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ARMS RACE BETWEEN PHYTOPATHOGENS AND HOST PLANTS: A SIMILITUDE OF TWO NATIONS AT WAR

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ABSTRACT

Phytopathogens have coexisted with their host plants from the beginning of their existence, relying on them for shelter and nutrients to survive, therefore leading to a constant attack of host plants. On the other hand, host plants have evolved several strategies to defend themselves against pathogen attack. The attacks and counter-attacks can be likened to an "arms race", between two nations at war. Pathogens evolved strategies (arms) which include exertion of mechanical forces and secretion of biochemical substances such as enzymes and growth regulators, to attack susceptible host plants. These strategies are facilitated by the inability of the host plants to recognize certain molecules, known as PAMPs (Pathogen-Associated Molecular Patterns) from the pathogens. In a counter attack, host plants have developed receptors to recognize the presence of pathogens and PAMPs. This recognition activates PAMP-triggered immunity (PTI), thereby making the hitherto host plants resistant. Pathogens have further evolved another set of "arms", known as secretion systems and effector genes, whose gene products suppress PTI, thus causing diseases. As the arms race continued, plants have evolved effector-triggered immunity (ETI) by developing intracellular resistance genes, thereby resulting to a hypersensitive reaction that follows the gene-for-gene concept. Presently, some pathogens have developed mechanisms for the suppression of ETI. In some plant-pathosystems, the plants are winning, while in others, the pathogens are winning. Phytopathological research should therefore be directed towards assisting susceptible plants to defeat phytopathogens, in order to ensure food security.

Keywords: Arms race, Phytopathogen, Strategies, MAMP, PAMP, PTI, ETI

INTRODUCTION

Phytopathogens have coexisted with their host plants from the beginning of their existence. Plant pathogens rely entirely on their hosts for shelter and nutrients to survive. This total reliance has led to a constant attack of host plants by phytopathogens, resulting to physiological dysfunction in host plants, leading to a diseased condition **(Shittu *et al.*, 2017)**. On the other end of the spectrum, host plants also deploy a myriad of structural and biochemical defence mechanisms to prevent or ameliorate the effects of phytopathogens. The resultant outcome of these attacks by phytopathogens and counter defence mechanisms by host plants has led to complex and dynamic interactions between plants and phytopathogens. This interaction is a battle that has existed over evolutionary periods and could be likened to a biological "arms race" **(Holub, 2001)**. The idea of this "arms race" is that one species evolves survival strategies in response to changes in another competing species **(Dawkins and Krebs, 1979)**, comparable to two nations at war with each other.

ARMS RACE BETWEEN PLANTS AND PHYTOPATHOGENS

During evolutionary periods, phytopathogens have learnt to rely entirely on plants for shelter and nutrients. This total reliance has led phytopathogens to develop strategies (arms) to constantly attack host plants. Plants have also developed strategies (arms) to defend themselves against pathogen attack, thus leading to constant and dynamic attacks-counter attacks, known as "arms race". This "arms race" plays a vital role in the evolution of new and well adapted species of phytopathogens and plants.

Pathogen attack: Evolution of physical and biochemical weapons by phytopathogens

Phytopathogens have developed strategies to break the protective barrier of host plants. Pathogens that penetrate the host plant directly exert a mechanical force on the host surface to enhance their penetration. For example, the formation of appressorium by some fungal phytopathogens such as *Colletotrichum*, *Verticillium* and the powdery mildew fungi enhanced the penetration of the host plant **(Agrios, 2005)**. The successful penetration of the host plant is immediately followed by colonization, thereby resulting to a diseased condition. Accumulation of melanin in the appressorial wall of some fungi such as *Cochliobolus*, *Colletotrichum*, *Gaeumannomyces* and *Magnaporthe* enhances the penetration of the host plant **(Agrios, 2005)**. In *Magnaporthe grisea*, single gene mutations at loci encoding melanin biosynthetic enzymes resulted in non-melanized appressoria that are unable to generate turgor, thereby making the organism non-pathogenic **(Howard and Valent, 1996; Money and Howard, 1996)**. Pathogenicity of a non-melanin producing albino mutant of the cucumber pathogen, *Colletotrichum lagenarium*, could be restored by transformation with a melanin biosynthetic gene **(Kubo *et al.*, 1991)**. Also, appressoria from melanin mutant strains of *Magnaporthe. grisea* as well as wild-type appressoria after treatment with a melanin synthesis inhibitor displayed much lower glycerol levels **(De Jong *et al.*, 1997)**. Therefore, glycerol appears to be the major compound generating the turgor pressure and melanization is required for efficient build-up of turgor by rendering the appressorial walls impermeable to glycerol. Parasitic nematode such as *Meloidogyne javanica* that causes root knot diseases physically penetrates the host surface by thrusting its stylet to and fro **(Agrios, 2005)**. The nematode first sticks to the plant surface by the pressure that develops when its fused lips contact with the host plant. Unlike fungal phytopathogens that produce appressoria, bacteria phytopathogens are unable to penetrate the host plant directly, but rather enter the host usually through natural openings such as the stomata, and they also secrete cell wall-softening and degradative enzymes.

The biochemical weapons of phytopathogens include secretion of enzymes, growth regulators, polysaccharides and toxins **(Shittu *et al.*, 2017)**. Enzymes such as cutinases, cellulases, pectolytic enzymes and xylanase are secreted by phytopathogens to enhance the penetration of the host plant **(Agrios, 2005)**. The secretion of enzymes seems to be the most important attack strategy (arms) in some diseases such as the soft rot, caused by *Erwinia carotovora* **(Agrios, 2005)**. Growth regulators are synthesized endogenously by host plants and they are effective at minute concentrations. Elevated levels of these growth factors caused by pathogens often create an imbalance in the hormonal system of the host plant, thereby bringing about abnormal growth responses incompatible with the healthy development of a plant **(Agrios, 2005)**. The most important groups of plant growth regulators that are implicated in plants diseases include auxins, gibberellins,

cytokinins and ethylene. Increased auxin levels have been reported in plants infected with some phytopathogens such as *A. tumefaciens* (causing crown gall), *Ustilago maydis* (causing corn smut), and *Fusarium oxysporum f. cubense* (causing banana wilt) **(Agrios, 2005)**. These increases in growth regulators could be attributed to direct production by the pathogens, interference with the plant regulatory system or inhibition of enzymes such as IAA oxidase by the pathogens **(Agrios, 2005)**. The foolish seedling disease of rice, in which rice seedlings infected with the fungus *Gibberella fujikuroi* grow rapidly and become abnormally taller than the healthy plant could be attributed to a considerable extent, to the gibberellin secreted by the pathogen **(Agrios, 2005)**. Ethylene is produced by several plant pathogenic fungi and bacteria such as *Ralstonia solanacearum* **(Agrios, 2005)**.

Infection by phytopathogens using physical and biochemical weapons is aided by the inability of the host plants to recognize the presence of pathogen-associated molecular patterns (PAMPs). PAMPs are structures that are conserved over an evolutionary period and also give the organism an adaptive advantage **(Boyd *et al*., 2018)**. These structures are also referred to as microbe-associated molecular patterns (MAMP) because they could be inherent in non-pathogenic microbe. Examples of PAMPs include bacterial flagellin, bacterial elongation factor (EF-Tu), glucans and glycoproteins from oomycetes, chitin from fungal cell wall, lipopolysaccharides of gram-negative bacteria and cold shock proteins, with the latter two known as orphan PAMPs **(Boyd *et al*., 2018)** Some PAMPs are able to suppress defence response in the host plant, for example, lipopolysaccharide (LPS) suppresses plant defence by chelating calcium (Ca^{2+}) ions which plays a very important role in host plant defence and signal transduction **(Newman *et al*., 2007)**.

Plant counter attack: Development of PAMP-triggered immunity (PTI)

In a counter attack, host plants have developed receptors known as pattern recognition receptors (PRRs) localized in the plasma membrane to recognize or perceive the presence of PAMPs in order to activate a defence response **(Couto and Zipfel, 2016)**. Examples of PRRs include flagellin sensitive 2 (FLS2), elongation factor receptor (EFR), chitin elicitor binding protein and glucan binding protein which recognize a conserved epitope of flagellin (flg22), bacterial elongation factor (EF-Tu), chitin and glucan respectively **(Zhang and Zhou, 2010)**. The first two are Leucine-Rich-Repeat (LRR) receptor-like kinases that are capable of auto phosphorylation **(Zhang and Zhou, 2010)**. Plants possessing the appropriate PRR are able to detect the presence of the pathogen at very low concentrations **(Boller and Felix, 2009)**. Recognition of PAMPs serves as an early system warning for the presence of a potential pathogen and it is followed by the activation of a form of immunity known as PAMP-triggered immunity (PTI).

PTI is the first line of innate immunity in the host plant defence that is activated following PAMP or pathogen perception and it is also known as basal resistance **(Nicaise *et al*., 2009)**. Different plant species respond to different PAMPs. For example, tobacco responds to cold-shock protein, while *Arabidopsis* does not, and only members of the Brassicaceae have so far been shown to respond to EF-Tu **(Felix and Boller, 2003)**. The possibility of engineering plants to possess different PRRs that recognizes different PAMPs could therefore play a role in plant improvement against phytopathogens. The most well-known PAMP–PRR system involves the perception of a stretch of the bacterial flagellin through a 22-amino acid epitope known as flg22 by the cognate PRR, FLS2 **(Gomez-Gomez and Boller, 2000)**. A good example of a plant that uses this mechanism to defend itself against pathogen attack is *Arabidopsis* plant. A mutant *Arabidopsis* plant in FLS2 is more susceptible to infections with the pathogenic bacterium, *Pseudomonas syringae* -pv. tomato DC3000 (Pto DC3000), when surface-inoculated **(Zhang *et al*., 2010; Zipfel *et al*., 2004)**.

Mechanisms of PAMP triggered immunity (PTI) and the response of host plants to PAMPs recognition

Flagellin perception is the most characterized in plants. It is recognized by a leucine-rich repeat receptor like kinase known as flagellin sensitive 2 (FLS2) localized on the plasma membrane of the host plant **(Boyd *et al*., 2018)**. The binding of bacterial flagellin to the extracellular domain of FLS2 leads to activation of signaling cascades that result in the activation of a defence response in the host plant **(Pieterse *et al*., 2009)**. FLS2 activation involves its association with other proteins including BR11 associated kinase 1 (BAK1) and localization to less mobile areas, probably lipid raft on the plasma membrane in a ligand-dependent manner to activate PTI **(Ali and Reddy, 2008)**.

The defense responses that occur during PTI include molecular, morphological and physiological changes. Early changes occurring within seconds to minutes include ion-flux across the plasma membrane **(Jabs *et al*., 1997)**, an oxidative burst (accumulation of reactive oxygen species, ROS, largely produced by the activity of the membrane-localized NADPH oxidases), mitogen activated protein (MAP) kinase activation and protein phosphorylation **(Shen *et al*., 2017)**. This is followed by transcriptional reprogramming of some genes most especially the defence-related genes including the pathogenesis-related genes and the signal transduction genes **(Zhang and Zhou, 2010)**. Later changes include callose deposition and cell wall thickening, which serves as a physical barrier at infection sites, phytoalexin accumulation and stomatal closure **(Schwessinger, and Zipfel, 2008)**. Stomata provide a major entry point for many plant pathogens and *A. thaliana* stomata has been shown to close within an hour in response to PAMP **(Melotto *et al*., 2006)**. During PAMP-triggered immunity (PTI), mitogen activated protein kinase (MAPK) activation leads to the phosphorylation of the WRKY transcription factors which is a key regulator of plant defence-related genes **(Pandey and Somssich, 2009)**. This phosphorylation leads to the activation of the WRKY transcription factor, thus leading to increase in the expression of the associated genes.

The ability of the host plant to induce stomatal closure following PAMP perception shows that the guard cells associated with the stomata are able to perceive the presence of PAMP **(Zipfel *et al*., 2006)**. The efflux of H^+, K^+, NO_3^- and Cl^- following PAMP perception tend to reduce the solute concentration of the guard cells thereby increasing the water potential **(Melotto *et al*., 2006)**. This leads to the flaccidity of the guard cells, thus leading to stomatal closure. Stomatal closure following PAMP perception is also mechanistically linked to abscisic acid (ABA) signaling in the guard cells **(Melotto *et al*., 2006)**.

Pathogen attack: Evolution of secretion systems and effectors to suppress PAMP-triggered immunity

As the "arms race" continued, successful pathogens have evolved secretion systems and effector proteins to suppress PTI, thus leading to effector-triggered susceptibility in the host plant **(Henry *et al*., 2012)**. Examples of phytopathogens that use this strategy to infect the host plant include *Pseudomonas syringae* pv *lycopercisi* and *Agrobacterium tumefaciens*. There are four basic types of secretion pathways, which include type I, II, III and IV. Type I and II pathways secrete effector proteins to the host extracellular space, whereas type III and IV pathways can deliver effectors directly into host cell **(Bhat and Shahnaz, 2014)**. With the aid of the latter two pathways, phytopathogens are able to deliver all the aforementioned biochemical weapons into the host plant to cause a disease condition. The type III secretion system (TTSS) in phytopathogenic bacteria is encoded by the *hrp* genes (hypersensitive response and pathogenicity genes) and it is the most studied because of its importance in pathogenicity **(Wei and Collmer, 2017)**. The secretion via this pathway is a one step process with no intermediary in the periplasm. Examples of TTSS effectors include HopA11 and HopG1 secreted by *Pseudomonas syringae* **(Bhat and Shahnaz, 2014)**. The Vir D2/T-DNA nucleoprotein complex from *A. tumefaciens* is transported directly into the host cell through the type IV pathway and it is the only pathway that is known to translocate both proteins and nucleic acids **(Bhat and Shahnaz, 2014)**.

Pathogens secrete molecules that manipulate host cell structure and function when they enter the host plants and these molecules are referred to as effectors **(Keller *et al*., 2016)**. These effectors are derived from the expression of the *avr* genes. Effectors are secreted into the host plants from pathogen secretion systems and they could be apoplastic or cytoplasmic, depending on whether they are secreted to the host extracellular space or cytoplasm, respectively **(Bhat and Shahnaz, 2014)**. When effectors enter the host plant cell, they can either act as toxins as elicitors **(Bhat and Shahnaz, 2014)**. They may trip the wire by acting as elicitors if the host plant has a cognate resistance gene that encodes a resistance protein, thus activating a strong hypersensitive reaction that follows the gene-for-gene concept **(Flor, 1955 and Jones and Dangl, 2006)**.

Mechanisms of effector-triggered susceptibility

Effectors enhance the virulence of the pathogen by suppressing the host defence response and also increasing the availability of nutrients to the pathogen. They may act by carrying out enzymatic activity or binding to other proteins to modify their function. Effector traffic to different cell compartments upon entering the host cell and they may have more than one target in the host plant. AvrRpt2 is a TTSS effector with proteolytic activity against at least five *Arabidopsis* proteins **(Takemoto and Jones, 2005)**. Some effectors proteins such as Avr2, Avr4, and ECP6 secreted by the tomato fungal pathogen *Cladosporium fulvum* act in the extracellular space where they interfere with apoplastic plant defences **(Kamoun, 2006)**. Phytopathogens must first overcome the apoplastic immunity before a successful colonization of the host plant to cause a diseased condition. The apoplast therefore serves as a battle ground in plant-pathogen interaction **(Gupta *et al*., 2015)**. Some cytoplasmic effectors such as *Xanthomonad* transcription activator-like (TAL) effectors, *Phytoplasma* SAP11, and *Phytophthora* Crinklers (CRNs) are capable of being localized in the nucleus after entering the host plant with the help of the nuclear localization signals that they possess, thus altering the transcription of defence-related genes during infection **(Bai *et al*., 2009; Mak *et al*., 2013; Schornack *et al*., 2010 and Van den *et al*., 1996)**. The type-III effector, HopG1, secreted by *P. syringae* targets plant mitochondria, whereas HopI1 is transported to plant chloroplasts **(Block *et al*., 2010 and Jelenska *et al*., 2007)**. HopG1 targets mitochondria; disrupt plant development and suppresses plant innate immunity **(Block *et al*., 2010)** HopZ1a disorders microtubule networks, thereby interfering with the plant secretory pathway and suppressing plant defence brought about by the cell wall. **(Bhat and Shahnaz, 2014)**. T3SS effectors of the plant pathogenic bacteria *Pseudomonas syringae* and *Xanthomonas campestris* have been reported to target secretory pathways, which indicates that protein trafficking plays a significant role in plant innate immunity **(Wang *et al*., 2016)**.

Protein trafficking helps to transport the necessary molecules (arms) needed to protect the host plant following pathogen perception **(Thordal-Christensen, 2009)**. Therefore, one of the virulence strategies of an invading phytopathogen is interference with protein trafficking pathway. *Pseudomonas syringae* secretes HopM1 which is restricted to the trans-Golgi network/early endosome where it destabilizes the host protein, AtMIN7, which has a crucial role in vesicle trafficking **(Nomura *et al.*, 2006)** Some effectors are capable of blocking the PAMP signaling pathway by targeting the important signaling molecules. For example, HopA11, a phosphothreonine lyase secreted by several strains of *Pseudomonas syringae* targets mitogen activated protein kinases which are important signaling molecules, thus causing a blockage in PAMP signaling pathway. Some effectors alter plant behaviour and development, for example, during infection *Pseudomonas* produce coronatine, a jasmonic acid (JA) mimic that contributes to virulence by silencing salicylic acid (SA) mediated defence response to trigger stomatal reopening in *Arabidopsis* and thereby facilitating bacterial entry into the host plant **(Melotto *et al.*, 2006)**. This suggests that the stomata play an important function in plant innate immunity against bacterial invasion. *Xanthomonas* effectors of AvrBs3 family are known to induce cellular division and enlargement in susceptible host plants **(Boch and Bonas, 2010)**. Other morphological changes include galls and witches broom induced by *Agrobacterium* spp. and other bacteria such as *phytoplasmas* respectively **(Christie, 2004 and Hoshi *et al.*, 2009**). *Pseudomonas syringae* secretes effectors -such as HopU1, AvrRpt2, AvrB, HopAO1, HopE1, HopAM1, AvrRpm1, AvrPtoB and HopX1s which are capable of inhibiting PAMP-induced callose and it may also negatively alters defence gene expression **(Cui *et al.*, 2009)**.

Plant counter attack: Development of effector-triggered immunity

Continuing the arms race, the plants never surrender to the attempted devastating effect of the pathogens. Some successful and well-adapted plants have evolved another set of receptors known as intracellular resistance proteins encoded by the *R* genes to recognize pathogen effectors **(Cui *et al.*, 2015)**. An example of a plant that uses this strategy to defend itself against pathogen attack is *Arabidopsis* **(Zhang *et al.*, 2010)**. These resistance proteins are immune-receptors characterized by two domains; a nucleotide binding site (NBS) and leucine-rich repeats (LRR). The NBS domain located approximately in the middle of the R protein comprises several conserved amino acid motifs that are believed to function in ATP hydrolysis **(Tameling *et al.*, 2002)**. The LRR domain comprises a core of about 26 amino acid repeats found at the C-terminal end of the protein and it is predicted to modulate direct or indirect interactions between the R protein and its corresponding effector molecule **(McHale *et al.*, 2006)**. Pathogen effectors are recognized in a specific manner by intracellular resistance proteins which could be direct or indirect. The direct recognition follows the ligand receptor model, where the *R* gene products act as receptors that directly interact with the *avr* gene products (effectors) **((Pieterse *et al.*, 2009)**. This interaction activates a resistant reaction that follows the gene-for-gene concept. Indirect recognition of effector proteins could follow either the guard model or the decoy model. According to the guard model, the effector target is monitored by the R protein and these results in the change of the target, therefore, activating the resistance proteins to trigger a hypersensitive reaction in the host plant as observed in *Pseudomonas syringae* **(Dangl and Jones, 2001)**. The perception of AvrPto effector protein produced by *Pseudomonas syringae* by the tomato proteins, Pto and Prf is in accordance with the guard model **(Tang *et al.*, 1996)**. The decoy model is similar to the guard model, however, the manipulation of the guarded effector target by the effector facilitates pathogen fitness in the absence of the corresponding *R* gene **(Van Der Hoorn and Kamoun, 2008)**. The recognition of the effector protein by the cognate resistance gene product prevents the blockage of PAMP signaling, thus activating effector-triggered immunity (ETI) in the host plant, and hence turning the table on the pathogen by making the effectors liabilities **(Pieterse *et al.*, 2009)**. Therefore, the presence of effectors in phytopathogens could either be an advantage or disadvantage to the pathogen depending on the absence or presence of a cognate resistance protein that recognizes the effector protein. Pathogenesis caused by pathogen effectors can be overcome by the activation of ETI as explained using the gene-for-gene concept **(Cui *et al.*, 2015; Flor, 1955)**. ETI is a stronger form of immunity compared to PTI. ETI may be accompanied by the induction of a hypersensitive response (HR), which is a form of programmed cell death and this is a defence response effective against biotrophic phytopathogens **(Hammond-Kosack and Jones, 1996)**. The HR effectively prevents the biotrophic pathogen to access healthy tissues which it utilizes as a nutritional source and also prevents the further spread of the pathogen **(Greenberg, and Yao, 2004)**. HR also triggers a wide variety of defence responses, which include systemic acquired resistance (SAR), which serves as a warning throughout the plant of the invasion by the pathogen and this is also accompanied by the accumulation of some signaling molecules such as salicylic acid and ethylene **(Fu and Dong, 2013)**.

The gene-for-gene concept

Genetic analysis of the interactions between flax (*Linum usitatissimum*) and flax rust (*Melampsora lini*) led Herod H Flor to the formulation of a gene-for-gene concept **(Flor, 1955)**. He found that the resistance of flax to specific flax rust strain could be inherited monogenetically by the next generation. The gene-for-gene concept proposes that the *avr* gene products are specifically recognized by the cognate resistance gene product of the host plant to activate a strong defence response known as effector-triggered immunity, thereby making the host plant resistant to a non-adapted pathogen **(Nobuta and Meyers, 2005)**. This type of resistance is also known as cultivar-specific resistance **(Agrios, 2005)**. In rice, the *LRR-RK*, *Xa21* confers resistance to *Xanthomonas oryzae* pv *oryzae* strains carrying the *avr* gene, *AvrXa2* **(Song *et al.*, 1995)**. According to the gene-for-gene hypothesis, pathogens may be virulent for several reasons, (a) the pathogen does not produce an avirulent protein, (b) the plant lacks a factor, for example, a resistance protein that would enable it to detect the avirulent protein, and (c) the pathogen and plant respectively lack both the avirulent and the corresponding resistance protein **(Nobuta and Meyers, 2005)**.

Pathogen attack: Development of mechanisms to suppress ETI

At present, some well-adapted pathogens have developed mechanisms to suppress effector-triggered immunity. One of these mechanisms is the silencing of effector genes by endogenous pathogen sRNA to escape R protein recognition **(Wang *et al.*, 2015)**. In this case, recognitions of effector proteins by the cognate resistance proteins activate effector-triggered immunity, thereby making the effectors a liability to the pathogen. Silencing of the pathogen effector gene is a mechanism to escape recognition by resistance proteins, thereby hindering the activation of any immune response and thus making the host plant susceptible. The silencing is sRNA-mediated in a phenomenon known as RNA interference (RNAi) **(Das *et al.*, 2011)**. Effector genes of some phytopathogens such as *Phytophthora* are usually located in the transposable element (TE) rich regions that give rise to sRNAs **(Ventukuri *et al.*, 2012; Whisson *et al.*, 2012)**. An example of a pathogen that uses this mechanism to infect the host plant is *Phytophthora sojae*. **Qutob *et al.* (2013)** observed sRNA mediated silencing of an effector gene *PsAvr3a* in a virulent strain of *Phytophthora sojae* ACR10, when infecting plants carrying the *Rps3a* resistance gene; but this silencing was absent in the avirulent strain, P7076. Another attack strategy developed by pathogens to silence host immune genes is by sRNA effectors (cross-kingdom RNAi) **(Wang *et al.*, 2015)**. Some phytopathogens have evolved mechanisms for silencing of the host immune genes in order to enhance infection. They achieve this by producing sRNA effectors, which they secrete into the host plants to silence their defence-related genes **(Wang *et al.*, 2015)**. The sRNA effectors are small interfering RNA molecules secreted by the pathogen to silence the immune genes of the host plant in a phenomenon known as RNA interference (RNAi). This type of RNAi that describes a phenomenon in which a donor organism produces an RNA trigger that moves into a recipient organism of different kingdom to cause gene silencing is known as cross-kingdom RNA interference **(Wang *et al.*, 2015)**. Cross-kingdom RNAi indicates that gene silencing signal can travel extracellularly over long distance, even across the cell wall in the case of plant-pathogen interaction. *Botrytis cinerea* is a very virulent pathogen that produces sRNA effector to silence the host immune gene **(Weiberg *et al.*, 2013)**. Cross-kingdom RNAi has also been observed in the interaction between *Verticillium dahlia* and the model plant *Arabidopsis thaliana* **(Wang *et al.*, 2015)**.

CONCLUSION

The “arms race” between plants and phytopathogens is an ongoing process that has been in existence over evolutionary periods. The plants seem to be winning in some plant-pathosystems, while in others, the pathogens are winning. Research efforts in the field of phytopathology, such as screening for resistance gene and disease tolerant plants, should therefore, be geared towards assisting the susceptible plants to overcome the deleterious effects of phytopathogens in order to ensure food security.

REFERENCES

Agrios, G. N. (2005). *Plant Pathology*. Fifth Edition. Elsevier Academic Press, London, 922p.

Ali, G. S. & Reddy, A. S. N. (2008). PAMP-triggered immunity. *Plant Signaling and Behaviour*, 13(6), 423 – 426. https://doi.org/10.1093/pcp/pcm132

Bai, X., Correa, V. R., Toruno, T. Y., Ammarel, D., Kamoun, S. & Hogenhout, S. A. (2009). AY-WB *Phytoplasma* secretes a protein that targets cell nuclei. *Molecular Plant-Microbe Interaction*, 22, 18 – 30. https://doi.org/10.1094/MPMI-22-1-0018

Bhat, B. S. & Shahnaz, F. (2014). Effectors role in host-pathogen interaction. Journal of Agriculture and Environmental Sciences, **3**(2), 265 – 285.

Block, A., Guo, M., Li, G., Elowsky, C., Clemente, T. E. & Alfano, J. R. (2010). The *Pseudomonas syringae* type III effector, HopG1 targets mitochondria, alters plant development and suppresses plant innate immunity. *Cellular Microbiology*, 2, 1 – 13. https://doi.org/10.1111/j.1462-5822.2009.01396.x

Boller, T. & Felix, G. (2009). A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. *Annual Review Plant Biology*, 60, 379 – 406. https://doi.org/10.1146/annurev.arplant.57.032905.105346

Boyd, L. A., Ridout, C., Sullivan, D. M., Leach, J. E. & Laung, H. (2018). Plant-pathogen interactions: disease resistance in modern agriculture. *Trends in Genetics*, In Press. http://dx.doi.org/10.1016/j.tig.2012.10.011

Christie, P. (2004). Type VI secretion: The *Agrobacterium* VirB/D4 and relate congution systems. *Biochem Biophys Acta* 1694, 219 – 234. https://doi.org/10.1016/j.bbamcr.2004.02.013

Couto, D. & Zipfel, C. (2016). Regulation of pattern recognition receptor signaling in plants. *Nature Review Immunology*, 16, 537 – 552. https://doi:10.1038/nri.2016.77

Cui, H., Xiang, T. & Zhou, J. (2009). Plant immunity: A lesson from pathogenic bacterial effector proteins. *Cellular Microbiology*, 11(10), 1453 – 1461. https://doi.org/10.1111/j.1462-5822.2009.01359.x

Cui, H., Tsuda, K. & Parker, J. E. (2015). Effector-triggered immunity: from pathogen perception to robust defence. *Annual Review Plant Biology*, 66, 487 – 511. https://doi.org/10.1146/annurev-arplant-050213-040012

Dang, J. L. & Jones, J. D. G. (2001). Plant pathogen and integrated response to infection. *Nature*, 411, 826 – 833. https://doi.org/10.1038/35081161.

Das, S., Marwal, A., Choudhary, D. K., Gupta, V. K. & Gaur, R. K. (2011). Mechanism of RNA interference: Current concept. *International Conference on Food Engineering and Biotechnology*, *9*, 240 – 246.

Dawkins, R. & Krebs, J. R. (1979). Arms races between and within species. *Proceedings of the Royal Society of London*, 205, 489 – 511. https://doi.org/10.1098/rspb.1979.0081

De Jong, J. C., McCormack, B. J., Smirnoff, N. and Talbot, N. J. (1997). Glycerol generates turgor in rice blast**.** *Nature*, 389, 244 – 245.

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. *Journal of Biological Chemistry*, 278, 6201 – 6208.

Flor, H. H. (1955). Host-parasite interaction in flax rust. Its genetics and other implications. *Phytopathology*, 45**,** 680 – 685.

Fu, Z. Q. & Dang, X. (2013). Systemic acquired resistance: turning local infection into global defence. *Annual review Plant Biology*, 64, 839 – 863. https://doi.org/10.1146/annurev-arplant-042811-105606

Gomez-Gomez, L. & Boller, T. (2000). FLS2: An LRR receptor-like kinase involved in the perception of the bacterial elicitor flagellin in *Arabidopsis*. *Molecular Cell*, 5, 1003 – 1011. https://doi.org/10.1016/s1097-2765(00)80265-8

Greenberg, J. T. & Yao, N. (2004). The role and regulation of programmed cell death in
plant-pathogen interactions. *Cell Microbiology*, 6, 201 – 211. https://doi.org/10.1111/j.1462-5822.2004.00361.x

Gupta, R., Lee, S. E., Agrawal, G. K., Rakwal, R., Park, S., Wang, Y. & Kim, S. T. (2015). Understanding theplant-pathogen interactions in the context of proteomics-generated apoplastic proteins inventory. *Frontiers in Plant Science*, 6, 1 – 7. https://doi.org/10.3389/fpls.2015.00352

Hammond-kosack, K. E. & Jones, J. D. G. (1996). Resistant gene-dependent plant defence responses. The Plant Cell, 8, 1773 – 1791. https://doi.org/10.1105/tpc.8.10.1773

Henry, G., Thonart, P. & Ongena, M. (2012). PAMPs, MAMPs, DAMPs and other: an update in the diversity of plant immunity elicitors. *Biotechnology Agricultural Science Environment*, 16(2), 257 – 268.

Hoshi, A., Oshima, K., Kakizawa, S., Ishi, Y., Ozeki, J., Hashimoto, M., Komatsu, K., Kagiwala, S., Yamaji, Y. & Namba, S. (2009). A unique virulence factor for proliferation and dwarfism in plants identified from a phytopathogenic bacteria. *Proceedings of the National Academy of Science*, 106(15), 6416 – 6421. https://doi.org/10.1073/pnas.0813038106

Holub, E. B. (2001). The arms race is ancient history in *Arabidopsis*, the wildflower. *Nature Reviews Genetics*, 2, 516 – 527. http://dx.doi.org/10.1038/35080508

Howard, R. J. & Valent, B. (1996). Breaking and entering: Host penetration by the fungal rice blast pathogen *Magnaporthe grisea*. *Annual Review of Microbiology*, 50, 491 – 512. https://doi.org/10.1146/annurev.micro.50.1.491

Jabs, T., Tschope, M., Colling, C., Hahlbrock, K. & Scheel, D. (1997). Elicitor-stimulated ion fluxes and O_2^- from the oxidative burst are essential components in triggering defence gene activation and phytoalexin synthesis in Parsley. *Proceedings of Natural Academy of Sciences*, USA. 99, 4800 – 4805.

Jelenska, J., Yao, N., Vinatzer, B. A., Wright, C. M., Brodsky, J. L. & Greenberg, J. T. (2007). A J domain virulence effector of *Pseudomonas syringae* remodels host chloroplasts and suppresses defence. *Current Biology*, 17, 499 – 508. https://doi.org/10.1016/j.cub.2007.02.028

Jones, J. D. G. & Dangl, J. L. (2006). The plant immune system. *Nature*, 444, 323 – 329. https://doi.org/10.1038/nature05286

Kamoun, S. (2006). A catalogue of the effector secretome of plant pathogenic oomycetes. *Annual Review of Phytopathology*, 44, 41 – 60. https://doi.org/10.1146/annurev.phyto.44.070505.143436

Keller, H., Boyer, L. & Abad, P. (2016). Disease susceptibility in the Zig-Zag model of host-microbe interactions: Only a consequence of immune suppression? *Molecular Plant Pathology*, 17(4), 475 – 479. https://doi.org/10.1111/mpp.12371

Kubo, Y., Nakamura, H., Kobayashi, K., Okuno, T. & Furusawa, I. (1991). Cloning of a melanin biosynthetic gene essential for appressorial penetration of *Colletotrichum lagenarium*. *Molecular Plant-Microbe Interaction*, 4, 440 – 445. https://doi.org/10.1007/978-1-4899-2635-7_9

Mak, A. N., Bradley, P., Bogdanove, A. & Stoddard, B. L. (2013). TAL effectors: Functions, structure, engineering and applications. *Current Opinion in Structural Biology*, 223, 93 – 99. https://doi.org/10.1016/j.sbi.2012.11.001

McHale, L., Tan, X., Koel, P. & Michelmore, R. W. (2006). Plant NBS-LRR proteins: adaptive guards. *Genome Biology*, 6(4), 212.1 – 212.10. https://doi.org/10.1186/gb-2006-7-4-212

Melottto, M., Underwood, W., Koczan, J., Nomura, K. & He, S. Y. (2006). Plant stomata function in innate immunity against bacterial invasion. *Cell*, 126, 969 – 980. https://doi.org/10.1016/j.cell.2006.06.054

Money, N. P. & Howard, R. J. (1996). Confirmation of a link between fungal pigmentation, turgor pressure and pathogenicity using a new method of turgor measurement**.** *Fungal Genetics Biology*, 20, 217 – 227. https://doi.org/10.1006/fgbi.1996.0037

Newman, M. A., Cow, J. M., Molinaro, A. & Parrili, M. (2007). Priming induction and modulation of plant defence responses by bacterial lipopolysaccharides. *Journal of Endotoxin Research*, 13, 69 – 84.

Nicaise, V., Roux, M. & Zipfel, C. (2009). Recent advances in PAMP-Triggered Immunity against bacteria: pattern recognition receptors watch over and raise the alarm. *Plant Physiology*, 150, 1638-1647. https://doi.org/10.1104/pp.109.139709

Nobuta, K. & Meyers, B. C. (2005). *Pseudomonas* versus *Arabidopsis*: Models for genomic research into plant disease resistance. *BioScience*, 55(8), 679 – 686.

Nomura, K., Debroy, S., Lee, Y. H., Pumplin, V., Jones, J. & He, S. Y. (2006). A bacteria virulence protein suppresses host innate immunity to cause plant disease. *Science*, 313, 220 – 223. https://doi.org/10.1126/science.1129523

Pandey, S. P. & Somssich, I. E. (2009). The role of WRKY transcription factors in plant immunity. *Plant Physiology*, 150, 1648 – 1655. https://doi.org/10.1104/pp.109.138990

Pieterse, C. M. J., Leon-Reyes, A., Van Der Ent, S. & Van Wees, S. C. M. (2009). Networking by small-molecule hormones in plant immunity. *Nature Chemical Biology*, 5, 308 – 316. https://doi.org/10.1038/nchembio.164

Qutob, D., PatrickChapman, B. & Gijzen, M. (2013). Transgenerational gene silencing causes gain of virulence in a plant pathogen. *Nature Communication* 4, 1 – 6. https://doi.org/10.1038/ncomms2354

Schornack, S., van Damme, M., Thines, M., Gaulin, E., Kamoun, S. & Huitema, E. (2010). Ancient class of translocated Oomycetes effectors targets the host nucleus. *National Academy of Science*, 107**,** 17421 – 17426. https://doi.org/10.1073/pnas.1008491107

Schwessinger, B. & Zipfel, C. (2008) News from the frontline: recent insights into PAMP-triggered immunity in plants. *Current Opinion in. Plant Biology*. 11, 389 – 395. https://doi.org/10.1016/j.pbi.2008.06.001

Shen, Y., Liu, N., Li, C., Wang, X., Xu, X., Chen, W., Xing, G. & Zheng, W. (2017). The early response during the interaction of fungal phytopathogen and host plant. *Open Biology* 7, 170057. http://dx.doi.org/10.1098/rsob.170057

Shittu, H. O., Aghogban, O. N. & Igiehon, E. (2017). Plant pathogen attack strategies. *Journal of Phytopathology and Plant Health*, 4(1), 1-9.

Song, W. Y., Wang, G. L., Chen, L. L., Kim, H. S., Pi, L. Y., Holsten, T., Gardener, J., Wang, B., Zhai, W. X., Zhu, L. H., Fauquet, C. & Ronald, P. (1995). A receptor kinase-like protein encoded by the rice disease resistant gene, Xa21. Science, 270(5243), 1804 – 1806. https://doi.org/ 10.1126/science.270.5243.1804

Tang, X., Frederick, R. D., Zhou, J., Halterman, D. A., Jia, Y. & Martin, G. B. (1996)
Initiation of plant disease resistance by physical interaction of avrPto and Pto kinase.*Science*, 274, 2060 – 2063. https://doi.org/10.1126/science.274.5295.2060

Takemoto, D., & Jones, D. A. (2005). Membrane release and destabilization of *Arabidopsis*
RIN4 following cleavage by *Pseudomonas syringae* AvrRpt2. *Molecular Plant-Microbe Interactions*, 18, 1258 – 1268. https://doi.org/10.1094/MPMI-18-1258

Tameling, W. I., Elzinga, S. D., Darmin, P. S., Vossen, J. H., Takken, F. L., Haring, M. A. & Cornelissen, B. J. (2002). The tomato R gene products 1-2 and m 1-1 are functional ATP binding proteins with ATPase activity. *Plant Cell*, 14, 2929 – 2939. https://doi.org/10.1105/tpc.005793

Thordal-Christensen H. (2009) Vesicle Trafficking in Plant Pathogen Defence. In: Mancuso S., Balu¿ka F. (eds) Signaling in Plants. Signaling and Communication in Plants. Springer, Berlin, Heidelberg

Van den, A. G., Mario, E., & Bonas, U. (1996). Recognition of the bacterial avirulence protein AvrBs3 occurs inside the host plant cell, *Cell*, 87, 1307 – 1316.

Van der Hoorn, R. A. L. & Kamoun S. (2008). From guard to decoy: A new model for perception of plant pathogen effectors. *The Plant Cell*, 20, 2009 – 2017. https://doi.org/10.1105/tpc.108.060194

Vetukuri, R. R., Asman, A. K., Tellgren-Roth, C., Jahan, S. N., Reimegard, J., Fogelqvist, J., Savenkov, E., Soderbom, F., Avrova, A. O., Whisson, S. C. & Dixelius, C. (2012). Evidence for small RNAs homologous to effector-encoding genes and transposable elements in the oomycete *Phytophthora infestans*. *PLoS ONE*, 7, e51399. https://doi.org/10.1371/journal.pone.0051399

Wang, M., Weiberg, A. & Jin, H. (2015). Pathogen small RNAs: A new class of effector for pathogen attacks. *Molecular Plant Pathology*, 16(3), 219 – 223. https://doi.org/ 10.1111/mpp.12233

Weiberg, A., Wang, M., Lin, F. M., Zhao, H., Zhang, Z., Kaloshian, I., Huang, H. D. & Jin, H. (2013) Fungal small RNAs suppress plant immunity by hijacking host RNA interference pathways. *Science*, 342, 118 – 123. https://doi.org/10.1126/science.1239705

Wang, W., Liu, P., Xu, Y. & Xiao, X. (2016). Protein trafficking during plant innate immunity. *Journal of Integrative Plant Biology*, 58, 284 – 298. https://doi.org/10.1111/jipb.12426

Whisson, S., Vetukuri, R., Avrova, A. & Dixelius, C**.** (2012) Can silencing of transposons contribute to variation in effector gene expression in *Phytophthora infestans*? *Mobile Genetic. Element* 2, 110 – 114. https://doi.org/10.4161/mge.20265

Zhang, J. & Zhou, J. M. (2010). Plant immunity triggered in microbial molecular signatures. *Molecular Plant*, 6, 1 – 11. https://doi.org/10.1093/mp/ssq035

Zhang, J., Lu, H., Li, X., Li, Y., Cui, H., Wen, C., Tang, X., Su, Z. & Zhou, J. (2010). Effector-triggered and pathogen-associated molecular pattern–triggered immunity differentially contribute to basal resistance to *Pseudomonas syringae*. *Phytopathology*, 23(7), 940 – 948. https://doi.org/10.1094/MPMI-23-7-0940

Zipfel, C., Robatzek, S., Navarro, L., Oakeley, E. J., Jones, J. D., Felix, G. and Boller, T (2004).
Bacterial disease resistance in *Arabidopsis* through flagellin perception. *Nature*, 428,764-767. https://doi.org/10.1038/nature02485

Zipfel, C., Kunze, G, Chinchilla, D., Caniard, A., Jones, J. D. & Felix, G. (2006). Perception of
the bacterial PAMP EF-Tu by the receptor EFR restricts *Agrobacterium*-mediated transformation. *Cell*, 125(4), 749 – 760. https://doi.org/10.1016/j.cell.2006.03.037