



e-Book on plant virus infection-a cell biology perspective.

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► To cite this version:

Jean-François Laliberté, Peter Moffett, Hélène Sanfaçon, Aiming Wang, Richard S Nelson, et al.. e-Book on plant virus infection-a cell biology perspective.. Frontiers in Plant Science, 2013, 4, pp.203. 10.3389/fpls.2013.00203 . pasteur-01131940

HAL Id: pasteur-01131940

<https://riip.hal.science/pasteur-01131940>

Submitted on 16 Mar 2015

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modify its target, the chloroplast and its membranes. In this study, Jang et al. (2013) further solidified a previously observed influence of AltMV TGB3 protein on chloroplast structure through EM studies. They also found that TGB3 interacts with a nuclear-encoded chloroplast protein that may result in a destabilization of the thylakoid membranes. While shedding light on an influence of a viral protein on another membrane system, this and the previous study on PVX indicate how conserved proteins may have diverged in function.

In addition to TGB3, TGB2 seems also to play a role in targeting the virus to chloroplasts. In another research paper, Cowan et al. (2012) demonstrated that TGB2 protein of potato mop-top virus (PTMV), the type virus of the genus *Pomovirus*, interacts with the ER, mobile granules, small round structures, and chloroplast envelopes. Protein-lipid interaction assays confirmed the association of TGB2 with lipids of chloroplasts. Consistently, EM data revealed abnormal chloroplasts with cytoplasmic inclusions and terminal projections. Viral coat protein (CP), genomic RNA, and labeled TGB2 were colocalized to chloroplasts in PTMV-infected tissues.

Viral cell-to-cell movement remains a hot topic for plant virologists. Xu and Zhou (2012) studied NSvc4, the movement protein of rice stripe virus (RSV), the type member of the *Tenunivirus* genus. They showed that NSvc4 traffics on the actin filament and ER network and that targeting of NSvc4 to PD requires the functional cytoskeleton. They also found that NSvc4 contains a chloroplast-targeting signal and localizes to chloroplasts in infected cells, suggesting NSvc4 may also be a multi-functional protein.

In their hypothesis and theory article, Krenz et al. (2012) discuss alteration in chloroplast structure induced by geminiviruses.

They provide evidence that abutilon mosaic virus (AbMV) infection induces a network of stromules that extend from the plastid to the cellular periphery. The stromules contain heat shock cognate 70 kDa protein, a plant chaperone that interacts with the AbMV movement protein. The authors discuss a model in which AbMV traffics along the stromule network to move into neighboring cells.

Cellular remodeling is also the consequence of molecular pathways being overpowered by viruses. Verchot (2012) explores the recruitment of host proteins, such as cellular chaperones, to membrane bound sites required for virus replication and cell-to-cell movement. She discusses the possibilities that cellular chaperones are acting within their normal context to enable viral protein folding, trafficking, and functioning, or whether they are diverted from their normal activities to provide novel contributions to virus infection.

Zhang and Wang (2012) summarize current knowledge about the unfolded protein response (UPR) in cells, which is a reaction to ER stress triggered by the accumulation of unfolded or misfolded proteins in the lumen of the endoplasmic reticulum. The UPR is an attempt by the cell to return to ER homeostasis. Both animal and plant viruses are capable of redirecting the cell to produce large amounts of viral proteins, which causes ER stress and sets in motion the UPR signaling pathways. Zhang and Wang (2012) discuss how viral infections activate the UPR and implications for host physiology.

Bujarski (2013) gives an overview of genetic recombination in plant positive-sense RNA viruses. Questions being raised are the identity of the host factors involved in RNA recombination and the intracellular location of this RNA–RNA template switching.

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Received: 22 May 2013; accepted: 31 May 2013; published online: 17 June 2013.

Citation: Laliberté J-F, Moffett P, Sanfaçon H, Wang A, Nelson RS and Schoelz JE (2013) e-Book on plant virus infection—a cell biology perspective. *Front. Plant Sci.* 4:203. doi: 10.3389/fpls.2013.00203

This article was submitted to *Frontiers in Plant-Microbe Interaction*, a specialty of *Frontiers in Plant Science*.

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