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Plant-Pest Interaction: An Overview

Rajaram Panneerselvam and Alagu Lakshmanan

Stress Physiology Lab, Department of Botany, Annamalai University,
Annamalainagar 608 002, Tamilnadu - India

Abstract: The potential regulatory role of ROI involved in pathogen infection leads to HR. During HR the ROI play in different roles to trigger the defense mechanism of plants to protect from the destruction nature of diseases. Many findings provide direct evidence for the involvement of ROI in mediating pathogen induced PCD. Programmed cell death (PCD, or apoptosis) is a basic process for elimination of redundant and damaged cells in multicellular organisms, which operates in the course of their development and in response to various stress-inducing stimuli. PCD in plant and animal kingdoms has a number of morphological features in common, yet PCD mechanisms are far better understood in animals than in plants. Here, we have reviewed the effects of pests on plant metabolisms which may/may not lead to PCD and other metabolic alteration. This mini-review describes some specific areas of plant-pest interaction, with special emphasis on regulatory pathways.

Key words: Plants · Pathogen · Pests · PCD

INTRODUCTION

Upon recognition of a pathogen, plants exhibit remarkable ability to differentiate pathogen and later based on the nature of pathogen mount a resistance response meant to cease pathogen growth and disease development [1-5]. The resistance responses of plants include activation of local and systemic defenses (e. g. expression of pathogenesis-related proteins) and induction of a localized plant cell death at the site of infection called the hypersensitive response (HR). The HR is a form of programmed cell death (PCD) and shares some molecular and biochemical similarities with animal apoptosis.). It is believed that the coordinated activation of PCD and defense mechanisms at the site of pathogen entry provides the plant with an efficient defense response that prevents pathogen proliferation and its possible consequence: systemic infection.

Saviors of Plant Defense: Salicylic acid (SA), nitric oxide (NO) and reactive oxygen species (ROS) (particularly H_2O_2) increase in abundance following pathogen recognition and each are important signaling molecules that promote and coordinate defense and HR responses [1-4]. There exists complex synergistic (and possibly antagonistic) interactions between SA, NO and ROS that define the responses to biotic stress [2-4]. The increase in ROS (the so-called oxidative burst) involves activation of a plasma membrane-localized nicotinamide adenine dinucleotide phosphate (NADPH) oxidase. During the HR, this is accompanied by an active down-regulation of ROS-scavenging systems to further promote ROS accumulation. It is hypothesized that plant mitochondria act in the perception of biotic stress and take part in initiating responses such as the HR.

Plants and Insects Interaction: The key mechanism used by plants to recognize herbivorous caterpillars is elicitors present in their oral secretions; however, these elicitors not only cause the induction of plant defenses but recent evidence suggests that they may also suppress plant responses. The absence of "expected changes" in induced defense responses of insect-infested plants has been attributed to hydrogen peroxide produced by caterpillar salivary glucose oxidase (GOX). Activity of this enzyme is variable among caterpillar species; it was detected in two generalist caterpillars, the beet armyworm (*Spodoptera exigua*) and the bertha armyworm (*Mamestra configurata*), but not in other generalist or specialist caterpillar species tested. In the beet armyworm, GOX activity fluctuated over larval development with high activity associated with the salivary glands of fourth instars. This implies that a factor in the diet is involved in the regulation of caterpillar salivary enzyme activity.

Corresponding Author: Dr. R. Panneerselvam, Department of Botany,
Annamalai University, Annamalainagar 608 002 Tamilnadu, India

Therefore, plant diet may be regulating caterpillar oral elicitors that are involved in the regulation of plant defense responses.

In response to herbivores, volatile terpenoid compounds are synthesized and released by many plant species. The synthesis of terpenoids affected by the herbivores at molecular level to control the expression of genes involved in metabolic pathway of terpenoid synthesis.

Plants Topical Suicide-A Mechanism to Protect: Localized activation of programmed cell death (PCD) in response to microbial attack is thought to act as a defense mechanism that inhibits the growth of pathogens within infected plant tissues. By killing cells at and around the site of infection this process generates a physical barrier composed of dead plant cells and limits the availability of nutrients to the pathogen because of rapid dehydration that accompanies tissue death, which subsequently accompanied by the induction of numerous antimicrobial defenses. Among these are pathogenesis-related (PR) proteins, such as glucanases and chitinases and phytoalexins. PCD that occurs during the HR is accompanied by an increase in the production of reactive oxygen intermediates (ROI). Recent studies indicated that ROI in the form of H_2O_2 and O^ may be key mediators of PCD during the HR. ROI also were implicated as signal transduction agents that lead to the induction of other defense mechanisms such as PR proteins, salicylic acid (SA) biosynthesis and systemic acquired resistance H_2O_2 was shown to induce PCD and defense mechanisms in bean and tobacco.

It is presumed that the recognition of an invading pathogen results in the activation of a plasma membrane-associated NAD(P)H oxidase and the excess production of O_2. Subsequent spontaneous or superoxide dismutase (SOD) catalyzed dismutation of O_2 leads to the accumulation of H_2O_2 that diffuses into cells and causes the activation of defense mechanisms and PCD. In accordance with this model, ROI scavenging mechanisms such as ascorbate peroxidase (APX) and catalase (CAT) were shown to be suppressed during the HR, a process that was suggested to potentiate the induction of PCD and other defenses because of the increase in ROI levels that results from the decreased capability of cells to scavenge H_2O_2 during the HR. Mitochondrial electron transport is associated with the generation of ROS such as super oxide and H_2O_2. Because ROS can damage macromolecules, their cellular levels are managed through avoidance and scavenging mechanisms [2, 3].

Destiny of Mitochondria and Chloroplast: The signaling roles for SA during biotic stress, suggested its direct impact in mitochondria. It was shown that SA disrupts mitochondrial function in a concentration-dependent manner in tobacco suspension cells [2-5]. At low concentrations, it acted as an uncoupler, whereas at higher concentrations it strongly inhibited electron flow. These effects were seen in both whole cells and isolated mitochondria and provide a rationale for studies showing that SA could dramatically inhibit ATP synthesis by tobacco cells [2-4]. AOX expression correlated with the ability of SA to disrupt mitochondrial function. Hence, AOX may represent an excellent 'reporter gene' to evaluate whether mitochondrial dysfunction is occurring during biotic stress. Another signal molecule during biotic stress is NO, which along with SA and ROS, has been shown to promote the HR Plant cyt oxidase is similarly sensitive to NO but whether the physiological

No concentrations generated during plant-pathogen interactions are sufficient to inhibit cyt oxidase and whether such inhibition contributes to defense responses or the HR remains unknown. Chloroplasts are a large source of ROS and so it is often assumed that the steady-state level of cellular ROS as well as the capacity of cellular ROS-scavenging systems is largely determined by this organelle.

Action of Host Versus Pathogen: An important set of virulence factors in pathogenic fungi is the so-called host-selective toxins that interact with host molecules to cause plant cell death and contribute to disease development. One such toxin, victorin, was shown to bind to and inhibit mitochondrial glycine decarboxylase (GDC), suggesting that GDC inhibition acted to promote cell death. Victorin treatment of oat leaves resulted in a loss of DCm, followed by an ability of victorin to gain access to the mitochondrial matrix. The cell death precedes access of victorin to the cell interior and that victorin likely interacts with a cell surface protein to initiate defense responses and cell death. In this respect, the virulence of the toxin may reside in its ability to elicit a plant PCD pathway. These results shed doubt on the importance of victorin-induced GDC inhibition in promoting cell death, but they do not preclude a role for the mitochondrion in this cell death. In particular, victorin induces a burst of mROS preceding death. Harpins are virulence factors produced by bacterial pathogens such as *Pseudomonas syringae.* At low concentrations, H_2O_2 acts as a signaling molecule in the plant [3, 4]. During incompatible plant-pathogen interactions, endogenous H_2O_2 levels increase and have

been implicated in localized cell death during the hypersensitive response of some plant species against invading pathogens (De Gara et *al.* 2003). It was shown that a large and early ROS burst associated with harpin treatment emanated specifically from the mitochondrion, suggesting the ETC as the likely source of ROS [3-5].

There is also evidence that the ROS-scavenging capacity of the mitochondrion is modulated in response to pathogen infection. In particular, increases in mitochondrial superoxide-scavenging capacity combined with decreases in the H_2O_2-scavenging components of the organelle were seen during *Botrytis cinerea* infection of tomato leaves and it was hypothesized that this could promote accumulation of mitochondrial H_2O_2 [4, 5]. Such results imply an active mechanism to ensure accumulation of specific ROS species at the mitochondrion.

Polyamines such as spermine are proposed to play a role during biotic stress responses. Spermine accumulates dramatically and in an N-gene-specific manner in the apoplast of tobacco mosaic virus (TMV)-infected tobacco because of up regulation of genes involved in spermine biosynthesis [6-8]. Accumulated polyamines are subsequently degraded in the apoplast by polyamine oxidase, generating H_2O_2 that may contribute to plant responses [5, 6]. Exogenous application of spermine to tobacco leaves could induce defense responses and cell death, mediated by a pathway involving activation of mitogen activated protein (MAP) kinases and resulting in increased expression of HR marker genes and transcription factors [6-9]. It was also shown that AOX induction and activation of the MAP kinases could be blocked by the antioxidant flavone and by the Ca^{21} channel blocker $La^{2\ 1}$ suggesting that increases in ROS and influx of Ca^{21} are cellular events.

The treatment of tobacco cells with AA resulted in the rapid expression of eight different genes, as identified using differential display. Only one of these genes encoded an ETC component (AOX), whereas the others encoded proteins more generally implicated in either senescence or (biotic) stress responses. All eight genes were also induced by treatment of cells with SA or H_2O_2. Each of the gene-inducing treatments was associated with increased cellular levels of ROS and gene induction could be partially blocked by antioxidants that lowered ROS levels, suggesting ROS as an important intermediary in gene induction. The authors also showed that pre-treatment of cells with BA(Bonkrekic acid) blocked induction of all eight genes regardless of whether AA (Antimycin A), SA or H_2O_2 was used as the inducing agent [10].

CONCLUSION

The potential regulatory role of ROI involved in pathogen infection leads to HR. During HR the ROI play in different roles to trigger the defense mechanism of plants to protect from the destruction nature of diseases. Many findings provide direct evidence for the involvement of ROI in mediating pathogen induced PCD. Thus we are optimistic that the high expectations rose by the recent reports on PCD mechanisms and plant-pest interactions for fully revealing all the steps and pathways can be met in the not too distant future.

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