MOLECULAR ASPECTS OF PLANT VIRUS MOVEMENT THROUGH PLANTS

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ABSTRACT

The movement of viruses through plants plays a key role in viral infection spread and establishment of the virus. Viruses have developed many strategies to ensure their efficient spread through plants and within cells to promote protein expression, replication, and production of the next generation. The movement of viruses is mainly mediated by movement proteins (MP) and to a lesser extent by capsid proteins (CP). Short-distance movement is promoted by the interactions of MP and/or CP with the host cellular cytoskeleton, which directs targeting of viral particles to the plasmodesmata. Plasmodesmata allow free intercellular passage of small molecules and limit movement of viruses due to their exclusion size. Viruses use different strategies to expand the diameter of the plasmodesmataand to enable spread of infectious viral particles over short distances. Viruses have also adapted to interact with particular host factors to promote their long-distance movements through carbohydrate fluxes. Different viral species interact with various host factors to promote their movement. The main plant regulators of viral movement along plants are callose deposits and the RNA interference mechanism. The study of viral movement is important for agriculture, for medicine, and for vector engineering to obtain recombinant products.

Key words: cell-to-cell movement, movement, movement protein, phloem channels, plant viruses, plasmodesmata, callose

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INTRODUCTION

Viruses are very small infectious agents which are able to replicate and express proteins only inside of a host cell. Viruses are pathogens of any type of an organism living on Earth: bacteria, archaea, fungi, plants and animals. The first discovered virus was of a plant origin, tobacco mosaic virus (TMV), and despite that fact plant viruses are not being deeply studied as human or animal pathogens have been. That is because plant viruses are unable to infect humans and the symptoms of plant viral infection are often invisible. However, there are over thousands of plant viruses, which affect the agricultural industry in a negative way. It is quite hard to measure impact caused by plant viruses, because the data varies among location, season and plant species. The most wide-spread plants infecting viruses are mosaic viruses of tobacco (TMV) and cauliflower (CMV), and representatives of *Bromoviridae*, *Bunyaviridae*, *Closteroviridae*, *Luteoviridae*, *Rhabdoviridae* and *Potyviridae* viral families. Indeed, the most significant plant pathogen is *Geminiviridae*, because it has a wide host range and was found in many countries throughout the world [1].

Top 10 plant viruses were described to their scientific and economic extents in 2011. These plant viruses include: Tobacco mosaic virus (TMV); Tomato spotted wilt virus (TSWV); Tomato yellow leaf curl virus (TYLCV); Cucumber mosaic virus (CMV); Potato virus Y (PVY); Cauliflower mosaic virus(CaMV); African cassava mosaic virus (ACMV); Plum pox virus (PPV); Brome mosaic virus (BMV); Potato virus X (PVX) [2].

All viruses share similar characteristics in morphology [3]. They consist of a nucleic acid and a protective coat made from proteins. Most plant viruses such as *Bromoviridae*and *Closteroviridae*contain positive sense single-stranded RNA molecules. Positive sense RNA molecule allows direct translation of viral proteins once it enters the host cell. Relatively few plant viruses such as *Bunyaviridae*and *Rhabdoviridae*possess negative-sense RNA genomes. That means that viruses should firstly synthesize positive-sense RNA, and then express proteins. *Reoviridae*, *Caulimoviridae andGeminiviridae*are viral families, which possess DNA genomes of single or double strands [4].

The first stage of plant infection is the viral penetration into the host, which can be performed by different ways including transmission by mechanical means, seeds, vegetative propagation, soil, pollen grains or vector mediated transmission. After initial entrance, the symptoms of the disease cannot be observed, because entered virus amount is not enough to cause visible symptoms. The virus should firstly produce sufficient progeny (second stage of infection), at the

same time moving through the plant and infecting new cells (the third stage). So, the aggressiveness and efficiency of viral infection upon plants depend not only on nucleic acid type or proteins it expresses, but on molecular interactions between a virus and a host cell. All consequences of virus infection depend on success of the short and distant movement of that particular virus within a plant. Short distance movement recruits components such as movement proteins (MP), coat proteins (CP), whereas potiviruses additionally recruit HC proteinases and viral-genome linked proteins (VPg) [5]. Movement protein is the most important protein in short distance viral moving, all viruses encode this protein but with slight variations in content, numbers and properties of virus-host interactions. Plant viruses recruit MP for moving between cells through plasmodesmata by two strategies: the first strategy is the movement of ribonucleoprotein (RNP) consisting of the viral genome and MP and the second strategy is the movement of viral particle [6]. Long distance movement of plant viruses is carried out by phloem channels. This type of movement is more complex and less investigated in comparison with cell-to-cell movement. This type of systemic movement requires viral translocation from mesophyll cells to sieve elements, and only afterwards the viral particle can be translocated to new cells by phloem sap. The process of virus loading into phloem involves different steps consisting of crossing of successive borders such as mesophyll cells/bundle sheath, bundle sheath/vascular parenchyma, vascular parenchyma/companion cells, companion cells/sieve elements. After reaching new layer of cells viruses can be able to enter these cells, overcoming successive borders in reverse order.

Plants have developed adaptations during evolutionary course to withstand virus infection, but viruses have adapted too and have also developed efficient strategies to disperse and move within a plant to cover bigger areas for infection and to numerously replicate. The rate of virus movement depends on both host and viral factors such as plasmodesmata exclusion limit, plants proteins, callose in particular, virus size, and viral proteins expressed by cells translation machinery. The mechanisms of viral movement through the plant, factors affecting it and examples of plants defense mechanisms are going to be discussed in the following subunits.

The study of how viruses move, infect plant cells and are transmitted by vectors would allow the development of more efficient defense methods against them and decreasing productivity losses due to infections. Investigation of plant viruses leads to the development of resistant plants to particular range of viruses without needs to apply drugs or chemicals.

Plant viruses

As it was discussed earlier, there are many species of viruses infecting plants. Most of them are carriers of positive-strand RNA, and some species carry single-stranded or double-stranded DNA. Viruses encode a particular set of proteins, and that set is in direct dependence with the genome size. Viruses often use a cellular machinery to translate proteins, especially, when the virus genome is small and is limited in expressing proteins.

One of the most common plant viruses are of the family *Bromoviridae*. The examples of *Bromoviridae* family are Anulavirus, Bromovirus, Oleavirus and Cucumber mosaic virus (CMV) of Cucumoviruses.

Cucumber mosaic virus is of a non-enveloped icosahedral capsid structure with T=3 symmetry. Its diameter is 29 nanometers, and logically, it cannot move through the plant plasmodesmata in the form of virion without any additional modifications. Some viruses have segmented genomes such as CMV along with Closteroviruses and Partitiviruses. In order to cause disease symptoms an infecting cell should obtain at least one copy of each segment. CMV contains a positive single stranded RNA genome, which promotes direct translation of viral proteins and nucleic acid replication. The set of proteins encoded by CMV are a capping enzyme, a replicase, a helicase, a silencing suppressor, an RNA polymerase, a movement protein (MP) and a coat protein (CP). So, as the CMV is unable to passage through plasmodesmata in the form of virion, it has developed another strategy to move along the plant. Its MP enlarges the diameter of plasmodesmata in order to allow intercellular passage in the form of MP:RNA complex. The interesting and distinguishing feature of CMV is the presence of a silencing suppressor, a 2b protein, which functions as a suppressor of post-transcriptional gene silencing or RNA-interference. RNA-interference is a significant plant adaptation mechanism against viral infections, which recognizes and breaks foreign genetic material, and 2b protein guards RNA of CMV from RNA-interference functioning. Other examples of Bromoviruses are peanut stant virus, tomato aspermy virus and brome mosaic virus [3].

Closteroviridae family is another example of plants infecting viruses. It is also known as beet yellows viral group, because its most common symptoms are the yellowing of plant parts and the formation of necrotic areas. The most suffocating part of a plant is the phloem, and this observation leads to the assumption that viruses are able to replicate in carbohydrate flux and cause severe symptoms as the infectious virion particles and proteins are being synthesized. Closteroviruses are also carriers of positive single-stranded RNA with segmented genome. Their sets of encoding proteins include a major capsid protein, a minor capsid protein, a movement protein, a replicase protein and a silencing suppressor. The diameter of closteroviruses is approximately 10-13 nm in diameter, which means that the virion is able to move through the plasmodesmata with only slight changes. The passage along the plant is promoted by the interactions of MP with microtubules. Its transmission from infected to non-infected plants occurs via mechanical inoculation, aphids and mealybug transmission.

Tobacco mosaic virus was the first virus identified in the end of XIX century and described in the first half of XX century. It is a common example of rod-shaped viruses. The genetic material of tobacco mosaic virus is a positive sense single-stranded RNA, which is able to directly translate viral proteins. The set of proteins encoded by TMV are a

replicase with methyltransferase and helicase domains, an RNA-dependent RNA polymerase, a movement p30 kDa protein and a capsid protein [1].

TMV is transmitted by aphids and grasshopper vectors and due to mechanical inoculation, rubbing or direct contact of infected plant parts with non-infected ones due to human or wind activities. The transmittance within the plant is promoted by the p30 protein movement protein, which is able to enlarge plasmodesmata's diameter.

Strategies of plant viruses spreading

Viruses cannot move autonomously in order to infect new victims, so they always need a carrier to transport them. Viruses can be transported by both biotic and abiotic factors. Semi-abiotic transmission is promoted by the parts of infected plants via wind or water transmission, and purely biotic transmission is promoted by insects, aphids in particular, fungi or animals. 88% of plant RNA viruses use arthropods to spread within the environment [6]. Biotic carriers are also called vectors and they are very efficient due to their ability to enter cytoplasm of deep plant tissues by breaking through cells walls of epidermis cells. Currently, plant viruses are divided on two types, depending on abilities to maintain and spread in vector body. First type is circulative viruses and another is non-circulative viruses. Circulative viruses can exploit both circulative propagative transmission and circulative non-propagative transmission [7]. Circulative viruses can reside in vector body. Moreover, circulative non-propagative viruses (family *Luteoviridae*) acquired by vectors can move from gut to salivary glands and then are released within saliva into tissues of a healthy plants. Circulative propagative viruses are able to amplify within vector cells and mostly belong to *Rhabdoviridae, Bunyaviridae, Reoviridae* families. Non-circulative viruses do not have a life cycle within vector body. Non-propagative viruses interact only with receptors located on the alimentary or salivary canal of the mouth. Arthropods and nematodes use foregut region for non-propagative virus spreading [8,9].

Some viruses such as Potato Virus X (PVX) and TMV can be transmitted by parts of mechanically injured tissues by human or animal activity to non-infected plants. Grapevine virus A from *Vitivirus* genus can be transmitted by grafting and mealybug vectors [10,11].

Many of plant viruses are carried by aphid, a wide insect family. The transmission by aphids is very efficient and the most frequent due to many advantages. Aphids have a special anatomy, an organ called stylet, which allows direct and deep penetration into the plant tissues, and since aphids feed from phloem carbohydrates flux, the transmitted viruses are able to enter the flux without crossing mesophyll layers. In addition, aphids might carry any type of plant viruses despite the tissue specificity, and this feature widens the host range. Cauliflower mosaic virus (CaMV) is transmitted at the tip of the aphid's stylet [12]. Genera Cucumovirus, Alfamovirus, Carlavirus and Crinivirus use their CP to retain within the insect vector.GeneraPotyvirus and Caulimovirus use the "helper mode" of transmission, when two independent domains interact with CP with the use of aphid molecular factors [13-14].

Leafhoppers and planthoppers transmit *Rhabdoviridae* family such as Cytorhabdovirus and Nucleorhabdovirus, which have the ability to bud from cytoplasmic membranes or from nuclear membranes, respectively [15-17]**.**

Phytoreoviruses such as rice dwarf virus and Luteoviruses such as barley yellow dwarf virus spread by the means of aphids by retaining within mitgut and filter chamber [18].

Some viruses have adapted strategies to be transmitted to plants by Nematoda vectors. For example, tobraviruses are transmitted by *Trichodoridae* family. The grapevine funleaf virus is transmitted by *Longidaridae* family [19].

Intracellular movement of viruses

Once the virus enters cellular cytosol, it should reach a particular site for proteins translation and replication, where all required molecules and ATP accumulate. For some viruses it is a nucleus, for others - a defined location in the cytosol. To promote this type of movement, viruses have developed several strategies such as intracellular movement via cellular vesicles or microtubules [3].

In many cases, viruses move in the form of virions due to endosomal vesicles derived from Goldi apparatus or endoplasmatic reticulum. Vesicles are also used to transport newly synthesized molecules to virion assembly site. In some viral species, capsid interacts with host cellular factors to promote its movement intercellularly. The mechanism of this movement type lies in the interactions with microtubules. Viral movement proteins interact with microtubule-dependent motor called dynein and an adaptive complex called dynacin. The dynein carries viral molecules along chains of microtubules from initial binding site to the defined location by the guidance of cellular factors. That feature is a significant advantage for viruses, because dynein molecules carry viral particles through all cytoplasmic obstacles in the form of sites with many accumulated molecules and between organelles. Dynactin plays a role in forming interactions between viral proteins and dynein domains, thus facilitating the movement [3].

CP is also involved in intracellular movement of viruses through the interactions with host factors. For example, red clover necrotic mosaic virus (RCNMV) which infects species of *Trifoliumincarnatum*uses its CP during virion assembly within the cytoplasm and during intracellular movement. CP of RCNMV participates in the formation of viral replication "factories", which are membrane-bound. These vesicle-like structures contain both host and viral multiple factors in order to promote virus replication using cellular machinery. CP interacts with ADP ribosylation factor 1 (Arf1) and forms complex I (COPI) on Golgi membranes [20] to destabilize cellular membranes while exiting cells. The multiple viral factors are moved intracellularly in Golgi-derived vesicles very efficiently, in direct dependence with cellular cytoskeleton and transport machinery.

Short distance movement through plasmodesmata

Plasmodesmata are specific channels of plants which connect cytoplasms of neighbouring cells. Plasmodesmata's function is to allow passage of different molecules from one cell to another, thus regulating vital functions of a plant. For example, the formation of root hairs strongly depends on regulation by molecular factors such as TRANSPARENT TESTA GLABRA (TTG1), GLABRA 3 (GL3), enhancer of GLABRA 2 (EGL3), and GLABRA 2 (GLA2), which move through plasmodesmata and regulate development and differentiation of cells [21].

Plasmodesmata consist of plasma membrane layer and a tube of compact endoplasmatic reticulum called desmotubule. Small molecules such as RNA or water move inside the space between membrane layer and a desmotubule. Special proteins are located on both surfaces within the plasmodesmata to regulate the passage of different molecules. Viruses have adapted different strategies to promote passage of their molecules within interplasmodesmatic space because it is a convenient way to move, see figure 1.

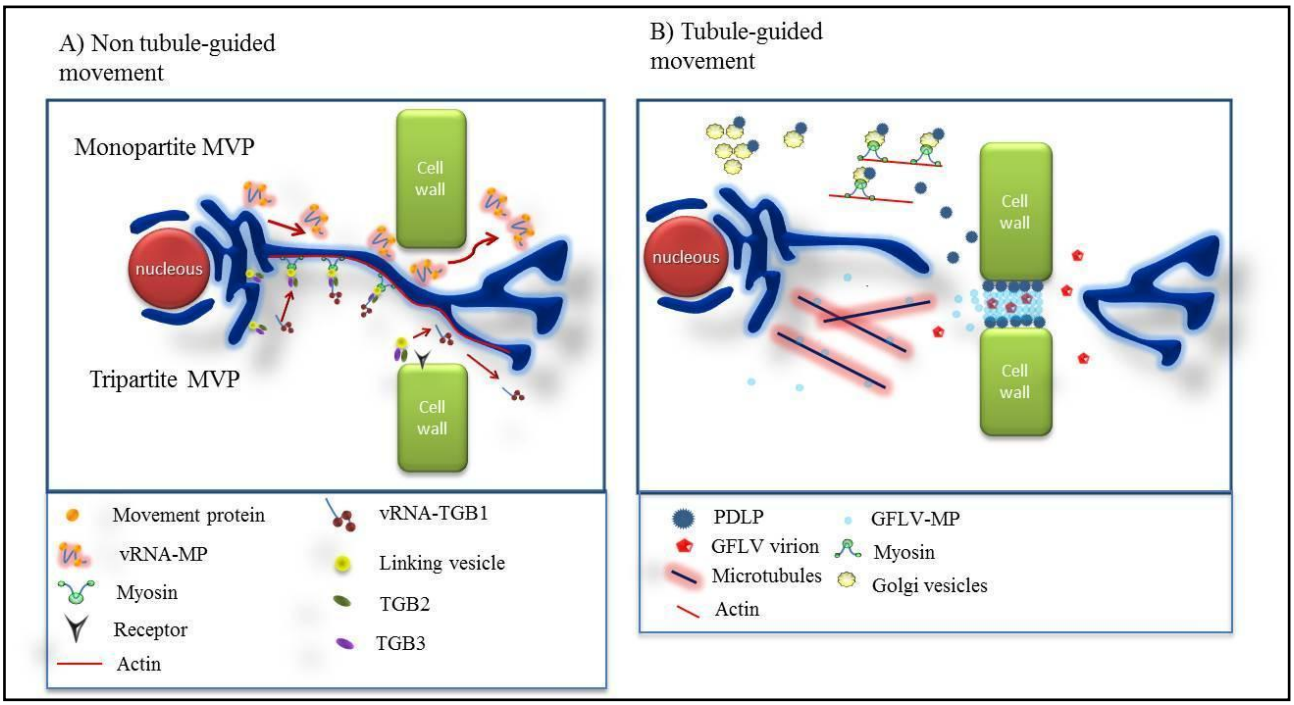

Fig.1. Short distance movement (plasmodesmata)

Viruses have adapted to use intracellular transport machinery to move from one cell into another. One of the most significant issues regulating the passage of viruses is the size of plasmodesmata aperture. Size exclusion limit of the plasmodesmata is under strict control and changes during plant development or due to specific responses such as deviations from common environmental conditions [21].

Viruses are sometimes distinguished by their mode of movement. They are able to passage from one cell to another in virion or non-virion forms, and this mostly depends on viral species [21].

Most viruses encode a typical set of proteins. One of the most common proteins of viruses is the movement protein (MP), which is responsible for viral movement to neighboring cells. It was shown that MP is able to manipulate the size of plasmodesmata exclusion limit by interacting with callose deposits and thus increasing the permeability through plasmodesmata.

There are two main types of short-distance mode of viral movement, which are promoted by MP interactions: tubule-guided movement and non-tubule guided. In tubule-guided mode of movement, MP form tubules to ensure structural modifications of the plasmodesmata. MP forms a tubule, which is then inserted into the plasmodesmata to destabilize the desmotubule within the plasmodesmata and eventually destroy it. As desmotubule becomes destabilized, plasmodesmata's size exclusion limit increases and pores dilate. It was shown that many viruses with single-stranded RNA such as bromoviruses, comoviruses and nepoviruses (Grapevine fanleaf virus (GFLV)) and cauliviruses with double-stranded DNA possess this type of short distance movement.

Non-tubule guided transport implies that MP does not form tubules but hijacks cellular transport machinery to transport viral particles. Viruses such as TMV, tobamoviruses and tombusviruses form nucleic acid:MP complexes and move it through cellular cytoskeletal network. However, plasmodesmata's permeability still increases due to formed

interactions of these complexes with cell wall pectin methylesterases. It was also shown, that cellular cytoskeleton, cellular kinases and plasmodesmata anchored proteins facilitate the spread of TMV infection throughout the plant [22].

Tobacco mosaic virus (TMV) uses its MP to move RNA from one cell to another in a non-virion form. MP has the ability to independently bind single-stranded nucleic acids of viruses and then transport through plasmodesmata. It is believed, that TMV infection is spread by this way. RNA binds to MP, forming a non-encapsidatedribonucleocomplex of TMV, and moves along the plasmodesmata. RNA:MP complex associates with viral replication complexes (VRCs), which include viral replicases [21]. Microscopicalobsevations show that VRC accumulate near endoplasmatic reticulum whose tubes prolong through plasmodesmata. In addition, movement protein has a high affinity to microtubules, especially tubulin, and thus facilitates the anchorage of VRC to desmotubule in order to pass it into neighboring cell. VRC that do not move but stay anchored to microtubule-associated ER-sites are grow into viral factories within host cells. These complexes produce high levels of coat proteins, replicase, movement protein and viral RNA, which are then moved to yet non-infected cells.

TMV does not need its capsid protein (CP) to promote movement through plasmodesmata, whereas bromoviruses like Brome mosaic virus (BMV) and Cucumber mosaic virus (CMV) and potexviruses need it.

Long-distance transport

The transport of viruses through plasmodesmata and cellular components allows short-distance spread of infection, but it is not efficient for long-distance transport. When the virus enters the plant, it may be localized in only one particular place such as a single leaf or a small portion of a stem. In order to widespead within a plant, the virus should use a different type of movement strategy. One of the most common pathways a virus uses is a structure of a plant phloem. Phloem's function is to transport photosynthesis products such as glucose to all parts located below. Viruses have adapted strategies to move along the phloem in order to widespread within all plants.

In order to reach phloem, viruses should firstly replicate in the initial host cell, and then enter the plasmodesmata, cross the layers of bundle sheath, vascular parenchymal cells and companion cells, which together form mesophyll tissue of plants (see figure 2).After those successive crossings, viruses are able to enter sieve elements and enter the flux of sugars through the phloem.

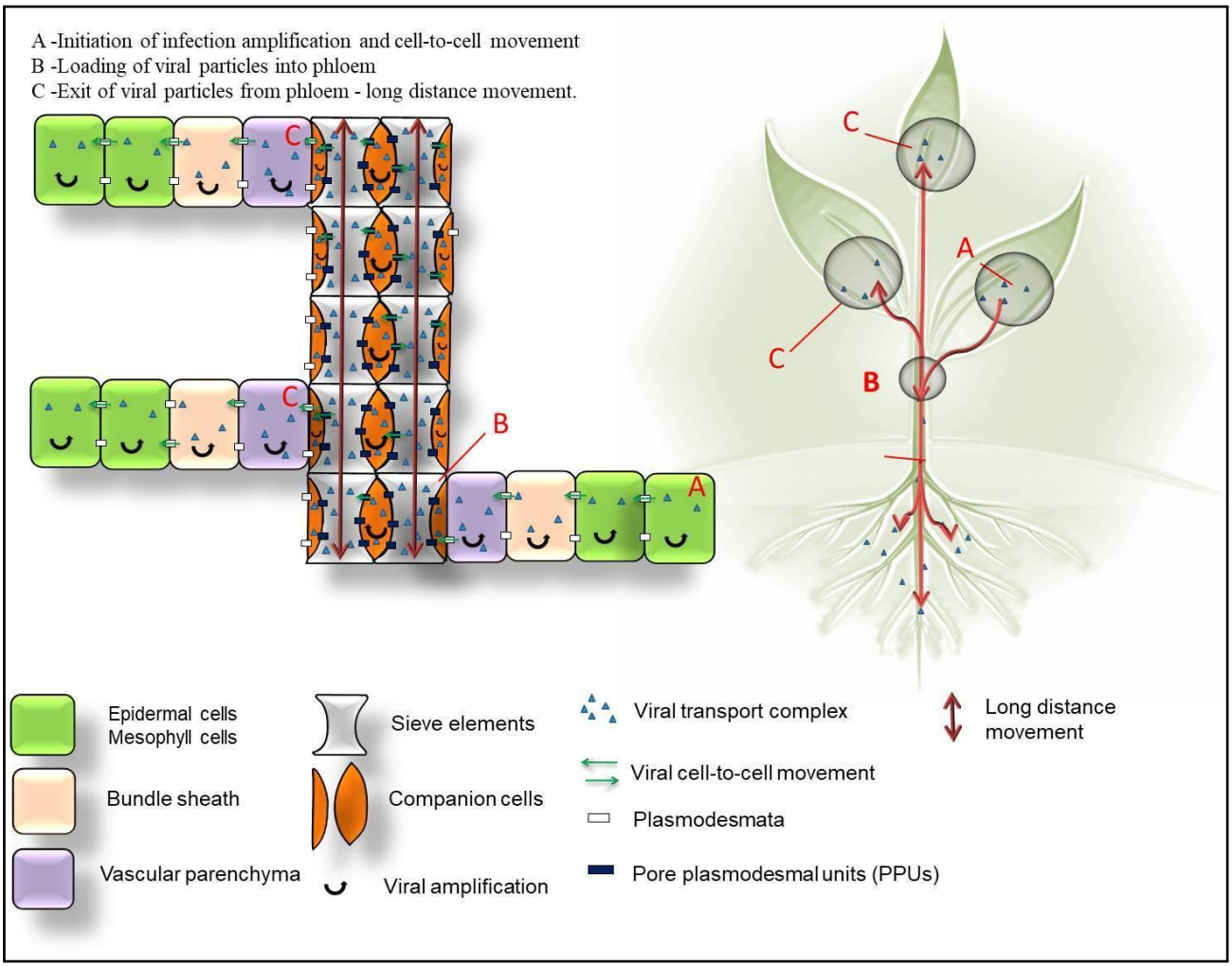

Fig.2. Long-distance movement of plant viruses

Plants have regions, where plasmodesmata's size exclusion limit is lower than in other cells. Those specialized plasmodesmata are called as pore plasmodesmal units (PPU) and can be found in cells with high metabolic activity [22]. Companion cells of plants are close neighbours of sieve elements of a phloem and possess a high metabolic activity and PPU. Viruses are able to rapidly replicate in companion cells, generating a high input of movement and coat proteins along with RNA replication. Then, due to pore plasmodesmal units proteins of viruses easily enter the flux of carbohydrates and disseminate within plant tissues to widespread within a whole plant without specific modification of the plasmodesmata [23-24]. However, viral particles and rribonucleic complexes cannot move that way, because size exclusion limit is still applies to them.

Most viruses move through phloem and sieve elements in the form of viral complexes, which composition depends on viral species. The examples of those virus genera are Potexvirus, Begomovirus, Necrovirus, Tobamovirus, Sobemovirus, and Benyvirus. For other viral species such as Potexvirus, Cucumovirus, Bromovirus, Closterovirus, and Potyvirus genera it is still unknown, whether their virions or CP-asociated viral complexes are involved in intercellular transport or not. It was experimentally shown that Bean Golden Mosaic virus (BGMV, begomovirus) moves along the carbohydrates flux in a binary mode of transport: in both CP-dependent (in the form of virions) and CP-independent manner. It should be noted, that CP-independent mode of intercellular passage is present in beans and is not so efficient [14-26]**.**

Potato mosaic virus (PVX) has developed a strategy for movement to use both CP and MP for both short and long-distance spread [27]. It was shown by live cell imaging of both viral RNA and proteins that virus moves along the phloem as an assembled viral particle. Three proteins of PVX accumulate at distinct plasmodesmata sites to perform different transportational functions. TGB2 and TGB3 accumulate at plasmodesmatic pores to remodel membranes and form plasmodemata-anchored viral RNA replication sites. TGB1 facilitates the deposition of CP inside the plasmodesmata. Thus, as viruses replicate and form virion particles, they cross mesophyl layers through plasmodesmata and enter carbohydrate flux to move longitudinally [28].

Regulatory factors involved in viral movement throughout the plants.Regulation factors of a plantCallose deposits

Callose is a biomolecule which is of a beta1,3-glucan nature. It accumulates at the neck region of the plasmodesmata and controls the size exclusion limit of plasmodesmata. When the amount of callose is high at these neck regions, it decreases the diameter of plasmodesmata and alters the trafficking of molecules. So, callose acts as a sphincter by constricting and expanding plasmodesmata.

It was described that TMV moves along the plant in the form of RNA: MP complex. This complex is relatively big and requires the expansion of the plasmodesmata to be able to pass through it. Movement protein of TMV has been shown to interact with plant host factors, such as cytoskeletal elements, calreticulin, pectin methylesterases, and DnaJ chaperones; in order to target the RNA: MP complex to the plasmodesmata [29].

It is believed that MP fristly interacts with plant ankyrin repeat-containing protein ANK, which accumulates at the cytoplasmic regions. The complex of MP:ANK leads to a decrease in Pdcallose levels and enhances cell-to-cell movement of TMV MP by expanding size exclusion limit of plasmodesmata [30].

RNA silencing suppressors

RNA silencing mechanism is a plant adaptation against viral infections. RNA silencing is the production of virus-specific short interfering RNA by a plant cell to recognize and cut foreign genetic material- sequences. These siRNA are obtained from double-stranded RNA (dsRNA) by restriction of Dicer enzymes (DCL). siRNA is then recruited by RNA-induced silencing complexes (RISC), whichcontain an ARGONAUTE (AGO) effector protein. AGO 1 protein is responsible for cutting the sequence-specific homologous targets [31]**.**

So, RNA silencing mechanism recognizes foreign nucleic complexes once they enter cells. The RNA silencing may restrict viral further movement and spread of infection to other neighbouring cells by AGO1 cleavage.

Actin and myosin

Actin and myosin are proteins which form cellular cytoskeleton. They are responsible for transport of molecules within cells and they are also involved in the passage of molecules from one cell to another. Evidence shows, that some parts of microfilaments localize at plasmodesmata, possibly facilitating intercellular movement [32-33]. That is especially true for actin microfilaments, because it plays a crucial role in targeting viral proteins to the plasmodesmata. It was proved by addition of actin-destabiliming polymer into the plant, after which the predicted increase of plasmodesmata's permeability was observed.

It was also shown that movement proteins of CMV and TMC also interact with actin microfilaments by destabilizing its structure and expanding the diameter of plasmodesmata. The real occurrence of this inhibiton was checked by introduction of actin-stabilizing molecule phalloidin into the plant. In the result, movement proteins could

not destabilize cell cytoskeleton and increase size exclusion limit of the plasmodesmata, leading to poor infection spread [34].

Another virus which was shown to directly interact with microfilaments is Beet Yellow virus (BYV) of closterovirus family. BYV encode protein p65 with high homology to cellular heat shock protein family (HSP), which are mostly responsible for protein folding and managing stress conditions. The p65 protein firstly binds to microtubules, which facilitate its targeting to the plasmodesmata. Then p65 forms a transport-competent complex with the viral genome and facilitates the passage through plasmodesmata due to microtubules [35-36].

Viral factors involved in movementCoat protein

The main function of a coat protein is to provide a protective shell from the envorinment. The coat of viruses is usually made from one (Bromoviridae) or two different types (minor and major capsid proteins in Closteroviruses) of coat proteins, which interact with host factors for different purposes such as virus entering and exiting, disassembling, movement and symptom development [37].

CP plays a role in transportation, but not the most significant one. Rather, it functions as a security tool to facilitate long or short distance movement. The CP of Tomato Mosaic Virus (ToMV) interacts with protein IP-L to secure long-distance transportation in both in vivo and in vitro conditions. ToMV infection increases the level of IP-L mRNA in tomato, and as IP-L is silenced, the systemic symptoms of ToMV infection are delayed to occur in *N. benthamiana* plants [38].

In many cases, as in Potexvirus movement mecahnisms, CP interacts with MP to facilitate intercellular movement. The C-terminal of MP of Prunus necrotic illarvirus (PNRSV) interacts with C-terminus of CP, and the interaction is responsible for targeting the complex to the plasmodemata. However, MP of PNRSV was shown to be capable of intercellular movement in both CP-dependent and CP-independent modes [39]. Alfalfa mosaic virus (AMV) moves intercellarly either as mature virions if plasmodesmata exclusion size allows that, or as RNA-CP complexes which are transported due to MP–CP interactions with host cellular cytoskeleton [40].

Coat protein can also serve as a mediator in vector transportation of viruses. Its domains may interact with different host molecular factors, which promote viral retention inside insects gut, midgut or stylet, or to receptors via binding to ligands in nematoda vectors [38]. The translation of coat and movement proteins by TMV down regulates the translation of host proteins, suggesting that viral products directly interact with cellular translation machinery. It was shown that CP in particular down regulates plant defense responses by reducing the expression of PR-1 and RDR-1 protein pathways [41].

The translation of CP may be inhibited by RNA interference, a plants resistance mechanism against viral infections. The inhibition is promoted by co-activity of chaperone CPIP and heat shock proteins (HSP70), which regulate the production of viral CP [42].

Apparently, the coat protein plays an important role in viral movement. Mutations within coding regions of CP nucleotide sequence result in abnormal functioning of the protein, such as disruption of virus retention within animal vectors and viral movement through plasmodesmata and phloem.

Movement protein

Movement proteins (MP) are encoded by all viral species, because they are responsible for all kinds of movement: intracellular, intercellular and long-distance spread through phloemic carbohydrate flux or within bodies of animal vectors. There are many functions of viral movement protein: formation of viral replication factories, formation of interacting pathways with host cellular factors promoting replication, movement, dissemination and virions assembling, direct movement of viral proteins via interactions with cytoskeleton, direct and indirect expanding of plasmodesmata, etc. So, the movement protein plays a crucial role in life establishment of any virus, because it is responsible for any kind of movement.

MP of Tobacco mosaic virus plays a crucial role in virus spread throughout the plant. Point mutation in the middle cistron of MP impairs all kinds of viral movement and thus prevents disease spread [43]. Molecular weight of MP is 30 kD, so it is usually referred as 30K protein [44].

MP of many plant viral species resembles each other by homologous nucleotide sequences, which possibly are key points in proper functioning of the protein. So, there are several families of MP, most common of which are double-block, triple-block, porline-rich tymovirus like MP, quintuple-block and 30K [45].These superfamilies are formed in order to facilitate classification of properties, features, molecular arrangement, and nucleotide sequences of MP derived from different plant viral studies [46]. Unique feature of 30K superfamily includes the ability to cooperatively bind to single-stranded nucleic acids to facilitate formation of non-virion ribonucleoprotein of viral genome, which is a carrier unit in many viral species [47]. Examples of viruses bearing 30K superfamily MP are Cilevirus, Citrivirus, Ourmiavirus, Sadwavirus, Ophioviridae and Rhabdoviridae [48].

MP plays a crucial role in movement by its active role in regulating size exclusion limit of plasmodesmata. Some viral MP directly interacts with cellular cytoskeleton, actin microfilaments in particular, to enlarge plasmodesmata's diameter [33]. In rare cases, MP self-interacts to form hollow tubular structures like cellular microfilaments to promote movement of viral particles from cytoplasm directly to plasmodesmata and otherwise [50].

CONCLUSION

All kinds of movement are vital for plant viruses. Virus is able to replicate and express its proteins due to intracellular movement through cellular cytoskeleton. The virus should reach nucleus or specialized sites from initial entrance site in order to hijack host cellular protein expression machinery in its localization. Newly synthesized viral particles in the form of virions move intercellularly to find new sources of energy and molecules required for their replication cycle, which also involves viral protein synthesis. Viruses move intercellularly through plasmodesmata, which allow free passage of molecules of particular size range. These strategies have been adapted by viruses to move on short distances, whereas long distance is promoted through the carbohydrate flux.

There is a constant struggle to survive between viruses and plants. As plants develop a certain mechanism to withstand virus infection, virus adapts to overcome the obstacles. That is the case of RNA interference mechanism in plants and the ability of viruses to overcome it by inhibiting RNA synthesis in plants to promote its infection.

There is also a set of regulators from both viral and plant sites to coordinate movement of molecules throughout the plant. The main plant regulator of movement is the deposition of callose in neck regions of plasmodesmata to regulate its exclusion size. As callose accumulates, plasmodesmata becomes narrower and viruses are no longer able to move through it, as well as some large molecules (ATP, carhohydrates, or lipids) involved in crucial metabolic pathways. So, viruses have adapted their MP protein to interact with callose to promote its degradation, leading to expansion of plasmodesmata diameter and thus allowing viral particles to move freely through it.Another viral strategy is the formation of interaction between MP and cellular microfilaments: in one case plasmodesmata size exclusion limit increases, and in another microfilaments directly target viral particles to the plasmodesmata, facilitating the movement.

MP and CP are main factors of viruses, which promote its movement. Mutations within coding regions of these proteins disable their proper functioning, thus leading to poor virus infection spread. As viruses poorly spread, they also produce ineffective generation, and the cycle of poor infection repeats itself.

So, the virus movement depends on both plant and viral factors. As CP and MP promote viral movement due to interactions with host factors such as microfilaments and cellular expression machinery, plant factors resist to viral spread by different strategies such as limiting plasmodesmata size exclusion limit by callose deposits and adapting RNA interference mechanism to recognize infectious viral particles.

The study of how viruses move throughout the plant is crucial for agriculture, because there are big losses of harvest due to viral infections. Description of main factors involved in virus movement of various viral genera and species enables to develop more effective vaccines against infections. As movement of viral particles is studies, the possibility of using plant viruses as important vectors increases. The plant vectors can be manipulated to achieve high protein expression within target cells, especially when target cells are located deep inside the plants such as mesophyl layers.

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ВИРУСТАРДЫҢ ӨСІМДІКТЕР ІШІНДЕГІ ҚОЗҒАЛЫСЫНЫҢ МОЛЕКУЛАЛЫҚ АСПЕКТІЛЕРІ

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ТҮЙІН

Вирустардың қозғалысы олардың өміріне және инфекциясының таралуында маңызды рөл атқарады. Өсімдіктердің ішіне таралу үшін әртүрлі ақуыздарды синтездеуде, репликациясын жасау мен келесі үрпаққа өмір беру үшін вирустар эволюциялық жолмен арнайы стратегиялар әзірлеген. Вирустардың қозғалысы көп жағдайда қозғалыс ақуыздар арқылы жүзеге асырылып, ал кейде вирустар капсид ақуыздар арқылы қозғалады.

Қозғалыс пен капсид ақуыздар жасушаның цитоскелетімен әрекеттесіп, қысқа қашықтыққа қозғалысын жеңілдетеді. Жасушаның цитоскелеті сол молекулардың және вирустардың бөлшектері көрші жасушаларға плазмодесма арқылы жолдайды. Плазмодесма арқылы әр-түрлі молекулалар ешқандай энергияны пайдаланбай қозғалады, бірақ сол плазмодесма молекулалардың көлемі бойынша жүк ағынын шектейді. Вирустар инфекциялары таралу үшін әр-турлі стратегияларды пайдаланып, плазмодесманы ұлғайта алады. Вирустар өсімдіктер жасушаларының молекулалық факторлармен қатынастар орнатып, ұзақ қашықтыққа флоэма арқылы жылжи алады.

Вирус түрлері қозғалу үшін өсімдіктердің әртүрлі факторлармен қатынастар жасайды. Вирустардың қозғалысының ең басты реттеушілері - каллозаның қабаттары мен РНҚ интерференция механизмі. Вирустардың қозғалысы ауыл шаруашылығы, медицина және векторлардың инженерлеу салалары үшін өте маңызды.

Негізгі сөздер: жасушалардың арасындағы қозғалыс, қозғалыс ақуыз, қозғалыс, өсімдіктердің вирустары, плазмодесма, каллоза