A. Pathotoxins

1. These are the toxins which play a major role in disease production and produce all or most of the symptoms characteristic of the disease in susceptible plants.

2. Most of these toxins are produced by pathogens during pathogenesis.

Ex: Victorin: *Cochliobolus victoriae (Helminthosporium victoriae)*, the causal agent of Victoria blight of oats. This is a host specific toxin.

Classification based on specificity of toxins

1. Host specific / Host selective toxins:- These are the metabolic products of the pathogens which are selectively toxic only to the susceptible host of the pathogen.

Ex:- Victorin, T-toxin, Phyto-alternarin, Amylovorin.

2. Non-specific/Non-selective toxin:- These are the metabolic products of the pathogen, but do not have host specificity and affect the protoplasm of many unrelated plant species that are normally not infected by the pathogen.

Ex: Ten-toxin, Tab-toxin, Fusaric acid, Piricularin, Lycomarasmin and Alternaric acid

(Scheffer, 1983)

Differentiate host – specific and non-host specific toxins

Host specific.	Non-host specific.
1. Selectively toxic only to susceptible host of the pathogen.	1. No host specificity and can also affect the physiology of those plant that are normally not infected by the pathogen.
2. Primary determinants of disease.	2. Secondary determinants of disease.
3. Produce all the essential symptoms of the disease.	3. Produce few or none of the symptoms of the disease.
Ex: Victorin, T- toxin	Ex: Tentoxin, Tabtoxin

A. Host specific:-

T-toxin: *Helminthosporium maydis*
HC-toxin: *Helminthosporium carbonum.*
HS- toxin: *Helminthosporium sacchari.*
Phytoalternarin: *Alternaria kikuchiana*
PC- toxin: *Periconia circinata*

B. Non-host specific:-

Tentoxin: *Alternaria tenuis.*
Tabtoxin or wild fire toxin: *Pseudomonas tabaci.*
Phaseolotoxin: *Pseudomonas syringae* pv. *Phaseolicola.*

Effect of toxins on host tissues

1. Changes in cell wall permeability:

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

(Singh, 2001)

2.Disruption of normal metabolic processes:-

- Increase in respiration due to disturbed salt balance.
- Malfunctioning of enzyme system

(Singh, 2001)

3. Other mechanisms:-

- Interfere with the growth regulatory system of the host plant.

- Some toxins inhibit root growth.

Ex:-*Fusarium moniliforme* produces a thermostable toxin even in soil around the root which causes browning of the root and their restricted development.

(Singh, 2001)

The selective (Host-specific) Toxins

- **T-toxin or *Helminthosporium maydis* race T-toxin (HMT-toxin)** Is produced by the fungus *Helminthosporium maydis* (*Cochiobolus heterostrophus*). The pathogen cause leaf blight of maize.

- T-toxin is the disrupting the function of the mitochondria of Tcms maize.

- T-toxin bind to inner mitochondrial membrane protein (URF-13), the product of the T-URF 13, gene to create a pore on the membrane, cause leakage of small molecules, and subsequently inhibit ATP synthesis, resulting in cell death.

Victorin or HV-toxin:

- Is one of the most impotent and selective Pathotoxins. it is the first-well documented and widely recognised host-specific toxin.
- This toxin is produced by *Cochliobolus victoriae*, the fungus that causes victoria blight of oat.
- The disease is characterised by necrosis of the root and stem base and blighting of leaves.
- It is highly mobile in the plant.

HC- toxin or *Helminthosporium carbonum* toxin

- The fungus causes leaf spot of maize. The fungus species has tow races, 1 and 2. only race 1 produces the HC-toxin.

- Race 1 is also weak pathogenic on most lines but is highly virulent on maize that is homozygous recessive at the Mendelian loci Hm1 and Hm2.

PC-toxin or periconia toxin:

- Milo disease or **periconia blight** caused by ***periconia circinata*** attacks only milo type of grain sorghum.

- The pathogen is soil-borne and invade the root and lower internodes of the plant.

- The toxin causes rapid loss of potassium ions and other materials through leakage of the plasma membrane of susceptible but not resistance tissues.

Non-specific/Non-selective toxin:

- **Tabtoxin or Wildfire toxin:-** Tabtoxin is produced by *Pseudomonas syringae* pv. *tabaci* the causal bacterium of tobacco wildfire disease.

- In tobacco wildfire disease the necrotic lesion on the leaves are surrounded by a yellow halo.

- Most of the toxins produced by plant pathogens are pleiotropic that is, they have more than one effect on the host cell, but most bacterial toxin, include tabtoxin, are monotropic, having single effect

Phaseolotoxin

Phaseolotoxin: *Pseudomonas syringae* pv. *Phaseolicola*, causes halo blight of bean and some other legumes.

- The chlorotic halos are accompanied by ornithine accumulation in the tissues.
- Both race 1 and race 2 of *P. phaseolicola* can produce phaseolotoxin, but they differ in host range, suggesting that the toxin is not involved in host specificity.

Tentoxin:

- Is Produced by *Alternaria alternata.* causes leaf spots.
- That bind to inactivate the protein.
- Also inhibits phosphorylation of ADP to ATP.
- Leading to disruption of chlorophyll synthesis

Introduction

Antimicrobial compounds from plants are broadly classified into two categories: phytoantipicins and phytoalexins (Mansfield, 1999). Phytoanticipins; Phytoanticipins are described as "low molecular weight, antimicrobial compounds that are present in plants before challenge by micro-organisms, or are produced after infection solely, from pre-existing precursors". They include primarily the saponins, avenacin and tomatine. One saponin, avenacin A-1, is localized in the epidermis of oat roots and another saponin, a-tomatine, is produced in tomato and has antimicrobial activity against many fungi. Phytoalexins; Phytoalexins are toxic antimicrobial substances produced in appreciable amounts in plants only after stimulation by various types of phytopathogenic microorganisms or by chemical and mechanical injury. It was proposed in 1940 by Muller and Borger in the study of the interaction between potato and Phytophthora infestans. Most known phytoalexins are toxic to and inhibit the growth of fungi pathogenic to plants, but some are also toxic to bacteria, nematodes, and other organisms. More than 350 phytoalexins have been chemically characterized from approximately 30 plant families. The greatest number 130 have been characterized from the Leguminosae (Joseph, 1995). The chemical structures of

phytoalexins produced by plants of a family are usually quite similar; e.g., in most legumes, phytoalexins are is oflavonoids, and in the Solanaceae they are terpenoids. Phytoalexins represents one component of a battery of induced defence mechanisms used by plants including lytic enzymes such as chitinases and glucanases, oxidizing agents, cell wall lignifications and a number of pathogenesis-related (PR) proteins and transcripts of unknown functions (Dixon and Lamb, 1990; Lamb et al.,, 1989). It is important to recognize that phytoalexin accumulation may be part of a co-ordinated defence strategy, in which any one factor may alone be unable to account for restriction of the potential pathogen (Mansfield, 1999).

Chemical Diversity of Phytoalexins

Most phytoalexins produced by the Leguminosae belong to six isoflavonoid classes: isoflavones, isoflavanones, pterocarpans, pterocarpenes, isoflavans and coumestans (Table 1). Some pterocarpan phytoalexins are especially well known: pisatin, phaseollin, glyceollin, medicarpin and maackiain. Pisatin was the first phytoalexin to be isolated and characterized from garden pea, Pisum sativum (Cruickshank and Perrin 1960). Besides these compounds, a small number of legumes also produce non-isoflavonoid phytoalexins such as furanoacetylenes and stilbenes (Table 1).

Elicitors of phytoalexin accumulation

The molecules that signal plants to begin the process of phytoalexin synthesis are called elicitors. Elicitors of biotic origin may be involved in the interaction of plants and potential pathogens, whereas abiotic elicitors are not involved in normal host-pathogen interactions (Darvill and Albersheim, 1984).

In natural conditions, the stimulus is provided by the presence of the micro-organism and its perception by the host initiates the chain of events leading to phytoalexin synthesis. Biotic elicitors may originate in the invading organism, in which case they are referred to as "exogenous", whereas "endogenous" elicitors are of plant origin and are generated by the interaction between micro-organism and plant. Molecules with elicitor activity have been identified across a wide range of structural types including polysaccharides, glycoproteins, lipids, lipopolysaccharides, oligosaccharides and even enzymes, though their activity can be attributed to their effect in releasing elicitor-active components from the cell walls of the pathogen or host (Anderson, 1989; Blein et al.,,1991; Alghisi and Favaron, 1995). Abiotic elicitors form a diverse collection of molecules that are not derived from natural sources, such as the tissues of the pathogen or host. Under normal circumstances, they would not be encountered by the plant. The group includes compounds such as fungicides; salts of heavy metals, for example Cu2+ and Hg2+; the detergents, basic molecules such as polylysine and histone; reagents that are intercalated DNA (Darvill and Albersheim, 1984). Treatment of plant tissues with factors that cause stress, for example repeated freezing and thawing, wounding or exposure to UV light (Kodama et al., 1992; Mert Turk et al., 1998) can also induce phytoalexin synthesis.

Phytoalexin in disease resistance

Phytoalexins accumulate at infection sites and they inhibit the growth of fungi and bacteria in vitro therefore, it is logical to consider them as possible plant-defence compounds against diseases caused by fungi and bacteria. Phytoalexins are considerably less toxic than chemical fungicides. Phytoalexin fungitoxicity is clearly evidenced by the inhibition of germ-tube elongation, radial mycelial growth and mycelia dry weight increase, as best illustrated by the

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action of resveratrol on B. cinerea, the causal agent for gray mold in grapevine. Phytoalexins may also exert some effects on the cytological, morphological and physiological characteristics of fungal cells. The activity of four phytoalexins from the Solanaceae family (rishitin, phytuberin, anhydro-β-rotunol and solavetivone) on three Phytophthora species resulted in loss of motility of the zoospores, rounding-up of the cells associated with some level of swelling, cytoplasmic granulation and bursting of the cell membrane (Harris and Dannis, 1997).

Table.1 Phytoalexins from different plant families

S. No.	Plant Families	Types of Phytoalexins
1.	Amaryllidaceae	Flavans
2.	Brassicaceae	Indole phytoalexins/camalexin Sulfur-containing phytoalexins/brassinin
3.	Chenopodiaceae	Flavanones/betagarin Isoflavones/betavulgarin
4.	Compositae	Polyacetylenes/safynol
5.	Convolvulaceae	Furanosesquiterpenes/Ipomeamarone
6.	Euphorbiaceae	Diterpenes/casbene
7.	Poaceae	Diterpenoids: Momilactones; Oryzalexins; Zealexins; Phytocassanes; Kauralexins Deoxyanthocyanidins/luteolinidin and apigeninidin Flavanones/sakuranetin Phenylamides
8.	Leguminosae	Isoflavones, Isoflavanones, Isoflavans, Coumestans Pterocarpans/pisatin, phaseollin, glyceollin and maiackiain Furanoacetylenes/wyerone Stilbenes/resveratrol Pterocarpens
9.	Linaceae	Phenylpropanoids/coniferyl alcohol
10.	Malvaceae	Terpenoids naphtaldehydes/gossypol
11.	Moraceae	Furanopterocarpans/moracins A-H
12.	Orchidaceae	Dihydrophenanthrenes/loroglossol
13.	Rutaceae	Methylated phenolic compounds/xanthoxylin
14.	Umbelliferae	Polyacetylenes/falcarinol Phenolics: xanthotoxin 6-methoxymellein
15.	Vitaceae	Stilbenes/resveratrol
16.	Rosaceae	Biphenyls/auarperin Dibenzofurans/cotonefurans
17.	Solanaceae	Phenylpropanoid related compounds Steroid glycoalkaloids Norsequi and sesquiterpenoids Coumarins Polyacetylenic derivatives

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Concerning the accumulation of pisatin in pea and phaseollin in green bean, it was apparent that the phytoalexins accumulated to fungitoxic concentrations not only in inoculum droplets placed on opened pea or bean pods but also in the tissues immediately below the inoculum droplets (Cruickshank and Perrin, 1968). Asymetric growth of the germ tube resulting in the production of "curved-germ tubes" has also been observed in B. cinerea conidia treated with sub-lethal doses of resveratrol. This cytological abnormality suggests that stilbenic compounds may interact with tubulin polymerization, the mode of action of many synthetic fungicides and anticancer agents (woods et al., 1995). Moreover, phytoalexins may affect glucose uptake by fungal cells as reported in the interactions between phaseollin or kievitone and Rhizoctonia solani. Conidia of B. cinerea showed a complete disorganization of mitochondria and disruption of the plasma membrane upon treatment with the stilbene phytoalexins, resveratrol and pterostilbene. Camalexin has recently been involved in the induction of fungal apoptotic programmed cell death in B. cinerea (Shlezinger et al., 2011). The efficaciousness in vivo of some phytoalexins, namely the coumarin phytoalexin, scopoletin on the reduction of green mold symptoms caused by Penicillium digitatum on oranges was shown (Sanzani et al.,, 2014). In the same way, phenolic phytoalexins (resveratrol, scopoletin, scoparone and umbelliferone) were shown to significantly inhibit the growth of Penicillium expansum and patulin accumulation in apples. Beside their antifungal activity, phytoalexins possess some antibacterial activity. Rishitin for instance decreased the viability of cells of Erwinia atroseptica by around 100% at a dose of 360 µg/L (Lyon and Bayliss, 1975). Resveratrol also exerts some activity against numerous bacteria affecting humans: Chlamydia, Helicobacter, Staphylococcus, Enterococcus, Pseudomonas and Neisseria. It is thus clear that phytoalexins exhibit toxicity across much of the biological spectrum, prokaryotic and eukaryotic.

In conclusion, phytoalexins are only one component of the complex mechanisms for disease resistance in plants. Studies on phytoalexins alone have contributed a great deal to plant biochemistry and molecular biology. Phytoalexin production and accumulation occur in healthy plant cells surrounding wounded or infected cells and are stimulated by alarm substances produced and released by the damaged cells and diffusing into the adjacent healthy cells. Phytoalexins are not produced during compatible biotrophic infections. Most phytoalexin elicitors are generally high molecular weight substances that are constituents of the fungal cell wall, such as glucans, chitosan, glycoproteins and polysaccharides. The elicitor molecules are released from the fungal cell wall by host plant enzymes. Most such elicitors are nonspecific, i.e., they are present in both compatible and incompatible races of the pathogen and induce phytoalexin accumulation irrespective of the plant cultivar. A few phytoalexin elicitors however are specific as the accumulation of phytoalexin they cause on certain compatible and incompatible cultivars parallels the phytoalexin accumulation caused by the pathogen races themselves. Some of the better studied phytoalexins include phaseollin in bean; pisatin in pea; glyceollin in soybean; alfalfa and clover; rishitin in potato; gossypol in cotton and capsidiol in pepper.

References

Alghisi P and Favaron F. 1995. Pectin-degrading enzymes and plant parasite interactions. Euro J Plant Pathol. 101: 365-375.

Anderson AJ. 1989. The biology of glycoproteins as elicitors. In: Plant-Microbe Interactions.