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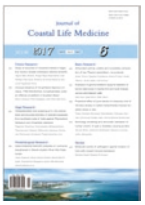

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Molecular events of pathogenic agents invasion of host cells: toward induced resistance

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ABSTRACT

Recognition of pathogens by their hosts is the result of a highly regulated interaction between both host and pathogen elicitors. Therefore, mechanical defence lead to an increase in the production of substances capable of inhibiting pathogen invasion. Furthermore, chemical materials [antimicrobial (phytoalexins)] released from hosts lead to an increase in the activity of host defense. Recent studies showed that elicitors have been widely used in controlling pathogenic plant diseases showing mostly impressive results and less adverse effect on the environment. The current article explains information about molecular events occurring at the host-pathogen interactions and how these events influence the organization and activation of resistance responses and prospects of advance toward induced resistance in the host.

1. Introduction

Plant pathogenic agents include microbes and parasites that attack host plants. These parasitic agents attack plants and cause crop yield losses even in interactions which do not end up with disease or death of the plant[1]. So, the programs of phytopathogenic management depend on knowing what exactly occurs between the pathogenic microorganisms and their host plants[2]. The genetic makeup of the host plant determines its susceptibility to disease. Susceptibility depends on various physical and biochemical factors, which are released through the host plant after infection[3]. For instance, growth habit, cuticle thickness and shape of stomata that allows water, oxygen, and carbon dioxide in and out of plant tissues are a few physical factors that affect disease occurrence and development[4,5]. Additionally, pathogenic agent causes diseases when a host plant is invaded by a microorganism. Pathogen infection leads to changes in secondary metabolism based on the induction of defence programmes as well as to changes in primary metabolism which influences plant growth[6,7]. Lately, phytopathological studies took into consideration the physiological status of the infected plant tissues to elucidate the fine-tuned infection mechanisms[8]. Yet, plants must continuously defend themselves against attacks from pathogenic microorganisms, *e.g.*, fungi, bacteria, viruses, viroid and even other pests. Actually, infectious disease mechanisms between plant and pathogen interaction are complex. Therefore, the application of molecular genetic techniques has resulted in major advances in explaining the mechanisms that regulate gene expression, and in identifying components of many signal transduction pathways in diverse physiological systems[9]. Though many disease resistant genes of many host plants and signaling mechanisms are now characterized, it is still equivocal whether and how they can be engineered to enhance disease resistance[10]. Finally, hosts can protect themselves by producing natural compounds called secondary metabolites (phytoalexins), such as terpenes, phenolics, nitrogen (N), and sulphur (S), which defend plants against attacks from a variety of pathogenic microorganisms[11].

In this review, the new understanding of the defense system of host plants against pathogen, including perception of molecular event will be reviewed. A discussion of how modern technologies can be applied to help further understand the mechanisms of infectious diseases is presented. This may lead to formulating new

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strategies for the development of plant disease control approaches toward induced resistance.

2. Molecular characteristic of virus invasion of host living cells

2.1. Events of infected plant cell after recognition with virus

The sites of infection from virus to plant are determined through few basic steps which are called the viral life cycle. The process of adherence includes: (a) A virus particle attaches to a susceptible cell; (b) The viral particle injects its DNA or RNA into the cell; (c) The invading DNA or RNA takes over the cell and obligates the host cell produce enzymes; (d) These cellular enzymes start making new virus particles to replicate in the host cell; (e) Viruses created by the cell come together to form new viruses; in addition they kill the host cell and search for a new host cell[12]. At the molecular level, infection occurs after the attachment between the viral particles and the host cells; these are not simply taken into cells. They must attach to a receptor on the host plant cell surface. Each virus has a specific receptor and it is usually a vital component of the cell surface. Virus distributes the receptor molecules on plant cells, and these molecules select the virus favorite cells for infection[13]. After control is established and the suitable environment is set for the virus to make copies by generating multiple progeny genomes, it usually consumes the content of the host cell[14]. This happening declares the end of the viral life cycle as the host plant cell often dies and the newly produced viruses must find a new host[15]. For example, in the case of positive strand RNA plant viruses, the infecting genome must not only be replicated but also serves as an mRNA for the production of the replication associated proteins. Also, it has become evident that in all the stages of the infection cycle, positive strand RNA viruses are intertwined[16].

2.2. Developmental events of pathogenic virus through infected cells

The interference occurring between plant development and antiviral defence processes, leads to the interference among the common points of their signaling pathways which can trigger pathological symptoms[17]. Infection by a specific virus in a host plant can induce more than 4 000 different genes. So, the different viruses do have varying responses in a common host[18]. On the other hand, the number of genes whose expression is affected by different viruses seems to be logical with the variation of the symptoms they cause[19]. Viral pathogenic process reveals that the metabolic processes for the host plant cells of its defence after viral infections are not only high, but are also diverse in form[20].

3. Molecular characteristic of bacterial invasion of host cells

3.1. Events of infected plant cell after recognition with bacteria

After the contact between the host cell and bacteria, a particular chain of events is produced. There are different ways of possible interactions after this contact. Firstly, the plant is provided by a receptor that interacts with bacterial protein, which is developed as a quick protective reaction. In such a state, the bacteria is called avirulent for a given plant genotype[21]. Secondly, proteins of the pathogenic bacteria are virulent for the given plant genotype. Hence, virulence genes may be sheltered in different replicons, independent replicating units, such as spread throughout the chromosomes or in specialized areas called genomic or pathogenicity islands[22]. On the other side, the plant is affected by the pathogen, while protective mechanisms are activated more slowly[23]. Generally, the cell wall of host cells is strengthened with the start of pathogenesis gene transcription. In the infection by pathogen, the active forms of oxygen are formed causing the death of infected cells[24].

3.2. Developmental events for pathogenic bacteria inside the host cells

Genetically, development in the application of the molecular tools aids in understanding the relationship between host cells and bacterial pathogen. This understanding is important especially in the HR hypersensitive reaction stage (cell death). In such important phase, the pathogen penetrates the cells and pathogenesis related genes (PR-genes) are activated. These genes encode a protein secretion system that has the potential to transfer virulence proteins into host cells[25]. The necrogenic bacteria have diverse pathogens with a wondrous array of symptoms and host specificities. Such symptoms vary according to the specialization of the host. There is a growing evidence that the hrp genes are widespread in these pathogens, controlling early interactions with plants, and provide a unifying entry point for exploring bacterial phytopathogenicity[26]. Likewise, recent studies of chemical signaling, between pathogenic bacteria and host plants, are beginning to provide evidence that plants are affected by these signals via low molecular weight compounds[27]. Also, plants produce compounds which interact with bacterial regulatory proteins, which then affect gene expression. Bacterial elite sensing signals resulting in a range of functional responses in host plants and probably the occurrence of infection[28].

4. Molecular characteristic of fungal invasion of host cells

4.1. Events of infected plant cell after recognition with fungi

There are four crucial steps to the establishment of plant infection by pathogenic fungi. Firstly, the pathogen attaches to the host cells surface. Secondly, germination occurs into the plant surface and infection structure is formed. Thirdly, the hypha penetrate into the host plant cells. Fourthly, colonization is made by the fungi of the infected host tissue[29]. At the site of the hypha penetration, appressoria are often formed that may have walls and develop high distention pressure to support the penetration process. The penetration of hypha accumulates components of the cytoskeleton in the tip and secretes numerous enzymes to cell wall-degrading in a highly regulated fashion in order to penetrate the cuticle and the plant cell wall[30]. So, the numerous enzymes are needed to degrade the cell wall of complex web of carbohydrates, glycoproteins, and phenolic compounds of the plant cell wall[31]. Finally, the infection mechanism is determined by the interaction between host plants and pathogenic fungi, which is called recognition[32].

4.2. Developmental events for pathogenic fungi inside host cells

The molecular recognition events between pathogenic fungi and host plants that lead to expression of defensive reactions have been attributed to signal-sensor reactions consisting of three different steps[33]. The first step, the elicitor of the pathogen, a low molecular weight substance, sends a signal originating from the pathogen. The second step, the receptor of the plant specifically binds the elicitor. The third step, the effector designates one or more substances formed as a result of the recognition and binding between elicitor and receptor[10]. Subsequently, effectors comprise all the products participating in signal transduction for triggering expression of defensive reactions by the host plant. Hence, the process of elicitor-receptor interaction between pathogenic fungi and host cells is called elicitation. The elicitor acts like a ligand binding to the receptor. Elicitation and the formation of effectors release the expression of both, active basic resistance and race-specific resistance[34]. Furthermore, the physical resistance of the host plant presented by the strength and integrity of the cell walls and intercellular spaces is the host's first line of defense. Interestingly, nutrients in the plant play an important role in developing strong cell walls and other tissues[35]. On the other side, the germination of fungal spores depends on the stimulating compounds exuded by the plants, like sugars and amino acids that promote the establishment of the fungus[36,37]. Hence, the plant becomes infected by a fungus, and its natural defenses are triggered. The infection causes increased production of fungus inhibiting elements in the plant like, phenolic compounds and flavonoids, which occur in the site of infection to resist the pathogen[38].

5. Role of molecular parasitic events in host cells to make accuracy management

To protect themselves, plants accumulate an arsenal of antimicrobial secondary metabolites. Some metabolites represent constitutional chemical barriers to microbial attack and other inducible antimicrobials[39]. On the other side, an alternate approach would be the production of higher quantity of phytoalexin (antimicrobial compounds) by plants. This can be done either by spraying with phytoalexin elicitors, or by pre-immunization through a non-pathogen inoculation, or alternatively by genetic transformation. One problem of the first approach is that continuous production of phytoalexins results in stunted plants which produce a yield as poor as the infected plants[40]. Therefore, an enough regulation of the production of antimicrobial compounds could help resolve the problem. There must be some natural cases of induced resistance to protect hosts from disease. Elicitors activate chemical defense in the infected host plants. Several biosynthetic pathways are activated in treated host plants depending on the constituent used[41]. Commonly tested chemical elicitors are salicylic acid, methyl salicylate and also production of phenolic compounds and activation of various defense related enzymes in hosts[42,43]. Plant responses to the effect of pathogenic stresses are complex and involve various physiological, molecular, and cellular adaptations. This stress may be interactive and/or antagonistic and include among others the involvement of phytohormones, transcription factors, kinase cascades, and reactive oxygen species. In certain cases, this can lead to the enhancement of plants resistance against pathogens[44]. Several studies reported the involvement of induced host defence, *e.g*., using *Trichoderma harzianum* to control *Botritis cinerea* in some economic crops, which resulted in a 25%–100% reduction of grey form symptoms and suppression of lesion formation[45]. Finally, the protection of host plants against pathogenic microorganisms through induced resistance can be made through the use of exogenous substances. It is probable that stimulated resistance against diseases either through chemical activators or other means will become an important component of pest management, specifically in cases where modern control means are less effective[34].

6. Conclusion

This review gave an insight into cross-interaction between host plant and pathogenic agent stress, focusing on the molecular events of infectious diseases to induce resistance. Whereas host plant secondary metabolites (phytoalexins) reviewed here demonstrate that they constitute an important mechanism to stop spreading of pathogens by acting as antimicrobials themselves or/and as elicitors of other defence responses. Additionally, activation of resistance occurs to increase or enhance the infected plant to produce secondary metabolites or chemical activators through the use of exogenous substances, especially in the cases that current control measures are less effective.

Conflict of interest statement

I declare that I have no conflict of interest.

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