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A central role of multivesicular bodies in plant immunity

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Membrane trafficking is responsible for transport of membrane vesicles and their cargo between intracellular compartments and the plasma membrane. An important compartment is the multivesicular body (MVB), also named the late endosome. It contains intraluminal vesicles, which are generated by the highly complex ESCRT system on its surface. When MVBs fuse with the vacuolar membrane, their cargo including the intraluminal vesicles become degraded. When MVBs fuse with the plasma membrane, the cargo is secreted and the intraluminal vesicles become extracellular vesicles, which we call exosomes.

In recent years, we have found MVBs to be involved in a number of processes related to plant immunity. From studies of plants interacting with the powdery mildew fungus, we have found that pre-invasive immunity involves exosome secretion, and we have uncovered endosome and plasma membrane components involved in this process, including PEN1 and GNOM. This indicated to us that MVBs are involved. In a detailed study of well-known MVB components, we surprisingly found them not to be involved in this pre-invasive immunity mechanism. Rather, we found that these components have a major contribution to post-invasive, encasement-based immunity. This suggests existence of more than one MVB pathway in immunity.

In a separate study, we have revealed that an MVB-associated de-ubiquitinase is required for effector-triggered immunity (ETI) mediated by a subset of CC-NB-LRR-type resistance (R-) proteins, but not by TIR-NB-LRR-type R-proteins. Evidence suggests that this de-ubiquitinase requirement is due to a role of MVBs in these specific ETIs. The same study has exposed that a double edged sword is present here: The de-ubiquitinase is on one hand positively required for PTI mediated by CC-NB-LRR. However, on the other hand it appears to be a negative regulator of PTI mediated by TIR-NB-LRRs. The latter observation suggests that MVB components are monitored by one or more TIR-NB-LRRs, which may be the reason that ESCRT complex mutations occasionally are lethal.