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Unraveling the Role of Bacterial Toxins in Plant Disease Development: Mechanisms, Impacts, and Mitigation Strategies

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ABSTRACT: Bacterial toxins can play a significant role in the development of plant diseases, particularly those caused by pathogenic bacteria. These toxins are typically produced by the bacteria as part of their virulence mechanism to facilitate colonization, infection, and disease progression. These toxins may work as cell wall degrading agents, phytotoxins interfering with cellular processes, toxins inducing host defense responses and by facilitating virulence. Toxins contribute to disease symptoms development, impact on disease severity and spread and they also cause ecological implications as on plant communities and ecosystems. Development of novel antimicrobial agents targeting toxin synthesis or activity and biocontrol agents targeting toxin production may reduce the effects of toxins on disease development.

KEYWORDS: Bacteria, disease, fungi, symptoms, toxins.

I. INTRODUCTION

Phyto-bacterial toxins play a pivotal role in the pathogenesis of plant diseases by facilitating bacterial colonization, host tissue damage, and disease progression. Understanding the diversity and mechanisms of action of these toxins is essential for elucidating host-pathogen interactions and developing effective strategies for disease management. This research paper provides an overview of the various types of phyto-bacterial toxins, their mechanisms of action, and their implications in plant disease development. Bacterial toxins may contribute to plant disease development by following ways.

Cell Wall Degradation: Many bacterial toxins target the cell walls of plant tissues, causing degradation and weakening of the cell structure. This breakdown of cell walls facilitates bacterial entry and spread within the plant host.

Cellular Disruption: Bacterial toxins often interfere with cellular processes within plant tissues. They may disrupt important cellular functions such as protein synthesis, cell signaling, or membrane integrity. This disruption can lead to cell death and tissue necrosis, characteristic symptoms of many bacterial plant diseases.

Induction of Plant Defense Responses: Some bacterial toxins trigger plant defense responses, which can paradoxically contribute to disease development. For example, they may induce the production of reactive oxygen species (ROS) or activate defense signaling pathways that inadvertently promote bacterial colonization and disease spread.

Toxin-Mediated Virulence: Bacterial toxins directly contribute to the virulence of pathogenic bacteria by enhancing their ability to overcome plant defenses and establish infection. Toxins may aid in the suppression of host immune responses or manipulate host physiology to create a more favorable environment for bacterial growth and survival.

Symptom Development: Bacterial toxins often cause specific disease symptoms in infected plants. These symptoms may include wilting, leaf spots, necrosis, chlorosis, or other visible signs of disease. The toxins can directly induce these symptoms or contribute to their development by exacerbating tissue damage and stress.

Host Specificity: Some bacterial toxins exhibit host specificity, meaning they target specific plant species or genotypes. This specificity can determine the range of plant species susceptible to infection by particular bacterial pathogens and influence the severity of disease outbreaks in agricultural or natural ecosystems.

II. DISEASE DEVELOPMENT

Plant diseases are the result of biochemical reactions between substances secreted by the pathogens and host tissues and plant body. These substances are toxins, enzymes, polysaccharides, antibiotics and growth regulators which can vary in their pathogenicity. Almost all disease causal agents produce these chemical substances except viruses. Several toxins have been shown to be involved in disease development (Graniti, 1991). Guamann (1954) stated that microorganisms are pathogenic only if they are toxigenic. Toxins are extremely poisonous and effective in very low

concentrations. Toxins injure host cells either by affecting osmotic relations or by affecting metabolic reaction disrupting physiological process in the plant. Toxins may be endotoxins which are also known as intercellular toxins formed in bacterial cells and ectotoxins which are also known as extracellular toxins which diffuse from living bacterial cells and interact with the site of infection or tissues distant from the site of infection. Endotoxins are liberated following the lysis of bacterial pathogen cells. Bacterial endotoxins are lipopolysaccharides (LPS) in nature which are present in the outer membrane of Gram negative bacteria such as *Pseudomonas* (Wheeler and Luke, 1963). Bacterial toxins or mycotoxins synthesized by fungal pathogens act as major virulence factor of a variety of microorganisms (Upadhyay et al., 2015).

Bacterial toxins show extreme diversity in their structure, mode of action, intracellular receptor recognition, enzymatic activity and mode of secretion. In addition to their role in disease development, bacterial toxins may be used as tools for diagnosis, prevention and therapy of diseases (Popoff, 2018). The toxins are characterized by high efficiency, high potency and high specificity. These are frequently used biological and pharmacological tools (Aktories and Schmidt, 2015). Many of the fungal toxins induce permeability changes depending on the extent to injury to cells or tissues. Toxins also showed the inhibitory effects upon respiration and respiratory enzymes which act against the microbes (Sadasivan, 1968). Toxins are different from enzymes in that they affect the metabolic process but do not attack the structural integrity of the host tissue.

TYPES OF TOXINS

Toxins have been classified on the basis of their chemical characteristics and may be classified on their biological activity, antimetabolites, membrane affecting compounds and enzyme inhibitor efficacy. However, toxins broadly classified on the basis of their host selectivity and on general roles in disease developments (Durbin, 2012). In their disease development toxins are divided in three classes namely pathotoxins, vivotoxins and phytotoxins.

Pathotoxins

Pathotoxins may be regarded as chemopathogens of biological origin including toxic substances of the pathogen, the host or the interaction between them. Meehan and Murphy (1946) firstly described the toxic product produced by *Helminthosporium victoriae* a fungal pathogen causing Victoria blight of oats (Wheeler and Luke, 1963). This toxic substance later termed as 'victorin' toxin by wheeler and Luke (1954). Victorin toxin found to increase the permeability of susceptible tissues (Wheeler, 1961). Most of the pathotoxins are produced by pathogens during pathogenesis. Keck and Hodges (1972) also observed that toxins produced by *H. victoriae* increased the permeability of plasma membrane and tonoplast of root cells in susceptible cultivar of *Avena sativa* while it could not affect a resistant cultivar. Spot blotch disease in wheat and barley is caused by *Bipolaris sorokiniana*. The causal agent produces toxin BS-toxin, a sesquiterpenoid which is found to be degraded in the samples treated with bioagent *Chaetomium globosum* (Aggarwal et al., 2011).

Host selective pathotoxins play important role in disease outbreaks and severity of plant diseases. Most of the host selective pathotoxins belongs to two genera *Helminthosporium* and *Alternaria* such as *H. victoriae* on oats, *H. maydis* on Texas male sterile corn, *H. Carbonum* on sugarcane, *Alternaria mali* on apple, *A. alternata* on tomato and strawberry and *A. citri* on rough lemon and mandarin orange (Daly et al., 2013). The wildfire disease of tobacco is caused by *Pseudomonas tabaci* characterized by chlorotic halos. The bacterial pathogen produces wildfire toxin which satisfy the all the criterion to be in the class of pathotoxins. Evidences suggested that wildfire toxin is a single entity which has the ability to produce wildfire disease symptoms in susceptible host but not in resistant cultivars (Wheeler and Luke, 1963). Tobacco wildfire toxin has been found to inhibit the growth of *Chlorella vulgaris* and induce chlorotic symptoms in higher plants. It also causes convulsions identical to those induced by methionine sulphoximine in animals. These both compounds also inhibit cerebral glutamine synthetase (Wheeler and Luke, 1963, Sinden and Durbin, 1968).

Vivotoxins

Vivotoxins are chemical substances which functions to produce at least a portion of disease but it not itself the initiating causal agent of the infection. They are usually non specific and generally induce only part of the symptoms of disease (Wheeler and Luke, 1963, Srivastava et al. 2020). Dimond and Waggoner (1953) coined the term vivotoxin and described it as a substance produced in the infected host by the causal agent and or by its hosts or by both which leads to develop a disease but is not itself the initial inciting agent for symptoms development.

Vivotoxin shows three specific characteristic for its vivotoxicity. First is the reproducible segregation from the infected host plant, second its purification and last is reproduction of at least a portion of the disease symptoms by inoculating

the toxin in same healthy host plant. Fusaric acid was observed highest powerful toxin but didn't produce symptoms during the initiation of disease development because it was found that it alienates at above 6.0 pH (host plant tissue sap pH is 6.2 during initial execute of infection) and it is produced in little amount during initial stage of infection. Fusaric acid is a pyridine carboxylic acid and chemically it is a 5-n-butyl-picolinic acid. It was found to inhibit the growth of bacteria, affecting permeability of green algae *Spirogyra nitida*, affected germination of basidiospores of *Ustilago maydis* at specific concentrations (Srivastava *et al.* 2020). Vivotoxins generally categorized into two groups, one is 'Ipomeamarone' which is produced by the host in response to infection and another is 'Piricularin' which is produced by invading pathogens. The ipomeamarone is produced by the host due to infection of two unrelated fungal pathogens *Ceratostomella fimbriata*, the causal agent of the sweet potato black rot and *Helicobasidium mompa*, which causes violet root rot of sweet potatoes (Ingham, 1972). Seedling symptoms and leaf desiccations of rice in blast disease have been attributed to toxins α picolinic acid and piricularin isolated from cultures of pathogen and from diseased plants. Piricularin considered as semi specific in nature as it affect resistant varieties less than susceptible ones (Wheeler and Luke, 1963).

Phytotoxins

Phytotoxins are non specific in nature and may not show any relation between toxin production and pathogenicity. Most of the toxic substances which are not qualify as pathotoxins or vivotoxins fall in the phytotoxin category. Phytotoxins are produced by the invading causal agent upon infection of higher plants and these may be responsible for development of disease symptoms. The symptoms may vary from wilting, chlorosis, and necrosis to growth abnormalities to death of host plant. Phytotoxins inhibit specific diverse enzymes by a complex series of metabolic dysfunctions (Durbin, 1991). The toxic effects of 'fusicochin', the toxin produced by *Fusicoccum amygdale* on almond trees was believed to interfere with the function of leaf plasmalemma (Harborne, 1999). *Xanthomonas*, *Pseudomonas*, *Rhizobium* and *Cornetbacterium* are the most common bacterial causal agents of plant diseases and produce phytotoxins (Strobel, 1982). *Pseudomonas* spp. produce a wide range of non host specific phytotoxins. *Pseudomonas aeruginosa* produces Pseudomonas Exotoxins (PE) which has enzymatic activity and belongs to the mono ADP ribosyltransferase family (Marta and Phillip, 2015). Coronatine phytotoxine secreted by *Pseudomonas syringae* is a specific virulence factor for inducing disease symptoms. The bacterial pathogen invade through stomata and grows in apoplast (Geng *et al.* 2012). Different Pathovars of *P. syringae* are known to produce coronatine, phaseolotoxin, tabtoxin and syringomycin and syringopeptin (causes various symptoms) (Hu *et al.* 1998, Cazorla *et al.* 1998, Bender *et al.* 1999, Trigiano, Windham and Windham, 2004, Agrios, 2005, Geng *et al.* 2012, Gallarato *et al.* 2012). These phytotoxins may enhance the virulence of producing pathogens, even though some disease can occur in their absence. Visual assessment of phytotoxin production in plants can be observed by their reaction to phytotoxins (chlorosis, necrosis, stunting, blighting and wilting symptoms). Some phytotoxins may instead act by changing metabolic processes in the host in such a way that the deleterious activity might be manifested only at the biochemical level. Tabtoxin is chemically a monocyclic β -lactam which is produced by *P. syringae* pv. *tabaci*, *coronafaciens*, and *garcae*. It had also been detected in some *P. syringae* pv. *tomato* strains. This toxin act to inhibit the target enzyme glutamine synthetase, which induces abnormal accumulation of ammonia in plant cells, causing the characteristic chlorotic symptom in susceptible host plants. It was also observed that *P. syringae* has evolved to survive in choline-rich environment as choline and metabolic derivatives increased tabtoxin production in *P. syringae* (Gallarato *et al.* 2012). These toxins are chemically not related to each other and act in different ways to alter plant metabolism. Their activities were demonstrated by the production of distinctive chlorotic or necrotic symptoms following their diffusion into plant tissues showed successful pathogenicity (Hu *et al.* 1998).

Pathogenic microorganisms produce a number of chemical substances to facilitate their invasion and growth in the host plants. Their mechanism to induce pathogenesis is specific for each microbe and in different host plants. However, the correlations between production of toxins and pathogenicity have not been observed.

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