



Microbial Toxins and Their Role in Plant Disease Development

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Microbial toxins are important chemical substances produced by plant-pathogenic microorganisms that contribute to the development and expression of disease in plants. They may be produced by fungi and bacteria and can interfere with normal physiological and biochemical processes of the host. Unlike enzymes, which generally act as biological catalysts and participate in specific biochemical reactions, toxins primarily produce harmful effects on host tissues and may contribute directly to symptom development. Depending on their origin, biological activity, and relationship with the host, microbial toxins can influence membrane function, metabolism, growth, and other essential cellular processes. Toxins associated with plant diseases can be broadly considered as pathotoxins, vivotoxins, and phytotoxins. They also differ in their host specificity. Some toxins affect only particular plant species or cultivars and are therefore referred to as selective toxins, whereas others act on a wider range of plant hosts and are considered non-selective toxins. The biological effects produced by these compounds are closely associated with the ability of the pathogen to establish disease and produce characteristic symptoms.

Classification of Plant Pathogenic Toxins

Plant pathogenic toxins can be classified according to their relationship with the pathogen and host. Pathotoxins are toxins associated with pathogenic microorganisms and their disease-producing activities. Vivotoxins are substances produced by pathogens that contribute to disease development in living host tissues. Phytotoxins are toxic substances of microbial origin that adversely affect plants. These categories emphasize the importance of toxic metabolites in the interaction between a pathogen and its host. Another important classification is based on host specificity. Selective toxins show activity against particular plant species, varieties, or cultivars and are closely associated with the susceptibility of the host. In contrast, non-selective toxins can affect a broader range of plant tissues or hosts and generally produce toxicity without the same degree of host specificity. Both groups can contribute substantially to disease development, but their effects on host plants may differ according to their biological properties.

Effects of Toxins on Host Tissues

The effects of microbial toxins on plants are diverse because toxins can interfere with several physiological and biochemical processes. One important effect is alteration of cell membrane permeability. Changes in membrane properties can disturb the normal movement of water, ions, and other cellular constituents, ultimately affecting cellular integrity and normal plant function. Toxins can also cause metabolic disruption within host tissues. Normal biochemical processes depend on the coordinated functioning of enzymes, membranes, metabolites, and cellular structures. Interference with these processes can reduce normal cellular activity and contribute to the development of disease symptoms. In addition, some toxins have growth-

regulatory effects, resulting in changes in plant growth and development. Thus, toxins may act at different physiological levels and collectively contribute to visible symptoms associated with pathogen infection. The importance of toxins in disease development therefore extends beyond their direct toxicity. Their effects on membranes, metabolism, and growth can weaken host tissues and create conditions favorable for the development and progression of disease.

Selective Toxins

Selective toxins are associated with diseases in which the toxin exhibits specificity toward particular hosts or host genotypes. These toxins are particularly important because their biological activity may be closely related to host susceptibility. Several important selective toxins described in the source material include Victorin, T-toxin, HC-toxin, HS-toxin, PC-toxin, and AK-toxin.

Victorin or HV-toxin is produced by *Cochliobolus victoriae*, the causal fungus of Victoria blight of oat. The disease is characterized by necrosis of the roots and stem base along with blighting of the leaves. Victorin is a peptide toxin and is associated with pathogenicity on susceptible oat cultivars. The toxin is highly mobile within the plant. Its effects include inhibition of the glycine permeability enzyme complex involved in photorespiration and loss of chlorophyll, which can trigger premature senescence.

T-Toxin, also known as *Helminthosporium maydis* race T-toxin, is produced by *Helminthosporium maydis*, the pathogen responsible for leaf blight of maize. The toxin is particularly associated with Texas cytoplasmic male-sterile (T-CMS) maize. T-toxin disrupts the function of mitochondria in susceptible T-CMS maize, thereby contributing to disease development.

HC-toxin is produced by the fungus *Helminthosporium carbonum*, which causes leaf spot of maize. The fungus has four races, designated 1, 2, 3, and 4, of which race 1 produces HC-toxin. Certain maize inbred lines and their hybrids are highly susceptible to *H. carbonum* and its toxin.

HS-toxin is produced by *Helminthosporium sacchari*, the causal organism of eyespot disease of sugarcane. The disease is particularly severe in seedlings of certain sugarcane cultivars. The host-specific toxin produces reddish-brown stripes when introduced into susceptible leaves. It also causes changes in chloroplasts and cell-wall permeability, contributing to the development of disease symptoms.

PC-toxin, also called Periconia toxin, is produced by *Periconia circinata*, which causes Milo disease or Periconia blight of grain sorghum. The disease primarily attacks Milo-type grain sorghum. The pathogen is soil-borne and invades the roots and lower internodes. PC-toxin causes rapid loss of potassium ions and other cellular materials through leakage across the plasma membrane of susceptible tissues, whereas resistant tissues are not affected in the same manner.

AK-toxin, also referred to as alternarin, is a host-specific toxin produced by *Alternaria kikuchiana*, the causal fungus of black spot of Japanese pear. Three phytoalternarins, designated A, B, and C, have been obtained from culture filtrates of the fungus. The toxin causes direct damage to the plasma membrane, resulting in rapid loss of electrolytes from susceptible tissues.

Non-Selective Toxins

Non-selective or non-specific toxins differ from host-specific toxins because they do not show the same degree of host specificity. According to the source material, they can affect the physiology of plants that are not normally infected by the pathogen. The PPT groups these toxins into pathotoxins, vivotoxins, and phytotoxins. Examples include tentoxin, tabtoxin, fusaric acid, pyricularin, and alternaric acid.

Tabtoxin, also known as wildfire toxin, is produced by *Pseudomonas syringae* pv. *tabaci*, the causal bacterium of tobacco wildfire disease. The disease is characterized by necrotic lesions on leaves surrounded by a yellow halo. Tabtoxin has a single major effect and inhibits

the enzyme glutamine synthetase. This results in accumulation of toxic concentrations of ammonia, which disrupts the carbon and energy fixation components of photosynthesis and damages chloroplast membranes.

Phaseolotoxin is produced by *Pseudomonas syringae* pv. *phaseolicola*, which causes halo blight of bean and some other legumes. The disease is associated with chlorotic halos and accumulation of ornithine in affected tissues. The functional toxin in the plant is octicidin, which is more potent than intact phaseolotoxin. Octicidin blocks the conversion of ornithine into citrulline, a precursor of arginine. The resulting depletion of arginine in developing leaves contributes to chlorosis.

Tentoxin is produced by *Alternaria alternata*. The toxin and its producing pathogen cause striking variegated chlorosis in seedlings of cucumber, cotton, citrus, and other plants. Tentoxin inhibits the phosphorylation of ADP to ATP and causes a substantial reduction in chlorophyll content. These effects interfere with normal energy metabolism and contribute to the development of chlorotic symptoms.

Cercosporin is produced by species of *Cercospora* and is classified as a non-selective toxin. The toxin is activated by light, and the plant cell membrane is an important target of its activity. Its light-dependent activity contributes to damage of plant tissues and consequently to disease development.

Pyricularin is produced by *Pyricularia grisea*, the causal organism of rice blast. According to the source material, symptoms of the disease have been attributed to two toxins, α -picolinic acid and pyricularin. Pyricularin is toxic to conidial germination. The fungus produces a pyricularin-binding protein, described in the PPT as copper oxidase, which binds pyricularin and destroys its fungitoxicity but not its phytotoxicity. At low concentrations, pyricularin affects respiration and growth, whereas at higher concentrations it inhibits these processes.

Conclusion

Microbial toxins are important factors in the development and expression of plant diseases. They can interfere with essential host processes, particularly membrane function, metabolism, and plant growth, thereby contributing to physiological disturbance and symptom development. The classification of toxins into selective and non-selective groups highlights the diversity of toxin-mediated interactions between pathogens and plants. Selective toxins such as victorin, T-toxin, HC-toxin, HS-toxin, PC-toxin, and AK-toxin are associated with specific host-pathogen interactions, whereas non-selective toxins such as tabtoxin, phaseolotoxin, tentoxin, cercosporin, and pyricularin have broader effects on plant tissues. Overall, toxins form an important part of the mechanisms through which microbial pathogens disturb normal plant physiology and promote disease development. Understanding their biological effects and their relationship with host susceptibility is therefore essential for a clear understanding of plant-pathogen interactions and the mechanisms underlying disease expression.