AgriCos e-Newsletter
ISSN: 2582-7049
Volume : 02 Issue : 01 January 2021 Article No: 03

An Overview on Molecular Mechanism of Host Virus Interactions

Rajneesh Thakur
Ph.D. Scholar, Department of Plant Pathology, College of Horticulture, Dr. Yashwant Singh Parmar University of Horticulture and Forestry, Nauni, Solan, Himachal Pradesh

SUMMARY

Viruses are small pathogens composed of nucleic acid and protein coat, obligate parasites and require host machinery for their reproduction and causal agents for many common plant diseases. They lead to heavy economic losses in crop production and quality in different parts of the world. They make their passive entry into plant cells through the wounds caused by either physical injury, through environmental factors, or by the vectors. Viral RNA disassembles, replicates, and converts its mRNA to proteins in the host cytoplasm using energy and proteins from the host cell. Once viruses enter the host, they move from infected cells to healthy neighboring cells locally. Long-distance transport via the vascular system for systemic infection is also the key feature of plant viruses. In response to the infection by viruses, plants also develop certain defense mechanisms. Many molecular mechanisms develop Coat protein mediated, RNA interference, cRNA or antisense RNA, R Gene–mediated responses and Hypersensitive and necrotic resistance responses to virus infection.

INTRODUCTION

Plant viruses are diverse and unusual groups of plant pathogens that infect and cause disease in many crop plants. These pathogens depend on the normal cellular machinery of their plant host for reproduction; it is difficult to eliminate them without damaging the host plant. Plants possess active and passive means of preventing virus infection. Passive defenses are due to the failure of the plant to produce one or more host factors required for virus reproduction and spread within the host. Active defenses include detection and destruction of the virus-infected cells due to the function of specific resistance genes in the plant. Plant defense system, which is called RNA silencing, detects and degrades viral RNAs. Recognition of specific pathogen effectors, either through direct binding or by recognition of the effector's alteration of a host protein. These virulence factors drove co-evolution of plant resistant genes to combat the pathogens' Avr (avirulent) genes. (Collier and Moffett, 2009; Gururani *et al*., 2012). Depending on the particular combination of virus and host, and on environmental conditions, a plant's response to infection may range from a symptomless condition to severe disease and plant death.

Molecular mechanism of host virus interactions:

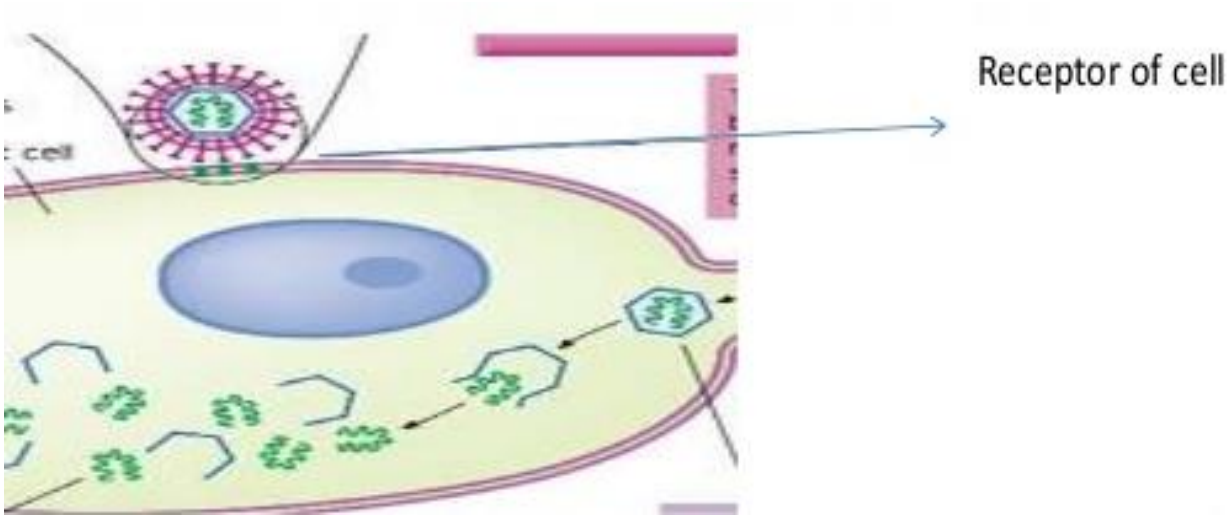
Viral replication is dependent on a host; with their small genomes, viruses need to hijack host cellular machinery to complete their life cycle. The coevolution of intimate virus-host relationships has led to many viruses being able to successfully propagate without a significant detrimental effect to the host. HR response is initiated by Avr/R protein interactions that lead to metabolic changes in defence hormone levels, such as salicylic acid (SA), Jasmonic acid (JA), and nitric oxide (NO), and the accumulation of reactive oxygen species (ROS), such as O2 and hydrogen peroxide (Carr *et al*., 2010; Pallas and García, 2011; Mandadi and Scholthof, 2012).

Coat protein mediated resistance:

The use of viral CP as a transgene for producing virus resistant plants is one of the most spectacular successes achieved in plant biotechnology. Numerous crops have been transformed to express viral CP and have been reported to show high levels of resistance in comparison to untransformed plants. Powell-Abel *et al*. reported resistance against TMV in transgenic tobacco expressing the TMV CP gene.

Mechanism of Coat Protein:

There may be some kind of receptor or uncoating site within the cell that may be responsible for initiating the disassembly of virus. CP blocks this site prior to infection, prevent virus to attach the cell. The presence of transgenic-derived coat protein in the cytoplasm may tip the disassembly- assembly equilibrium of incoming virus thus make Challenging virus is unable to uncoat.

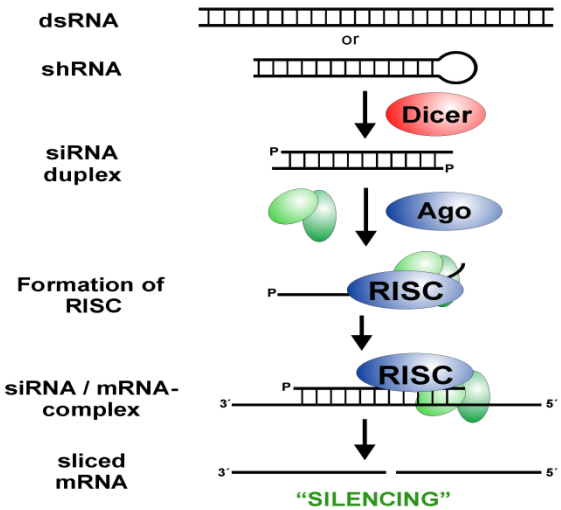


Replicase (Rep) mediated resistance

Other strategies also give resistance but causes net necrosis. On the contrary, replicase-mediated resistance gives resistance but no net necrosis. In potato, the expression of the CP gene from PLRV (potato leaf roll virus) affords only partial resistance. Transformation of plants with viral replicase has been shown to produce very effective and stable resistance.

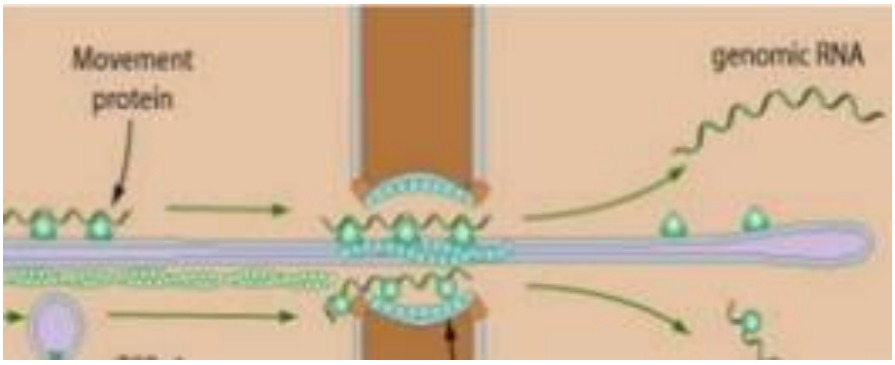
Interference:

RNA interference (RNAi) is a molecular mechanism in which fragments of double- stranded ribonucleic acid (dsRNA) interfere with the expression of a particular gene that shares a homologous sequence with the dsRNA. An intensive research effort has facilitated the rapid movement of RNAi from a relatively obscure biological phenomenon to a valuable tool used to silence target gene expression and perform large- scale functional genomics screens. Other names of RNA interference: RNA silencing, Gene silencing , Post-Transcriptional Gene silencing (PTGS). In certain fungi: quelling, RNAi can spread throughout certain organisms (C. elegans, plants).



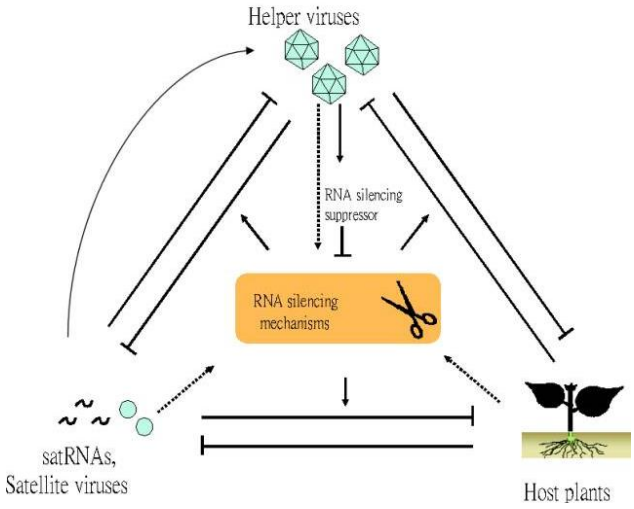
Movement protein mediated resistance:

Movement proteins (MP) are essential for cell-to-cell movement of plant viruses. These proteins have been shown to modify the gating function of plasmodesmata, thereby allowing the virus particles or their nucleoprotein derivatives to spread to adjacent cells. This phenomenon was first used to engineer resistance against TMV in tobacco by producing modified MP which is partially active as a transgene.



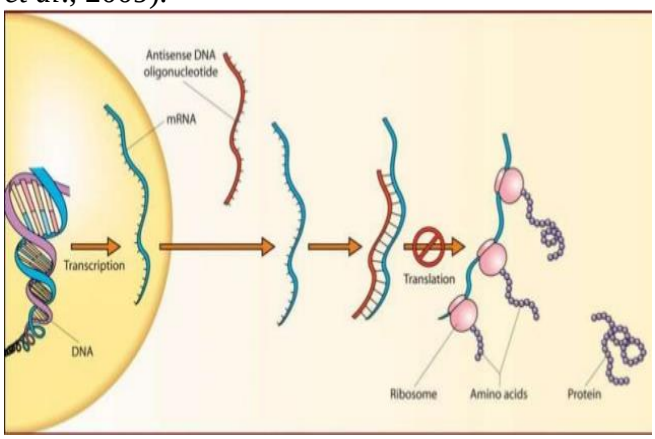
Satellite RNA:

It is strategy exploiting the use of satellite RNA associated with certain viruses received great attention. CMV sat RNA depends on its helper virus (HV) CMV for replication, movement within the plant, encapsidation and transmission. The presence of sat- RNA modulates the symptoms induced by the HV and often depresses HV accumulation in different host species. Thus, transgenic tobacco plants expressing multiple or partial copies of CMV sat-RNA showed attenuated symptoms when challenged with CMV. In addition, tobacco plants transformed with anti-sense sat-RNA also showed delayed symptom development with the cognate virus.



cRNA Or Antisense RNA Strategies:

Antisense RNA (asRNA) is a single-stranded RNA that is complementary to a messenger RNA (mRNA) strand transcribed within a cell. The use of RNA complementary to part of the viral genome (antisense RNA) is another potential pathogen-derived resistance strategy that may have some utility for protecting plants from systemic virus infection. Host interactions at almost every stage of antiviral defence, both in plants and animals (Gao and Luo, 2006; Citovsky *et al*., 2009).



Management of Virus:

The strategy for management will depend on the means by which a particular virus enters a crop, how the virus is transmitted between plants within a crop, and how the virus survives when the crop is not being grown. Preventative measures may include use of certified virus-free seed or vegetative stocks, elimination of the virus reservoirs in the surrounding wild vegetation, and modification of planting and harvesting practices. If the virus is known to be transmitted by a particular vector, control or avoidance of this vector is important. For instance, insect, nematode or fungal vectors can be controlled by insecticides, nematicides, or fungicides, respectively.

CONCLUSION

The interaction between Host plant and pathogen are specific, complex and dynamic. Signals for activation of various defenses initiate in response to recognition. Plant resistance molecular mechanisms to plant virus's interaction. The viral genetic resistance which is naturally occur-ring primarily comprises of antiviral RNA silencing, R-gene-mediated resistance and recessive resistance. As viruses are intracellular parasites consisting of a small genome of RNA or DNA packed in a capsid thus RNA silencing is considered as a major antiviral mechanism.

REFERENCES

Carr, J.P., Lewsey, M.G., and Palukaitis, P.(2010). Signaling ininduced resistance. Adv. Virus Res.76:57–121

Citovsky, V., Zaltsman, A., Kozlovsky, S.V., Gafni, Y., andKrichevsky, A.(2009). Proteasomal degradation in plant-pathogeninteractions. Semin. Cell Dev. Biol.20:1048–1054.

Collier S.M., Moffett P. (2009). NB-LRRs work a "bait and switch" on pathogens. Trends Plant Sci. 14: 521–529

Gao, G., and Luo, H.(2006). The ubiquitin-proteasome pathway inviral infections. Can. J. Physiol. Pharmacol.84:5–14.

Gururani M.A., Venkatesh J., Upadhyaya C.P., Nookaraju A., Pandey S.K., Park S.W. (2012). Plant disease resistance genes: Current status and future directions. Physiol. Mol. Plant Pathol. 78: 51–65

Mandadi, K.K., and Scholthof, K.-B.G.(2012). Characterization ofa viral synergism in the monocotBrachypodium distachyonrevealsdistinctly altered host molecular processes associated with disease.Plant Physiol.160:1432–1452

Pallas, V., and García, J.A.(2011). How do plant viruses inducedisease? Interactions and interference with host components. J.Gen. Virol.92:2691–2705.