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Towards a dynamic analysis of protein trafficking in plant cells

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Plants feed the world with the secretory materials that they produce and storage. Secretory materials are synthesized on the surface of the endoplasmic reticulum (ER)1. They are then shipped from the ER to the Golgi apparatus to be sorted either back to the ER or to distal secretory compartments such as vacuoles and plasma membrane. The ER and Golgi are closely associated in plant cells2,3. How these two organelles communicate with each other is an important question that remains largely unanswered in plant cells. To provide further understanding of the regulation of protein export from the plant ER, we have explored the mechanisms of protein trafficking between the ER and the Golgi apparatus using live cell imaging techniques. It appears that plant cells contain multiple mobile Golgi stacks distributed over the entire cytoplasm. These stacks move with the ER by means of actin-myosin motors3,4. The domains of the ER dedicated to the export of proteins, the ER export sites (ERESs), form secretory units that move along the surface of the ER together with the Golgi stacks4,5. We also found that the integrity of Golgi and ERESs is regulated by the activity of specific GTPases, such as Sar1 and Arf14,5. Our results indicate that in plant cells the ER and Golgi form a dynamic membrane system whose components continuously cycle through the ER via a regulated membrane trafficking pathway. Interestingly, we found that the integrity of Golgi membranes depends on an active ER protein export as disruption of protein secretion from the ER determines collapse of Golgi stacks. Finally, we determined the existence of a stringent signal-regulated mechanisms for ER export of multispanning, type I and type II membrane proteins6. For example, we found that mutations of a specific di-acidic motif (DXE) in the cytosolic tail of proteins such as CASP, a Golgi matrix protein with a type II membrane topology7, cause a reduction of the export of this protein from the ER6. ER export of type I and multispanning membrane proteins is similarly regulated6. These data open interesting avenues of research into the characterization of the mechanisms that regulate protein secretion at the ER/Golgi interface.

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