

INTERACTION OF PLANT VIRUSES IN MIXED INFECTION: A REVIEW

**B. Muhammad*¹, M.D. Alegbejo¹, A. Abdullahi¹, H. Badamasi², M.A. Ubale²,
A. Kabir.,¹ I. Yakubu¹ and B.D. Abdul-Hadi¹**

¹Department of Crop Protection, Faculty of Agriculture/Institute for Agricultural Research,
Ahmadu Bello University, PMB 1044, Zaria.

²Pest Management Technology Programme, Samaru College of Agriculture,
Division of Agricultural Colleges, Ahmadu Bello University, PMB 1044, Zaria.
abuidris24@gmail.com +2348034591888

ABSTRACT

Plant viruses are obligate parasites infecting specific plant hosts. They infect their hosts either singly or in combination, concurrently or at intervals. The latter condition is referred to as mixed or multiple infection and it is most common phenomenon in nature, where a single host is found to have many viruses infecting it at a point in time. The viruses in mixed infection phenomenon may be homogenous in nature, sharing similar characteristics in common; a situation that often results in antagonism between or among the viruses. On the other hand, synergism occurs when heterologous viruses with dissimilar traits infect a host plant. Plants defend themselves when infected by plant viruses by using pre-existing and induced structural and biochemical defence mechanisms such as hypersensitive response (HR) and Ribonucleic acid (RNA) silencing among others. The details of the afore mentioned highlighted items and insights for further research have been discussed.

INTRODUCTION

Plant viruses are obligate intracellular parasites that get into their hosts through wounds created by insects, man, mechanical injury between plants and so on. It is common for more than a virus to infect a host at a given period, a phenomenon referred to as mixed infection or multiple infection. (Syller, 2012). The mixed infection phenomenon does not stop at plant viruses but also extends to animal viruses, including humans (Waner, 1994; Lidsky *et al.*, 2009). The viruses may get into the host plant simultaneously or just a while after the invasion of the first one, the second one moves in, a phenomenon referred to as co-infection (Alegbejo and Nelson, 1982;1983a; 1983b;1983c). Super-infection on the other hand, is the situation when viruses or their strains infect a host at entirely different periods (Miralles *et al.*, 2001; Saldaña *et al.*, 2003). Plant viruses are generalists, having diverse forms which is due to the ease of recombination, error prone replication and mutation (Stobbe and Roossinck, 2016). These attributes make it easy for them to infect both

domestic and wild plants, with many plants having more than a virus at a time (Syller and Grupa, 2016). Many researches concentrate on the studies of a single virus infection in the earlier periods of plant virology; however, a substantial number of scientists have engaged in the study of behaviour of viruses in mixed infection which is a natural phenomenon (Alegbejo and Nelson, 1982;1983a;1983b;1983c; Syller, 2012). The interaction of viruses in mixed infection results in two major scenarios, antagonistic and synergistic interplay (Syller, 2012).

Virus-Host interaction

When a plant virus gets into a plant, after a wound is created by other agents, the plants respond in different ways based on their inherent nature. The different responses are: immunity, when the virus cannot replicate in the plant cell it first infected; resistance, in a situation that the virus multiplies in the initially infected cells or go beyond that to the subsequent cells, causing local necrotic lesions only; and susceptibility, the condition that virus

replicates and move systemically to most or parts of the host plant inducing visible symptoms or causing no symptoms (Agrios, 2005;; Hull, 2002; Green, 1991).

Virus-host relationship was also explained in terms of compatibility or non-compatibility with the host plant. In compatible interaction, there is manifestation of external symptoms, local or systemic symptoms as was found in *Tobacco mosaic virus* (TMV) infected-tobacco plants. It may lead to internal or micro symptoms, in which, the virus invades the host plant cell but unable to induce any visible symptoms but intracellular symptoms only, as is known with Potyviruses inclusion bodies. No symptoms (internal or external) are observed in virus-host incompatible relationship. This has been ascribed to the interaction of host resistant genes and the pathogen's gene (Gaur *et al.*, 2014; Takács *et al.*, 2014)

Plants in nature defend themselves from infection by a pathogen due to the interaction of their genes and that of the pathogens. Host plants devise so many mechanisms to defend themselves against the invasion of plant viruses based on the inherent character. A virus must cross numerous hurdles before establishing itself in the host it attacks, both preexisting structural and biochemical blockades (Agrios, 2005; Pallas and García, 2011).

Gene-for-gene model is among the strategies used by plants for defense, in which, the host resistant genes (*R*) compliment the avirulence (*avr*) gene of the virus to confer resistance to the plants. This leads to either local restrain of the pathogen in the initially infected cell, or systemic defense in the entire host plant, a process referred to as hypersensitive response (HR), which is a form of programmed cell death (Agrios, 2005; Hull, 2002; Syller and Grupa, 2016).

A virus synthesizes double-stranded RNA upon entering a plant cell with the aid of RNA dependent RNA polymerase (RdRP). The virus then moves from cell-to-cell through plasmodesmata, that is

short-distance and transported in the phloem in long-distance movements, in all cases by aid of virus movement protein (MP) (Syller and Grupa, 2016).

Plants defend themselves from the intruding virus(es) by means of RNA silencing, in which case the plant targets the viral RNA and degrade it, a process referred to as RNA interference (RNAi), which is a sequence-specific mechanism. The process begins basically by the action intracellular double-stranded RNA (dsRNA), which leads to down-regulation of genes expressing considerable sequence homologies with the virus dsRNA, the trigger (Gaur *et al.*, 2014; Syller and Grupa, 2016).

The RNA silencing involves the coalition of some protein molecules; Dicer-like protein (DCL), double-stranded RNA-binding proteins (DRBPs), and Argonaute proteins (AGOs), that come together in forming RNA-induced silencing complexes (RISCs), as well as enzymes; RdRp and RNA helicase (Pallas and García, 2011). Virus dsRNA induce DLS to produce virus-derived short interfering RNAs (vsiRNA), which are carried on the AGOs, thereafter turns on RISC complexes to degrade or prevent the translation of the single-stranded RNA of the invading virus (Syller and Grupa, 2016).

To evade the plant antiviral mechanism, viruses employ RNA silencing suppressor, thereby disrupting the processes involved in the RNA silencing; hence, finding it convenient to cause disease. A lot of damages are caused in the process of suppressing the plant's RNA silencing system, such as the disruption of the host's physiological system, decrease rate of photosynthesis, increase. or decrease rate of respiration, decrease in the quantity of growth hormone among others (Agrios, 2005; Pallas and García, 2011; Syller and Grupa, 2016).

Antagonistic relationship

Under antagonistic situation, one virus takes the advantage of the mixed infection and derives more benefit from the host plant, while the activity of the other one(s) is suppressed or drastically reduced.

Antagonistic interaction mostly results due to the infection of cognate viruses (Syller, 2012; Syller and Grupa, 2016). Antagonistic interaction in mixed infection takes two forms in manifesting itself; cross-protection or super-infection exclusion or homologous interference and mutual exclusion (Syller, 2012).

A phenomenon whereby a mild strain of a virus is used to inoculate a host plant with aim of preventing the invasion of the virulent strain of the virus is referred to as cross-protection. Farmers had used this method in controlling plant virus infection (Abel *et al.*, 1986). Cross-protection occurs naturally when an already established virus variants in a host plant prevents a second similar virus from invading the host or properly establishing itself as the former. In this case, the former virus is referred to as protecting while the latter is termed as challenging virus respectively (Syller, 2012). The protecting virus inhibits the symptom expression of the challenging one or masks its effect by employing various methods. The coat protein of the protecting virus may be expressed to disallow the challenging one decodes its gene. The challenging virus could not uncoat its RNA, more or less of causing disease symptoms on the host. Thus, the primary virus or the protecting virus provides resistance against the secondary or challenging virus (Sherwood and Fulton, 1982; Abel *et al.*, 1986). Antagonistic interaction is also observed when the protecting virus induce the RNA silencing machineries of the host, which leads to disruption of the challenging virus RNA, and hence averting its invasion into the host (Ratcliff *et al.*, 1997; Fagoaga *et al.*, 2006; Ratcliff, 1999). Cross protection has been employed over the years in the management of viral diseases in plants (Syller, 2012). An outstanding achievement in cross protection was recorded in the control of *Citrus tristeza virus* (CTV) using the attenuated strain; though the resistance was compromised when different virulent strain of the virus was found attacking the crop, suggesting that the protection is only achieved when the protecting and the challenging virus are similar strains. Super-infection exclusion only occurs at same or closely related strains of the viruses (Folimonova *et al.*,

2010). Cross protection is therefore limited to super-infection conditions. Mutual exclusion on the other hand occurs when there is co-infection of two or more viruses in a host. When the viruses get in, they all suppress each other or one another, thereby either inducing mild symptoms or completely masking the symptom expression and setting the host free of infection to the extent that no virus is detected after a while, but the actual mechanism of mutual exclusion remains terra incognita (Syller, 2012). Many researchers have worked on mutual exclusion studies; for example, Takeshita *et al.* (2004) has demonstrated the exclusion of two *Cucumber mosaic virus* (CMV) strains, when they co-infected Subgroup I and II strains of CMV. The exclusion was not only observed in the inoculated leaves but also the subsequent top leaves formed after inoculation, and it was sustained in serially inoculated plants.

Synergistic Interaction

When two or more heterologous plant viruses attack a host plant, the commonest relationship that exists between or among the viruses is synergism. Under this condition, the viruses multiply more and cause some conditions that a single virus would not be able to induce in terms of symptoms development and yield reduction (Syller, 2012).

A study conducted by Valverde *et al.* (2007) clearly showed how the viruses interact synergistically and aggravate the symptom severity and yield loss in sweet potatoes. The viruses involved were *Sweet potato chlorotic stunt virus* (SPCSV) and *Sweet potato feathery mottle virus* (SPFMV). Potyviruses are among the group of viruses with high synergistic relationship among themselves and with other plant virus genera. For instance, the interaction between *Potato virus X* (PVX) and *Potato virus Y* (PVY) resulted in an exponential symptom development and higher PVX titre in the infected tobacco plants, maintaining the PVY titre level at constant in both cases of single and coinfection (Vance, 1991; Syller, 2012). In another scenario, Cucumoviruses, CMV, was involved in synergistic interaction with PVY, worsening the disease severity and raising the virus titre. Tomato crops were found with lethal necrotic symptoms



because of mixed infection of an alien CMV variant in Italy, CMV-PG or CMV-Tfn and PVY (Mascia and Gallitelli, 2014). Hence, synergistic interaction among plant viruses could be intra- or inter-genus.

Another form of synergism is helper-dependence interaction between plant viruses, in which case one of the parties relies at some points in its life for survival on the other party. The virus that provides the assistance, is referred to as helper, while the beneficiary virus is called dependent virus (Syller, 2012). An excellent example of helper-dependent scenario is the interaction of Umbraviruses, which are not transmissible by vertebrate vectors and Luteoviruses that are aphid-transmissible. A luteovirus, *Groundnut rosette assistor virus* (GRAV) encapsidates the RNA of an Umbravirus, *Groundnut rosette virus* (GRV) and the GRV's satellite RNA, which completely depends on GRV for its multiplication, to cause a serious disease known as groundnut rosette disease (GRD) on

groundnuts (Anitha *et al.*, 2014). The GRV lacks expression of coat protein gene, hence, totally relies on the coat protein of GRAV for coating and subsequent transmission to other groundnut plants by aphids. The three components must be present for the complex of the disease to be established, even though GRAV and GRV are infectious in single infections. Dependent virus separate infection causes symptom to appear while helper virus only hardly causes any reasonable disease condition (Zhang *et al.*, 2000; Hull, 2002; Syller, 2012).

CONCLUSION

Plant virus mixed infection is a very important phenomenon that deserves due diligence any time viral disease detection is to be embarked upon for a crop. Independent studies should be carried out to determine the effects of viruses found in mixed infection on plants' yield, physiology and gene expressions using the modern techniques of molecular biology.

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