was inhibited by the reduction in VPS29 (see figure). Indeed, in *mag1-1* mutants, a substantial amount of VSR1 was transported to the lytic vacuole, rather than being recycled from the prevacuolar compartment to the trans-Golgi network. Therefore, this work provides an important perspective, highlighting the unique nature of protein trafficking to different compartments in plants and the important contribution of the retromer complex in recycling plant transport proteins.

Jennifer Mach  
Science Editor  
jmach@aspb.org



Mutation of VPS29 affects trafficking of VSRs from the postvacuolar compartment to the trans-Golgi network. Overlap of tagged VSR1 (red) with GFP:SYP61 (green), a marker for the trans-Golgi network (TGN), indicates efficiency of trafficking. Latrunculin B (LatB) inhibits actin filament assembly, preventing movement of VSR1 from the TGN to the prevacuolar compartment. In the wild type, prevacuolar compartment to TGN movement causes accumulation of VSR1 in the TGN (left); this does not occur in *mag1-1* mutants (right). (Reprinted from Kang et al. [2012], Figure 5.)

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Jennifer Mach

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