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Review

Plant viruses spread by diffusion on ER-associated movement-protein-rafts through plasmodesmata gated by viral induced host β-1,3-glucanases

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ABSTRACT

Plant viruses spread cell-to-cell by exploiting and modifying plasmodesmata, coaxial membranous channels that cross cell walls and interlink the cytoplasm, endoplasmic reticulum and plasma-membranes of contiguous cells. To facilitate viral spread, viruses encode for one or more movement proteins that interact with ER and ER derived membranes, bind vRNA and target to Pd. Mounting evidence suggests that RNA viruses that do not spread as virions employ the same basic mechanism to facilitate cell-to-cell spread. In light of the research reviewed here, we propose a general functional model for the cell-to-cell spread of these viruses. This model posits that MPs have multiple functions: one function involves directing virus induced β-1,3-glucanases which accumulate in ER derived vesicles to the cell wall to hydrolyze Pd associated callose in order to gate open the Pd; independently, the MPs form ER-associated protein rafts which transport bound vRNA by diffusion along ER to adjacent cells via the ER component of the plasmodesmata. The driving force for spread is the diffusion gradient between infected and non-infected adjacent cells.

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Contents

1. Viruses exploit plasmodesmata to overcome the host wall barrier

Viral infection and spread depends on the successful introduction of a virus into a cell of a compatible host, followed by replication, cell-to-cell spread and entry into the vascular system in order to allow systemic infection. Since plant viruses are simple pathogens that apparently are unable to lyse cell walls, they must somehow overcome the cell wall barrier. To do this, they exploit and modify plasmodesmata (Pd), co-axial membranous channels that cross cell-walls of contiguous plant cells, linking their cytoplasm, plasma membranes (PM) and endoplasmic reticulum (ER) [1–4]. Normally, these channels function in the direct cytoplasmic cell-to-cell transport of both small molecules and macromolecules [4] and can serve as conduits for the transport of ER-associated membrane proteins [5] and possibly RNAs [6]. At present there in no evidence for cell-to-cell transport of PM associated proteins via the PM component of Pd.

2. Pd structure and its relevance for viral spread

The basic structure of a simple Pd consists of two coaxial membrane sheaths surrounded by a specialized cell-wall-sheath (Fig. 1).

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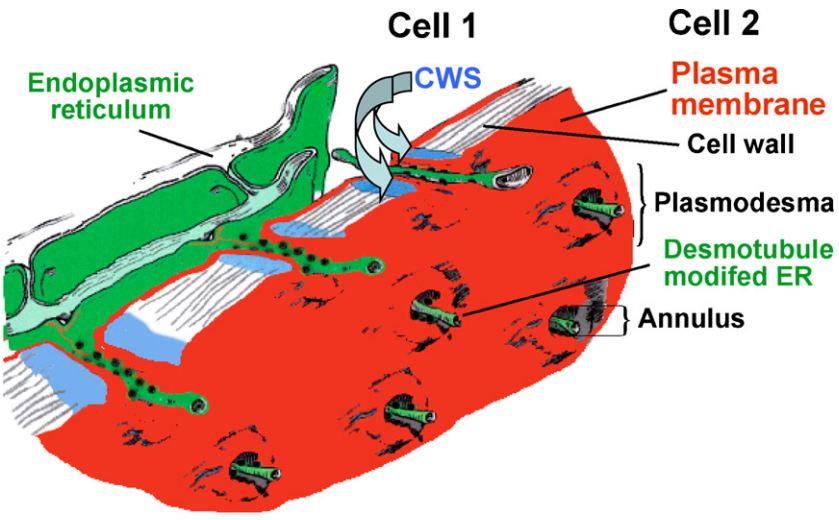

Fig. 1. A schematic model of the substructure of a simple plasmodesma. The outer coaxial membrane lining the plasmodesma consists of modified plasma-membrane (red) that is continuous between adjoining cells. The cell wall-sheath surrounding the Pd (blue) is devoid of cellulose and hemicellulose and is composed in part of non-esterified pectin, callose, other non-cellulosic polyglucans and probably as yet uncharacterized proteins. The inner coaxial component, the desmotubule (green) consists of modified cortical ER and may be constricted with no lumen or it may be tubular with open lumen. Particles present within cytoplasmic sleeve may be embedded within plasma-membrane, desmotubule or present entirely within the cytoplasmic sleeve. Taken from Heinlein and Epel [4] with modification with permission of publisher Elsevier.

The inner axial membrane which consists of modified ER and is termed the desmotubule (ER^{DT}), is continuous between cells, interconnecting the ER of bordering cell. The outer coaxial membrane is continuous with and interconnects the PM of adjacent cells. Between the coaxial membrane sheaths is a sleeve, the cytoplasmic sleeve (CS) that interconnects the cytoplasm of the neighbouring cells. Within this sleeve, soluble molecules whose size is within the size constraints of the sleeve move cell-to-cell [7]. Surrounding the Pd is a specialized cell-wall sheath (CWS^{Pd}) that is devoid of cellulose and is composed of callose, non-esterified pectin and hemicellulose polyglucans [4]. The CWS^{Pd} is a dynamic entity that controls Pd conductivity and plays a role in developmental and defence processes [8–13]. Much evidence is available for Pd conductivity changes that are due to alteration in the composition of CWS^{Pd}, mediated in part by the synthesis and hydrolysis of callose [4,5,14–16]. It has now becoming apparent that regulation of callose levels in the CWS^{Pd} is of major importance in the mechanism of viral spread and arises as a result of host–parasite interaction (see below).

3. Virus spread is facilitated by virus encoded movement proteins

An accepted paradigm for years has been that viruses spread cell-to-cell via the cytoplasmic sleeve between the coaxial membranes of the Pd that transverse the wall. If this was indeed the case, the virus would have to modify this passageway, as it is too small to facilitate the free diffusion of whole virus particles or of ribo-nuclear complexes in the cytoplasm [7]. Since unfacilitated transport of cytoplasmic soluble molecules is limited to molecules with a Stokes radius of less than about 3 nm (equivalent to a globular protein with a molecular weight of less that ~50,000), an active or facilitated transport mechanism is invoked. To facilitate transport, many viruses encode for one or more proteins, designated movement proteins (MPs) that target to and dilate Pd [1,4,17,18]. Furthermore, it is uncertain that free diffusion in the cytoplasmic sleeve, even if dilated, would be feasible given the constraints of Pd diameter [5,7]. Various hypotheses have been proposed to explain the movement mechanism, ranging from microtubule mediated intracellular transport of a virus replication complex to the Pd, microfilament mediated transport to and through the cytoplasmic sleeve, and diffusional transport of a MP vRNA complex in the ER membranes interconnecting contiguous cells [4,5,19–22]. In this paper we will review present knowledge only about spread of RNA viruses which do not spread as virions and we will propose a unified functional model for virus cell-to-cell spread for such viruses.

3.1. Viruses have developed varied movement mechanisms

At a superficial level, the various virus groups have apparently developed a range of different movement strategies. According to a classification based on the number of viral encoded MPs involved in viral cell–cell spread, three to four viral movement strategies have been distinguished.

3.1.1. Cell-to-cell transport mediated by a single movement protein

The Tobamoviruses, Dianthoviruses, Umbravirues, Bromovirues and Cucumoviruses encode for only a single MP that is ER associated, binds RNA non-specifically, targets to Pd and increases the SEL of Pd [18,23]. The Bromoviruses and Cucumoviruses may also employ CP as an ancillary protein, but CP involvement is not absolute since a deletion in the C-terminus of their MP eliminates the requirement for CP [18]. Since the requirement for CP can be overcome by a mutation in MP, it has been suggested that in this case, CP may be involved in some host response rather than being directly involved in the movement mechanism [18].

3.1.1.1. The TMV movement protein has multiple functions. The MP of *Tobacco mosaic virus* MP^{TMV}, the arch type of the group, was the first viral protein shown to have a movement function [24,25]. MP^{TMV} localizes to the Pd [26–29], modifies the Pd size exclusion limit [10], binds RNA non-specifically [30] and associates with the ER or ER derived membranes [31–34]. It has been suggested that the MP^{TMV} is an integral ER membrane protein [31]. However, conclusive data for this conjecture remains to be obtained. Based on most tools for predicting membrane protein topology, MP^{TMV} is not predicted to be an integral membrane protein. Nevertheless, there is biochemical data which suggests that MP^{TMV} is an integral membrane protein [31,32]. Based on this assumption, a model was proposed suggesting that MP^{TMV} has two transmembrane segments with the N- and C-termini in the cytoplasm and the peptide-sequences bounded by the two transmembrane segments within the ER lumen. This

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