Role of Toxins in Pant Disease Development

Toxins are compounds that are produced by the pathogens and cause part or all of the symptoms of a disease. Genetic and biochemical studies revealed that at least in part of the plant-pathogen interactions toxins are the determinants of specificity. In such cases, resistance or susceptibility to the fungus correlates with insensitivity or sensitivity to the toxin (Slavov, 2005). Microbial toxins have been the objects of extensive studies as possible pathogenicity or virulence factors for the producer pathogens. Toxins are considered to be the special weapons of the plant pathogens to evade or overcome the inherent resistance strategies of host plants (Kimura *et al.*, 2001).

According to Gaumann in 1954, "Micro-organisms are pathogenic only if they are toxicogenic: in other words, the agents responsible for diseases can damage their hosts only if they form toxins – microbial poisons that penetrate into host tissue".

According to Ludwig in 1960, "A toxin as a product of a microorganism or of a microorganism host complex which acts on living host protoplast to influence disease development or symptoms".

Toxin Hypothesis

The toxin hypothesis states that a toxic substance (X) is directly responsible for the symptoms of disease (Y). Even more briefly, X causes Y. When X is a toxin produced by a microbial pathogen, the hypothesis predicts:

(a) the toxin will produce all symptoms characteristic of the disease

(b) sensitivity to the toxin will be correlated with susceptibility to the pathogen

(c) toxin production by the pathogen will be directly related to its ability to cause disease.

These requirements were clearly met for the first time by a toxin produced by *Helminthosporium victoriae*, the fungus which causes Victoria blight of Oats (Meehan and Murphy, 1947; Luke and Wheeler, 1955).

Effect of Toxins on Host Tissues

Changes in cell permeability

Toxins kill plant cells by altering the permeability of plasma membrane, thus permitting loss of water and electrolytes and also the unrestricted entry of substances including toxins. The cellular transport system, especially, H^+ /K^+ exchange at the cell membrane is affected.

Disruption of normal metabolic processes

Due to changes in cell wall permeability many physiological activities of the host cells disrupted. The disturbed salt balance in the protoplasm causes increase in respiration. The loss of water and other substances causes malfunctioning of the enzyme system finally resulting in death of cell. Such as pyricularin toxin inhibits polyphenol oxidase system and victorin acts by uncoupling of oxidative phosphorylation.

Other mechanisms

Interfere with the growth regulatory system of host plant may cause stimulation of growth of plant parts. *Fusarium moniliforme* produces a thermo-stable toxin even in soil around the roots which

causes browning roots and their restricted development. Stomatal dysfunction has also been reported for certain phytotoxins. Physical blocking effect of large molecule is also a mechanism of phytotoxicity by toxins.

Classification of Toxins:

There are three broad classes of toxins namely, phytotoxins, vivotoxins and pathotoxins according to their sources of origin.

Phytotoxins

Phytotoxins are products of plant pathogens or of the host–pathogen interaction that directly injure plant cells and influence the course of disease development or symptoms (Bender et al., 1999).

Any compound produced by a microorganism which is toxic to plants is a phytotoxin. Phytotoxins are nonspecific, incite few or none of the symptoms that are incited by the pathogen, and as happens in most cases, and show no relation between toxin production and pathogenicity. Altenaric acid produced by *Alternaria solani* is a typical example of phytotoxin.

Vivotoxins

It is defined as a substance produced in the infected host by the pathogen and/or its hosts which functions in the production of the disease but is not itself the initial inciting agent of the disease. The criteria were suggested for a vivotoxin: reproducible separation of the toxin from the sick plant, purification or chemical characterization, and induction of at least a part of the disease syndrome by placing the toxin in a healthy plant. Fusaric acid is a typical example of vivotoxin.

Pathotoxins

Pathotoxins play a major causal role in disease development, and produce symptoms characteristic of the disease in susceptible plants. Pathotoxins may be produced by the pathogen, host, or interaction between them. Victorin produced by *Cochliobolus victoriae* (*Helminthosporium victoriae*) is a typical example of pathotoxin.

According the specificity the toxin can be divided into two types

Non-specific (Non-selective) toxin

Host-specific (Selective) toxins

Non-specific (Non-selective) toxin which can affect the protoplasm of many unrelated to plant species in addition to the main host of the pathogen producing toxin. Such as Tabtoxin, Phaseolotoxin, Tentoxin, Fusaric acid, Lycomarasmin, Pyricularin etc.

Tabtoxin

- Tabtoxin is produced by the bacterium *Pseudomonas syringae* pv. *tabaci*, which causes the wildfire disease of tobacco.
- Tabtoxin is a dipeptide composed of the common amino acid threonine and the previously unknown amino acid tabtoxinine.
- Tabtoxin as such is not toxic, but in the cell it becomes hydrolyzed and releases tabtoxinine, which is the active toxin.

- Toxin-producing strains cause necrotic spots on leaves, with each spot surrounded by a yellow halo.

Tentoxin

- Tentoxin is produced by the fungus *Alternaria alternata* (previously called *A. tenuis*), which causes spots and chlorosis in plants of many species.
- Tentoxin is a cyclic tetrapeptide that binds to and inactivates a protein (chloroplast-coupling factor) involved in energy transfer into chloroplasts.
- The toxin also inhibits the light-dependent phosphorylation of ADP to ATP. In sensitive species, tentoxin interferes with normal chloroplast development and results in chlorosis by disrupting chlorophyll synthesis.
- An additional but apparently unrelated effect of tentoxin on sensitive plants is that it inhibits the activity of polyphenol oxidases, enzymes involved in several resistance mechanisms of plants.

Host-specific (Selective) toxins which adversely affect only the specific host of the pathogen. The letter are very active and can produce their effect even in extremely low quantities. Such as Victorin or HV-toxin, T-toxin/*Helminthosporium maydis* race T-toxin, HC-toxin/ *Helminthosporium carbonum* toxin, HS-toxin/ *Helminthosporoside,* AK-toxin/ *Phytoalternarin,* PC-toxin/ *Periconia* toxin, AM-toxin/ *Alternaria mali* toxin etc.

Victorin, or HV Toxin

- Victorin, or HV-toxin is produced by the fungus *Cochliobolus* (*Helminthosporium*) victoriae, which causes Victoria blight of oats.
- This fungus appeared in 1945 after the introduction and widespread use of the oat variety
- *C. victoriae* infects the basal portions of susceptible oat plants and produces a toxin that is carried to the leaves, causes a leaf blight, and destroys the entire plant.
- Victorin is a complex chlorinated, partially cyclic pentapeptide.
- The primary target of the toxin seems to be the cell plasma membrane, where victorin seems to bind to several proteins.
- Victorin also functions as an elicitor that induces components of a resistance response that include many of the features of hypersensitive response and lead to programmed cell death.

T Toxin [HMT Toxin]

- T toxin is produced by race T of *Cochliobolus heterostrophus* (anamorph: *Bipolaris maydis*, earlier called *Helminthosporium maydis*), the cause of southern Corn leaf blight.
- First appeared in the United States in 1968, it spread throughout the Corn Belt by 1970, attacking only corn that had the Texas male-sterile (Tms) cytoplasm.
- The ability of *C. heterostrophus* race T to produce T toxin and its virulence to corn with Tms cytoplasm are controlled by one and the same gene.
- T toxin does not seem to be necessary for the pathogenicity of *C. heterostrophus* race T, but it increases the virulence of the pathogen.
- The T toxin apparently acts specifically on mitochondria of susceptible cells, which are rendered nonfunctional, and inhibits ATP synthesis.