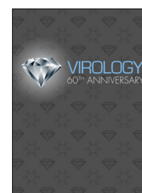
Virology 479-480 (2015) 657–671

Contents lists available at ScienceDirect

Virology

journal homepage: www.elsevier.com/locate/yviro



Review

# Plant virus replication and movement

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ARTICLE INFO

*Article history:*
Received 5 December 2014
Returned to author for revisions
19 January 2015
Accepted 28 January 2015
Available online 3 March 2015

*Keywords:*
Plant virus
TMV
TuMV
RCNMV
PVX
Membranes
Cytoskeleton
Plasmodesmata
Viral replication complex
Movement protein

ABSTRACT

Replication and intercellular spread of viruses depend on host mechanisms supporting the formation, transport and turnover of functional complexes between viral genomes, virus-encoded products and cellular factors. To enhance these processes, viruses assemble and replicate in membrane-associated complexes that may develop into "virus factories" or "viroplasms" in which viral components and host factors required for replication are concentrated. Many plant viruses replicate in association with the cortical ER-actin network that is continuous between cells through plasmodesmata. The replication complexes can be highly organized and supported by network interactions between the viral genome and the virus-encoded proteins. Intracellular PD targeting of replication complexes links the process of movement to replication and provides specificity for transport of the viral genome by the virus-encoded movement proteins. The formation and trafficking of replication complexes and also the development and anchorage of replication factories involves important roles of the cortical cytoskeleton and associated motor proteins.

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## Introduction

Plant viruses replicate and then move between cells through plasmodesmata (PD), gatable symplasmic channels in the walls of adjoining cells. Although the process of virus movement can be complex and requires support by the coordinated activity of several virus-and host-encoded proteins, many viruses achieve

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http://dx.doi.org/10.1016/j.virol.2015.01.025
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their movement with the help of classical, virus-encoded 'movement proteins (MP)' that bind nucleic acids and target and dilate PD (Lucas, 2006). Upon introduction into cells by microinjection, or by transient expression using microparticle bombardment or agroinfiltration, MPs interact with PD, move between cells and may co-transport co-injected nucleic acids (Waigmann et al., 1994; Ding et al., 1995; Fujiwara et al., 1993; Lough et al., 1998), thus indicating that their function is independent of infection. The ability of MPs to bind nucleic acids is sequence-independent, which raises the important question of how these proteins find their viral RNA cargo in infected cells. Since the MPs are produced by translation of the viral genome, they may simply attach co-translationally to viral genomes in their vicinity. Consistently, the MPs of several RNA viruses, including *Tobacco mosaic virus* (TMV; Asurmendi et al., 2004; Heinlein et al., 1998; Kawakami et al., 2004), *Potato virus X* (PVX; Bamunusinghe et al., 2009; Tilsner et al., 2013; Tilsner et al., 2012), *Brome mosaic virus* (BMV; Dohi et al., 2001) and *Red clover necrotic mosaic virus* (RCNMV; Kaido et al., 2009) colocalize with membrane-associated inclusions that harbor their viral replication complexes (VRC). As will be discussed below, VRCs can be highly organized structures involving membranes and cytoskeletal elements, and in which the binding of MP to viral RNA involves specific mechanisms, for example interaction with other viral proteins, as exemplified by VRCs formed by RCNMV. Another important question is whether MPs transport viral genomes as part of subcomplexes that are derived from anchored VRCs or whether infection moves between cells in the form of mobile VRCs. Studies with TMV and other viruses have shown that VRCs develop at multiple sites and can be mobile, and that their subcellular formation and movement are associated with the spread of the virus (e.g. Boyko et al., 2007). Moreover, VRCs may associate with PD and load replicated viral genome into PD for movement (e.g. Tilsner et al., 2013), or move through PD as an intact VRC to spread infection (Kawakami et al., 2004; Grangeon et al., 2013). The following paragraphs are aimed to summarize current knowledge about the mechanisms of VRC formation and trafficking and thus the membrane- and cytoskeleton-associated processes through which virus replication and movement are mechanistically connected. Additional information and alternative interpretations of findings can be found in several other reviews published elsewhere (e.g. Laliberté and Zheng, 2014; Liu and Nelson, 2013; Park et al., 2013; Tilsner and Oparka, 2012; Niehl and Heinlein, 2011; Heinlein, 2015).

## Organization of *Tobacco mosaic virus* movement-competent VRCs and virus factories by MP

MP-deficient viruses that replicate but do not move can often be complemented for movement by virus-specific or functionally related MPs produced ectopically by a transgene or by transient expression (Deom et al., 1987; Holt and Beachy, 1991; Morozov et al., 1997; Niehl et al., 2014). This suggests the existence of active mechanisms by which MPs and replicated viral genomes are brought into close proximity despite that the MP is not produced within replication complexes. One possibility to achieve proximity is by active targeting of the same subcellular site. During in vivo studies with TMV the same subcellular localization patterns were shown for its MP irrespective whether produced during infection or transiently (Boutant et al., 2010; Heinlein et al., 1998). Thus, this MP has intrinsic features that allow targeting to sites in the cell at which the virus replicates. The MP associates with the cytosolic face of the endoplasmic reticulum (ER) membrane (Peiro et al., 2013), interacts with microtubules (Ashby et al., 2006; Boyko et al., 2000a; Heinlein et al., 1995), and targets microtubule-ER junctions (Sambade et al., 2008) that may also be targeted by the virus. Moreover, the MP has the capacity to recruit ER membranes to microtubules (Ferralli et al., 2006) and may thus contribute to the formation of ER membrane inclusions that are seen upon ectopic MP expression as well as in the form of 'virus factories' during TMV infection (Heinlein et al., 1998; Niehl et al., 2013b; Reichel and Beachy, 1998). Experiments using microinjection of in vitro-coated, Cy5-labeled TMV RNA have demonstrated that also viral RNA associates with the ER, by virtue of its 5'CAP (Christensen et al., 2009). Interestingly, in the presence of ectopically expressed MP, the introduced viral genome was capable to move and spread infection only after producing progeny without the tag, thus after replication (Christensen et al., 2009). This indicates that the initially ER-attached viral genome remains at the attachment site and that only progeny viral RNA is available for the formation of movement complexes. This observation is remarkable as it demonstrates that the movement process is linked to replication and that, therefore, the formation of a ribonucleoprotein (vRNP) consisting only of MP and viral RNA is not sufficient for movement. Since replication of the TMV genome depends on the translation of the 183k replicase, it appears plausible that the vRNP complex that moves between cells is a VRC containing the viral genome, replicase, MP and host factors. The VRC may also contain the 126k protein, which is produced by termination of 183k translation at a leaky amber stop codon within the 183k open reading frame. This shorter protein acts as a replication co-factor as it lacks the RNA-dependent RNA polymerase (RdRp) domain present in the full length 183k protein but interacts with the 183k protein (Goregaoker et al., 2001) and functions as the viral suppressor of RNA silencing (VSR) (Ding et al., 2004; Vogler et al., 2007). Supporting evidence for the presence of the 126k protein in the movement complex comes from domain swapping experiments indicating a role of the non-conserved region and the RNA helicase domain of the 126k/183k replicase in TMV movement (Hirashima and Watanabe, 2001, 2003). Moreover, virus evolution experiments correlated the efficient movement of evolved TMV lineages in certain Arabidopsis mutants with adaptive mutations in this protein (Peña et al., 2014). It is clear that the movement in the form of a VRC rather than as a simple MP:vRNA complex would represent an efficient means to accelerate the spread of infection as the viral genome would be already associated with proteins required to rapidly initiate replication in recipient cells. Consistently, the speed of cell-to-cell movement was reported to increase significantly after viral exit from the first infected cells (Kawakami et al., 2004). VRC movement could also explain observations suggesting a role of replicase (or replicating virus) in contributing to the degradation of PD-associated callose induced by MP to facilitate movement (Guenoune-Gelbart et al., 2008). The observation that the localization of ectopically expressed MP mimics the localizations of MP produced during infection indicates that the MP, in addition to being part of the VRC that spreads between cells, provides the microenvironment for VRC maturation. As already mentioned and as will be described further below, the MP may employ a microtubule-dependent aggregation mechanism by which it attracts and holds together ER membranes and associated factors.

Taken together, the above observations suggest the following model: Following initial stages of replication by the replicase produced from the ER-associated viral genome, the MP is produced from subgenomic RNA. The MP does not interact with the replicase proteins (Hirashima and Watanabe, 2003) but may remain associated with the VRC through the binding to the viral RNA and, indirectly, with the RNA-bound replicase. VRC formation and stability may also be supported by the ER membrane, to which the viral genome and the MP, and perhaps also the replicase, have affinity. The spread of infection has been visually correlated with the occurrence of punctate MP-complexes (Fig. 1A) that show mobility along microtubules and the cortical ER-actin network in cells at the leading front of infections sites in leaves. These mobile MP complexes contain RNA and may represent "early VRCs" that serve to transport infection into

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