Host-Parasite Interaction during Development of Major Seed-Borne Bacterial Diseases

10

Sudheer K. Yadav, Jai S. Patel, Gagan Kumar, Arpan Mukherjee, Anupam Maharshi, Surendra Singh, Harikesh B. Singh, and Birinchi K. Sarma

Abstract

Parasitic species demonstrate a wide range of population structures and life cycle plan, including various transmission modes, life cycle complication, survivability, and dispersal ability with and without the presence of their hosts. A prominent feature of hosts and parasites is based on their genetics which can be regulated by coevolution. Infections measured under laboratory conditions have shown that the environment in which hosts and parasites interact might substantially affect the strength and specificity of selection. An effective defense response is the precursor of evolution in plant immunity which restricts the potential onset of disease by microbial pathogens (parasites). In plants, the primary immune response, pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI), is one of the best examples of evolution to acknowledge general characteristics of microbial pathogens. Such type of coevolution was manifested in host-parasite interactions, but the knowledge is very less. The behavior of parasite and environmental factors also affects the host-parasite interactions. The environmental conditions such as moisture content, temperature, wind velocity, and availability of food are major factors in host-parasite interaction. The environment provides a suitable condition for the establishment of host and their parasite. In this book chapter, we are focusing on coevolution, environmental effect, and specificity during host-parasite interactions.

S. K. Yadav · J. S. Patel · S. Singh
Department of Botany, Banaras Hindu University, Varanasi, India

G. Kumar · A. Mukherjee · A. Maharshi · H. B. Singh · B. K. Sarma (✉)
Department of Mycology and Plant Pathology, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi, India
e-mail: birinchi@bhu.ac.in

R. Kumar, A. Gupta (eds.), *Seed-Borne Diseases of Agricultural Crops: Detection, Diagnosis & Management*,
https://doi.org/10.1007/978-981-32-9046-4_10

10.1 Introduction

Plants always interact with various microbes without having a somatically adaptive immune system against them. However, in presence of these microbes, the plant populations do not frequently subject to devastating disease epidemics. Disease development through seed-borne inoculum is the best result of host (plant) and parasite (pathogen) interaction in the nature. The study of these interactions is known as epidemiology. Seed-borne diseases are one of the main factors for the great economic loss in agriculture in various forms. Losses might start early from reduced germination, and it may include inadequate seedling vigor due to immature seed and crop damage at the level of seedling growth, development to harvest, and storage. In many instances, the losses occur due to previous crop produced from infested seeds. In that case the pathogens survive in soil and on debris of crop and weeds which results in further attack on subsequent susceptible crops. Sometimes, the inoculum is available in very poor quantity and unable to infect plants. Therefore, it takes too much time to accomplish a detectable level of disease in the crops (Sheppard 1998). To overcome these losses, plants need to show disease resistance against various seed-borne diseases. It is a very complex procedure that provides various potential barriers to inhibit the pathogen invasion. The disease resistance (R) genes are one of the main defense mechanisms which activate the defense responses, namely, localized cell death during the encounter with pathogenic microbes carrying respective avirulence genes (Dangl and Jones 2001; Allen et al. 2004).

The coevolution in host-parasite interaction is reciprocal natural selection between host resistance and parasite infection potential (Thompson 1999). The hypothesis proposed that the reciprocal natural selection totally relies on frequency (Bell and Smith 1987; Hamilton et al. 1990), in which the parasites are picked out to minimize the common host's resistance, and accordingly the hosts having rare resistance genes are favored for selection (Carius et al. 2001). The other interaction of nearby species provides crude materials for coevolutionary change (Thompson 1999). The local host-parasite interactions are also dependent on adjacent populations because these populations have different strategies for their resistance and virulence genes. Therefore, the new resistance and virulence genes may enter in local populations through gene flow (Gandon et al. 1998). Local extinctions, founder effects, and genetic drift also have the ability to determine the interactions and lead to shape a large-scale picture (Thrall and Burdon 1997; Burdon and Thrall 1999; Thompson 1999; Carius et al. 2001).

10.1.1 *Pseudomonas syringae* pv. *tomato*

The bacterial speck disease caused by *Pseudomonas syringae* pv. *tomato* is generally found growing epiphytically. The host range of the pathogen is very broad even though its population falls in absence of susceptible hosts (Preston 2000). Several economically important diseases are caused by *Pseudomonas syringae* in number of

its host plants. *P. syringae* pv. *tomato* can survive on seeds of tomato inside the seed cavities and lead to the infection of whole tomato fruit (Devash et al. 1980). Generally infested seeds develop visible symptoms and sometimes remain symptomless. However, infested plants growing in high relative humidity develop high amount of pathogen population and serve as source of infection (McCarter et al. 1983). A number of reports suggest that it is a seed-borne disease (Bashan et al. 1982). Each strain of *P. syringae* has some sort of host specificity, and only few of the plant species get infected, sometimes only few cultivars of a plant species (Xin and He 2013). Moisture, cool condition, and temperature around 12–25 °C favors the disease spread; however, this can depend on pathovar of the pathogen. The disease is seed-borne, and rain splashes are majorly involved in its dispersal to other noninfected plants (Hirano and Upper 1990). Though being a plant pathogen, *P. syringae* can facilitate itself as saprophyte around the phyllosphere in the unfavorable condition (Hirano and Upper 2000). *P. syringae* strains, which are saprophytic in nature, have the ability to act as biocontrol agent in postharvest rot disease (Janisiewicz and Marchi 1992). The ability of pathogen to cause disease can be divided into a number of categories such as overcoming the host resistance, ability to go inside the plant, forming biofilm, and producing some proteins having ice-nucleating properties (Ichinose et al. 2013). *P. syringae* is planktonic in nature, able to invade the plant, and with the help of pili and flagella can move toward its host. The main insertion site for *P. syringae* is through natural opening sites and wounds, and it is also able to break the cell wall. The way of movement of bacteria toward the plant is not well-studied, but some of the studies showed the chemotactic movement toward the plant and caused the infection (Ichinose et al. 2013). *P. syringae* isolates adopt type III secretion system (T3SS) for its virulence. The effector proteins of T3SS are a major cause to modify the host immune system in favor of the pathogen for infection. The hrp gene clusters are the major group of T3SS effector proteins and codes Hrp secretion apparatus. Some other ways like production of phytotoxins such as coronatine of pathovar Pto and Pg have the ability to suppress the host immunity and invade the host plant (Ichinose et al. 2013).

Certain polysaccharides produced by *P. syringae* causes the adherence of the pathogen to the plant cell surface. The communicating phenomenon 'quorum sensing' is reported for the bacteria to communicate the other bacterial cells nearby. When the level of quorum sensing molecules crosses the threshold, the bacteria starts biofilm formation and expression of genes related to virulence. Some highly viscous compounds such as DNA and polysaccharides are secreted by *P. syringae*, which create the protective environment for growth of the pathogen (Ichinose et al. 2013). *P. syringae* majorly causes cell wall damage by chilling in plants greater than any other organisms or minerals. The plants which are devoid of any antifreeze proteins are majorly damaged by −4 to −12 °C temperature, and in this condition, water remains as supercooled liquid in plants. *P. syringae* have ability to cause freezing of water on slightly higher temperature, i.e., −1.8 °C (28.8 °F) (Maki et al. 1974), but ice nucleation is generally reported at lower temperature (−8 °C). The epithelial cells are injured by freezing, which causes nutrient availability to bacteria from plants. Certain ice-nucleating genes coding ice-nucleating proteins

translocated to bacterial membrane and act as nuclei for ice formation (Fall and Wolber 1995). Two races of the *P. syringae* in tomato (0 and 1) have been described around the world (Lawton and MacNeill 1986). Due to development of resistant tomato varieties, a selection pressure created on race 0 causes appearance of new race 1 even on heterozygous nature of Pto gene in tomato (Buonaurio et al. 1996). The appearance of new race of *P. syringae* due to newer resistant varieties of tomato plant is a well-known complicated mechanism of plant-pathogen interaction (Mew et al. 1992). The development of newer race is advantageous for the pathogen (Pohronezny et al. 1992). The selection pressure increases due to introduction of newer imported resistant varieties and hybrids. Certain countries like the Balkan Peninsula and the Mediterranean, where tomato is grown intensively, observe the seed-borne nature of the *P. syringae*. However, unwillingly introduction of the pathogen with imported infected seeds cannot be excluded for the newer race development as it was reported earlier in other countries. The host plant resistance increases due to introduction of resistance genes in plants to improve control of *P. syringae* (Milijašević et al. 2009). A report found that ABA signaling pathway is the major target of effectors secreted by pathogen. Modulation of PP2C gene expression affected hypersensitive reaction toward ABA, which is otherwise helpful to bacterial multiplication. ABA level is found to be increased during bacterial colonization. However, exogenous application of ABA enhances susceptibility reaction. As per the data shown by de Torres-Zabala et al. (2007), the virulence strategy of pathogen due to presence of effector protein leads to suppresion in host defense responses.

10.1.2 *Xanthomonas campestris* pv. *campestris*

The strains of genus *Xanthomonas* infect up to 124 monocot and 268 dicot plants and create severe economic damage in the warm and humid region (Chan and Goodwin 1999). The black rot disease is caused by *Xanthomonas campestris* pv. *campestris* (Xcc) and also considered as one of the most devastating diseases of crucifers worldwide infecting most varieties of brassicas including broccoli, cabbage, kale, oilseed rape, cauliflower, turnip, radish, and mustard along with model plant *Arabidopsis thaliana* (Williams 1980). For all these conditions, Xcc-infected seeds are the basic source of inoculum. Throughout the germination epicotyl of seedling gets infected (Alvarez 2000) and the developing cotyledons became black at the margin, wilt, and fall down. The pathogen strides out through vascular system of the plants to young stem and leaves. The disease appears in V-shaped chlorotic to necrotic lesions and extends from the margin of leaves. During the humid environment, the bacteria oozes out and forms a droplet by the process of guttation through hydathodes and these droplets may spread through wind, rain, and/or through mechanical damage to their neighboring plants. The pathogen (Xcc) gains entry through the wounded leaves and plant roots due to insect damage or through hydathodes which is natural route of infection. Sometimes but rarely the infection also occurs through stomata. The hydathodes provide a straight path for pathogen to enter from leaf margin to vascular system of plant and hence systemic infection in

host. Attack through the suture vein leads to the formation of Xcc-infected seed. Xcc has the ability to survive in plant debris present in soil up to 2 years, but it hardly remains in free soil up to 6 months (Alvarez 2000), and it serves as a source of secondary inoculum. In a significant development, bean flowers inoculated with *X. campestris* pv. *campestris* led to the production of higher level of infested seeds and are also carried efficiently to seedlings of bean plants (during incompatible interaction) (Darrasse et al. 2010). This kind of floral pathway might allow the production of contaminated seeds by cohort of different bacteria and also sometimes includes biocontrol agents (Fessehaie and Walcott 2005). Similarly, the type III secretion system mutants of *X. citri* pv. *phaseoli* var. *fuscans* leads to the production of infested seeds through the entry from floral pathway and supports the previous hypothesis strongly (Darsonval et al. 2008). However, the contrasting result was found when these mutants were applied through vascular system of plants and no infested seeds were found, as found for wild-type strain.

The interactions between seeds or seedlings of the plants with the bacteria allowed multiplication of bacteria without any negative result on plants during the commensal interaction. During the compatible interactions, the seed-borne xanthomonads colonize on the surface of seedling and have no negative impact on early endophytic development (Gilbertson and Maxwell 1992). The bacteria do not require a molecular crosstalk with plants to colonize efficiently because rich amount of nutrients are available during germinating seeds and seedlings (Nelson 2004). Practically, the similar results were observed for *X. campestris* pv. *campestris*, *E. coli*, and *X. citri* pv. *phaseoli* var. *fuscans* in bean seed imbibition to get 14-day-old seedlings representing incompatible, null interactions and compatible interactions, respectively. Apart from this the bacterial colonization also does not need a functional T3SS in spermosphere, suggesting that nutrients are not major limiting factors and T3SS genes are not induced in the presence of nutrient-rich medium (Valls et al. 2006). This result was reversed in the phyllosphere as the nutrient-rich medium is a limiting factor (Mercier and Lindow 2000). The bacteria need the expression of T3SS gene to colonize efficiently in phyllosphere. Both the results suggest that for effective colonization on seedling, bacteria need different strains than phyllosphere multiplication, and these environmental parameters are also different for bacteria to adopt. Certain special interactions between bean plants and bacteria *X. citri* pv. *phaseoli* var. *fuscans* have occurred during the multiplication in phyllosphere (Darsonval et al. 2009), where nutrient availability is a major limiting factor for bacterial colonization. Additionally, the *X. citri* pv. *phaseoli* var. *fuscans* downregulate the expression of PR-3 gene in leaves indicating the suppression of plant defense. This is the sequential action of T3SS effectors to inhibit the defense response stimulated by PAMPs during compatible interactions (Mishina and Zeier 2007). During the incompatible interaction, no enhanced defense response induction was observed by *X. campestris* pv. *campestris* in bean seedlings. Therefore, these results indicate that defense responses are induced in early-stage plantlets when *X. campestris* pv. *campestris* and acibenzolar-S-methyl were applied on seedling and leaves as inoculum. The similar defense response induction was also observed earlier in melon and cowpea seedling (Buzi et al. 2004). This clearly

showed that wild-type strain *X. campestris* pv. *campestris* induces defense responses following the infiltration inoculation of seedlings, while the mutant in T3SS *X. campestris* pv. *campestris* is unable to induce defense response.

10.2 Differentiation of Molecular Pattern for Microbe Identification

Similar to animal system, plants also have the innate immune response which is turned on subsequently after the identification of invading microbes (Nürnberger et al. 2004; Akira et al. 2006; Spoel and Dong 2012). Some of these microbes have no targeted effect on plant growth and development. However, among them huge variety of microbes present in plant's microbiome are either beneficial or pathogenic (Berendsen et al. 2012). Beneficial interaction with microbes provides root colonization, plant growth promotion, and yield and also induces plant defense either directly or indirectly. Since the beneficial microflora were initially thought to be alien organisms that modify the immune system of plants for a successful establishment of mutual relationship with their host (Zamioudis and Pieterse, 2012), it is important for the plants to not only distinguish the microbes but also to possess the ability to differentiate them as good or bad and thus react accordingly. This process is very important during the plant-microbe interaction for their better development and protection, and also it needs to maximize the same. Similarly, the reverse action is needed for microbe to regulate host immune system to avoid an array of effective defense according to their interaction and relationship (Pel and Pieterse 2013).

Identification and differentiation between self and foreign molecule is a very crucial first step to initiate the effective immune response through pattern recognition receptors (PRRs) in the host plant cells. The PRRs identify these microbe-associated molecular patterns (MAMPs), which were earlier known as pathogen-associated molecular patterns (PAMPs) (Boller and Felix 2009). After recognition of PAMPs through the plant's PRRs, plants respond with an enhanced immune response known as PAMP-triggered immunity (PTI). The PTI is a first line of defense in plants effective against their various non-accommodated pathogens (Jones and Dangl 2006). The most prominent examples of MAMP perception in plants are recognition of conserved RNA-binding motif of bacterial cold-shock proteins (Felix and Boller 2003) and 17 amino acid-conserved domain of the Ax21 protein of *Xanthomonas* through Xa21 receptor of rice (Song et al. 1995; Lee et al. 2009). Pathogens are able to escape themselves from recognition through their evolutionary adaptations of MAMPs. Apart from this, pathogens also have the capability to secrete certain proteins known as effector proteins which modulate the plant's defense mechanisms and make them susceptible for pathogenesis. The type III secreted proteins AvrPto and AvrPtoB of *P. syringae* are well-studied case of these effector proteins. AvrPto usually binds with kinase domain of EFR, FLS2, RLKs, BAK1, and CERK1, while AvrPtoB binds FLS2 and degrades them. Hence the MAMP signaling is blocked (Göhre and Robatzek 2008; Shan et al. 2008; Xiang et al. 2008; Gimenez-Ibanez et al. 2009). In the course of coevolution

between hosts and pathogens, the plants develop resistance (R) proteins for the recognition of particular effectors of pathogens leading toward secondary immune response known as effector-triggered immunity (ETI) (Fig. 10.1). Hence, the final result of the combat between hosts and pathogens is known as zigzag model (Jones and Dangl 2006). It totally depends on balance battle between the capability of pathogens to inhibit the host's immune response and the capability of host plant to identify the pathogens and activate an array of effective defense accordingly. The recent studies discovered proteins other than effector proteins that inhibit the primary immune response of infected tissue of host plant. These proteins are present in both fungal and bacterial pathogens which escape the identification of MAMPs and interfere before microbe recognition by host plants. In addition to this, certain microbes modify their MAMPs structure to suppress the MAMPs identification. Bacteria also adopt similar escape plan from recognition by the host plants. During the search of TLR5 signaling of antagonism, type I secreted alkaline protease AprA was recognized in the supernatant of *Pseudomonas aeruginosa* (Bardoel et al. 2011). The zinc metalloprotease AprA belongs to the serralysin family in Gram-negative bacteria, and most of the members of this family are virulence factors (Stocker et al. 1995). In human cells, the addition of AprA before flagellin treatment results in weaker induced immune responses, while addition of higher

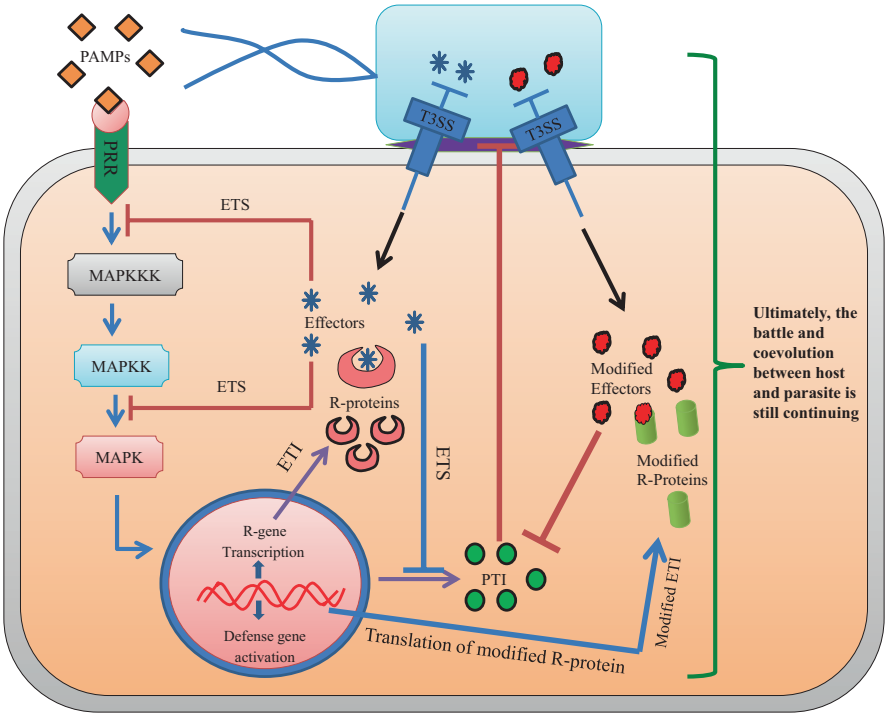

Fig. 10.1 Diagrammatic representation of battle between host and parasite which leads the coevolution in both

concentration of AprA shows no flagellin-induced immune responses. Similarly, in plants the treatment of *P. aeruginosa* aprA mutants to *A. thaliana* shows faster stomatal closure to that of induced stomatal closure through wild-type bacteria. Thus, flagellin degradation by AprA enhances the ability of *P. aeruginosa* to avoid the recognition by host immune system in both human and plants (Bardoel et al. 2011). Although various MAMPs are widely distributed and conserved in pathogenic microbes, beneficial microbes also have similar MAMPs-like pathogens. Therefore, it is important for plants to discriminate between beneficial and pathogenic microbes to get benefitted in presence of beneficial microbes. Studies are exploring good evidences which proposed that beneficial microbes are also recognized as potential invaders at the beginning and plant activate their immune response (Zamioudis and Pieterse 2012). An example of *Rhizobium*, a beneficial bacterium that forms symbiotic interaction with leguminous plants where they form nodules to reside and fix atmospheric nitrogen, is well-known. Initially, the plants recognize them as a pathogen, resulting in the stimulation and activation of defense gene expression (Kouchi et al. 2004; Lohar et al. 2006; Zamioudis and Pieterse 2012). For the successful symbiotic interaction, rhizobia involve themselves to avoid recognition in a similar fashion like pathogen. *Sinorhizobium meliloti* and *Mesorhizobium loti* bring out flagellin molecules that do not induce defense responses of plants (Felix et al. 1999; Lopez-Gomez et al. 2012). This recent study supports the importance of avoiding recognition for beneficial microorganisms. Apart from this, in various *Rhizobium* species, homologues of AprA are also found to prevent the recognition. Further, throughout the afterward stages of interaction, rhizobial colonization in plants and downregulation of expression of defense-related genes indicate that *Rhizobium* bacteria become successful to reduce host plants' defense responses (El Yahyaoui et al. 2004; Kouchi et al. 2004; Lohar et al. 2006; Moreau et al. 2011). LPS is one of the bacterial molecules from *S. meliloti* having the capability to reduce the defense response of the host plant. The cell culture of plant treated with LPS of *S. meliloti* initiates a much diluted defense response in host plants *Medicago sativa*. In another experiment, the simultaneous application of LPS of *S. meliloti* and defense elicitors from yeast shows suppressed early- and late-induced defense responses in *M. sativa*. This restrictive capability of LPS appears very narrow to *S. meliloti-M. sativa* association because nonhost plants show usual response toward the LPS of *S. meliloti* (Albus et al. 2001; Scheidle et al. 2005; Tellstrom et al. 2007). Plant growth-promoting rhizobacteria (PGPRs) form nonsymbiotic interaction with plants and enhance plant growth (Lugtenberg and Kamilova 2009). In similar trend of rhizobia, PGPRs also stimulate PTI response in plants (Bakker et al. 2007; Van Wees et al. 2008). Thus, PGPRs should reduce the degree of identification by host plants for insignificant stimulation of host defense arsenal (Millet et al. 2010). Phase variation is one of the possible strategies for PGPRs to reduce the recognition during root colonization of host plants. The reversible switching of two phenotypic stages in bacteria as per requirement according to environmental condition is known as phase variation (Davidson and Surette 2008; Van der Woude 2011). Regarding the soft interactions in between the plant roots and soil-borne mutualistic microbes, lots of more

mutualistic microbial effectors are still unexplored that have the capability to modulate the immune response of host for successful interaction of plants and beneficial microbes. In the last few years, the researcher proved that hormone-regulated network of signaling in plants is the first aim of both pathogenic and beneficial microbe (Jacobs et al. 2011; Kloppholz et al. 2011; Plett et al. 2011; Pieterse et al. 2012; Zamioudis and Pieterse 2012).

10.2.1 Plant Defense

The evolution of immune system in plant cell reaches to high level that is able to prevent the attack of the pathogenic microorganism. The common property pathogenic microorganisms are firstly detected by the primary immune response known as PAMP-triggered immunity (PTI) by the plant cell. Pathogen acquires some protein through coevolution of host-microbes interaction to suppress the PAMP-triggered immunity (PTI) and enhance the growth of pathogen and disease. To monitor the pathogen effector protein either directly or indirectly, plant cell acquired surveillance proteins (R proteins) (Chisholm et al. 2006). Pathogen has to overcome three defense systems to infect the plant: (1) shaped physical barriers, (2), a cell outer surface investigating system that finds conserved molecules of pathogen, (3) and a surveillance system that detects effector molecule that pushes into intracellular host by pathogenic organism.

Bacterial pathogen defeats the primary layer either through natural opening or wounds or by the help of enzymatic activity through damaging the layer of plant cell surface. The second layer of defense system is overcome by injecting effectors protein. The third layer is overcome either by modifying or eliminating existing effectors or by developing new effector. Wilts, galls, specks, spots, cankers, and chlorosis (yellowing) are produced by pathogenic bacterial infection during disease. For example, water and nutrient supply is blocked during wilt causing bacterial infection in vascular tissue. Recently, *Xylella fastidiosa* are most studied plant pathogenic bacteria responsible for major decrease in the production of grapes in California. To break the barriers, some plant pathogenic microbes introduced a wide range of extracellular virulence components like cutin-degrading enzymes, cell wall-degrading enzymes, etc. These enzymes are released by a type II secretary path. Cell wall-degrading enzymes cellulases, pectinases, and endoglucanases are also released via the same process. These enzymes are important for causing soft rot in plant cell by bacteria specially the *Erwinia* genus (Ade et al. 2007).

The plants initiate signal-transduction cascades after the sensation of PAMPs that start basal defenses. The deposition of callose and silicone for cell wall strenghtening, production of ethylene and reactive oxygen species (ROS), trancriptional activation of different defence gene carrying PR-genes, and post-transcriptional inhibition of auxin signaling are the various mechanisms that come under basal defence responses. The bacterial flagellin is a well-described molecule that induces basal defense in host plants. The bacterial flagellin (flg22) is a 22 amino acid long peptide which activates basal defense response in plants, namely, generation of ROS

and alkalinization of the extracellular matrix (Felix et al. 1999). In *Arabidopsis* the basal response induced by flg22 also includes closure of stomata to inhibit the bacterial entrance. In *Arabidopsis*, the flg22 is recognized by a transmembrane receptor kinase PRR FLS2. The fls2 mutant *Arabidopsis* plants are more sensitive to *Pseudomonas syringae* pv. *tomato* (PstDC3000) infection, when bacteria are used on leaf surface for infection in comparison to infiltration of bacteria into intercellular spaces of leaf (Zipfel et al. 2004). These experiments suggest that FLS2 is one of the factors responsible for the activation of initial defense responses and inhibits the entry of bacterial pathogen *Pseudomonas syringae* pv. *tomato* into plants. The recent reports also indicate that flg22 inhibit the expression of mRNA of certain auxin receptor genes (Navarro et al. 2006). It is quite interesting that study showing downregulation was proved through micro (RNA) miR393 via posttranscriptional modifications. The flg22 enhances the expression of miR393, and downstream action of miR393 results in the degradation of mRNAs responsible for the action of auxin receptor genes, AFB2, AFB3, and TIR1. *P. syringae* is well-known for the production of auxin for their own benefit. The inhibition of auxin signaling leads to basal resistance because overexpression of auxin makes it more sensitive to *P. syringae* (Navarro et al. 2006). Apart from flagellin, the elongation factor Tu (EF-Tu) present in profuse amount in increasing bacterial population also functions as activator of basal defense response in plants (Kunze et al. 2004). EF-Tu is 43 kDa protein and 18 amino acids present at N-terminus (elf18) of this, also able to induce basal defense response by itself. In *Arabidopsis*, elf18 is recognized by EFTu receptor (EFR), a receptor-like kinase (RLK). The intracellular kinase domain and extracellular leucine-rich repeats (LRR) domain of EFR has structural similarities with FLS2 (Zipfel et al. 2006). Hence, it is clear that the perception and recognition of bacterial pathogens by plants and subsequent initiation of basal defense is not relying on a single factor. The basal defense response can also be induced by various other factors. The plants using multiple PAMPs during the perception of microbes are totally dependent on the recognition of pathogens and activation of defense response. The initiation of defense responses in plants is triggered by the perception of PAMPs; therefore, it is known as PAMP-triggered immunity (PTI) (Jones and Dangl 2006).

The pathogenic bacteria adopt one strategy to overcome the defense of plants which is the secretion of coronatine (COR), a small molecule and virulence factor that mimics as jasmonic acid hormone secreted by plants (Melotto et al. 2006). COR-mediated inhibition of stomatal closure is induced by suppression of PAMP-stimulated abscisic acid (ABA) signaling within guard cell. The bacterial mutant lacking coronatine shows diminished virulence to that of wild-type bacteria during the inoculation onto the plant leaf surface for infection. The colonization on host plants and inhibition of basal defense response through particular effector proteins is another strategy for causing disease also reported in bacterial pathogens. These effectors are normally translocated straight into the host cell from bacterial cell employing type III secretion system (T3SS), most common in *Ralstonia solanacearum*, *Erwinia*, all *Pseudomonas* pathovars, and *Xanthomonas* (Hueck 1998).

When T3SS effectors minimize or stop basal defense signaling, plants adopt secondary defense response and recognize the existing T3SS effectors within the plant cell, a best example of coevolution (Fig. 10.1). This recognition system uses the receptors present inside the plant cell, encoded by resistance (R) gene. Most of the R proteins present in plants include nucleotide-binding site and leucine-rich repeat (NBS-LRR). The NBS-LRR proteins regulate resistance against majority of plant pathogens. Stimulation of R protein by effector protein of pathogen triggers the initiation of programmed cell death (PCD) at the site of pathogen infection and surrounding tissues in plants. This R protein-regulated resistance is known as effector-triggered immunity (ETI), and localized PCD is known as hypersensitive response (HR). Apart from the stimulation of PCD, R protein-regulated resistance also includes the generation nitric oxide (NO) and reactive oxygen species (ROS) which further have the capability of signaling molecule and antimicrobial activity. ROS along with NO induce the transcriptional activation of defense genes and HR in plants. The superoxide anion (O_2^-) and NO in combination form highly toxic peroxynitrite ($ONOO^-$), which participates either directly or indirectly in death of pathogens and host plant's cell (Saito et al. 2006). Here the pathogens evolve themselves for self-defense by secreting various enzymes, free radical scavengers, and production of antioxidants to fight against abovementioned plant defense molecules. An emerging new theory explores the capability of certain R proteins to trigger gene silencing through small interfering RNA (siRNA) in pathogens. The RPS2 is a R protein in *Arabidopsis* which has been experimentally proven to enhance siRNA nat-siRNAATGB2 to silence the PPRL gene (Katiyar-Agarwal et al. 2006). The RPS2-mediated disease resistance is diminished by overexpression of PPRL gene in *P. syringae*. Therefore, it seems that the signaling pathway mediated by RPS2 downregulates the expression of PPRL, and full elicitation of RPS2-mediated signaling needs the degradation of PPRL mRNA through siRNA. Now, the bacterial pathogens had any coevolution to enhance their pathogenesis against siRNA, and miRNA-regulated resistance is still yet to be explored. The recognition of bacterial effector molecule by plant R proteins at molecular level might be either direct or indirect. The model representing direct recognition is also known as "ligand-receptor model." This model hypothesized that the R proteins act as receptor and bind to ligand effector proteins of the pathogen directly (Deslandes et al. 2003). The model representing indirect recognition is known as "guard model" which shows the spots of changes occur in host plant protein by effectors. This model is very common in bacterial pathogens and often has been explored for effector molecules like AvrPtoB, AvrRpt2, AvrRpm1/AvrB, and AvrPphB in *P. syringae* (Kim et al. 2002; Mackey et al. 2002; Axtell and Staskawicz 2003; Ade et al. 2007).

Indirect recognition model is supported by the interaction of two R proteins RPM1 and RPS2 with RIN 4 protein in *Arabidopsis*. Especially RPM1 interacts with the phosphorylated form of RIN4 induced by the interaction with AvrRpm1 and AvrB effector proteins of the *P. syringae* (Mackey et al. 2002). Additionally, indirect recognition model can also be elaborated with the interaction of RPS5 protein of *Arabidopsis* with PSB1 protein. PSB1 protein is also part of *Arabidopsis*

itself cleaved by a cysteine protease effector AvrPphB of *P. syringae*. Cleaved product of PBS1 is recognized by R protein RPS5 leading to activation of HR (Ade et al. 2007).

These traits related to resistance are introduced into the plants through resistance breeding. Pathogens also have the mechanism to overcome such type of resistance by developing new pathogenic strains that leads to the next round of resistance breeding. This everlasting battle during host-pathogen interaction has received much more attention from researchers. Recently, various studies provided the insight lying behind this phenomenon. Similar to suppression of PTI, bacteria also have developed the mechanism to suppress ETI. The development of a new effector is one of these mechanisms to suppress R gene-mediated hypersensitive response related to disease resistance. Hop-AB1 and HopZ3 are two effector proteins of *P. syringae* pv. *syringae* which are able to suppress programmed cell death initiated by some other effector molecules in *N. benthamiana* (Vinatzer et al. 2006). HopAB1 and HopZ3 mutants from *P. syringae* pv. *syringae* restores its capability to provoke the HR. Similarly, some other effectors from Pst DC3000 such as AvrPtoB, HopPtoE, AvrPphEPto, AvrPpiB1Pto, and HopPtoF are capable to retard effector-triggered programmed cell death in tobacco. *P. syringae* pv. *phaseolicola* also has the ability to suppress the effector-triggered PCD via altered effector in bean in a cultivar-specific manner. Recently immunity-associated protein AtMIN7 was found to be destroyed by an effector HopM1 from *P. syringae* leading to the development of disease in the model plant *Arabidopsis* (Nomura et al. 2005). Coevolution of Avr gene in context to host R gene defines the suppression of effector-triggered HR, an important component of ETI leading to development of disease by bacterial pathogen *P. syringae.*

10.2.2 Host Specificity

Host specificity of pathogens basically depends upon growth, colonization, and infection ability to their respective hosts (Kirzinger and Stavrinides 2012). Bacterial pathogens show various mechanisms of host specificity by modulating their genome-like duplication, point mutation and horizontal gene transfer (HGT), etc. Certain bacterial pathogens have very broad host range, and the earlier studies of symbiotic bacteria determine a single regulatory gene for the host specificity (Mandel et al. 2009). Divergent gene expression of bacterial single regulatory gene expands the successful colonization on various host species. The regulation of gene expression might be the key factor for host specificity in bacterial pathogens contributing to the emergence of new disease due to development of new pathogenic strains. The molecular interactions of hosts and pathogens are the key factors that demonstrate host specificity in bacterial pathogens (Pan et al. 2014). Very small changes in host-pathogen interactions might cause great modification in host range and state of disease intensity in bacterial strains (Killiny and Almeida 2011). The interaction of bacteria with other organisms is greatly diversified from the biofilm formation to mutual interaction and up to pathogenic associations. The formation

and synthesis of certain specific proteins play an important role in such plant pathogen interactions. Among them certain proteins are toxic and enter within host cells and modify their physiology and colonization for the production of effector proteins leading to disease development (Tseng et al. 2009). The allelic difference in effector molecules in all pathogens is also an important factor in host specificity (Alfano and Collmer 2004). The genetic engineering strategies are not so easy due to plant defense response, inhibition of bacterial "vir" factors, and production of certain antimicrobial compounds for effective disease management required for the protection of host (Melchers and Stuiver 2000). A considerable amount of protection against various diseases has been accomplished, but the resistance against broad range remains to be explored (Rivero et al. 2012).

10.2.3 Environmental Effects

A prominent aspect of hosts and parasites basically depends on genetics of organisms, and hence it can be determined by coevolution. In laboratory conditions the evaluation of infection is determined by the environment in which hosts and parasites interact with other. The interaction of hosts and parasites is also regulated by environmental conditions for the strength and specificity. Additionally, the various constituents of interaction for host-parasite fitness are differentially manipulated by environment. In spite of all these conditions, the environmental variations are not frequently included in experiments studying the coevolution and theoretical models. However, most of the interactions of host-parasite occur in heterogeneous environments; hence it is important to include fluctuating environments during the theoretical and experimental studies of host-parasite coevolution (Wolinska and King 2009).

The traits of life history are not only dependent on specific genotype but also mediated by environmental conditions. The phenomena "reaction norm" result into versatile phenotypes from a single genotype according to environmental conditions (Stearns 1992). During extreme cases or non parallel slopes of reaction norm, if genotype A is better than genotype B under one set of environmental conditions and the case is opposite in another set of environmental conditions, the cross is known as genotype-by-environment interaction. Subsequently, the genotypic variation is conserved in population, if there is any possibility of adaptation under different environmental conditions throughout time and/or space (Byers 2005). Several studies have proposed continuously that the environmental conditions modify the strength of selection during host-parasite interactions and the host genotype experience less or more problem during infection according to environmental settings (Sandland and Minchella 2003). In 1960, the concept of "disease triangle" was suggested for consequence of disease in hosts (plants). Three factors basically involved in this concept for the regulation are the hosts, the parasites, and the environment. Before 20 years, the scientists of plant breeding group recognized another term "genotype-by-environment interactions" in the research field of host-parasite interactions. This term advocated that comparative resistance of cultivars to their

particular parasitic strains is governed by environmental conditions (Browder 1985). One of the best examples of this concept is in oat cultivar which gets fewer infections by rust fungus during the winter season, while high infection rate is found in summer season. However, this conventional process is entirely reversed during the infection of the same cultivar with another fungal strain.

The experimental studies are going on in the field of host-genotype-environment (H-G-E) interactions and parasite-genotype-environment (P-G-E) interactions under various conditions along with different genotypes of hosts and parasites (HG-PG-E). The statistical tests of these three-way interactions were conducted in barley growth and aphid reproduction. The various genotypes and presence of rhizobacterium (the rhizobacterium is taken as "environment" in this experiment) both affect the growth in barley and reproduction in aphid resulting in substantial HG-PG-E interactions (Tetard-Jones et al. 2007). The environment (rhizobacterium here) substantially changed the selection specificity in 31 out of 92 performed tests.

The nutrient availability and temperature are environmental factors tested most frequently during host-parasite interactions. Temperature greatly affects the physiological, biochemical, and behavioral processes in both hosts and parasites. In several studies, the temperature enhances development of parasites and exploitation of hosts and therefore disease occurrence (Thomas and Blanford 2003). The nutrient variability is able to alter specificity of hosts and parasites. For example, certain susceptible host genotypes can use the supplementary food during nutrient variability for their defense, while the rich nutrient availability might provide more suitable environment for parasite infection in other hosts (Laine 2007).

10.2.4 Epigenetics and Transgenerational Resistance

Plants can achieve immunity within their own lifetime, and pathogen interaction with plants also results in epigenetic modifications in cell that lead to immunization in the next generations. A study shows that the treatment of *Arabidopsis* with the β-amino-butyric acid (BABA), a SAR inducer component, and the avirulent strains of *P. syringae* pv. *tomato* inoculated on tomato plant led to enhanced disease resistance up to the next generation with faster and stronger expression of SA-dependent defense-related genes (Luna et al. 2012; Slaughter et al. 2012). Offspring from primed plants also shows enhanced resistance to the biotrophic oomycete *Hyaloperonospora arabidopsidis*. This phenotype persists in further one stress-free generation followed by increased resistance during pathogen attack in the next upcoming generations (Slaughter et al. 2012). Mutation in the npr1 gene can block the transgenerational resistance (Luna et al. 2012).

Transgenerational resistance of plants is regulated by certain epigenetic modifications such as DNA methylation and chromatin rearrangements. Control of transgenerational stress memory takes place through somatic homologous recombination of gene. *Arabidopsis* plants treated with flg22 or ultraviolet C show enhanced somatic homologous recombination in both parental and in the next four subsequent

generations (Molinier et al. 2006). Further studies focusing on the molecular mechanism of passing immunological memory to its subsequent generations are still in progress for better disease control mechanism in plants (Henry et al. 2013).

10.3 Conclusion

Plants have various cell surface receptors to discriminate the different PAMPs common in most of the microbes. Apart from isolating bacterial PAMP receptors, the research in host-parasite coevolution field is rapidly progressing toward the recognition of PAMPs and their respective receptors. Several PAMPs have been identified with their own receptor. An individual microorganism will efficiently be distinguished by several PAMP receptors. Many labs are specifying the enzymatic activities of effector molecules and recognizing their targets in host. The recognition of effector molecule targets will open the molecular action of PTI because the primary action of effector molecules is inhibition of PTI. Apart from this, the pathogen effector molecules may also contain virulence components which can be necessary for disease incidence. The battle between host-parasite interactions is a continuous process for their coevolution. In the coming years, there may be some new mechanisms revealed about host-parasite interactions. Explanation of mechanisms involved in controlling the coevolution of host-parasite interactions will be greatly affected by new techniques including rapid genome sequencing, gene editing, and development of computational methods along with bioinformatics to study the available genome information. Concurrent researches that make use of post-genomic technologies including system biology perspective will finally provide understanding of expression of all genes and proteins in hosts and parasites that are expressed during the battle of resistance simultaneously. These technologies will be very helpful to explore the complicated interactions between various pathways expressed during coevolution of hosts and parasites. Finally, the complete perception of molecular basis of host-parasite interactions will permit execution of these explored researches to make the hosts more resistant by adding novel combination of genes that are durable and identify various range of parasites.

References

Ade J, DeYoung BJ, Golstein C et al (2007) Indirect activation of a plant nucleotide binding site-leucine-rich repeat protein by a bacterial protease. Proc Natl Acad Sci U S A 104(7):2531–2536

Akira S, Uematsu S, Takeuchi O (2006) Pathogen recognition and innate immunity. Cell 124(4):783–801

Albus U, Baier R, Holst O et al (2001) Suppression of an elicitor-induced oxidative burst reaction in *Medicago sativa* cell cultures by *Sinorhizobium meliloti* lipopolysaccharides. New Phytol 151(3):597–606

Alfano JR, Collmer A (2004) Type III secretion system effector proteins: double agents in bacterial disease and plant defense. Annu Rev Phytopathol 42:385–414

Allen RL, Bittner-Eddy PD, Grenville-Briggs LJ et al (2004) Host-parasite coevolutionary conflict between *Arabidopsis* and downy mildew. Science 306(5703):1957–1960

Alvarez AM (2000) Black rot of crucifers. In: Slusarenko AJ, Fraser RSS, van Loon LC (eds) Mechanisms of resistance to plant diseases. Kluwer Academic Publishers, Dordrecht, pp 21–52
Axtell MJ, Staskawicz BJ (2003) Initiation of RPS2-specified disease resistance in *Arabidopsis* is coupled to the AvrRpt2-directed elimination of RIN4. Cell 112(3):369–377
Bakker PA, Pieterse CM, van Loon LC (2007) Induced systemic resistance by fluorescent *Pseudomonas* spp. Phytopathology 97(2):239–243
Bardoel BW, van der Ent S, Pel MJ et al (2011) *Pseudomonas* evades immune recognition of flagellin in both mammals and plants. PLoS Pathog 7(8):e1002206
Bashan Y, Okon Y, Henis Y (1982) Long-term survival of *Pseudomonas syringae* pv. *tomato* and *Xanthomonas campestris* pv. *vesicatoria* in tomato and pepper seeds. Phytopathology 72(9):1143–1144
Bell G, Smith JM (1987) Short-term selection for recombination among mutually antagonistic species. Nature 328(6125):66–68
Berendsen RL, Pieterse CM, Bakker PA (2012) The rhizosphere microbiome and plant health. Trends Plant Sci 17(8):478–486
Boller T, Felix G (2009) A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. Annu Rev Plant Biol 60:379–406
Browder L (1985) Parasite: host: environment specificity in the cereal rusts. Annu Rev Phytopathol 23(1):201–222
Buonaurio R, Stravato V, Cappelli C (1996) Occurrence of *Pseudomonas syringae* pv. *tomato* race 1 in Italy on Pto gene-bearing tomato plants. J Phytopathol 144(9–10):437–440
Burdon J, Thrall P (1999) Spatial and temporal patterns in coevolving plant and pathogen associations. Am Nat 153(S5):S15–S33
Buzi A, Chilosi G, Magro P (2004) Induction of resistance in melon seedlings against soil-borne fungal pathogens by gaseous treatments with methyl jasmonate and ethylene. J Phytopathol 152(8–9):491–497
Byers DL (2005) Evolution in heterogeneous environments and the potential of maintenance of genetic variation in traits of adaptive significance. Genetica 123(1–2):107–124
Carius HJ, Little TJ, Ebert D (2001) Genetic variation in a host-parasite association: potential for coevolution and frequency-dependent selection. Evolution 55(6):1136–1145
Chan JW, Goodwin PH (1999) The molecular genetics of virulence of *Xanthomonas campestris*. Biotechnol Adv 17(6):489–508
Chisholm ST, Coaker G, Day B et al (2006) Host-microbe interactions: shaping the evolution of the plant immune response. Cell 124(4):803–814
Dangl JL, Jones JDG (2001) Plant pathogens and integrated defence responses to infection. Nature 411(6839):826–833
Darrasse A, Darsonval A, Boureau T et al (2010) Transmission of plant-pathogenic bacteria by nonhost seeds without induction of an associated defense reaction at emergence. Appl Environ Microbiol 76(20):6787–6796
Darsonval A, Darrasse A, Meyer D et al (2008) The type III secretion system of *Xanthomonas fuscans* subsp. *fuscans* is involved in the phyllosphere colonization process and in transmission to seeds of susceptible beans. Appl Environ Microbiol 74(9):2669–2678
Darsonval A, Darrasse A, Durand K et al (2009) Adhesion and fitness in the bean phyllosphere and transmission to seed of *Xanthomonas fuscans* subsp. *fuscans*. Mol Plant-Microbe Interact 22:747–757
Davidson CJ, Surette MG (2008) Individuality in bacteria. Annu Rev Genet 42:253–268
de Torres-Zabala M, Truman W, Bennett MH et al (2007) *Pseudomonas syringae* pv. *tomato* hijacks the *Arabidopsis* abscisic acid signalling pathway to cause disease. EMBO J 26(5):1434–1443
Deslandes L, Olivier J, Peeters N et al (2003) Physical interaction between RRS1-R, a protein conferring resistance to bacterial wilt, and PopP2, a type III effector targeted to the plant nucleus. Proc Natl Acad Sci U S A 100(13):8024–8029
Devash Y, Okon Y, Henis Y (1980) Survival of *Pseudomonas tomato* in soil and seeds. J Phytopathol 99(2):175–185

El Yahyaoui F, Küster H, Amor BB et al (2004) Expression profiling in *Medicago truncatula* identifies more than 750 genes differentially expressed during nodulation, including many potential regulators of the symbiotic program. Plant Physiol 136(2):3159–3176

Fall R, Wolber PK (1995) Biochemistry of bacterial ice nuclei. In: Lee RE, Warren GJ, Gusta LV (eds) Biological ice nucleation and its applications. American Phytopathological Society, St. Paul, pp 63–83

Felix G, Boller T (2003) Molecular sensing of bacteria in plants-the highly conserved RNA-binding motif RNP-1 of bacterial cold shock proteins is recognized as an elicitor signal in tobacco. J Biol Chem 278(8):6201–6208

Felix G, Duran JD, Volko S et al (1999) Plants have a sensitive perception system for the most conserved domain of bacterial flagellin. Plant J 18(3):265–276

Fessehaie A, Walcott RR (2005) Biological control to protect watermelon blossoms and seed from infection by *Acidovorax avenae* subsp *citrulli*. Phytopathology 95(4):413–419

Gandon S, Ebert D, Olivieri I et al (1998) Differential adaptation in spatially heterogeneous environments and host-parasite coevolution. In: Mopper S, Strauss SY (eds) Genetic structure and local adaptation in natural insect populations: effects of ecology, life history, and behavior. Chapman and Hall, New York, pp 325–342

Gilbertson RL, Maxwell DP (1992) Common bacterial blight of bean. In: Chaube HS, Singh US, Mukhopadhay AN (eds) Diseases of international importance, vol 2. Prentice Hall, Englewood Cliffs, pp 18–39

Gimenez-Ibanez S, Hann DR, Ntoukakls V et al (2009) AvrPtoB targets the LysM receptor kinase CERK1 to promote bacterial virulence on plants. Curr Biol 19(5):423–429

Gohre V, Robatzek S (2008) Breaking the barriers: microbial effector molecules subvert plant immunity. Annu Rev Phytopathol 46:189–215

Hamilton WD, Axelrod R, Tanese R (1990) Sexual reproduction as an adaptation to resist parasites (a review). Proc Natl Acad Sci U S A 87(9):3566–3573

Henry E, Yadeta KA, Coaker G (2013) Recognition of bacterial plant pathogens: local, systemic and transgenerational immunity. New Phytol 199(4):908–915

Hirano SS, Upper CD (1990) Population biology and epidemiology of *Pseudomonas syringae*. Annu Rev Phytopathol 28:155–177

Hirano SS, Upper CD (2000) Bacteria in the leaf ecosystem with emphasis on *Pseudomonas syringae*-a pathogen, ice nucleus, and epiphyte. Microbiol Mol Biol Rev 64(3):624–653

Hueck CJ (1998) Type III protein secretion systems in bacterial pathogens of animals and plants. Microbiol Mol Biol Rev 62(2):379–433

Ichinose Y, Taguchi F, Mukaihara T (2013) Pathogenicity and virulence factors of *Pseudomonas syringae*. J Gen Plant Pathol 79(5):285–296

Jacobs S, Zechmann B, Molitor A et al (2011) Broad-spectrum suppression of innate immunity is required for colonization of *Arabidopsis* roots by the fungus *Piriformospora indica*. Plant Physiol 157(1):531–531

Janisiewicz WJ, Marchi A (1992) Control of storage rots on various pear cultivars with a saprophytic strain of *Pseudomonas syringae*. Plant Dis 76:555–560

Jones JDG, Dangl JL (2006) The plant immune system. Nature 444(7117):323–329

Katiyar-Agarwal S, Morgan R, Dahlbeck D et al (2006) A pathogen-inducible endogenous siRNA in plant immunity. Proc Natl Acad Sci U S A 103(47):18002–18007

Killiny N, Almeida RP (2011) Gene regulation mediates host specificity of a bacterial pathogen. Environ Microbiol Rep 3(6):791–797

Kim YJ, Lin NC, Martin GB (2002) Two distinct *Pseudomonas* effector proteins interact with the Pto kinase and activate plant immunity. Cell 109(5):589–598

Kirzinger MWB, Stavrinides J (2012) Host specificity determinants as a genetic continuum. Trends Microbiol 20(2):88–93

Kloppholz S, Kuhn H, Requena N (2011) A secreted fungal effector of *Glomus intraradices* promotes symbiotic biotrophy. Curr Biol 21(14):1204–1209

Kouchi H, Shimomura K, Hata S et al (2004) Large-scale analysis of gene expression profiles during early stages of root nodule formation in a model legume, *Lotus japonicus*. DNA Res 11(4):263–274

Kunze G, Zipfel C, Robatzek S et al (2004) The N terminus of bacterial elongation factor Tu elicits innate immunity in *Arabidopsis* plants. Plant Cell 16(12):3496–3507

Laine AL (2007) Pathogen fitness components and genotypes differ in their sensitivity to nutrient and temperature variation in a wild plant-pathogen association. J Evol Biol 20(6):2371–2378

Lawton M, MacNeill B (1986) Occurrence of race 1 of *Pseudomonas syringae* pv. *tomato* on field tomato in southwestern Ontario. Can J Plant Pathol 8(1):85–88

Lee SW, Han SW, Sririyanum M et al (2009) A type I–secreted, sulfated peptide triggers XA21-mediated innate immunity. Science 326(5954):850–853

Lohar DP, Sharopova N, Endre G et al (2006) Transcript analysis of early nodulation events in *Medicago truncatula*. Plant Physiol 140(1):221–234

Lopez-Gomez M, Sandal N, Stougaard J et al (2012) Interplay of flg22-induced defence responses and nodulation in *Lotus japonicus*. J Exp Bot 63(1):393–401

Lugtenberg B, Kamilova F (2009) Plant-growth-promoting rhizobacteria. Annu Rev Microbiol 63:541–556

Luna E, Bruce TJ, Roberts MR et al (2012) Next-generation systemic acquired resistance. Plant Physiol 158(2):844–853

Mackey D, Holt BF, Wiig A et al (2002) RIN4 interacts with *Pseudomonas syringae* type III effector molecules and is required for RPM1-mediated resistance in *Arabidopsis*. Cell 108(6):743–754

Maki LR, Galyan EL, Chang-Chien MM et al (1974) Ice nucleation induced by *Pseudomonas syringae*. Appl Microbiol 28(3):456–459

Mandel MJ, Wollenberg MS, Stabb EV et al (2009) A single regulatory gene is sufficient to alter bacterial host range. Nature 458(7235):215–218

McCarter S, Jones J, Gitaitis R et al (1983) Survival of *Pseudomonas syringae* pv. *tomato* in association with tomato seed, soil, host tissue, and epiphytic weed hosts in Georgia. Phytopathology 73(10):1393–1398

Melchers LS, Stuiver MH (2000) Novel genes for disease-resistance breeding. Curr Opin Plant Biol 3(2):147–152

Melotto M, Underwood W, Koczan J et al (2006) Plant stomata function in innate immunity against bacterial invasion. Cell 126(5):969–980

Mercier J, Lindow SE (2000) Role of leaf surface sugars in colonization of plants by bacterial epiphytes. Appl Environ Microbiol 66(1):369–374

Mew T, Vera Cruz C, Medalla E (1992) Changes in race frequency of *Xanthomonas oryzae* pv. *oryzae* in response to rice cultivars planted in the Philippines. Plant Dis 76(10):1029–1032

Milijašević S, Todorović B, Rekanović E et al (2009) Races and hosts of *Pseudomonas syringae* pv. *tomato* in Serbia. Arch Biol Sci Belgrad 61:93–102

Millet YA, Danna CH, Clay NK et al (2010) Innate immune responses activated in *Arabidopsis* roots by microbe-associated molecular patterns. Plant Cell 22(3):973–990

Mishina TE, Zeier J (2007) Pathogen-associated molecular pattern recognition rather than development of tissue necrosis contributes to bacterial induction of systemic acquired resistance in *Arabidopsis*. Plant J 50(3):500–513

Molinier J, Ries G, Zipfel C et al (2006) Transgeneration memory of stress in plants. Nature 442(7106):1046–1049

Moreau S, Verdenaud M, Ott T et al (2011) Transcription reprogramming during root nodule development in *Medicago truncatula*. PLoS One 6(1):e16463

Navarro L, Dunoyer P, Jay F et al (2006) A plant miRNA contributes to antibacterial resistance by repressing auxin signaling. Science 312(5772):436–439

Nelson EB (2004) Microbial dynamics and interactions in the spermosphere. Annu Rev Phytopathol 42:271–309

Nomura K, Melotto M, He SY (2005) Suppression of host defense in compatible plant–*Pseudomonas syringae* interactions. Curr Opin Plant Biol 8(4):361–368

Nürnberger T, Brunner F, Kemmerling B et al (2004) Innate immunity in plants and animals: striking similarities and obvious differences. Immunol Rev 198(1):249–266
Pan X, Yang Y, Zhang JR (2014) Molecular basis of host specificity in human pathogenic bacteria. Emerg Microbes Infect 3(3):e23
Pel MJ, Pieterse CM (2013) Microbial recognition and evasion of host immunity. J Exp Bot 64(5):1237–1248
Pieterse CM, Van der Does D, Zamioudis C et al (2012) Hormonal modulation of plant immunity. Annu Rev Cell Dev Biol 28:489–521
Plett JM, Kemppainen M, Kale SD et al (2011) A secreted effector protein of *Laccaria bicolor* is required for symbiosis development. Curr Biol 21(14):1197–1203
Pohronezny K, Stall RE, Canteros BI et al (1992) Sudden shift in the prevalent race of *Xanthomonas campestris* pv. *vesicatoria* in pepper fields in southern Florida. Plant Dis 76(2):118–120
Preston GM (2000) *Pseudomonas syringae* pv. *tomato*: the right pathogen, of the right plant, at the right time. Mol Plant Pathol 1(5):263–275
Rivero M, Furman N, Mencacci N et al (2012) Stacking of antimicrobial genes in potato transgenic plants confers increased resistance to bacterial and fungal pathogens. J Biotechnol 157(2):334–343
Saito S, Yamamoto-Katou A, Yoshioka H et al (2006) Peroxynitrite generation and tyrosine nitration in defense responses in tobacco BY-2 cells. Plant Cell Physiol 47(6):689–697
Sandland GJ, Minchella DJ (2003) Costs of immune defense: an enigma wrapped in an environmental cloak? Trends Parasitol 19(12):571–574
Scheidle H, Groß A, Niehaus K (2005) The lipid A substructure of the *Sinorhizobium meliloti* lipopolysaccharides is sufficient to suppress the oxidative burst in host plants. New Phytol 165(2):559–566
Shan L, He P, Li J et al (2008) Bacterial effectors target the common signaling partner BAK1 to disrupt multiple MAMP receptor-signaling complexes and impede plant immunity. Cell Host Microbe 4(1):17–27
Sheppard J (1998) Seed-borne pathogens of vegetable and flower seeds: their devastation, identification and control. Seed Technol 20(2):187–197
Slaughter A, Daniel X, Flors V et al (2012) Descendants of primed *Arabidopsis* plants exhibit resistance to biotic stress. Plant Physiol 158(2):835–843
Song WY, Wang GL, Chen LL et al (1995) A receptor kinase-like protein encoded by the rice disease resistance gene, Xa21. Science 270(5243):1804
Spoel SH, Dong X (2012) How do plants achieve immunity? Defence without specialized immune cells. Nat Rev Immunol 12(2):89–100
Stearns SC (1992) The evolution of life histories. Oxford University Press, Oxford
Stöcker W, Grams F, Reinemer P et al (1995) The metzincins-topological and sequential relations between the astacins, adamalysins, serralysins, and matrixins (collagenases) define a super family of zinc-peptidases. Protein Sci 4(5):823–840
Tellström V, Usadel B, Thimm O et al (2007) The lipopolysaccharide of *Sinorhizobium meliloti* suppresses defense-associated gene expression in cell cultures of the host plant *Medicago truncatula*. Plant Physiol 143(2):825–837
Tétard-Jones C, Kertesz MA, Gallois P et al (2007) Genotype-by-genotype interactions modified by a third species in a plant-insect system. Am Nat 170(3):492–499
Thomas MB, Blanford S (2003) Thermal biology in insect-parasite interactions. Trends Ecol Evol 18(7):344–350
Thompson JN (1999) Specific hypotheses on the geographic mosaic of coevolution. Am Nat 153(S5):S1–S14
Thrall PH, Burdon JJ (1997) Host-pathogen dynamics in a metapopulation context: the ecological and evolutionary consequences of being spatial. J Ecol 85(6):743–753
Tseng TT, Tyler BM, Setubal JC (2009) Protein secretion systems in bacterial-host associations, and their description in the gene ontology. BMC Microbiol 9(1):1
Valls M, Genin S, Boucher C (2006) Integrated regulation of the type III secretion system and other virulence determinants in *Ralstonia solanacearum*. PLoS Pathog 2(8):e82

Van der Woude MW (2011) Phase variation: how to create and coordinate population diversity. Curr Opin Microbiol 14(2):205–211

Van Wees SC, Van der Ent S, Pieterse CM (2008) Plant immune responses triggered by beneficial microbes. Curr Opin Plant Biol 11(4):443–448

Vinatzer BA, Teitzel GM, Lee MW et al (2006) The type III effector repertoire of *Pseudomonas syringae* pv. *syringae* B728a and its role in survival and disease on host and non-host plants. Mol Microbiol 62(1):26–44

Williams PH (1980) Black rot: a continuing threat to world crucifers. Plant Dis 64(8):736–742

Wolinska J, King KC (2009) Environment can alter selection in host–parasite interactions. Trends Parasitol 25(5):236–244

Xiang T, Zong N, Zou Y et al (2008) *Pseudomonas syringae* effector AvrPto blocks innate immunity by targeting receptor kinases. Curr Biol 18(1):74–80

Xin XF, He SY (2013) *Pseudomonas syringae* pv. *tomato* DC3000: a model pathogen for probing disease susceptibility and hormone signaling in plants. Annu Rev Phytopathol 51:473–498

Zamioudis C, Pieterse CM (2012) Modulation of host immunity by beneficial microbes. Mol Plant-Microbe Interact 25(2):139–150

Zipfel C, Robatzek S, Navarro L et al (2004) Bacterial disease resistance in *Arabidopsis* through flagellin perception. Nature 428(6984):764–767

Zipfel C, Kunze G, Chinchilla D et al (2006) Perception of the bacterial PAMP EF-Tu by the receptor EFR restricts *Agrobacterium*-mediated transformation. Cell 125(4):749–760