INTERNATIONAL JOURNAL OF AGRICULTURE & BIOLOGY
ISSN Print: 1560–8530; ISSN Online: 1814–9596
18–1824/2019/22–1–178–194
DOI: 10.17957/IJAB/15.1048
http://www.fspublishers.org

Review Article

Genetic Basis for Biotic Stress Resistance in Plants from Solanaceae Family: A Review

Ijaz Rasul[1†], Farheen Zafar[1†], Muhammad Amjad Ali[2], Habibullah Nadeem[1], Muhammad Hussnain Siddique[1], Muhammad Shahid[1], Usman Ali Ashfaq[1] and Farrukh Azeem[1,3*]

[1]*Department of Bioinformatics and Biotechnology, Government College University, Faisalabad, Pakistan*
[2]*Department of Plant Pathology, University of Agriculture Faisalabad, Pakistan*
[3]*Haikou Experimental Station, Chinese Academy of Tropical Agricultural Sciences (CATAS) - Hainan Key Laboratory of Banana Genetic Improvement, Haikou, Hainan Province, China*
[*]For correspondence: azeuaf@hotmail.com
[†] Contributed equally as first co-author

Abstract

Solanaceous crops are ranked among the most important crops of the world after the staple crops. However, these plants are under a constant threat of various pathogens. Disease management of plants is very important to minimize the damage and economic losses caused by plant pathogens. Various strategies are used to manage the diseases in solanaceous crops stretching from cultural practices to transgenic approaches. However, manipulation of host resistance is considered the most effective and economical strategy for controlling these diseases. Plants employ a number of host resistance approaches against the invading pathogens ranging from basal resistance to hypersensitive response. Numerous resistance genes have been identified and well characterized in Solanaceae species. Such genes have long been conferring resistance against notorious phyto-pathogens. This review gives a comprehensive description of various resistance mechanisms employed by plants to resist pathogens. The emphasis of this review is on the resistance genes that have been investigated in solanaceous plants. Furthermore, a number of known homologous resistance genes within the Solanaceae family have also been discussed. The review will be helpful in understanding the genetic knowledge of virulence in pathogens and resistance in plants. The knowledge of gene-for-gene interaction of plant-pathogen interaction at solanaceous crop interface will also aid in the effectual deployment of resistance genes in Solanaceae species in alternate forms. © 2019 Friends Science Publishers

Keywords: Solanaceae; Phyto-pathogens; Resistance genes; Host resistance; Homologous resistance

Introduction

Plant diseases cause severe threats for food security which needs to be addressed on preferential basis to allow a continuous accessibility to sufficient and nutritious food (Strange and Scott, 2005; Hameed *et al.*, 2016). In developing countries, the concept of food security is a major trepidation because of rapidly increasing populations. It has been estimated that over 800 million people around the world do not have access to sufficient food (Strange and Scott, 2005). The food shortage caused by the plant diseases is an important factor to be pondered about. It is believed that 20 to 40% of agricultural production is lost globally due to the direct yield losses caused by plant diseases, animals and weeds (Savary *et al.*, 2012; Bouwmeester *et al.*, 2016). Plants pathogens and pests not only damage crops but are also affecting native plants and trees (Mumford *et al.*, 2016).

Estimations suggest that the major part of food for human consumption is provided by 14 crop plants belonging to different families and genera (Strange and Scott, 2005). One of them is the Solanaceae family that comprises of the most important vegetable crops from genus *Solanum*. These solanaceous species not only fulfill the nutritional requirements but are also a source of drugs, ornamentals and medicines (Knapp *et al.*, 2004; Mueller *et al.*, 2005; Samuels, 2015; Yadav *et al.*, 2016). These crop plants are attacked by major groups of plant pathogens *viz*., viruses, bacteria, fungi, nematodes, oomycetes and parasites (Strange and Scott, 2005).

In this context, plant disease management seems very important to minimize the damage and financial loss caused by plant pathogens. General strategies to control plant diseases primarily rely on four methods: exclusion, eradication, protection and resistance. Exclusion is to prevent the entry of pathogens into the cropping area. Eradication is the removal of a pathogen after it enters into the planting area. Protection is to create a barrier between host plant and pathogen using chemicals like herbicides, insecticides or fungicides. Resistance is to enhance the ability of plant to withstand the plant diseases caused by pathogens. Resistance

To cite this paper: Rasul, I., F. Zafar, M.A. Ali, H. Nadeem, M.H. Siddique, M. Shahid, U.A. Ashfaq and F. Azeem, 2019. Genetic basis for biotic stress resistance in plants from Solanaceae family: A review. *Intl. J. Agric. Biol*., 22: 178–194

is considered as the most effective way of controlling disease and it will be illustrated in this review. Moreover, this article is focused on prevailing diseases in plants of Solanaceae family, the defense mechanisms and the resistance genes that have long been proved effective in conferring significant resistance against these diseases.

Importance of the Solanaceae Family

Solanaceae family belongs to the class angiosperms (flowering plants). It is considered as one of the most crucial flowering families for human beings, which consists of approximately 100 genera and more than 3500 species (Samuels, 2015). Eight genera are of significant importance, which contain more than 60% of the Solanaceae species (Yadav *et al.*, 2016). *Solanum* is the most diverse and largest genus with approximately 1330 species that constitutes 50% of the species of Solanaceae family. The remaining seven genera are Lycianthes with 200 species, Cestrum with 150 species, Nolana with 89 species, Physalis and Lycium with 85 species each and *Nicotiana* and *Brunfelsia* with 76 and 45 species, respectively (Yadav *et al.*, 2016). The diversity of *Solanum* genus has broadened its significance as it includes edible, medicinal and ornamental plant species (Table 1). Economically, the Solanaceae family is the 3rd most significant plant taxon, and is considered as the most variable and valuable in terms of agricultural utility and vegetable crops, respectively (Knapp *et al.*, 2004; Mueller *et al.*, 2005; Perveen and Qaiser, 2007; Samuels, 2015).

Immune Responses in Plants Against different Pathogens at Solanaceous Interface

Plants respond to pathogen attacks by activating different immune responses. The type of activated defense response depends on respective pathogen. The environment in which plants are evolving is rich in microorganisms (Glazebrook, 2005). A number of microorganisms can induce plant diseases (Ali *et al*., 2015). However, plants have developed extravagant and active defense mechanisms to avoid and minimize the loss occurring due to pathogenic infections. Plant disease resistance is very crucial to plants by providing effective and durable immunity to many plant pathogens (Soosaar *et al.*, 2005).

Generally the disease resistance in plants has been divided into two categories: non-host resistance and host resistance (Kang *et al.*, 2005). Many plant species show complete resistance against number of pathogens. The plant species that show resistance to all members of a given pathogen are referred as non-host plants and the conferred resistance is termed as non-host resistance (Nürnberger and Lipka, 2005). The resistance is evident when the pathogen is unable to cause disease upon contact with plant (Gururani *et al.*, 2012). The plant pathogens that are unable to cause any disease in non-host plants are termed as "heterologous pathogens". Such pathogens exhibit a phenomenon of "heterologous plant-microbe interaction". The non-host resistance comprises both constitutive physical and biochemical barriers and inducible reactions. Rigid cell wall, wax layers and antimicrobial secary metabolites on plant surface acts as constitutive barriers (physical and chemical barriers constitutively) for invading pathogens. If a pathogen succeeds in breaking these barriers, then the plasma membrane of plant cell aids in pathogen recognition and activates the inducible reactions in plant. The pathogen-associated molecular patterns (PAMP) recognition actually triggers the receptor-mediated defense mechanisms in non-host plants often regarded as PAMP triggered immunity (PTI) (Nürnberger and Lipka, 2005). Still, the heterologous plant-microbe interactions need various explanations. Hence, an improved understanding would prove to be an important breakthrough in the development of broad-spectrum disease resistance plants (Kang *et al.*, 2005).

Both host and non-host resistances are the result of plant innate-immune responses. However, extensive investigations have been carried out on host resistance compared to non-host resistance as the latter is comprised of multiple unidentified pathways (Gill *et al.*, 2015). Host resistance (also referred as genotypic resistance, specific resistance or cultivar resistance) can easily be studied genetically which is largely controlled by gene-for-gene resistance (Kang *et al*., 2005; Gill *et al*., 2015). Likewise in non-host resistance, basal defense is the first line of defense of host resistance to a range of plant pests and pathogens (Gururani *et al.*, 2012). The sec response (may be considered as first active response) involves the PAMP recognition that results in PTI also known as primary immune response (Nürnberger and Lipka, 2005; Chisholm *et al.*, 2006), the failure of which ultimately leads to the gene-for-gene interaction defense phenomenon mainly know as effector triggered immunity (ETI). In ETI, resistance (R) proteins from the plants interact with effector proteins from the pathogens. The pathogenic effector proteins suppress the primary immune response in plants lacking particular resistance genes. The R genes in plant either directly or indirectly monitor the presence of effector proteins, thus protect the plant against pathogens. This pair wise association of effector recognition by surveillance genes is termed as gene-for-gene interaction (Chisholm *et al.*, 2006; Jones *et al.*, 2014). Several plant immune responses that have proved effective in conferring resistance to plants under different situations have been discussed below.

Basal Resistance

Basal resistance is the first line of passive defense of both host and non-host resistance. This type of resistance includes: physical barriers (rigid cell wall, thick and waxy cuticles) and chemical barriers (phytoanticipins that acts as antimicrobial secary metabolite on the plant cell surface). These barriers help in restricting the penetration and establishment of pathogens on plant surfaces (Kaloshian,

2004; Nürnberger and Lipka, 2005; Kang *et al*., 2005; Chisholm *et al*., 2006; Gill *et al*., 2015).

In basal resistance, microbe-associated molecular patterns (MAMPs) or pathogen-associated molecular patterns (PAMPs) like chitin, lip polysaccharides, flagellins, glucans and plant cell wall derived components (*i.e*., results of enzyme hydrolytic activities) acts as elicitors for plants. These elicitors are secreted by invading pathogens. Basal defense can be triggered by pathogens/non-pathogens as both exhibit such molecular components in their cells. These elicitors are primarily involved in PTI as first line of defense in plants. However, basal resistance is compromised during the release of effector proteins by adapted microbes that results in the suppression of these defense responses (Gururani *et al.*, 2012).

R Gene-mediated Resistance Responses

Avirulence (*Avr*) or effector proteins are secreted by plant pathogens into the plant cell during initial infection. These effectors facilitate pathogen establishment on the plants by suppressing the plant defense mechanisms. However, plants have also modified their defense mechanisms by acquiring resistance proteins encoded by host resistance genes (R-genes) that confer significant resistance against particular pathogen races. These R proteins are involved in a gene-for-gene (*R-Avr*) interaction for recognizing plant pathogens (Collmer, 1998; Hammond-Kosack and Kanyuka, 2007; Mundt, 2014). Plant-pathogen interaction is a pairwise association in which the plant harboring R-genes confer surveillance against pathogen races carrying the corresponding *Avr* genes. *Avr* genes are found in each class of plant pathogens including viruses, bacteria, nematodes, fungus, and oomycetes, where these genes elicit a resistance response in the host plants. This resistance response is associated with another defense response termed as hypersensitive reaction (HR) (Gururani *et al*., 2012).

According to the guard hypothesis of gene-for-gene interaction, indirect interaction occurs between resistance protein (R-protein) and pathogen *Avr* effector protein. The *Avr* determinant of pathogen initially interacts with another host plant protein and causes conformational changes in the target host protein. The target host protein might be considered as a part of general plant defense. The R protein monitors the *Avr* protein, target host protein interaction and detect the conformational alteration in host protein caused by *Avr* determinant. This conformational alteration of the target host protein allows the R- protein to bind with *Avr*-host protein complex so that R-protein may trigger the resistance response against particular pathogen (Glazebrook, 2005; Gururani *et al.*, 2012). A prominent example of such interaction between *Arabidopsis thaliana* R-gene *RPS5* and *Pseudomonas syringae-Avr* gene *AvrPphB* provides the most convincing evidence to the support guard hypothesis (Kaloshian, 2004). However, plants devoid of R-genes are more susceptible to disease-causing phyto-pathogens as pathogen virulence determinants suppress the plant defense mechanisms in the absence of R genes (Glazebrook, 2005).

Systemic Acquired Response (SAR)

Systemic acquired response (SAR) has emerged as an important category of host resistance that provides durable resistance to diverse pathogens (Kang *et al.*, 2005). The phenomenon was observed in 1960 by Ross when it was found that the affected tobacco(*Nicotiana tabacum*) plants have developed resistance to secary infection of TMV in distal tissues (Durrant and Dong, 2004). The increased level of Salicylic acid (SA) in systemic leaves might be considered as a signal for the initiation of SAR. SA is then imported from infected leaves through phloem to other parts of plants and is being synthesized de novo. The conversion of SA to methyl salicylate (volatile compound) acts as a signaling factor and induces resistance in nearby healthy plants (Durrant and Dong, 2004; Glazebrook, 2005).

Increase in SA level is associated with the activation of defense related genes. Two of them namely *PAD4* and *EDS1* are responsible for SA accumulation in plant tissues. *PAD4* activates the defense reaction in response to SA signaling through the production of phytoalexins. However, two genes *i.e*., *EDS5* and *SID2* are needed for the biosynthesis of SA. *SID2* initiates SA production by isochorismate pathway while *EDS5* triggers the production of SA upon pathogen attack. Pathogen induced expression of *EDS5* requires *PAD4* and *EDS1* (Glazebrook, 2005). The accumulation of H_2O_2 in plant tissues is also responsible for the production of SA during HR (Hammond-Kosack and Jones, 1996). It is complicated to arrange SA signaling events in order as the pathway comprises many feedback loops. Cell death during HR response causes SA production, which accelerates cell death. In the same way, *EDS1* and *PAD4* genes are required for SA accumulation and on the other hand the SA accumulation also enhances the expression of these genes (Glazebrook, 2005).

Systemic acquired response is also associated with the activation of large number of pathogenesis-related genes (PRs) in both local and distal tissues (Ryals *et al.*, 1994). PRs are defined as plant proteins that are produced, more specifically, induced in pathological or related situations. These proteins were first described in 1970s by Loon in infected tobacco plants being unaware of their role as anti-pathogenic (Sugawara and Nikaido, 2014). Pathogenesis-related genes may be considered as stress response genes as their expression is inducible by various factors. Among these factors, pathogenic infections caused by viruses, bacteria, nematodes and fungi are dominant. PR proteins together form a set of pathogen-induced proteins that possess anti-pathogenic activities (Loon, 1997). These proteins are acidic in nature and have been recognized as chitinases and glucanases and are able to hydrolyze microbial cell constituents (Ryals *et al.*, 1994; Hammond-Kosack and Jones, 1996). However, a number of attempts

have been made to clone PR genes but they failed to enhance resistance by inducible glucanases and chitinases against fungal pathogens (Ryals *et al*., 1994).

Hypersensitive Response (HR)

A number of plant pathogens are biotrophs, which means these pathogens depend on living host tissues for their nutritional requirements (Govrin and Levine, 2000). Others are necrotrophs that fulfill their nutritional needs from dying or dead cells. Hemi-biotrophs are the pathogens that derive their nutrients from either living or dying cells and induce different defense mechanisms in plants (Glazebrook, 2005). The HR is one of the defense responses that act against biotrophic pathogens. The response is referred as one of the most important aspects in hampering growth of biotrophic pathogens by restricting their access to nutrients and water, which is accompanied by the localized and programmed plant cell death (Hiruma *et al*., 2013). During hypersensitive response, specific pathogen-attack recognition by gene-for-gene interaction, prompts the generation of reactive oxygen intermediates (ROIs) and causes the death of local cells. Generation of ROIs (Superoxide and Hydrogen peroxide) within hours of pathogen attack is considered an important component of HR defense mechanism (Govrin and Levine, 2000). Superoxide (O_2^-) and hydrogen peroxide (H_2O_2) are both moderately reactive in original form and cause cellular damage upon conversion into more reactive forms often known as "Reactive Oxygen Species (ROS). The O_2^- is converted into HO_2 (hydroperoxyl) radical upon protonation of O_2^- at low pH. The HO_2 is less polar as compared to O_2^-, therefore, crosses the biological membrane quite efficiently. It acts on fatty acids directly and converts linoleic, linolenic and arachidonic acids to lipid peroxides. Generation of O_2^- under optimum conditions causes cellular damage by affecting cell membranes and by producing signaling molecules in the form of lipid peroxides. H_2O_2 enters into cell cytoplasm in sufficient concentration and somehow manages to reach nucleus of either plant or pathogen where it is converted into OH^- upon reaction with intracellular metal ions, which causes self-perpetuating lipid peroxidation. In this way, HR response cause local damage to plants and pathogens and activates other defense mechanisms of plants against pathogen infection (Hammond-Kosack and Jones, 1996). In addition to lipid peroxidation, H_2O_2 acts as toxic substrate to microbes at specific levels in plants. H_2O_2 also initiates the synthesis of salicylic acid, which is an important signaling molecule of systemic acquired response (SAR) (Hammond-Kosack and Jones, 1996). However, sometimes, plant pathogens like nematodes are able to modulate the ROS pathway to develop compatible interaction with plants (Reviewed by Ali *et al*., 2015).

RNA Silencing Pathways

"RNA silencing" is another sophisticated genetic defense mechanism which provides resistance specifically against viral pathogens. Plants affected with viral infections grow new asymptomatic healthy leaves. These leaves show resistance to secary infection by the same or related pathogens (Liang *et al*., 2012). This phenomenon was previously known as "Recovery". Years of research then resolved the mystery of recovery mechanism and the phenomenon was named as RNA silencing mechanism (Baulcombe, 2004; Soosaar *et al*., 2005)

RNA silencing pathway is initiated when virus invades host plant and integrates its viral single stranded RNA (ssRNA). Virus genome encodes enzymes named "RNA-dependent RNA Polymerases" (RDRPs). These proteins facilitate the mechanism of RNA silencing (Wang *et al*., 2012). The RNA polymerase produces the antisense copies of viral genome thus helps in creating the double stranded viral RNA (dsRNA). Double stranded RNA is a key regulator in gene expression that initiates gene silencing collectively known as RNA interference of RNA silencing. The dsRNA is further processed by dicer into small RNAs (sRNAs) comprising 21-24 nucleotides. Dicer is an RNAase-III type endonuclease having the RNAase-III catalytic activity and double stranded RNA (dsRNA) binding site. In *Arabidopsis thaliana*, four types of dicer-like proteins are present, named DCL1-DCL4. Three of them are involved in processing dsRNAs from different sources. Double stranded RNA is processed by Dicer into small interfering RNA (siRNA). The siRNA contains ribonucleoprotein particles that make complex with the members of protein-like family "Argonaute (Ago)". This complex is referred as RISC complex (RNA induced silencing complex). While assembling into a complex, the duplex is unwound by special enzymes into single stranded form and bound directly and tightly to Argonaute protein in a complex (Baulcombe, 2004; Meister and Tuschl, 2004). The siRNA in the complex then guides the complex to carry out degradation of RNA and/or inhibition of translation (Wang *et al*., 2012).

Major Resistance Genes for different Phyto-pathogens in Solanaceous Species

Plants are a natural source of nutrition in an environment, where microorganisms are present everywhere (Glazebrook, 2005; Leonard *et al*., 2017). Plants serve as host of several phyto-pathogens to fulfill their nutritional requirements. In plant pathology, a disease caused by pathogenic microbe depends on host-pathogen interaction and environmental conditions that favor the disease development. The environmental conditions are regarded as a most important factor in favoring the plant-microbe relationship and developing diseases in plants. Even the most susceptible plants do not develop disease when exposed to a large population of pathogens under unfavorable conditions. The environmental factors such as pH, light, temperature, heavy metals, water deficit, soil texture and moisture content

control plant-microbe interaction (Leonard *et al.*, 2017). Solanaceae species are susceptible to a wide range of plant pathogens (Table 1). However, only major groups of these pathogens have been discussed here that are causing devastating damage to some of the globally important Solanaceae species.

R Gene Family in Crop Plants

Plant genome contains several classes of genes that provide resistance against particular pathogens. These pathogens are harmful to host plants because of their complex host-pathogen interaction at molecular level (Meyers *et al.*, 2005). The resistant genes provide protection against diverse plant pathogens in a given environment. To date, above 140 R-genes have been introgressed and well characterized. The most of these R-genes (approx. 80%) belong to a class of genes called "nucleotide binding-site and leucine rich repeats" (NBS-LRR) (Wei *et al.*, 2016). R-genes play a crucial role in plant defense against pathogens and provide resistance against particular races of pathogens. Different crops may contain specific resistant genes against specific pathogen strains/races. Such genes are often transformed into different plant species to induce resistance against specific plant pathogens. Almost 70 specific R-genes have already been reported (Collinge *et al.*, 2008).

According to the structural characteristics and amino acid compositions, R-gene encoding protein products have been classified into 8 major classes (Table 2) (Gururani *et al.*, 2012). Most prevalent classes of R proteins are cytoplasmic proteins that contain nucleotide-binding site (NBS) and leucine rich repeats (LRR). These proteins are often associated with variable N-terminus and sometimes C-terminus domains (Tameling and Takken, 2008). In angiosperms, the N terminus of NBS-LRR proteins contains three domains, namely Toll/Interleukin-1 receptor (TIR), Coiled-coil (CC), and Resistance to powdery mildew8 (RPW8). Therefore, NBS-LRR genes are divided into TIR-NBS-LRR (TNL), CC-NBS-LRR (CNL), and RPW8-NBSLRR (RNL) subclasses. TNL and CNL proteins are involved in the recognition of specific pathogens, whereas RNL proteins serve as helpers in downstream defense signal transduction (Shao *et al.*, 2019).

Plant Nucleotide Binding Site Leucine-Rich Repeat (NBS-LRR) proteins can thus be categorized into two major classes: CC-NBS-LRR (CNL) and TIR-NBS-LRR (TNL). TNL is further subdivided into TIR and non-TIR proteins (DeYoung and Innes, 2006; McHale *et al.*, 2006). The TNL proteins encode amino-terminus toll-interleukin-1 like receptor domain and the CNL proteins encode alpha-helical coiled-coil (CC) domain at its N-terminus. CNL proteins constitute the class one and TNL proteins comprise the class two of R-genes (DeYoung and Innes, 2006; Tameling and Takken, 2008; Gururani *et al.*, 2012). The members of the class NBS-LRR are found abundantly in plants, for example, in *Arabidopsis*, these genes comprise 1% of its genome. The proteins of third and fourth class contain extracellular LRR region (eLRR) attached to a transmembrane domain (TrD), the latter also contains a receptor like protein kinase that is an intracellular serine-theorine kinase (KIN) domain. The fifth class comprises of resistant proteins having putative extracellular LRR (eLRR) attached to transmembrane domain (TrD), a Pro-Glu-Ser-Thr (PEST) domain and short protein motifs i-e; endocytosis cell signaling (ECS) domain. PEST domain functions for degradation of proteins. Class six R-proteins contain transmembrane domain (TrD) associated with a coiled coil (CC) domain. R-proteins of class seven has emerged as a new member of TNL class with addition of nuclear localization signal (NLS) at carboxy terminus and a WRKY domain at amino terminus. Enzymatic R-genes devoid of NBS and LRR domains constitute the class eighth (Gururani *et al.*, 2012).

Plant resistance genes mostly encode NBS-LRR proteins with variable amino and carboxy-terminal domains. To date, most of the R-genes cloned in plants belong to class NBS-LRR. These proteins are associated with the detection of diverse pathogens through direct and indirect interaction between NBS-LRR proteins and pathogen-derived molecules. The resistance conferred by NBS-LRR has found to be effective against biotrophs and hemi-biotrophs only (McHale *et al.*, 2006; DeYoung and Innes, 2006).

***R-Avr* Gene Interaction**

Plants and pathogens exhibit complex relationships. A specific pathogen can only infect a specific host plant. Plant species (that is not infected by a microorganism) is referred as non-host to that particular pathogen. In plant-pathogen interaction where the pathogen confers disease to host plant, the pathogen is termed virulent, the interaction is named compatible and the host plant is described as susceptible (Soosaar *et al.*, 2005). Many resistant (R) and avirulence (*Avr*) genes have been identified in past years. The function of *Avr* genes is to induce virulence in host plant that lack specific R genes against particular virulent strain. Avirulence genes encode elicitors that act as specific signal molecules for plant resistant genes. The resistant genes of plants contain receptors for these elicitors. This recognition of elicitors by host plant R genes stimulates a cascade of host genes that leads to hypersensitive response, sometimes followed by systemic acquired response (Staskawicz *et al.*, 1995).

Plant Viruses and R-genes used against these Viruses in Solanaceous Plants

Viruses are among the most harmful and detrimental plant pathogens. Almost 450 species of plant pathogenic viruses have been reported that are significantly limiting the crop production worldwide (Soosaar *et al.*, 2005; Navas-Castillo *et al.*, 2011). The unavailability of antiviral products have forced us to use disease control strategies like eradication, protection and genetic resistance (Hanssen *et al.*, 2010; Imran *et al.*, 2013).

Table 1: Plant pathogens affecting Solanaceae family plants

Species	Genus	Host Range	Disease/ Symptoms	References
Viral pathogens and their solanaceae hosts				
Tomato chlorotic leaf distortion virus (TCLDV)	*Bipartite Begomovirus*	Tomato, Pepper	Leaf deformation and chlorotic mottle	Zambrano *et al.* (2011)
Tomato yellow margin leaf curl virus (TYMLCV)	*Begomovirus*	Tomato	- - - - - - - - - - -	
Tomato Venezuela virus (ToVEV)	Partially characterized *Begomovirus*		- - - - - - - - - - -	
Tomato rugose mosaic virus (ToRMV)	*Begomovirus*	Tomato	Golden mosaic and leaf distortion (rugosity)	Fernandes *et al.* (2006)
Potato yellow mosaic virus (PYMV)/ Tomato yellow Mosaic Virus (TYMV)		Tomato, Potato	Yellow Mosaics	Morales *et al.* (2001)
Tomato yellow leaf curl virus (TYLCV)	*Begomovirus*	Tomato	Minor yellowing of leaflet margins in apical leaves during early infections and then upward curling of leaflets occur.	Moriones and Navas-Castillo (2000)
Tomato torrado virus (ToTV)	*Torradovirus*	Tomato, Tobacco, Pepper, Eggplant	Burn-like symptoms has been seen on infected tomato leaves. Marked mosaics and stunting in pepper but no necrosis.	Verbeek *et al*. (2007); Amari *et al.* (2008)
Tobacco rattle virus (TRV)	*Tobravirus*	Tobacco, Potato	Necrosis, chlorosis, mottling.	Canto *et al.* (2004); Macfarlane (2010)
Pepper ringspot virus (PepRSV)		Pepper	Ringspot on leaves.	
Pepino mosaic virus (PepMV)	*Potexvirus*	Tomato, Pepino	Yellow leaf spots and mosaic on leaves, chlorosis, brown streaking on stems, spotty ripening of fruit.	Jones *et al*. (1980); Vlugt *et al*. (2000)
Potato virus X (PVX)		Potato, Pepper, Tomato	Tuber necrotic ringspot disease in potatao/Apical necrosis	Green and Kim (1991); Davino *et al.* (2017)
Tobacco mosaic virus (TMV)	*Tobamovirus*	Tobacco, Pepper	Mosaic and mottle	Green and Kim(1991); Scholthof (2004)
Tomato spotted wilt virus (TSWV)	*Tospovirus*	Pepper	Chlorosis, chlorotic spots on leaves	Tsompana *et al*. (2004); Zheng *et al.* (2010)
Tomato necrotic ringspot virus.		Pepper, Tomato	- - - - - - - - - - -	Chiemsombat *et al*. (2010)
Cucumber mosaic virus (CMV)	*Cucumovirus*	Pepper	Leaf deformation, yellow discoloration, mosaic, mottle and necrosis	Green and Kim (1991); Tsai *et al*. (2008)
Chilli veinal mottle virus (ChiVMV)	*Potyvirus*	Pepper	Dark green mottle, leaf distortion and reduced leaf size.	
Pepper veinal mottle virus (PVMV)			Leaf abscission, fruit distortion, vein chlorosis and mottle.	
Potato virus Y (PVY)	*Potyvirus*	Pepper, Potato, Tobacco	Tuber necrotic ringspot disease in potato/Yellowing, veinclearing, mottle, leaf distortion, stunted plant growth and necrosis.	Beczner *et al*. (1984); Green and Kim (1991); Heuvel *et al*. (1994); Solomon-Blackburn and Barker (2001)
Potato virus S (PVS), Potato virus M (PVM)	*Carlaviruses*	Pepper, Potato	Tuber necrotic ringspot disease in potato.	Green and Kim (1991)
Potato mop-top virus (PMTV)	*Pomovirus*	Potato	Brown arcs and circles in the tuber flesh.	Barker *et al.* (1998)
Bacterial pathogens and their solanaceae Hosts				
Ralstonia solanacearum	*Ralstonia*	Tomato, Potato, Tobacco, Eggplant	Wilt Disease	Nguyen and Ranamukhaarachchiv (2010); Kumar *et al.* (2017)
Pseudomonas syringae pv.	*Pseudomonas*	Tomato	Bacterial speck	Martin (1999)
Erwinia carotovora	*Erwinia*	Tomato, Potato	Bacterial soft rot disease	Reddy (2016)
Xanthomonas campestris pv. Vesicatoria	*Xanthomonas*	Pepper, Tomato	Bacterial spot disease	Liu *et al*. (2008); Potnis *et al*. (2015)
Xanthomonas axonopodis pv. vesicatoria.	*Xanthomonas*	Pepper	Bacterial spot disease	Romero *et al*. (2010)
Xanthomonas euvesicatoria			Bacterial leaf spot	Ivey *et al*. (2016)
Oomycete pathogens and their solanaceae hosts				
Phytophthora infestans	*Phytophthora*	Potato, Tomato,Hairy nightshade, Cutleaf nightshade, Bittersweet, Tobacco	Late blight	Zimnoch-Guzowska *et al.* (2003); Potter *et al*. (2011); Akhtar *et al*. (2012); Fisher *et al*. (2012; Mendonca *et al*. (2015)
Phytophthora capsici	*Phytophthora*	Pepper, Tomato	Phytophthora blight and fruit rot/root and crown necrosis	Ozgonen and Erkilic (2007); Mendonca *et al*. (2015)

Table 1: Continued

Fungal pathogens and their solanaceae hosts				
Alternaria solani		Tomato	Early blight	Panthee and Chen (2010)
Rhizopus stolonifer, Botrytis cinerea, Alternaria alternata		Cherry tomato	Black rot by *Alternaria alternate*	Wang *et al*. (2008)
Macrophomina phaseolina	*Macrophomina*	Eggplant	Root-rot disease	Ramezani (2008)
C. acutatum, C. capsici, C. gloeosporioides, C. coccodes	*Colletotrichum*	*Capsicum* spp.	Chilli anthracnose disease/sunken necrotic tissues	Than *et al*. (2008)
Fusarium oxysporum f. spp. *melongenae*		Eggplant	Vascular disease	Toppino *et al*. (2008)
Chalara elegans		Tobacco	Black root-rot/ black lesions on the roots, stunting and late maturation	Bai *et al*. (1995)
Nematodes and their solanaceae hosts				
M. incognita	*Meloidogyne*	Eggplant	Root-knot disease/stunted growth, root deformation and leaf discoloration	Khan *et al*. (2012)
M. javanica		Eggplant, Potato		Vovlas *et al*. (2005); Khan *et al*. (2012)
Meloidogyne species		Tomato		Nombela *et al*. (2003); Jones *et al*. (2013)
M. euphorbiae	*Macrosiphum*	Tomato		
G. rostochiensis	*Globodera*	Potato	Potato cyst	Back *et al*. (2006); Almeida-Engler and Favery (2011)
G. pallida				Jones and Lettice (2016)
N. aberrans	*Nacobbus*	Potato	Falsa root-knot/cavities and lessions inside root tissues	Almeida-Engler and Favery (2011); Jones *et al*. (2013); Reddy (2016)
P. neglectus	*Pratylenchus*		Root Lession	

Table 2: Major classes of resistance gene-encoding R-proteins and their domain arrangements

Class	Arrangement of functional domains	Examples	
		Host	R-gene
CNL		Tomato	*I2, Mi-1.2*
TNL		Tobacco, *Arabidopsis*	*N gene, RPP5*
eLRR-TrD		Tomato	*Cf-9, Cf-4, Cf-2*
eLRR-TrD-Kinase		Rice	*Xa21*
LRR-TrD-PEST-ECS		Tomato	*Ve1, Ve2*
eLRR-CC		*Arabidopsis*	*RPW8*
TIR-NBS-LRR-NLS-WRKY		*Arabidopsis*	*RRS1-R*
Enzymatic R-genes		Tomato	*Pto*

(Adapted from (Gururani *et al.*, 2012) NBS: Nucleotide Binding Site; LRR: Leucine Rich Repeats; TrD: Transmembrane Domain; TIR: Toll-interleukin-1 receptor; eLRR: extracellular Leucine Rich Repeats; PEST: Pro-Glu-Ser-Thr (amino acid domain); CC: Coiled Coil; NLS: Nuclear Localization Signal domain; WRKY: conserved amino acid sequence WRKYGQK

Table 3: Host resistance genes against viral pathogens

Host	Pathogen	R-gene	Location in genome	Avr-gene	References
Solanum peruvianum	*Tomato spotted wilt virus (TSWV)*	*Sw-5*	Long arm of chr 9	----	Roselló *et al*. (1998, 2001); Brommonschenkel *et al*. (2000); Spassova *et al*. (2001)
		Sw-6	----	----	
	Tomato Mosaic Virus (ToMV)	*Tm-2*	----	----	Lanfermeijer *et al*. (2005)
		Tm-2^2	----	----	Lanfermeijer *et al*. (2003)
	Tomato yellow leaf curl virus (TYLCV)	*Ty-5*	----	----	Verlaan *et al*. (2013)
Solanum habrochaites	*Tomato Mosaic Virus (ToMV)*	*Tm-1*	----	----	Ishibashi *et al.* (2007)
Solanum habrochaites	*Tomato yellow leaf curl virus (TYLCV)*	*Ty-2*	----	----	Verlaan *et al*. (2013)
Solanum chilense		*Ty-, Ty-3, Ty-4*	----	----	
Solanum tuberosum spp. *Stoloniferum*	*Potato Virus Y*	*Ry*$_{sto}$	Chr 11	----	
Solanum tuberosum sspp. *Andigena*		*Ry*$_{adg}$	Chr 11	----	
Solanum chacoense		*Ry*$_{chc}$	----	----	
Solanum tuberosum		*Y-1*	----	----	Vidal *et al.* (2002)
Solanum tuberosum	*Potato virus X*	*Rx1*	Top arm of chr 12	*Coat protein*	Querci *et al*. (1995); Jong *et al*. (1997)
		Rx2	Upper arm of chr 5		
		Ry	----	*Nla proteinase*	Mestre *et al.* (2000)
		Nx, Nb	Chr 5	*Coat protein*	
Solanum lycopersicon	*Tobacco mosaic virus*	*Tm-1, Tm-2, Tm-2a*	----	----	Whitham *et al*. (1994, 1996)
Solanum lycopersicum spp. *Hirsutum*	*Potato virus Y (PVY), Tobacco etch virus*	*pot-1*	----	*Vpg*	Moury *et al*. (2004)
Nicotiana glutinosa L.	*Tobacco Mosaic Virus (TMV)*	*Metallothionein-like gene*	----	----	Choi *et al.* (1996)
Nicotiana benthamiana	*Tobacco Mosaic Virus (TMV)*	*N-gene*	----	----	Whitham *et al*. (1994)
Capsicum chinense	*Tobacco Mosaic Virus (TMV)*	*L*^{3}	----	*Coat protein*	Berzal-Herranz *et al*. (1995)
Capsicum chacoense		*L*^{4}	----	----	Boukema (1980)
Capsicum annum	*Turnip Mosaic Virus*	*L1, L2, L3*	----	*Coat protein*	Cruz *et al*. (1997); Gururani *et al*. (2012)
	Potato virus Y	*pvr2-eIF4E*	----	----	Ruffel *et al*. (2005)

Table 4: Host resistance genes against bacterial pathogens

Host	Pathogen	R-gene	Location in genome	Avr-gene	References
Solanum pimpinellifolium	*Pseudomonas syringae*	*Prf*	----	*avr-Pto*	Martin *et al.* (1993)
Capsicum chacoense	*Xanthomonas campestris*	*Bs2*	----	*Avr-Bs2*	Kearney and Staskawicz (1990)
Lycopersicum esculentum	*Pseudomonas syringae pv tomato*	*Pto*	short arm of chr 5	*Avre-Pto, Avre-PtoB*	Ronald *et al.* (1992); Salmeron *et al.* (1996)
Capsicum spp.	*Pseudomonas syringae*	*Pflp gene*	----	----	Liau *et al*. (2003)

Table 5: Host resistance genes against fungal pathogens

Host	Pathogen	R-gene	Location in genome	Avr-gene	References
Solanum Peruvianum	*Cladosporium fulvum*	*Cf-2*	short arm of Chr 6	*Avr2*	Dixon *et al*. (1998)
Solanum hirsutum	*C. fulvum*	*Cf-4*	short arm of chr 1	*Avr4*	
Solanum Cerasiforme	*C. fulvum*	*Cf-5*	short arm of Chr 6	*Avr5*	
Solanum	*C. fulvum*	*Cf-9*	short arm of chr 1	*Avr9*	Jones *et al*. (1994)
Pimpinellifolium	*Fusarium oxysporum*	*I-2*	----	*Avr1*	
Solanum lycopersicum	*Alternaria alternata*	*Asc*	----	----	Ori *et al*. (1997); Simons *et al.* (1998)
(Lycopersicum	*Verticillium dahliae*	*Ve1*	Chr 9	*Ave1*	Brandwagt *et al*. (2000); Gururani *et al*.
esculentum)	*Verticillium albo-atrum*	*Ve1, Ve2*	----	*Ave1, Ave2*	(2012); Song *et al*. (2017)
Nicotiana benthamiana	*Colletotrichum destructivum*	*NbGSTU1, NbGSTU2, NbGSTU3, NbGSTF1,*	----	----	Dean *et al*. (2005)
Solanum torvum	*Verticillium dahliae*	*StoVe1*	----	----	

Among important Solanaceae crop plants, tomato (*S. lycopersicum*) is susceptible to many viruses which include: tomato chlorotic leaf distortion virus (TCLDV), tomato rugose mosaic virus (ToRMV), tomato yellow leaf curl virus (TYLCV), tomato mosaic virus (ToMV), pepino mosaic virus (PepMV) and the newly emerging tomato torrado virus (ToTV) (Hanssen *et al.*, 2010; Imran *et al.*, 2013). Within a decade of its identification, the *Pepino Mosaic Virus* has emerged as the major disease-causing virus in greenhouse tomato crops. Other newly emerging viruses include: *Torradovirus* (specie: tomato torrado virus), *Begomovirus* (specie: tomato yellow leaf curl virus), *Crinivirus* (specie: tomato infectious chlorosis virus) and *Tospovirus* (specie: tomato yellow ring virus) (Hanssen *et al.*, 2010). Potato (*S. tuberosum*) is also under a constant threat of plant viruses including potato virus Y (PVY), potato virus X (PVX), potato virus A (PVA), potato virus V (PVV), potato leaf roll virus (PLRV), potato virus S (PVS),

potato virus M (PVM) and potato mop-top virus (PMTV). Among these viruses, PVY belongs to the largest group of plant viruses (*Potyvirus*), which is the most harmful to cultivated potatoes. In recent years, the agricultural losses caused by PVY have surpassed the potato leaf roll virus (PLRV). In Pakistan, PLRV, PVX and PVY diseases are more common (Hameed *et al.,* 2014). According to an estimate, PVY strains may cause up to 70% yield loss globally (Naveed *et al.,* 2017). However, variable yield losses (40- 83%) of potato have been reported due to viral diseases in Pakistan (Ahmad *et al*., 2011; Hameed *et al*., 2014). Tobacco is a well-known for valued drug or medicine preparations. Nicotine derived from tobacco is used as an active ingredient for many purposes (Scholthof, 2004). This plant is also affected by viral pathogens where the most eminent pathogen is Tobacco Mosaic Virus (TMV). TMV was the first ever described plant virus and is known to cause severe losses to the tobacco industry (Koch *et al.*, 2016).

To date, a number of plant resistance genes have been reported in Solanaceae species that confer resistance against several plant pathogens upon gene-for-gene interactions. Plants are more susceptible to viruses compared to any other pathogen; hence, more efforts have been made to identify viral resistance genes (Table 3). There are examples of many functional R-genes in genus *Solanum* that provide resistance to diverse range of plant viruses and have been cloned in other species to trigger resistance against particular viral pathogens. One of those viral resistance genes is *Sw-5* that was the first dominant resistance gene reported against tomato spotted wilt virus (TSWV) in *S. peruvianum* (Stevens *et al.,* 1992). The *Sw-5* confers resistance against isolates of TSWV, tomato chlorotic spot virus (TCSV) and groundnut ring spot virus (GRSV) (Boiteux and Giordano, 1993). Owing to high expression in *S. lycopersicum* and wide protection conferred by *Sw-5.* Breeders have extensively used this gene to introgress into other crops. Another R-gene named '*Sw-6*' was also identified later in tomato but the resistance conferred by this gene was found lower than *Sw-5* (Roselló *et al.,* 2001).

Another example of resistance gene against viral pathogen is *N*-gene of *N. benthamiana*. The *N*-gene belongs to a TNL class of R-proteins and confers HR response in tobacco and tomato plants against TMV and ToMV respectively (Whitham *et al.*, 1994). Members of the class NB-LRR show resistance to a range of pathogens like viruses, fungi and bacteria. N-gene has been cloned in tomato and pepper (*Capsicum annuum*) cultivars and has found to delimit the movement of TMV in surrounding cells by effectively inducing HR in transgenic tomato plants (Whitham *et al.,* 1996). Although, wild tomato and pepper plants contain resistance genes that confer resistance to TMV isolates (*Tm-1, Tm-2, Tm-2*^{2} genes from tomato and *L1, L2, L3* and *L4* genes from pepper), but no gene has been found to be as effective as compared to *N*-gene (Whitham *et al.*, 1996). Plants show either extreme resistance (ER) or hypersensitive resistance (HR) to viral pathogens. Plants with ER provide resistance to several strains where even one gene can confer resistance against two or more viruses. For instance; *S. hougassi* R-gene Ry_{hou} confers resistance to potato virus Y and A and Ry_{sto} gene from *S. stoloniferum* provides resistance against potato virus Y, A and V (Cockerham, 1970). The HR is strain specific and the genes associated with HR include: R^5 from *S. stoloniferum* and *Ny* from potatothat provide resistance to PVY (Panthee and Chen, 2010; Mendonca *et al*., 2015). Plants with ER show no or limited symptoms when inoculated with viruses, however, plants with HR develop symptoms in the form of either local necrotic lesions or systemic necrosis (Solomon-Blackburn and Barker, 2001).

Phyto-pathogenic Bacteria and R-gene Interactions at Solanaceous Interface

A number of bacterial species have been reported as noxious plant pathogens (Table 1). Bacterial pathogens cause considerable yield losses (Kumar *et al.*, 2017). According to an estimate, at least 350 species of the genus *Xanthomonas* are pathogenic to the plants (Strange and Scott, 2005). *Ralstonia solanacearum*, is the sec most vital soil-borne bacterial pathogen with wide host range of almost 200 plant species over 50 families. Wilt disease caused by bacteria affects Solanaceae crops including tomato, potato, tobacco, pepper and eggplant (*S. aethiopicum*) (Table 1). To date, no chemical product has proved effective against *Ralstonia* wilt disease (Chen *et al.*, 2013). Other common bacterial diseases of Solanaceae species include bacterial leaf spot of *Capsicum annum* (bell pepper) caused by *Xanthomonas euvesicatoria*, bacterial soft rot by *Erwinia carotovora* spp. *Carotovora* on tomato and potato and bacterial spot of tomato and pepper by different *Xanthomonas* spp. (Potnis *et al.*, 2015). Similarly, various species of *Xanthomonas* cause leaf blights in cotton and rice as well.

Most of the bacterial pathogens (especially gram-negative strains) are known to inject virulence proteins into the host cell cytoplasm by type-III system (T3S) (Kay *et al.*, 2007; Gururani *et al*., 2012). One of them is the plant pathogenic gram-negative bacteria of *Xanthomonas*, *X. campestris* pv. *vesicatory* (*Xcv*), the causal agent of bacterial spot disease in pepper, tomato and other solanaceous crops. *Xcv* delivers its avirulence gene in the host plant *via* type-III system that elicits the resistance response in host by activating its resistance genes (Kay *et al.*, 2007). For instance, in pepper, resistance to *Xcv* is conferred by three, single loci, resistance genes *viz*., *Bs1, Bs2* and *Bs3* (Table 4). These genes are activated when the corresponding avirulence genes (*avrBs1, avrBs2, avrBs3*) of *XcV* enters the host cytosol. However, Bs2 gene has proved durable in the field and specifically recognizes different strains of *Xcv*. The gene has been cloned in tomato and pepper. Functional expression in transgenic tomatoes supported its use as a source of resistance in other Solanaceae species. The experiments on *Bs2* gene also show that the gene provides significant

resistance against the *Xcv* strains in the field (Tai *et al.*, 1999).

Another example of a genetic resistance to *Pseudomonas syringae* pv. *tomato* depends on two tomato resistance genes in tomato *viz.*, *Pto* and *Prf*. Both genes belong to two different classes of R-proteins. The former belongs to the 8th class of R-proteins encoding the protein kinase and the later contains the nucleotide binding site (NBS) and leucine rich repeat (LRR) region (Biezen and Jones, 1998). *Pseudomonas syringae* pv. *tomato AvrPto* avirulence gene interacts with *Pto* gene of tomato thus initiates the protein kinase cascade that in turn activates the plant defense mechanisms (Mucyn *et al*., 2006). Elimination mutations of *Prf* caused the loss of *Pto* resistance and a loss of organophosphate insecticide Fenthion sensitivity. However, when *Prf* is over expressed, it enhances the resistance against viral and bacterial pathogens but does not affect the *Avr-Pto* specified resistance. Moreover, it also improves the Fenthion sensitivity, hence controls both phenotypes (Oldroyd and Staskawicz, 1998). This role of *Prf* gene is because of its association with the host *Pto* kinase gene cluster. The gene exists in the middle of *Pto* gene cluster that is 500 bp from the Fen gene and 24 kb from the *Pto* gene. An overexpression of *Prf* mRNA leads to the activation of SAR independent of *Avr-Pto* and Fenthion. However, *Avr-Pto* and Fenthion elicitors recognition causes cell death and the induction of SAR (Salmeron *et al.*, 1996).

Fungi and Oomycetes-plant Interactions with Emphasis on R-genes In Solanaceae

Fungal diseases have long been known to cause widespread threats to food security around the globe. Nineteenth and 20th centuries faced disastrous fungal disease epidemics like late blight of Irish potato, dutch elm disease of trees and chestnut blight caused by *Phytophthora infestans*, *Phytophthora ramorum* and *Cryphonetrica parasitica*, respectively (Potter *et al.*, 2011; Fisher *et al.*, 2012). Late blight disease is very common in potatoes and is a serious disease that globally affects potato yields (Zimnoch-Guzowska *et al.*, 2003). Similarly, late blight is also one of the most important and common diseases of tomato (Mendonca *et al.*, 2015). This disease mainly spreads during cold, rainy and foggy weather. It is called late blight because it is very difficult to detect the early infections and, when it is detected, it is often too late to protect the crops *via* chemical fungicides and other means (Panthee and Chen, 2010). Another fungal disease of tomato caused by necrophytic *Alternaria solani* is "Early blight". The pathogen attacks the cultivated tomatoes during extreme cold conditions, high moisture, heavy dew and frequent rain (Panthee and Chen, 2010). Other different fungal diseases of Solanaceous crops and respective pathogens have been discussed in Table 1.

Many fungal resistance genes have been reported so far that confer great resistance to the various fungal pathogens. For example, *Cf-9* from *S. pimpinellifolium* (Jones *et al.*, 1994)**,** *Cf-2* from *S. peruvianum* (Dixon *et al*., 1998)**,** *Cf-4* from *S. habrochaites* (Thomas *et al*., 1997) and *Cf-5* from *S. cerasiforme* (Dixon *et al.*, 1998) provide strong resistance during interactions between different species of *Lycopersicon* and fungus *Cladosporium fulvum*. This pathogen contains avirulence genes (shown in Table 5) that induce a HR response in tomato plants harboring particular resistance genes (Jones *et al.*, 1994).

Similarly, another prominent example of fungal resistance gene is *Verticillium* wilt resistance gene *Ve* from tomato. It confers resistance against *Verticillium dahliae* race 1. Among the most important cultivated plants of Solanaceae family, the tomato, potato and eggplant serve as a host for *Verticillium* spp. The *Ve* gene is a single dominant resistance gene against *Verticillium dahliae* (Diwan *et al*., 1999). Many studies have been carried out on Ve locus and it was found to consist of two closely linked and inverted genes *viz*., *Ve1* and *Ve2* in tomato (Liu *et al.*, 2012; Song *et al.*, 2017), but only *Ve1* was found to confer resistance in tomato against race 1 of *Verticillium dahliae* and *Verticilliumalbo-atrum*. Initially, it was mapped on different locations. Then in 1993, the exact location of *Ve* gene was found by Zamir and his colleagues and the gene was mapped on short arm of chromosome no 9. The location of *Ve* is tightly linked with RFLP marker GP39 on the genome of tomato (Diwan *et al*., 1999). So far, *Ve* gene has been cloned in many Solanaceae species like *N. glutinosa* (tobacco), potato and *S. torvum* (wild eggplant) and has found to confer effective resistance in transgenic plants against *Verticillium* spp.

Like fungi, pathogenic oomycetes are also responsible for economic losses of important Solanaceae crops (Gururani *et al.*, 2012). One of the most devastating oomycete pathogens, *Phytophthora infestans* has long been known to cause worldwide yield losses of potato. This pathogenic oomycete is rapidly emerging as more virulent and pesticide resistant (Haverkort *et al*., 2009). The pathogen also causes late blight disease in many other important species of Solanaceae family like tomato and tobacco (Vossen *et al.*, 2005). A number of resistance genes have been identified in potato against *P. infestans*. Eleven resistance genes (9 of which are given in Table 6) have been found in *S. demissum* and *S. stoloniferum* (Colon and Budding, 1988). All of these R-genes were found race specific by conferring hypersensitive resistance (HR) against different races of the fungus. Therefore, when these R-genes were introgressed in potato cultivars, these genes were unable to confer resistance against all *P. infestans* races and hence, were attacked by more virulent races of pathogens. The resistance conferred by these race-specific R-genes was observed to be short-lived (Song *et al.*, 2003). Gene stacking of these genes could be a way forward for durable and multigenic resistance against *P. infestans* in potato.

Helgeson *et al.* (1998) identified resistance gene in wild potato specie, *S. bulbocastanum* against late blight. This specie showed great resistance against all races of *P. infestans.* After the successful examination of the late blight

Table 6: Host resistance genes against Oomycetes

Host	Pathogen	R-gene	Location in genome	Avr-gene	References
Solanum demissum	*Phytophthora infestans*	*R1*	chr 5	----	Jo *et al*. (2015); Vossen *et al*. (2016)
		R2	chr 4	----	
		R3a, R3b, R4, R6, R7	chr 11	*Avr3a of R3a*	
		R8	long arm of chr 9	*Avr 8*	
		Rpi-abpt1	----	----	
Solanum bulbocastanum	*P. infestans*	*Rpi-blb1*	chr 8	----	van der Vossen *et al*. (2003)
		Rpi-blb2	chr 6	----	van der Vossen *et al*. (2003)
		Rpi-blb3	chr 4	----	
		Ipio, Ipib, Ipieo4	----	*RB*	Vossen *et al*. (2005); Golas *et al*. (2010)
Solanum dulcamara	*P. infestans*	*Rpi-dlc1*	long arm of chr 9	----	Gururani *et al*. (2012)
Solanum pinnatisectum	*P. infestans*	*Rpi1*	chr 7	----	Wang *et al*. (2014)
Solanum mochiquense	*P. infestans*	*Rpi-mcq1*	long arm of chr 9	----	Golas *et al*. (2010); Kuhl *et al*. (2001); Ruffel *et al*. (2005); Sliwka *et al*. (2006);
Solanum phureja	*P. infestans*	*Rpi-phu1*	long arm of chr 9	----	Foster *et al*. (2009); Pel *et al*. (2009)
Solanum venturii	*P. infestans*	*Rpi-vnt1.1 Rpi-vnt1.2 Rpi-vnt1.3*	chr 9	----	

Table 7: Host resistance genes against nematode pathogens

Host	Pathogen	R-gene	Location in genome	Avr-gene	References
Solanum peruvianum	*Meloidogyne* spp.	*Mi-1.2*	----	----	Milligan *et al*. (1998); Rossi *et al*. (1998); Vos *et al*. (1998); Nombela *et al*. (2003)
Solanum pimpinellifolium	*Globodera* spp.	*Hero*	----	----	Ernst *et al*. (2002)
Solanum tuberosum	*Globodera pallida*	*Gpa2*	chr 12	----	van der Vossen *et al*. (2000); Gebhardt and Valkonen (2001)
Solanum tuberosum	*Globodera rostochiensis*	*Gro1-4*	----	----	Paal *et al.* (2004)
Solanum spegazzinnii	*Globodera rostochiensis*	*Gro1*	Chr 7		Ernst *et al*. (2002)
Solanum tuberosum spp. *andigena*		*H1*	Chr 5	----	
Capsicum annum	*Meloidogyne incognita, Meloidogyne arenaria, Meloidogyne javanica*	*Me1, Me3, Me7, N*	chr 9	----	Barbary *et al*. (2014); Celik *et al*. (2016)
		CaMi	----	----	

Table 8: Homologous resistance genes in *solanaceae* species

R-genes			Homologous R-genes			References
Host	R-gene	Pathogen	Host	R-gene	Pathogen	
Solanum tuberosum	*Gro1-4*	*Globodera rostochiensis*	*Solanum lycopersicum*	*Hero*	*Globodera rostochiensis*	Paal *et al*. (2004)
Solanum tuberosum	*Y-1*	*Potato Virus Y (PVY)*	*Nicotiana benthimiana*	*N-gene*	*Tobacco Mosaic Virus (TMV)*	Vidal *et al*. (2002)
Solanum tuberosum	*Gpa2*	*Globodera pallida*	*Solanum tuberosum*	*Rx1*	*Potato virus X*	van der Vossen *et al*. (2000)
Solanum venturii	*Rpi-vnt1.1*	*Phytophthora infestans*	*Solanum lycopersicum*	*Tm-2*^{2}	*Tomato Mosaic Virus*	Foster *et al*. (2009)
Solanum lycopersicum	*Ve1*	*Verticillium dahliae*	*S. lycopersicoides*	*SlVe1*	*Verticillium dahliae*	Chai *et al*. (2003)
			S. tuberosum	*StVe1*		Simko *et al*. (2004)
			S. torvum	*StVe, StoVe1*		Fei *et al*. (2000); Liu *et al*. (2012)
Solanum peruvianum	*Sw-5*	*Tomato spotted wilt virus*	*Solanum demissum*	*R8*	*Phytophthora infestans*	Vossen *et al*. (2016)
			Solanum peruvianum	*Mi*	*Meloidogyne* spp.	Brommonschenkel *et al*. (2000)
Solanum tuberosum	*StBCE2*	*Phytophthora infestans*	*Nicotiana benthamiana*	*NbBCE2*	*Phytophthora infestans*	Wang *et al*. (2014)
Lycopersicum esculentum	*Ve1*	*Verticillium dahliae*	*Nicotiana glutinosa*	*Ve1 (ortholog)*	*Verticillium dahliae*	Zhang *et al.* (2013)
Solanum peruvianum	*Mi-1*	*Meloidogyne* spp. *Bemisia tabaci*	*Solanum bulbocastanum*	*Rpi-blb2*	*Phytophthora infestans*	Vossen *et al*. (2005)
Capsicum annum	*pvr2-eIF4E*	*Potyvirus*	*Solanum lycopersicum*	*pot-1 (ortholog)*	*Potyvirus*	Ruffel *et al*. (2005)

resistance, somatic hybrids retaining the high resistance of wild *S. bulbocastanum* were produced, as it was extremely difficult to cross the wild specie with potato directly. Thus, through somatic hybridization, the resistance was transferred into potato breeding lines (Helgeson *et al.*, 1998). Many Solanaceous species harboring resistance genes are known to display resistance to *P. infestans* (Table 6). *S. dulcamara* (bitter sweet) has long been known as a host of *P. infestans.* Under conditions highly favorable for disease, *S. dulcamara* hardly gets infected with late blight. The specie shows a low level of genetic variation thus, easy to cross with other cultivar species. Golas and his colleagues (2010) conducted a study on *S. dulcamara* and identified the first resistance gene, *Rpi-dlc 1*on long arm of chromosome no 9 against *P. infestans.* The resistance was conferred by a single locus. In addition, one population was tested for broadness of resistance that indicated the presence of other *Rpi* genes in *S. dulcamara* (Golas *et al.*, 2010).

Plant-nematodes Interactions at R-gene Level Resistance in Solanaceae Faimly

Plant-parasitic pathogens have long been known to cause diseases in many important vegetable crops aided by pathogens like fungi, oomycetes, aphids and nematodes (Back *et al.*, 2002). Over 4300 species of nematodes are parasitic to plants causing serious crop losses worldwide (Jones *et al*., 2013; Ali *et al*., 2015). The most economically crucial nematodes belong to *Meloidogyne, Heterodera, Globodera, Rotylenchulus, Aphelenchoides, Pratylenchus* and *Radopholus* genera (Jones *et al*., 2013; Reddy, 2016). Among these, the root-knot nematodes with in genus *Meloidogyne* are causing severe crop losses. Approximately 25% yield losses have been estimated due to invasions of root-knot nematodes in main crops around the globe (Reddy, 2016). The genus *Meloidogyne* includes 98 species and four of them being *M. incognita*, *M. javanica*, *M. arenaria* and *M. hapla* cause major yield losses in solaneceous crops (Jones *et al.*, 2013).Recently, *M. graminicola* has also been shown to cause yield losses up to 20% in rice (Jabbar *et al*., 2015).

Meloidogyne incognita is the causal agent of root-knot disease in several solaneceous vegetable crops like tomato, potato, pepper and eggplant. The pathogen attack, in addition to severe gall formation on the root systems, results in chlorosis and stunting that lead to extreme yield losses in these crops (Abolusoro *et al*., 2015). Similarly, species belonging to genera *Heterodera* and *Globodera* are also causing great economic losses to potatoes, cereals and soybean (Reviewed by Ali *et al*., 2015). Estimations suggest that potato cyst nematodes (*G. rostochiensis and G. pallida*) lead to 9% losses of potato production throughout the world (Jones *et al.*, 2013).

Several R genes have been identified against both *Meloidogyne* spp. and *Globodera* spp. (Table 7). We have recently reviewed the implications of R genes from various crops species during plant nematode interactions (Ali *et al*., 2015) Resistance to *Meloidogyne* spp. was first identified in *S. peruvianum* which was named as *Mi-1* gene that confers resistance to only 3 species of genus *Meloidogyne* namely, *M. incognita, M. arenaria, M. javanica*. The predicted protein structure of *Mi* gene encodes NB site and LRR region with CC domain at its N-terminus, thus belonging to the CNL class of R-proteins. The gene was observed to confer hypersensitive response against root-knot nematodes with localized necrotic lesions at the site of infection upon gene-for-gene interaction (Milligan *et al.*, 1998; Rossi *et al.*, 1998; Gururani *et al.*, 2012). Another example of root-cyst nematode resistance gene is *CaMi* from *C. annum*. The gene appeared to be highly expressed in flowers, leaves and roots. Like *Mi-1* gene of tomato, *CaMi* also initiates a hypersensitive response in transgenic plants and show durable resistance to root-knot nematodes (Chen *et al.*, 2007).

Many resistance genes have also been genetically characterized within *Solanum* species against potato cyst nematodes. Two species of genus *Globodera* named, *G. rostochiensis and G. pallida* have long been known to affect the potato yield. It incited the scientists to identify potato cyst resistance genes in wild potato species so that to induce resistance in potato cultivars. One of the genes termed '*H1'* was later identified in *S. tuberosum* spp. *Andigena*. The dominant gene was found on chromosome no 5 of wild potato specie conferring resistance to *G. rostochiensis* pathotypes Ro_1 and Ro_4. The gene was widely used in commercial potatoes to confer significant level of durable resistance against *Globodera* spp. Similarly, another R-gene *Gro1* from *S. spegazzinii* confers nematode resistance against *G. rostochiensis* pathotypes $Ro_{1\text{-}5}$ (Ernst *et al.*, 2002). Moreover, R-gene, *HeroA* from tomato was transformed into potato for enhancement of resistance against potato cyst nematodes, *G. pallida* and *G. rostochiensis*. The transgeninc expression of this gene led to hypersensitive response after the initiation of syncytia, which become abnormal and necrotic due to degeneration of surrounding cells (Sobczak *et al*., 2005). This suggests that R-genes and their products are one of the key strategies to be used for development of resistant solanaceous crop plants (Reviewed by Ali *et al*., 2015).

Homologous Resistance Genes

Many species in Solanaceae family have been found to contain resistance genes that show considerable homology with the resistance genes of the same or other specie members (Table 8). In plants, majority of R-genes exist as members of tightly linked gene clusters. Uneven recombination between members of the cluster results in partial deletion or duplication events or the formation of novel chimeric genes (Vossen *et al.*, 2000). The duplication and speciation events lead to two types of homologous genes designated as paralogues and orthologues, respectively. Orthologous genes are presumed to describe the similarities and differences in genome sequences of different species in a most accurate way as they trace back to an ancestral gene shared by two different species (Gabaldón and Koonin, 2013). Moreover, orthologous genes are considered to show equivalent functions that is the imprecise perspective as in some cases greater than expected differences between orthologues have been seen. However, orthologous genes appear to be functionally more similar relative to paralogues by a narrow margin (Gabaldón and Koonin, 2013).

An example of an orthologous gene exhibiting the similar function is the *Ve1* resistance gene that confer resistance to fungus *Verticillium dahliae* in both tomato and tobacco (Zhang *et al*., 2013). Tomato and tobacco belong to different genera (*Solanum* and *Nicotiana*, respectively); yet they are close relatives for the same gene family. The two species share a high degree of (coding) sequence homology. The *Ve1* R-gene was first reported in tomato plant. Later on, during the agro-infiltration of *N. glutinosa* with the *Ave1* gene revealed the presence of functional *Ve1* ortholog in it.

During the investigation, when *Ave1* gene of *Verticillium dahliae* was inserted in *N. glutinosa*, the avirulence gene induced HR response while no such response was seen upon infiltration of *Ve1* gene. This analysis suggested the presence of endogenous functional *Ve1* orthologous gene in *N. glutinosa* conferring resistance to race 1 *V. dahliae* (Zhang *et al.*, 2013).

Whole genome duplication triggers the evolutionary novelties (Harikrishnan *et al.*, 2015). The duplication event is the major contributor of specie divergence. Duplication events within a specie assigns new functions to duplicated genes also referred as paralogs and one such example is *Sw-5* gene in *S. peruvianum*. *Sw-5* is a member of multigene family and confers multiple resistances. The RFLP aided mapping indicated that the majority of *Sw-5* homologous were located on chromosome 9. *Sw-5.1-4* map on the telomeric region, *Sw-5.5* near centromeric region and *Sw-5.6* map down the long arm of chromosome 9. The *Gpa6* gene (confer resistance to *Globodera pallida*) is present in proximity of *Sw-5.1-3* genes. Similarly, the loci of *Sw-5.5* and *Sw-5.6* genes were mapped near an R gene (*Tm2*^{2}). On chromosome 12, the *SW-5.7* gene locus is present near the *Lv* gene (for powdery mildew disease resistance). Thus, the paralog members of *Sw-5* are dispersed in tomato genome at the sites where several virus, fungus and nematode resistance genes have been mapped (Brommonschenkel *et al.*, 2000). Similarly, Gpa-2 R-gene from potato was overexpressed in the same plant species to infer resistance against *G. pallida*. This resulted in the development of stagnated and translucent female nematodes on transformed plant roots (Vossen *et al*., 2000). To date, several resistance genes and their homologs, orthologs and paralogs have been found in different Solanaceae species that are conferring resistance to a broad range of pathogens (Table 8).

Conclusion and Future Prospects

Solanaceous crops are amongst the major crops of the world that are fulfilling the nutritional requirements of human beings since very long. A huge number of pathogens including viruses, bacteria, fungi, oomycetes and nematodes attack this family. These phyto-pathogens are provoking serious threats to the global food security. Practical applications of commercially synthesized antimicrobial drugs and chemical pathocides are losing their effectiveness as the diverse range of pathogens has developed resistance to such toxic chemicals. The pests and pathogens have evolved new mechanisms of inducing diseases in plants by delivering their avirulence genes in plants. On the other hand, during the coevolution of plant-microbe interactions, plants also acquired the resistance genes that have long been providing effective and durable resistance to the devastating diseases induced by the pathogens. A single plant resistance gene reacts to a particular avirulence gene. However, Solanaceae species also exhibit homologous resistance genes that are conferring resistance to the same pathogen isolates or to the different pathogens.

Due to the significant resistance conferred by resistance genes in plants, breeders and biotechnologists are using these genes. These have been cloned in susceptible species of plants and successful transgenic species have been obtained. However, many isolates of phyto-pathogens have evolved mechanisms to resist the effects of resistance genes. Therefore, more research is needed to find the durable and robust resistance genes. It will be useful to understand the underlying mechanisms that many pathogens have evolved to combat plant R genes. A rapid increase in genome sequencing projects is producing vast amount of raw information, which is being stored as databases. Based on novel techniques of bioinformatics and omics, databases have become important tools for genome-wide identification and prediction of new genes. The application of novel methods for the development of disease resistance genes databases could be crucial to gather more information for such gene families and orthologous genes in plants. Moreover, a critical focus should be on the understanding of molecular gene regulatory mechanisms for disease resistance genes. It will be helpful for the integration of a sustainable disease resistance in plants.

References

Abolusoro, S.A., M.O. Abe, P.F. Abolusoro and N.B. Izuogu, 2015. Control of nematode disease of eggplant (*Solanum aethiopicum*) using manure. *Arch. Phytopathol. Plant Prot.*, 48: 188–193

Ahmad, N., M.A. Khan, S. Ali, N.A. Khan, R. Binyamin and A.F. Sandhu, 2011. Epidemiological studies and management of potato germplasm against PVX and PVY. *Pak. J. Phytopathol*., 23: 159-165

Akhtar, K. P., M.Y. Saleem, M. Asghar, S. Ali, N. Sarwar and M.T. Elahi, 2012. Resistance of solanum species to *Phytophthora infestans* evaluated in the detached-leaf and whole-plant assays. *Pak. J. Bot.*, 44: 1141–1146

Ali, M.A., A. Abbas, F. Azeem, N. Javed and H. Bohlmann, 2015. Plant-nematode interactions: From genomics to metabolomics. *Intl. J. Agric. Biol.*, 17: 1071–1082

Almeida-Engler, J. and B. Favery, 2011. *Genomics and molecular genetics of plant-nematode interactions*. Springer Netherlands, Dordrecht.

Amari, K., D. Gonzalez-Ibeas, P. Gomez, R.N. Sempere, M.A. Sanchez-Pina, M.A. Aranda, J.A. Diaz-Pendon, J. Navas-Castillo, E. Moriones, J. Blanca, M.D. Hernandez-Gallardo and G. Anastasio, 2008. Tomato torrado virus is transmitted by *Bemisia tabaci* and infects pepper and eggplant in addition to tomato. *Plant Dis.*, 92: 1-6

Back, M.A., P.P.J. Haydock and P. Jenkinson, 2002. Disease complexes involving plant parasitic nematodes and soilborne pathogens. *Plant Pathol.*, 51: 683–697

Back, M., P. Haydock and P. Jenkinson, 2006. Interactions between the potato cyst nematode *Globodera rostochiensis* and diseases caused by *Rhizoctonia solani* AG3 in potatoes under field conditions. *Eur. J. Plant Pathol,*. 114: 215–223

Bai, D., R. Reeleder and J.E. Brandie, 1995. Identification of two RAPD markers tightly linked with the *Nicotiana debneyi* gene for resistance to black root rot of tobacco. *Theor. Appl. Genet*., 91: 1184–1189

Barbary, A., A. Palloix, A. Fazari, N. Marteu, P. Castagnone-Sereno and C. Djian-Caporalino, 2014. The plant genetic background affects the efficiency of the pepper major nematode resistance genes *Me1* and *Me3*. *Theor. Appl. Genet*., 127: 499–507

Barker, H., B. Reavy and K.D. McGeachy, 1998. High level of resistance in potato to potato mop-top virus induced by transformation with the coat protein gene. *Eur. J. Plant Pathol*., 104: 737–740

Baulcombe, D., 2004. RNA silencing in plants. *Nature*, 431: 356–363

Beczner, I., L., Horvath, J. Romhanyi and H. Forster, 1984. Studies on the etiology of tuber necrotic ringspot disease in potato. *Potato Res.*, 27: 339–352

Berzal-Herranz, A., A.D.L. Cruz, F. Tenllado, J.R. Diaz-Ruiz, L. Lopez, A.I. Sanz, C. Vaquero, M.T. Serrra and I. Garcia-Luque, 1995. The *Capsicum L3* gene-mediated resistance against the tobamoviruses is elicited by the coat protein. *Virology*, 209: 498–505

Biezen, E.A.V.D. and J.D.G. Jones, 1998. The NB-ARC domain: a novel signalling motif shared by plant resistance gene products and regulators of cell death in animals. *Curr. Biol.*, 8: 226–228

Boiteux, L.S. and L.D.B. Giordano, 1993. Genetic basis of resistance against two *Tospovirus* species in tomato (*Lycopersicon esculentum*). *Euphytica*, 71: 151-154

Boukema, I.W., 1980. Allelism of genes controlling resistance to TMV in *Capsicum* L. *Euphytica*, 29: 433–439

Bouwmeester, H., G.B.M. Heuvelink and J.J. Stoorvogel, 2016. Mapping crop diseases using survey data: The case of bacterial wilt in bananas in the East African highlands. *Eur. J. Agron.*, 74: 173–184

Brandwagt, B.F., L.A Mesbah, F.L.W. Takken, P.L. Laurent, T.J.A. Kneppers, J. Hille and H.J.J. Nijkamp, 2000. A longevity assurance gene homolog of tomato mediates resistance to *Alternaria alternata* f. sp. *lycopersici* toxins and fumonisin B1. *Proc. Natl. Acad. Sci. U.S.A.*, 97: 4961–4966

Brommonschenkel, S.H., A. Frary, A. Frary and S.D. Tanksley, 2000. The broad-spectrum tospovirus resistance gene Sw-5 of tomato is a homolog of the root-knot nematode resistance gene Mi. *Mol. Plant-Microb. Interact.*, 13: 1130–1138

Canto, T., S.A. MacFarlane and P. Palukaitis, 2004. ORF6 of *Tobacco mosaic virus* is a determinant of viral pathogenicity in *Nicotiana benthamiana*. *J. Gen. Virol*., 85: 3123–3133

Celik, I., M.A. Sogut, E. Ozkaynak, S. Doganlar and A. Frary, 2016. Physical mapping of *NBS*-coding resistance genes to the *Me*-gene cluster on chromosome P9 reveals markers tightly linked to the *N* gene for root-knot nematode resistance in pepper. *Mol. Breed.*, 36: 1-7

Chai, Y., L. Zhao, Z. Liao, X. Sun, K. Zuo, L. Zhang, S. Wang and K. Tang, 2003. Molecular cloning of a potential *Verticillium dahliae* resistance gene *SlVe1* with multi-site polyadenylation from *Solanum licopersicoides*. *DNA Seq*., 14: 375–384

Chen, R., H. Li, L. Zhang, J. Zhang, J. Xiao and Z. Ye, 2007. CaMi, a root-knot nematode resistance gene from hot pepper (*Capsicum annuum* L.) confers nematode resistance in tomato. *Plant Cell Rep*., 26: 895–905

Chen, Y., F. Yan, Y. Chai, H. Liu, R. Kolter, R. Losick and J. Guo, 2013. Biocontrol of tomato wilt disease by *Bacillus subtilis* isolates from natural environments depends on conserved genes mediating biofilm formation. *Environ. Microbiol.*, 15: 848–864

Chiemsombat, P., M. Sharman, K. Srivilai, P. Campbell, D. Persley and S. Attathom, 2010. A new tospovirus species infecting *Solanum esculentum* and *Capsicum annuum* in Thailand, Australas. *Plant Dis. Notes*, 5: 1-4

Chisholm, S.T., G. Coaker, B. Day and B.J. Staskawicz, 2006. Host-microbe interactions: shaping the evolution of the plant immune response. *Cell*, 124: 803–814

Choi, D., H.M. Kim, H.K. Yun, J.A. Park, W.T. Kim and S.H. Bok, 1996. Molecular cloning of a metallothionein-like gene from *Nicotiana glutinosa* L. and its induction by wounding and tobacco mosaic virus infection. *Plant Physiol*., 112: 353–359

Cockerham, G., 1970. Genetical studies on resistance to potato viruses X and Y. *Heredity*, 25: 309–348

Collinge, D.B., O.S. Lund and H. Thordal-Christensen, 2008. What are the prospects for genetically engineered, disease resistant plants? *Eur. J. Plant Pathol*., 121: 217–231

Collmer, A., 1998. Determinants of pathogenicity and avirulence in plant pathogenic bacteria. *Curr. Opin. Plant Biol*., 1: 329–335

Colon, L.T. and D.J. Budding, 1988. Resistance to late blight (*Phytophthora infestans*) in ten wild Solanum species. *Euphytica*, 39: 77–86

Cruz, I., L. Lopez, F. Tenllado, J.R. Diaz-Ruiz, A.I. Sanz, C. Vaquero, M.T. Serra and I. Garcia-Luque, 1997. The coat protein is required for the elicitation of the *Capsicum* L2 gene-mediated resistance against the tobamoviruses. *Mol. Plant–Microb. Interact.*, 10: 107–113

Davino, S., S. Panno, G. Iacono, L. Sabatino, F. D'Anna, G. Iapichino, A. Olmos, G. Scuderi, L. Rubio, L. Tomassoli, G. Capodici, F. Martinelli and M. Davino, 2017. Genetic variation and evolutionary analysis of Pepino mosaic virus in Sicily: insights into the dispersion and epidemiology. *Plant Pathol.*, 66: 368-375

Dean, J.D., P.H. Goodwin and T. Hsiang, 2005. Induction of *glutathione S-transferase* genes of *Nicotiana benthamiana* following infection by *Colletotrichum destructivum* and *C. orbiculare* and involvement of one in resistance. *J. Exp. Bot.*, 56: 1525–1533

DeYoung, B.J. and R.W. Innes, 2006. Plant NBS-LRR proteins in pathogen sensing and host defense. *Nat. Immunol*., 7: 1243–1249

Diwan, N., R. Fluhr, Y. Eshed, D. Zamir and S.D. Tanksley, 1999. Mapping of Ve in tomato: A gene conferring resistance to the broad-spectrum pathogen, *Verticillium dahliae* race 1. *Theor. Appl. Genet*., 98: 315–319

Dixon, M.S., K. Hatzixanthis, D.A. Jones, K. Harrison, J.D. Jones, J. Dixon, M.S., Hatzixanthis, K., Jones, D.A., Harrison and K. Jones, 1998. The tomato Cf-5 disease resistance gene and six homologs show pronounced allelic variation in leucine-rich repeat copy number. *Plant Cell*, 10: 1915-1925

Durrant, W.E. and X. Dong, 2004. Systemic acquired resistance. *Annu. Rev. Phytopathol*., 42: 185–209

Ernst, K., A. Kumar, D. Kriseleit, D.U. Kloos, M.S. Phillips and M.W. Ganal, 2002. The broad-spectrum potato cyst nematode resistance gene (Hero) from tomato is the only member of a large gene family of NBS-LRR genes with an unusual amino acid repeat in the LRR region. *Plant J.*, 31: 127–136

Fei, J., Y. Chai, J. Wang, J. Lin, X. Sun, C. Sun, K. Zuo and K. Tang, 2000. CDNA cloning and characterization of the *Ve* homologue gene *StVe* from *Solanum torvum* Swartz. *DNA Seq*., 15: 88–95

Fernandes, J.J., M.G. Carvalho, E.C. Andrade, S.H. Brommonschenkel, E.P.B. Fontes and F.M. Zerbini, 2006. Biological and molecular properties of Tomato rugose mosaic virus (ToRMV), a new tomato-infecting begomovirus from Brazil. *Plant Pathol*., 55: 513–522

Fisher, M.C., D. Henk, C.J. Briggs, J.S. Brownstein, L.C. Madoff, S.L. McCraw and S.J. Gurr, 2012. Emerging fungal threats to animal, plant and ecosystem health. *Nature*, 484: 186–194

Foster, S.J., T.H. Park, M. Pel, G. Brigneti, J. Śliwka, L. Jagger, E.V.D. Vossen and J.D.G. Jones, 2009. *Rpi-vnt1.1* , a *Tm-2 2* homolog from *Solanum venturii*, confers resistance to potato late blight. *Mol. Plant-Microb. Interact*., 22: 589–600

Gabaldón, T. and E.V. Koonin, 2013. Functional and evolutionary implications of gene orthology. *Nat. Rev. Genet.*, 14: 360–366

Gebhardt, C. and J.P. Valkonen, 2001. Organization of genes controlling disease resistance in the potato genome. *Annu. Rev. Phytopathol*., 39: 79–102

Gill, U.S., S. Lee and K.S. Mysore, 2015. Host versus nonhost resistance: distinct wars with similar arsenals. *Phytopathology*, 105: 580–587

Glazebrook, J., 2005. Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. *Annu. Rev. Phytopathol*., 43: 205–227

Golas, T.M., A. Sikkema, J. Gros, R.M.C. Feron, R.G.V.D. Berg, G.M. van der Weerden, C. Mariani and J.J.H.M. Allefs, 2010. Identification of a resistance gene Rpi-dlc1 to *Phytophthora infestans* in european accessions of *Solanum dulcamara*. *Theor. Appl. Genet*., 120: 797–808

Govrin, E.M. and A. Levine, 2000. The hypersensitive response facilitates plant infection by the necrotrophic pathogen *Botrytis cinerea*. *Curr. Biol.*, 10: 751–757

Green, J.S. and S.K. Kim, 1991. Characteristics and control of viruses infecting peppers : a literature review, (No. SB608. P5. G74 1991.). *Asian Veg. Res. Dev. Center*, 18: 1-60

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

Hameed, A., Z. Iqbal, S. Asad and S. Mansoor, 2014. Detection of multiple potato viruses in the field suggests synergistic interactions among potato viruses in Pakistan. *Plant Pathol. J*., 30: 407-415

Hameed, S., M.A. Nawaz, W. Ahmed, A. Shehzahi, F. Hussain, N. Munir, M.N. Khan and F. Hayat, 2016. Potential of horticultural crops to ensure food security in Pakistan. *J. Environ. Agric*., 1: 74–78

Hammond-Kosack, K.E. and J.D. Jones, 1996. Resistance gene-dependent plant defense responses. *Plant Cell*, 8: 1773–1791

Hammond-Kosack, K.E. and K. Kanyuka, 2007. *Resistance genes (R genes) in plants*. John Wiley and Sons, Ltd., Chichester, UK

Hanssen, I.M., M. Lapidot and B.P.H.J. Thomma, 2010. Emerging viral diseases of tomato crops. *Mol. Plant-Microb. Interact*., 23: 539–548

Harikrishnan, S.L., P. Pucholt and S. Berlin, 2015. Sequence and gene expression evolution of paralogous genes in willows. *Sci. Rep*., 5: 1-10

Haverkort, A.J., P.C. Struik, R.G.F. Visser and E. Jacobsen, 2009. Applied biotechnology to combat late blight in potato caused by *Phytophthora infestans. Potato Res.* 52: 249-264

Helgeson, J.P., J.D. Pohlman, S. Austin, G.T. Haberlach, S.M. Wielgus, D. Ronis, L. Zambolim, P. Tooley, J.M. McGrath, R.V. James and W.R. Stevenson, 1998. Somatic hybrids between *Solanum bulbocastanum* and potato: A new source of resistance to late blight. *Theor. Appl. Genet*., 96: 738–742

Hiruma, K., S. Fukunaga, P. Bednarek, M. Pislewska-Bednarek, S. Watanabe, Y. Narusaka, K. Shirasu and Y. Takano, 2013. Glutathione and tryptophan metabolism are required for *Arabidopsis* immunity during the hypersensitive response to hemibiotrophs. *Proc. Natl. Acad. Sci. U.S.A.*, 110: 9589–9594

Heuvel, J.F.J.M.V.D., R.A.A.V.D. Vlugt, M. Verbeek, P.T.D. Haan and H. Huttinga, 1994. Characteristics of a resistance-breaking isolate of potato virus Y causing potato tuber necrotic ringspot disease. *Eur. J. Plant Pathol.*, 100: 347–356

Imran, M., M.A. Khan, M. Fiaz, M. Azeem and M. Mustafa, 2013. Influence of environmental conditions on Tomato Mosaic Virus disease development under natural condition. *Pak. J. Phytopathol.*, 25: 117–122

Ishibashi, K., K. Masuda, S. Naito, T. Meshi and M. Ishikawa, 2007. An inhibitor of viral RNA replication is encoded by a plant resistance gene. *Proc. Natl. Acad. Sci. USA.* 104: 13833-13838

Ivey, M.L.L., A. Strayer, J.K. Sidhu and G. V. Minsavage, 2016. Bacterial leaf spot of bell pepper (*Capsicum annuum*) in Louisiana is Caused by *Xanthomonas euvesicatoria* Pepper Races 1 and 3. *Plant Dis.*, 100: 853–853

Jabbar, A., N. Javed, S.A. Khan and M.A. Ali, 2015. *Meloidogyne graminicola* an emerging threat to rice and wheat in Punjab province in Pakistan. *Pak. J. Nematol.*, 33: 227-228

Jo, K.R., R.G.F. Visser, E. Jacobsen and J.H. Vossen, 2015. Characterisation of the late blight resistance in potato differential MaR9 reveals a qualitative resistance gene, *R9a*, residing in a cluster of *Tm-2 (2)* homologs on chromosome IX. *Theor. Appl. Genet*., 128: 931–941

Jones, D.A., C.M. Thomas, K.E. Hammond-Kosack, P.J. Balint-Kurti and J.D.G. Jones, 1994. Isolation of the tomato *Cf-9* gene for resistance to *Cladosporium fulvum* by transposon tagging. *Science*, 266: 789–793

Jones, J.D.G., K. Witek, W. Verweij, F. Jupe, D. Cooke, S. Dorling, L. Tomlinson, M. Smoker, S. Perkins and S. Foster, 2014. Elevating crop disease resistance with cloned genes. *Phil. Trans. Roy. Soc. Lond. B Biol. Sci.*, 369: 1-7

Jones, J.T., A. Haegeman, E.G.J. Danchin, H.S. Gaur, J. Helder, M.G.K. Jones, T. Kikuchi, R. Manzanilla-López, J.E. Palomares-Rius, W.M.L. Wesemael and R.N. Perry, 2013. Top 10 plant-parasitic nematodes in molecular plant pathology. *Mol. Plant Pathol.*, 14: 946–961

Jones, P.W. and E.P. Lettice, 2016. Effect of soil and soil bacteria on hatching activity towards potato cyst nematodes (*Globodera* spp.). *Nematology*, 18: 803–810

Jones, R.A.C., R. Koenig and D.E. Lesemann, 1980. Pepino mosaic virus, a new potexvirus from pepino (*Solanurn muricatum*). *Ann. Appl. Biol.*, 94: 61–68

Jong, W.D., A. Forsyth, D. Leister, C. Gebhardt and D.C. Baulcombe, 1997. A potato hypersensitive resistance gene against potato virus X maps to a resistance gene cluster on chromosome 5. *Theor. Appl. Genet*., 95: 246–252

Kaloshian, I., 2004. Gene-for-gene disease resistance: bridging insect pest and pathogen defense. *J. Chem. Ecol.*, 30: 2419–2438

Kang, B.C., I. Yeam and M.M. Jahn, 2005. Genetics of plant virus resistance. *Annu. Rev. Phytopathol*., 43: 581–621

Kay, S., S. Hahn, E. Marois, G. Hause and U. Bonas, 2007. A bacterial effector acts as a plant transcription factor and induces a cell size regulator. *Science*, 318: 648–651

Kearney, B. and B.J. Staskawicz, 1990. Widespread distribution and fitness contribution of *Xanthomonas campestris* avirulence gene *avrBs2*. *Nature*, 346: 385–386

Khan, M.R., F.A. Mohiddin, M.N. Ejaz and M.M. Khan, 2012. Management of root-knot disease in eggplant through the application of biocontrol fungi and dry neem leaves. *Turk. J. Biol*., 36: 161–169

Knapp, S., L. Bohs, M. Nee, and D.M. Spooner. 2004. Solanaceae-a model for linking genomics with biodiversity. *Comp. Funct. Genom.*, 5: 285–291

Koch, C., F.J. Eber, C. Azucena, A. Förste, S. Walheim, T. Schimmel, A.M. Bittner, H. Jeske, H. Gliemann, S. Eiben, F.C. Geiger and C. Wege, 2016. Novel roles for well-known players: from tobacco mosaic virus pests to enzymatically active assemblies. *Beils. J. Nanotechnol*., 7: 613–629

Kuhl, M., J.C. Hanneman and R.E. Havey, 2001. Characterization and mapping of *Rpi1*, a late blight resistance locus from diploid (1EBN) Mexican *Solanum pinnatisectum. Mol. Genet. Genom.*, 265: 977–985

Kumar, R., A. Barman, T. Phukan, K. Kabyashree, N. Singh, G. Jha, R.V. Sonti, S. Genin and S.K. Ray, 2017. *Ralstonia solanacearum* virulence in tomato seedlings inoculated by leaf clipping. *Plant Pathol*., 66: 835-841

Lanfermeijer, F.C., J. Warmink and J. Hille, 2005. The products of the broken *Tm-2* and the durable *Tm-22* resistance genes from tomato differ in four amino acids. *J. Exp. Bot*., 56: 2925–2933

Lanfermeijer, F.C., J. Dijkhuis, M.J.G. Sturre, P.D. Haan and J. Hille, 2003. Cloning and characterization of the durable tomato mosaic virus resistance gene *Tm-2(2)* from *Lycopersicon esculentum*. *Plant Mol. Biol.*, 52: 1037–1049

Leonard, S., F. Hommais, W. Nasser and S. Reverchon, 2017. Plant-phytopathogen interactions: bacterial responses to environmental and plant stimuli. *Environ. Microbiol*., 19: 1689-1716

Liang, D., R.G. White and P.M. Waterhouse, 2012. Gene silencing in Arabidopsis spreads from the root to the shoot, through a gating barrier, by template-dependent, nonvascular, cell-to-cell movement. *Plant Physiol*., 159: 984-1000

Liau, C., J. Lu, V. Prasad, H. Hsiao, S. You, J. Lee, N. Yang, H. Huang, T. Feng, W. Chen and M.T. Chan, 2003. The sweet pepper ferredoxin-like protein (pflp) conferred resistance against soft rot disease in *Oncidium orchid. Trans. Res*., 12: 329–336

Liu, S., Y. Zhu, C. Xie, D. Jue, Y. Hong, M. Chen, A.K. Hubdar and Q. Yang, 2012. Transgenic potato plants expressing stove1 exhibit enhanced resistance to *Verticillium dahliae*. *Plant Mol. Biol. Rep.*, 30: 1032–1039

Liu, W., C. Harn, M. Jahn, B. Kim and B. Kang, 2008. *Compendium of transgenic crop plants*. John Wiley & Sons, Ltd., Chichester, U.K.

Loon, L.C.V., 1997. Induced resistance in plants and the role of pathogenesis-related proteins. *Eur. J. Plant Pathol*., 103: 753–765

Macfarlane, S.A., 2010. Tobraviruses--plant pathogens and tools for biotechnology. *Mol. Plant Pathol.*, 11: 577–583

Martin, G.B., 1999. Functional analysis of plant disease resistance genes and their downstream effectors. *Curr. Opin. Plant Biol*., 2: 273–279

Martin, S.D., G.B. Brommonschenkel, S.H. Chunwongse, J. Frary, A. Ganal, M.W. Spivey, R. Wu, T. Earle and E.D. Tanksley, 1993. Map-based cloning of a protein kinase gene conferring disease resistance in tomato. *Science*, 262: 1432–1436

McHale, L., X. Tan, P. Koehl and R.W. Michelmore, 2006. Plant NBS-LRR proteins: adaptable guards. *Genom. Biol*., 7: 212-223

Meister, G. and T. Tuschl, 2004. Mechanisms of gene silencing by double-stranded RNA. *Nature*, 431: 343–349

Mendonca, L.B.P., L. Coelho, J. Stracieri, J.B.F. Junior and N.D. Tebaldi, 2015. Chemical control of Phytophthora wilt in tomatoes. *Biosci. J.*, 31: 1015–1023

Mestre, D., P. Brigneti and G. Baulcombe, 2000. An Ry-mediated resistance response in potato requires the intact active site of the Nla proteinase from potato virus Y. *Plant J*., 23: 653–661

Meyers, B.C., S. Kaushik and R.S. Nandety, 2005. Evolving disease resistance genes. *Curr. Opin. Plant Biol.*, 8: 129–134

Milligan, S.B., J. Bodeau, J. Yaghoobi, I. Kaloshian, P. Zabel and V.M. Williamson, 1998. The root knot nematode resistance gene *Mi* from tomato is a member of the leucine zipper, nucleotide binding, leucine-rich repeat family of plant genes. *Plant Cell*, 10: 1307–1319

Morales, F.J., R. Lastra, R.C.D. Uzcátegui and L. Calvert, 2001. Potato yellow mosaic virus: a synonym of *tomato yellow mosaic virus*. *Arch. Virol.*, 146: 2249–2253

Moriones, E. and J. Navas-Castillo, 2000. Tomato yellow leaf curl virus, an emerging virus complex causing epidemics worldwide. *Virus Res.*, 71: 123–134

Moury, B., C. Morel, E. Johansen, L. Guilbaud, S. Souche, V. Ayme, C. Caranta, A. Palloix and M. Jacquemond, 2004. Mutations in potato virus Y genome-linked protein determine virulence toward recessive resistances in *Capsicum annuum* and *Lycopersicon hirsutum*. *Mol. Plant-Microb. Interact*., 17: 322–329

Mucyn, T.S., A. Clemente, V.M.E. Andriotis, A.L. Balmuth, G.E.D. Oldroyd, B.J. Staskawicz and J.P. Rathjen, 2006. The tomato NBARC-LRR protein Prf interacts with Pto kinase *in Vivo* to regulate specific plant immunity. *Plant Cell Onl*. 18: 2792–2806

Mueller, L.A., T.H. Solow, N. Taylor, B. Skwarecki, R. Buels, J. Binns, C. Lin, M.H. Wright, R. Ahrens, Y. Wang, E.V. Herbst, E.R. Keyder, N. Menda, D. Zamir and S.D. Tanksley, 2005. The SOL genomics network: A comparative resource for Solanaceae biology and beyond. *Plant Physiol.*, 138: 1310–1317

Mumford, R.A., R. Macarthur and N. Boonham, 2016. The role and challenges of new diagnostic technology in plant biosecurity. *Food Secur.*, 8: 103–109

Mundt, C.C., 2014. Durable resistance: a key to sustainable management of pathogens and pests. *Infect. Genet. Evol.*, 27: 446–455

Navas-Castillo, J., E. Fiallo-Olivé and S. Sánchez-Campos, 2011. Emerging virus diseases transmitted by whiteflies. *Annu. Rev. Phytopathol.*, 49: 219–248

Naveed, K., A. Abbas and L. Amrao, 2017. Potato virus Y: an evolving pathogen of potato worldwide. *Pak. J. Phytopathol*., 29: 187-191

Nguyen, M.T. and S.L. Ranamukhaarachchi, 2010. Soil-borne antagonists for biological control of bacterial wilt disease caused by *Ralstonia solanacearum* in tomato and pepper. *J. Plant Pathol*., 92: 395–406

Nombela, G., V.M. Williamson and M. Muñiz, 2003. The root-knot nematode resistance gene *Mi-1.2* of tomato is responsible for resistance against the whitefly *Bemisia tabaci*. *Mol. PlantMicrob. Interact.*, 16: 645–649

Nürnberger, T. and V. Lipka, 2005. Non-host resistance in plants: new insights into an old phenomenon. *Mol. Plant Pathol.*, 6: 335–345

Oldroyd, G.E. and B.J. Staskawicz, 1998. Genetically engineered broad-spectrum disease resistance in tomato. *Proc. Natl. Acad. Sci. U.S.A.*, 95: 10300–10305

Ori, N., Y. Eshed, I. Paran, G. Presting, D. Aviv, S. Tanksley, D. Zamir and R. Fluhr, 1997. The l2C family from the wilt disease resistance locus l2 belongs to the nucleotide binding, leucine-rich repeat superfamily of plant resistance genes. *Plant Cell*, 9: 521–532

Ozgonen, H. and A. Erkilic, 2007. Growth enhancement and phytophthora blight (*Phytophthora capsici* Leonian) control by arbuscular mycorrhizal fungal inoculation in pepper. *Crop Prot.*, 26: 1682–1688

Paal, J., H. Henselewski, J. Muth, K. Meksem, C.M. Menéndez, F. Salamini, A. Ballvora and C. Gebhardt, 2004. Molecular cloning of the potato *Gro1-4* gene conferring resistance to pathotype Ro1 of the root cyst nematode *Globodera rostochiensis*, based on a candidate gene approach. *Plant J.*, 38: 285–297

Panthee, D.R. and F. Chen, 2010. Genomics of fungal disease resistance in tomato. *Curr. Genom.*, 11: 30–39

Pel, M.A., S.J. Foster, T.H. Park, H. Rietman, G.V. Arkel, J.D.G. Jones, H.J.V. Eck, E. Jacobsen, R.G.F. Visser and E.A.G.V.D. Vossen, 2009. Mapping and cloning of late blight resistance genes from *Solanum venturii* using an interspecific candidate gene approach. *Mol. Plant-Microbe. Interact.*, 22: 601–615

Perveen, A. and M. Qaiser, 2007. Pollen morphology of family Solanaceae from Pakistan. *Pak. J. Bot*., 39: 2243–2256

Potnis, N., S. Timilsina, A. Strayer, D. Shantharaj, J.D. Barak, M.L. Paret, G.E. Vallad and J.B. Jones, 2015. Bacterial spot of tomato and pepper: diverse Xanthomonas species with a wide variety of virulence factors posing a worldwide challenge. *Mol. Plant Pathol.*, 16: 907–920

Potter, C., T. Harwood, J. Knight and I. Tomlinson, 2011. Learning from history, predicting the future: the UK Dutch elm disease outbreak in relation to contemporary tree disease threats. *Phil. Trans. Roy. Soc. Lond. B Biol. Sci*., 366: 1966–1974

Querci, M., D.C. Baulcombe, R.W. Goldbach and L.F. Salazar, 1995, Analysis of the resistance- breaking determinants of potato virus X (PVX) strain HB on different potato genotypes expressing extreme resistance to PVX. *Phytopathology*, 85: 1003–1010

Ramezani, H., 2008. Biological control of root-rot of eggplant caused by *Macrophomina phaseolina*. *Amer. J. Agric. Environ. Sci.*, 4: 218–220

Reddy, P.P., 2016. *Grafted vegetables for management of soilborne pathogens. In: sustainable crop protection under protected cultivation*, pp: 83–97. Springer Singapore

Romero, A.M. C.S. Kousik and D.F. Ritchie, 2010 Resistance to bacterial spot in bell pepper induced by Acibenzolar-S-Methyl. *Plant Dis*., 85: 189–194

Ronald, P.C., J.M. Salmeron, F.M. Carland and B.J. Staskawicz, 1992. The cloned avirulence gene *avrPto* induces disease resistance in tomato cultivars containing the *Pto* resistance gene. *J. Bacteriol*., 174: 1604–1611

Roselló, S., B. Ricarte, M.J. Díez and F. Nuez, 2001. Resistance to tomato spotted wilt virus introgressed from *Lycopersicon peruvianum* in line UPV 1 may be allelic to *Sw-5* and can be used to enhance the resistance of hybrids cultivars. *Euphytica*, 119: 357–367

Roselló, S., M.J. Díez and F. Nuez, 1998. Genetics of tomato spotted wilt virus resistance coming from *Lycopersicon peruvianum*. *Eur. J. Plant Pathol*., 104: 499–509

Rossi, M., F.L. Goggin, S.B. Milligan, I. Kaloshian, D.E. Ullman and V.M. Williamson, 1998. The nematode resistance gene *Mi* of tomato confers resistance against the potato aphid. *Proc. Natl. Acad. Sci. U.S.A.*, 95: 9750–9754

Ruffel, S., J.L. Gallois, M.L. Lesage and C. Caranta, 2005. The recessive potyvirus resistance gene *pot-1* is the tomato orthologue of the pepper *pvr2-eIF4E* gene. *Mol. Genet. Genom.*, 274: 346–353

Ryals, J., S. Uknes and E. Ward, 1994. Systemic acquired resistance. *Plant Physiol*., 104: 1109–1112

Salmeron, J.M., G.E. Oldroyd, C.M.T. Rommens, S.R. Scofield, H.S. Kim, D.T. Lavelle, D. Dahlbeck and B.J. Staskawicz, 1996. Tomato Prf is a member of the leucine-rich repeat class of plant disease resistance genes and lies embedded within the Pto kinase gene cluster. *Cell*, 86: 123–133

Samuels, J., 2015. Biodiversity of food species of the Solanaceae family: A preliminary taxonomic inventory of subfamily solanoideae. *Resources*, 4: 277–322

Savary, S., A. Ficke, J.N. Aubertot and C. Hollier, 2012. Crop losses due to diseases and their implications for global food production losses and food security. *Food Secur*., 4: 519–537

Scholthof, K.B.G., 2004. Tobacco mosaic virus: a model system for plant biology. *Annu. Rev. Phytopathol.*, 42: 13–34

Shao, Z.Q., J.Y. Xue, Q. Wang, B. Wang and J.Q. Chen, 2019. Revisiting the origin of plant NBS-LRR genes, *Trends Plant Sci.*, 24: 9-12

Simko, I., S. Costanzo, K.G. Haynes, B.J. Christ and R.W. Jones, 2004. Linkage disequilibrium mapping of a *Verticillium dahliae* resistance quantitative trait locus in tetraploid potato (*Solanum tuberosum*) through a candidate gene approach. *Theor. Appl. Genet*., 108: 217–224

Simons, G., J. Groenendijk, J. Wijbrandi, M. Reijans, J. Groenen, P. Diergaarde, T.V.D. Lee, M. Bleeker, J. Onstenk, M.D. Both, M. Haring, J. Mes, B. Cornelissen, M. Zabeau and P. Vos, 1998, Dissection of the fusarium *I2* gene cluster in tomato reveals six homologs and one active gene copy. *Plant Cell*, 10: 1055–1068

Sliwka, J., H. Jakuczun, R. Lebecka, W. Marczewski, C. Gebhardt and E. Zimnoch-Guzowska, 2006. The novel, major locus *Rpi-phu1* for late blight resistance maps to potato chromosome IX and is not correlated with long vegetation period. *Theor. Appl. Genet*., 113: 685–695

Sobczak, M., A. Avrova, J. Jupowicz, M.S. Phillips, K. Ernst and A. Kumar, 2005. Characterization of susceptibility and resistance responses to potato cyst nematode (*Globodera* spp.) infection of tomato lines in the absence and presence of the broad-spectrum nematode resistance *Hero* gene. *Mol. Plant-Microb. Interact.*, 18: 158–168

Solomon-Blackburn, R.M. and H. Barker, 2001. A review of host major-gene resistance to potato viruses X, Y, A and V in potato: genes, genetics and mapped locations. *Heredity*, 86: 8–16

Song, J., J.M. Bradeen, S.K. Naess, J.A. Raasch, S.M. Wielgus, G.T. Haberlach, J. Liu, H. Kuang, S. Austin-Phillips, C.R. Buell, J.P. Helgeson and J. Jiang, 2003. Gene RB cloned from *Solanum bulbocastanum* confers broad spectrum resistance to potato late blight. *Proc. Natl. Acad. Sci. U.S.A.*, 100: 9128–9133

Song, Y., Z. Zhang, M.F. Seidl, A. Majer, J. Jakse, B. Javornik and B.P.H.J. Thomma, 2017. Broad taxonomic characterization of Verticillium wilt resistance genes reveals an ancient origin of the tomato Ve1 immune receptor. *Mol. Plant Pathol.*, 18: 195–209

Soosaar, J.L.M., T.M. Burch-Smith and S.P. Dinesh-Kumar, 2005. Mechanisms of plant resistance to viruses. *Nat. Rev. Microbiol.*, 3: 789–798

Spassova, M.I., T.W. Prins, R.T. Folkertsma, R.M. Klein-Lankhorst, J. Hille, R.W. Goldbach and M. Prins, 2001. The tomato gene *Sw5* is a member of the coiled coil, nucleotide binding, leucine-rich repeat class of plant resistance genes and confers resistance to TSWV in tobacco. *Mol. Breed.*, 7: 151–161

Staskawicz, B.J., F.M. Ausubel, B.J. Baker, J.G. Ellis and J.D. Jones, 1995. Molecular genetics of plant disease resistance. *Science*, 268: 661-667

Stevens, M.R., S.J. Scott and R.C. Gergerich, 1992. Inheritance of a gene for resistance to tomato spotted wilt virus (TSWV) from *Lycopersicon peruvianum* Mill. *Euphytica*, 59: 9–17

Strange, R.N. and P.R. Scott, 2005. Plant disease: a threat to global food security. *Annu. Rev. Phytopathol.*, 43: 83–116

Sugawara, E. and H. Nikaido, 2014. Properties of Ade ABC and AdeIJK efflux systems of *Acinetobacter baumannii* compared with those of the AcrAB-TolC system of *Escherichia coli*. *Antimicrob. Agents Chemother.*, 58: 7250–7257

Tai, T.H., D. Dahlbeck, E.T. Clark, P. Gajiwala, R. Pasion, M.C. Whalen, R.E. Stall and B.J. Staskawicz, 1999. Expression of the *Bs2* pepper gene confers resistance to bacterial spot disease in tomato. *Proc. Natl. Acad. Sci. U.S.A.*, 96: 14153–14158

Tameling, W.I.L. and F.L.W. Takken, 2008. Resistance proteins: scouts of the plant innate immune system. *Eur. J. Plant Pathol.*, 121: 243–255

Than, P.P., H. Prihastuti, S. Phoulivong, P.W.J. Taylor and K.D. Hyde, 2008. Chilli anthracnose disease caused by Colletotrichum species. *J. Zhej. Univ. Sci. B*, 9: 764–78

Thomas, C.M., D.A. Jones, M. Parniske, K. Harrison, P.J. Balint-Kurti, K. Hatzixanthis and J.D.G. Jones, 1997. Characterization of the tomato *Cf-4* gene for resistance to *Cladosporium fulvum* identifies sequences that determine recognitional specificity in *Cf-4* and *Cf-9*. *Plant Cell*, 9: 2209–2224

Toppino, L., G. Valè and G.L. Rotino, 2008. Inheritance of Fusarium wilt resistance introgressed from *Solanum aethiopicum* Gilo and Aculeatum groups into cultivated eggplant (*S. melongena*) and development of associated PCR-based markers. *Mol. Breed.*, 22: 237–250

Tsai, W.S., Y.C. Huang, D.Y. Zhang, K. Reddy, S.H. Hidayat, W. Srithongchai, S.K. Green and F.J. Jan, 2008. Molecular characterization of the *CP* gene and 3'UTR of Chilli veinal mottle virus from South and Southeast Asia. *Plant Pathol.*, 57: 408–416

Tsompana, M., J. Abad, M. Purugganan and J.W. Moyer, 2004. The molecular population genetics of the tomato spotted wilt virus (TSWV) genome. *Mol. Ecol.*, 14: 53–66

van der Vossen, E., A. Sikkema, B.T. L. Hekkert, J. Gros, P. Stevens, M. Muskens, D. Wouters, A. Pereira, W. Stiekema and S. Allefs, 2003. An ancient *R* gene from the wild potato species *Solanum bulbocastanum* confers broad-spectrum resistance to *Phytophthora infestans* in cultivated potato and tomato. *Plant J.*, 36: 867–882

Verbeek, M., A.M. Dullemans, J.F.J.M.V.D. Heuvel, P.C. Maris and R.A.A.V.D. Vlugt, 2007. Identification and characterisation of tomato torrado virus, a new plant picorna-like virus from tomato. *Arch. Virol.*, 152: 881–890

Verlaan, M.G., S.F. Hutton, R.M. Ibrahem, R. Kormelink, R.G.F. Visser, J.W. Scott, J.D. Edwards and Y. Bai, 2013. The tomato yellow leaf curl virus resistance genes *Ty-1* and *Ty-3* are allelic and code for DFDGD-class RNA-dependent RNA polymerases. *PLoS Genet.* 9: e1003399

Vidal, V.J., S. Cabrera, S. Anderson and R.A. Fredreiksson, 2002. Potato gene *Y-1* is an N-gene homolg that confers cell death upon infection with potato virus Y. *Mol. Plant-Microb. Interact.*, 15: 717–727

Vlugt, R.A.A.V.D., C.C.M.M. Stijger, J.T.J. Verhoeven and D. Lesemann, 2000. First report of *Pepino Mosaic Virus* on tomato. *Plant Dis.*, 84: 103–103

Vos, P., G. Simons, T. Jesse, J. Wijbrandi, L. Heinen, R. Hogers, A. Frijters, J. Groenendijk, P. Diergaarde, M. Reijans, J. Fierens-Onstenk, M.D. Both, J. Peleman, T. Liharska, J. Hontelez and M. Zabeau, 1998. The tomato *Mi-1* gene confers resistance to both root-knot nematodes and potato aphids. *Nat. Biotechnol.*, 16: 1365–1369

Vossen, E.A.G., J. Gros, A. Sikkema, M. Muskens, D. Wouters, P. Wolters, A. Pereira and S. Allefs, 2005. The *Rpi-blb2* gene from *Solanum bulbocastanum* is an *Mi-1* gene homolog conferring broad-spectrum late blight resistance in potato. *Plant J.*, 44: 208–222

Vossen, E.A.V.D., J.N.V.D. Voort, K. Kanyuka, A. Bendahmane, H. Sandbrink, D.C. Baulcombe, J. Bakker, W.J. Stiekema and R.M. Klein-Lankhorst, 2000. Homologues of a single resistance-gene cluster in potato confer resistance to distinct pathogens: a virus and a nematode. *Plant J.*, 23: 567–576

Vossen, J.H., G.V. Arkel, M. Bergervoet, K.R. Jo, E. Jacobsen and R.G.F. Visser, 2016. The *Solanum demissum* R8 late blight resistance gene is an *Sw-5* homologue that has been deployed worldwide in late blight resistant varieties. *Theor. Appl. Genet.*, 129: 1785–1796

Vovlas, N., D. Mifsud, B.B. Landa and P. Castillo, 2005. Pathogenicity of the root-knot nematode *Meloidogyne javanica* on potato. *Plant Pathol.*, 54: 657–664

Wang, H., C. Sun, R. Jiang, Q. He, Y. Yang, Z. Tian, Z. Tian and C. Xie, 2014. The dihydrolipoyl acyltransferase gene *BCE2* participates in basal resistance against *Phytophthora infestans* in potato and *Nicotiana benthamiana*. *J. Plant Physiol.*, 171: 907–914

Wang, M.B., C. Masuta, N.A. Smith and H. Shimura, 2012. RNA silencing and plant viral diseases. *Mol. Plant-Microb. Interact.*, 25: 1275–1285

Wang, Y., Y. Bao, D. Shen, W. Feng, T. Yu, J. Zhang and X.D. Zheng, 2008. Biocontrol of *Alternaria alternata* on cherry tomato fruit by use of marine yeast *Rhodosporidium paludigenum* Fell & Tallman. *Intl. J. Food Microbiol.*, 123: 234–239

Wei, C., J. Chen and H. Kuang, 2016. Dramatic number variation of R genes in Solanaceae species accounted for by a sew R rene subfamilies. *PLoS One*, 11: e0148708

Whitham, S., S. McCormick and B. Baker, 1996. The N gene of tobacco confers resistance to tobacco mosaic virus in transgenic tomato. *Proc. Natl. Acad. Sci.*, 93: 8776–8781

Whitham, S., S.P. Dinesh-Kumar, D. Choi, R. Hehl, C. Corr and B. Baker, 1994. The product of the tobacco mosaic virus resistance gene N: Similarity to toll and the interleukin-1 receptor. *Cell*, 78: 1101–1115

Yadav, R., M. Rathi, A. Pednekar and Y. Rewachandani, 2016. A detailed review on Solanaceae family. *Eur. J. Pharm. Med. Res.*, 3: 369–378

Zambrano, K., F. Geraud-Pouey, D. Chirinos, G. Romay and E. Marys, 2011. Tomato chlorotic leaf distortion virus, a new bipartite begomovirus infecting *Solanum lycopersicum* and *Capsicum chinense* in Venezuela. *Arch. Virol.*, 156: 2263–2266

Zhang, Z., E. Fradin, R.D. Jonge, H.P.V. Esse, P. Smit, C.M. Liu and B.P.H.J. Thomma, 2013. Optimized agroinfiltration and virus-induced gene silencing to study Ve1-mediated Verticillium resistance in tobacco. *Mol. Plant-Microbe Interact.*, 26: 182–190

Zheng, Y.X., C.H. Huang, Y.H. Cheng, F.Y. Kuo and F.J. Jan, 2010. First report of tomato spotted wilt virus in sweet pepper in Taiwan. *Plant Dis.*, 94: 920–920

Zimnoch-Guzowska, E., R. Lebecka, A. Kryszczuk, U. Maciejewska, A. Szczerbakowa and B. Wielgat, 2003. Resistance to *Phytophthora infestans* in somatic hybrids of *Solanum nigrum* L. and diploid potato. *Theor. Appl. Genet.*, 107: 43–48

[Received 08 Dec 2018; Accepted 21 Jan 2019; Published (online) 26 Apr 2019]