Eco. Env. & Cons. 26 (4) : 2020; pp. (1716-1720)
Copyright@ EM International
ISSN 0971–765X

Plant resistance strategies against the viral pathogens

Siddharam Math[1], Hiralal Sonawane[1*] Vilas Patil[2], Sagar Arya[3,4] and Avinash Ade[5]

[1]Department of Botany, Prof. Ramkrishna More ACS College, Akurdi, Pune 411 044, India.
[2]Dr. B. N. Purandare Arts and Smt. S.G. Gupta Commerce and Science College, Lonavala, Pune 410 403, India
[3]Center for Innovation Research and Consultancy, Pune 411 018, India
[4]TERI-Deakin Nano Biotechnology Centre, Gurgaon 122 001, India
[5]Department of Botany, Savitribai Phule Pune University, Pune 411 007, India

(Received 5 February, 2020; Accepted 15 April, 2020)

ABSTRACT

Pests and pathogenic interaction might be beneficiary mostly for the pathogen only, in some case there may be a symbiotic association between them. Worldwide there is approximately 70,000 different types of pathogens that are damaging agricultural crops. The plants withstand against the pathogenic attack by expressing many genes and by signaling mechanism. In case of viral pathogens, the proteins like Viral Capsid Protein are majorly responsible for the hypersensitive response. RNA silencing have also been used as a tool for the inhibition of viral gene expression.

Key words : *Viral infection, Plant response, Resistance approach, Survival, RNA silencing*

Introduction

Plants make up the greater part of the earth's environments as a tree, grass, herbs, and shrubs and so on. The heterotrophic organisms including human beings, directly or indirectly depends on the plants. The plants are prone to many pests and pathogens which is responsible for the major loss in the plant development and growth. The diseases to the plants may be caused by the Fungi, Bacteria, Protozoa, Nematodes, and Viruses and so on. In this article we will discuss only on the viral pathogen and their infection to the plants and their disease establishment on the host plant (George, 2005).
Some of the viruses and their hosts are listed below

Initiation of Viral Infection

The exposure of the host plant to the virus particles will not results in to the infection unless the plant tissue is injured. Until and unless the tissue will not damage the viral particles are unable to infect. The damage area of the tissue is said to be a "Infectible Site"The infection phenomenon will consist of the following events.

- Construction of Infectible Area
- Differentiation of Infectible Area
- Conversion of Infectible Area to Infective Centre
- The Infection Spreading from The Infective Centre
- The Multiplication of the Plant Virus

Construction of the Infectible Area

The Infectible site is an area from where the viral particles will get entered. There are many types of Infectible sites, which are naturally developed by plants or may be through mechanically injury to the plants. The Stomatal Opening, Root Hairs etc. are considered to be a naturally developed Infectible Sites. The wounded or injured due to some abiotic factors results in the formation of Infectible Sites.

Table 1. Viral Types and their Host Plants

Shape of the Virus Particle	Genus Name	Type	Host Plant
Rod Shaped Particles	*Tobamovirus*	Tobacco Mosaic Virus Tomato Mosaic Virus	Tobacco, Tomato, and Most of the Solanaceae Members
	Tobravirus	Tobacco Rattle Virus	Tobacco and Potato
	Hordeivirus	Barley Stripe Mosaic Virus	Barley and many grain crops and wild grasses
	Furovirus	Soilborn Wheat Mosaic Virus	Wheat Plant
	Pecluvirus	Peanut Clump Virus	Peanut Plant
Filamentous Particles	*Allexivirus*	Shallot Virus	*Urginea* sp.
	Carlavirus	Carnation Latent Virus	Lily
	Foveavirus	Apple Stem Pitting Virus	Apple Plant
	Vitivirus	Grapevine Virus	Grape Plant
	Trichovirus	Chlorotic Leaf-spot Virus	Apple Plant
Isometric Particles	*Sobemovirus*	Southern Bean Mosaic Virus	Bean Plants and most of Leguminosae Family Members
	Marafivirus	Maize Rayado Fino Virus	Maize
	Umbravirus	Carrot Mottle Virus	Carrot Plant

Abiotic factors may be light intensity, temperature etc.

Bawden and Roberts carried out a simple experiment with a four different host and virus combinations, they kept these combination in dark for three days continuously and conclude that as compared to light exposed plants the plants which are incubated in dark show more number of the Infectible Sites. *Bawden & Robert* suggested that the photosynthesis leads to the accumulation of the inhibitory products, these products may reduce the Infectible Sites that are produced when leaves are abraded (Siegel and Zaitlin, 1964).

Differentiation of Infectible Area

There is no need to be a single mode of entry for infecting viral particles. Any wounded cell make a passage for the entrance and also for multiplication. *Hildebrand* described his observation which could serve as a model of how a plant cell get intake a viral particles through injured cells. He made an injury to the leaf of tomato plant, the injured tissue surface ware exude a drop of protoplasm which is drawn back in to the cell about 0.6 sec later, if this drop of protoplasm ware contaminated with virus while outside the cell, this viral particles also will get entered in to the cell. *Rawlins & Tompkins* have demonstrated that foreign particles can be introduced in to living epidermal calls by abrasion of the leaf surface (Siegel and Zaitlin, 1964).

The Infectible Sites are having a fixed lifespan. either they are converted into infective centers or they may be disappear. The phenomenon of the disappearance is studied by exposing these infective sites to high concentration of viral particles at different time interval of the injury. The number of infective sites present at the time of exposure to virus is detected by developing a subsequent lesions.

Conversion of Infectible Area to Infective Centre

A single viral particle is enough to initiate the formation of infective center. when the virus get entered in to the plant cell that will get undergoes a multiplication event, forming a more population of viral particles that viral particles containing cell is called as Infective Centre (Gachomo *et al.,* 2003).

The Infection Spreading from the Infective Centre

The viral agent translocated to next adjacent cell through Plasmodesmata. *Welkie& Pound* are studied on the Cucumber Mosaic Virus in Cowpea and found that the spreading is patently dependent on the temperature. At 24 ^{o}C the viral particle translocated to sub-epidermal tissue for this distance of spreading require 6 hours.

The Multiplication of the Plant Virus

One of the earliest event of the infection is the interacted virus particles releases the nucleic acid from the viral protein. The released viral DNA will get duplicated by using the host's synthesis machinery to multiply themselves, and also for the expression. (Mandadi and Scholthof, 2013).

Plant Response Against The Viral Infection

Virus is a kind of Supra-molecule which is a nucleoprotein that multiply only in living cell and has the ability to cause a disease. When the plant will get infected by the virus pathogen, there are two possible outcomes: either infection occurs or it does not (Fig. 1)(Kang *et al.,* 2005).

Susceptibility and Resistance

The infectible plants (Host) ware attacked by many pathogens, these plants may be develop a confrontation power to save themselves or may be unable to defend against pathogens these are said to be a susceptible plants (susceptible variety) (Gachomo *et al.,* 2003).

Susceptibility and Resistance are considered to be an opposite ends of the seesaw; more the susceptible plant is having a low resistance vice versa (Palukaitis and Carr, 2008).

Types of Resistance

Plants have developed many approaches to sustain against the viral infection

- Protein Mediated Resistance
- Nucleic Acid Mediated Resistance
- Systemic Acquired Resistance

Protein Mediated Resistance

Capsid Protein Mediated Resist ance (Viral Protein Mediated)

Capsid protein is the surrounding protective layer to the viral genome. The nucleotide sequence of viral which are involving in the expression of these capsid protein have been found to be useful in the development of resistance to viral pathogen. The incorporation and expression of viral capsid protein responsible gene in plants has provides a strongest viral resistance mechanism in plants (Koh *et al.,* 2014).

Examples of Capsid Protein Mediated Resistance

There have been a number of reports of Capsid Protein Coat Mediated Resistance in the plants of Solanaceae and Leguminosae

NBS-LRR Protein Mediated Resistance

The Plant resistance gene like R-Gene which are responsible for the expression of Proteins like TIR-NBS-LRR (Toll/Interleukin-1 Receptor - Nucleotide Binding Site - Leucine Rich Repeats) these proteins actively involved for response against the pathogen (Román *et al.,* 2020)

These Proteins are directly or indirectly interact

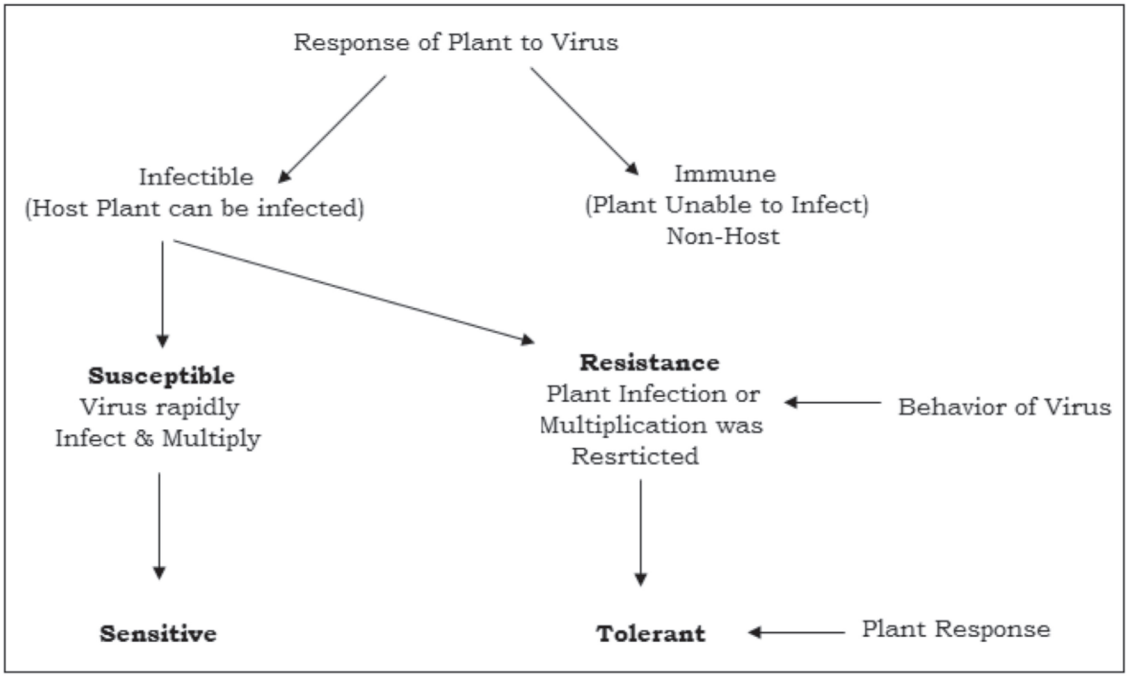

Fig. 1. Plant Response to the Viral Pathogen.

Table 2. Host plant and their resistance to viral pathogen

Host Plant	Resistance to Viral Pathogen
Alfalfa	Alfalfa Mosaic Virus
Cucumber	Cucumber Mosaic Virus
Potato	Potato Virus X
Tobacco	Tobacco Streak Virus Tobacco Etch Virus Tobacco Rattle VirusTobacco Mosaic Virus

with the helicase domain of TMV replicase (Kachroo *et al.*, 2006). This N gene mediated recognition of TMV responsible for the hypersensitive response development in the host, leads to the inhibition of viral activity (Marone *et al.*, 2013).

TIR-NBS-LRR protein is a subgroup of NBS-LRR. This Nucleotide Binding Site - Leucine Rich Repeats Protein is classified in to the two subgroups on the basis of N-terminal variations

1) TIR-NBS-LRR (Toll / Interleukin-1 Receptor - Nucleotide Binding Site - Leucine Rich Repeats)
2) CC-NBS-LRR (Coiled-Coil - Nucleotide Binding Site - Leucine Rich Repeats)

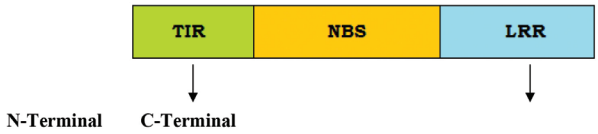

Fig. 2. TIR-NBS-LRR Protein

The NBS domains are involved in the accumulation of various plant hormones and signaling molecules like Jasmonic Acids, Salicylic acid and ethylene which are actively involved in the signaling transduction (Reina-Pinto and Yephremov, 2009).

Nucleic Acid Mediated Resistance

Viral defense mechanism by RNA Silencing

RNA silencing involved indirectly in the plant immunity mechanism against the pathogenic attack by double standard RNA. It referred as the suppression of specific sequence expression (Stavolone *et al.*, 2020).

RNA Silencing is a nucleotide sequence specific phenomenon which will resulting in to the degradation of Genetic Material or by inhibiting the translational or post translational procedure (Duan *et al.*, 2012).

The RNA Silencing pathway is consist of series of components:- dsRNA (Double Stranded RNA), processor or Dicer (Dicer like Protein) DCL, processor products: small RNA (siRNA and/or miRNA) which consist of 21-24 nucleotide sequence in length, Effecter Complex: RISC (RNA Inducing Silencing Complex) which consist of Argonaut (AGO) it is a key player in the destruction of genome (RISC is a sequence specific nuclease (Duan *et al.*, 2012).

Role of Signaling in the Resistance Induction

Systemic Acquired Resistance (SAR) is referred to be an unique term for the signal transduction pathway, which is an important ability of the plant to protect themselves against the pathogenic attack

After the construction of the infectible area the SAR pathway is activated which resulting in the formation of a specific systemic resistance

Biosynthesis and Involvement of Salicylic Acid during the plant viral interaction

Salicylic acid (SA) plays an important role in the signal transduction leads to be activation of SAR. Salicylic acid is responsible for the expression of many protein related genes, that are together known as PR protein (Pathogenic Related Protein) (Carr *et al.*, 2010) SA biosynthesis is started more strongly during lesions development. while the TMV induced hypersensitive response in tobacco; biosynthesis is initiated in the surrounding cells of infective center later on the entire infective cells also undergoes the synthesis of SA in response to SAR (Ryals *et al.*, 1996).

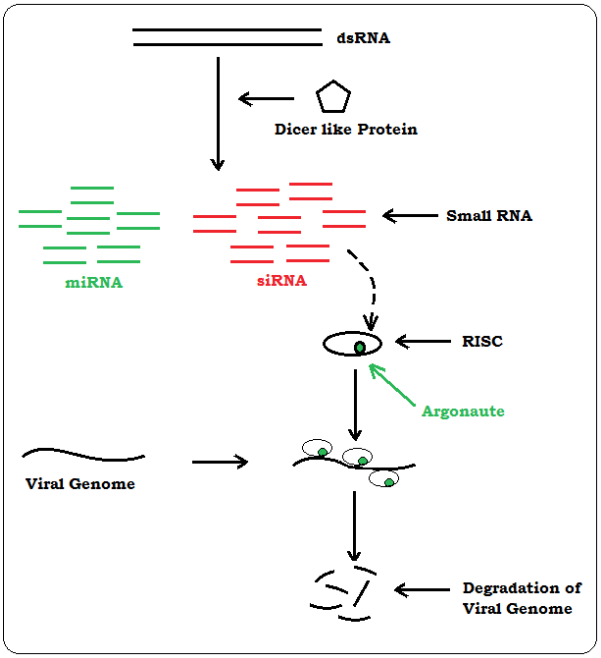

Fig. 3. RNA Silencing resulting in to the resistance to viral pathogen (Duan *et al.*, 2012)

The Synthesized SA is conjugated with the sugar molecules like glucose forming Salicylic acid-2-O-β-D-glucoside (SAG) which are stored in the vacuole. Kiessing suggested that SAG is the strongest form of the salicylic acid from which an active SA can be released in response to secondary infection expressing systemic acquired resistance in the plants (Carr *et al.,* 2010; Ryals *et al.,* 1996).

If the Salicylic Acid synthesis is suppressed by salicylic-degrading enzyme SA hydrolase (In nahG transgenic plant) or SA biosynthetic genes are blocked by a mutagenic agent the SA production will be decreased, resulting into the plants are highly susceptible for both virulent and a-virulent pathogens. Salicylic Acid is responsible for the maintenance of basal resistance and establishment of Systemic Acquired Resistance (Carr *et al.,* 2010).

Conclusion and Future prospectus

When a plant prone by any kind of pathogen a series of activities are induced to protect from the pathogenic attack. Mostly the activity of signaling molecules which will suppresses the growth and development of the pathogen

References

Carr, J.P., Lewsey, M.G. and Palukaitis, P. 2010. *Signaling in Induced Resistance*, Vol. 76. Elsevier Inc. 1st ed.

Duan, C.G., Wang, C.H. and Guo, H.S. 2012. Application of RNA silencing to plant disease resistance. *Silence*. 3(1) : 1.

Gachomo, E.W., Shonukan, O.O. and Kotchoni, S.O. 2003. The molecular initiation and subsequent acquisition of disease resistance in plants. *African J. Biotechnol.* 2(February):26–32

Kachroo, P., Chandra-Shekara, A.C. and Klessig, D.F. 2006. Plant Signal Transduction and Defense Against Viral Pathogens. *Adv. Virus Res.* 65(06) : 161-191.

Kang, B.C., Yeam, I. and Jahn, M.M. 2005. Genetics of Plant Virus Resistance. *Annu. Rev. Phytopathol.* 43(1) : 581–621.

Koh, K.W., Lu, H.C. and Chan, M.T. 2014. Virus resistance in orchids. *Plant Sci.* 228 : 26-38.

Mandadi, K.K. and Scholthof, K.B.G. 2013. Plant Immune Responses Against Viruses: How Does a Virus Cause Disease? *Plant Cell*. 25(5) : 1489–1505.

Marone, D., Russo, M.A., Laidò, G., De Leonardis, A.M. and Mastrangelo, A.M. 2013. Plant nucleotide binding site-leucine-rich repeat (NBS-LRR) genes: Active guardians in host defense responses. *Int. J. Mol. Sci.* 14(4) : 7302-7326.

Palukaitis, P. and Carr, J.P. 2008. Plant resistance responses to viruses. *J. Plant Pathol.* 90(2) : 153-171.

Reina-Pinto, J.J. and Yephremov, A. 2009. Surface lipids and plant defenses. *Plant Physiol. Biochem.* 47(6) : 540-549.

Román, B., Gómez, P., Picó, B. and Die, J. V. 2020. The NBS-LRR gene class is a small family in Cucurbita pepo

Ryals, J.A., Neuenschwander, U.H., Willits, M.G., Molina, A., Steiner, H.Y. and Hunt, M.D. 1996. Systemic Acquired Resistance. *Plant Cell*. 8 (10) : 1809-1819.

Siegel, A. and Zaitlin, M. 1964. Infection process in plant virus diseases. *Annu. Rev. Phytopathol.* 2(887) : 179–202.

Stavolone, L., Isabella, M. and Cillo, F. 2020. *Plant Viruses against RNA Silencing- Based Defenses/: Strategies And*. Elsevier Inc.

George, N. Agrios. 2005. *Plant Pathology*. Elsevier Academic Press