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Role of Phytoalexins in Plant Disease Resistance

Shridhar N. Banne and Shruti S. Kadam
Ph. D. Research Scholar, Department of Plant Pathology, Vasantrao Naik Marathwada Krishi Vidyapeeth, Parbhani-431402, (M.S.), India

ARTICLE ID: 50

Abstract

In response to the stress effect, plants protect themselves by carrying certain substances such as phytoalexins. The production of phytoalexins is one of the complex mechanisms by which plants show resistance to disease. Some observations, especially on phytoalexins, have broadened understanding in the fields of plant biochemistry and molecular biology. However, this review describes the interaction of toxins and phytoalexins in the plant-pathogenic cycle, the association of phytoalexins with plant disease resistance, and the role of phytoalexins in plant disease control.

Keywords: Complex mechanism, Phytoalexins, Plants, Plant disease

Introduction

One biochemical response closely related to defence is the accumulation of phytoalexins, characterized as low molecular weight antimicrobial compounds synthesized after infection (Jeandet *et al*., 2013). The possibility that defence mechanisms can be activated after disease confirmed the phytoalexin theory of Muller and Borger (1940), and the study of phytoalexins has been part of the structure of plant resistance ever since. Phytoalexins have received much attention in recent years. In this study, we present the main highlights of this diverse group of molecules, particularly the structures, biosynthesis, elicitors and regulatory mechanisms of these compounds.

Characteristic of Phytoalexins

- They are produced or activated only when the host cells come in contact with the pathogen/parasite.
- The defense reaction happens only in the living cells.
- The inhibitory material may possibly be considered as a product of necrobiosis of the host cells.
- They show non specificity in its toxicity towards fungi.
- The fundamental response that occurs in resistant and susceptible host is similar.

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- Resistant and susceptible host can be differentiated through the speed of formatior of phytoalexins.
- The resistant state is not innate (inherited).
- They are possibly fungistatic and not fungicidal.
- They are not produced in the compatible biotrophic infection; they are produced in incompatible reaction with the resistance in the plants.
- Phytoalexins are produced by the healthy cells adjacent to the damaged or necrotic cells in response to phenolics released by damaged cells.
- Elicitors of phytoalexins are of high molecular weight (i.e. chitosan, glucan, glycoprotein and polysaccharide).
- The phytoalexins produced by different members of the families have the similar structure.
- They are wound antibiotics, low molecular weight, 350 in number 30 in families.
- Example: Isoflavonoids in legumes, terpenoids in Solanaceae.
- Members of Orchifaceae are known for production of phytoalexins.

Chemical Diversity of Phytoalexins

- **Most phytoalexins produced by Leguminosae belong to six classes of isoflavonoids:** isoflavones, isoflavanones, pterocarpans, pterocarpenes, isoflavans and coumestanos.
- **Some pterocarpan phytoalexins are particularly well known:** pisatin, phaseolin, glisolin, medicarpin and maackiain. Pisatin was the first plant alexin isolated and characterized from the garden pea *Pisum sativum* (Cruickshank and Perrin 1960).
- In addition to these compounds, a small number of legumes also produce non-isoflavonoid phytoalexins such as furanoacetylenes and stilbenes.

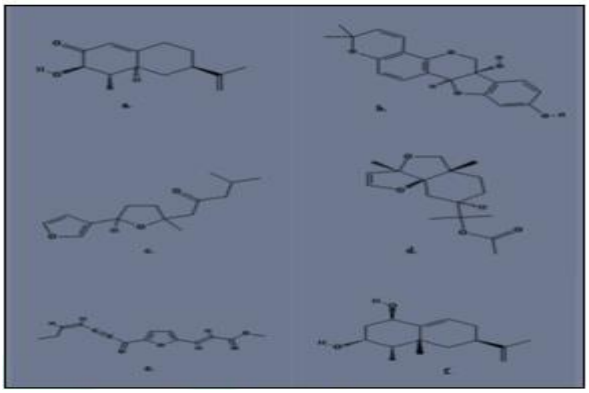

Figure 1. Chemical structures of some phytoalexins: Glutinosone (a), glyceollin (b), Ipomeamarone (c), phytuberin (d), wyerone (e) and capsidol (f)

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- Concentrations of several toxic chemicals have been reported to increase in response to infection, so that phytoalexins are now considered to be small molecule antimicrobial compounds produced in plants as a result of de novo infection or abiotic stress.
- It excludes existing phenols such as chlorogenic acid, caffeic acid and scopoletin.
- Phytoalexins have been demonstrated in several plants belonging to the families Gramineae (oats, rice, sorghum and sugarcane), Solanaceae, Leguminaceae, Chenopodiceae, Convolulaceae, Compositae, Malvaceae and Umbellifera (Table 1).

Table 1. List of Phytoalexins

Sr. No.	Host Plants	Phytoalexin
1.	Arabidopsis	Camalexin
2.	Soybean	Glyceollin
3.	Cotton	Gossypol
4.	Pepper	Capsidiol
5.	Potato	Rishitin
6.	Pea	Pisatin
7.	Bean	Phaseollin
8.	Sweet potato	Ipomeamarone
9.	Red clover	Trifolirihzin
10.	Carrot	Isocoumarin
11.	Alfalfa	Medicarpin
12.	Broad bean	Wyerone acid
13.	Chickpea	Cicerin
14.	Parsnip roots	Xanthotoxin

Phytoalexin in disease resistance

- Phytoalexins accumulate in infection sites and prevent the growth of fungi and bacteria in vitro, so it is logical to consider them as potential plant protection agents against diseases caused by fungi and bacteria.
- Phytoalexins are significantly less toxic than chemical fungicides. Phytoalexin fungitoxicity is clearly demonstrated by inhibition of germ tube elongation, radial mycelial growth and mycelial dry weight growth, best illustrated by the effect of resveratrol on *B. cinerea*, a grape gray mold pathogen.

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- Phytoalexins can also have some effect on the cytological, morphological and physiological characteristics of fungal cells.
- The activity of four phytoalexins from the Solanaceae family (ricitin, phytouberin, anhydro-β-rotunol and solavetivone) in three species of Phytophthora caused loss of zoospore motility, cell rounding and some swelling., cytoplasmic granulation and cell membrane disruption (Harris and Dannis, 1997).

Resistance to Fungi

- Available evidence for the effect of phytoalexins in limiting fungal growth at various stages of colonization includes e.g. Inhibition on plant surfaces: Fungal spores often do not germinate after landing on leaf surfaces (Friend, 2012).
- A striking example of this concern is the behavior of saprophytes in the phyllosphere. Ahuja (2012) described an increased growth of epiphytic fungi that coincides with the onset of senescence.
- The ability to produce phytoalexins decreases during aging (Friend, 2016) and it has been suggested that fungal growth in young leaves may be limited by phytoalexins produced by fungal metabolites spreading from germinating spores (VanWees *et al*., 2003).
- However, the limited evidence available does not support this attractive hypothesis. Thus, Mansfield *et al*. (1982) found that germination of the saprophytes *Aureobasidium pullalans*, *Cladosporium herbarum* and *Epicoccum nigrum* on pea leaves did not induce the formation of the phytoalexin pisatin.
- The apparent lack of effect of phytoalexins on fungal development in the pilosphere can be explained by the fact that the cuticle acts as a barrier that prevents the diffusion of phytoalexin biosynthetic compounds into the basal cells.
- *Botrytis* spp. and *Vicia faba*: Phytoalexins in *Vicia faba* tissues in response to *Botrytis* infection were studied for several years, before attention was paid to the precise timing of phytoalexin accumulation and fungal growth arrest during resistant responses (Mansfield, 1999).

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- The spores of the French bean *Colletotrichum lindemuthinum* germinate within 48 hours of inoculation and produce equal numbers of printers in resistant and susceptible plants (Wheeler, 2012).

Resistance to Bacteria

Studies in the role of phytoalexins in resistance to bacteria have been mainly concerned with the restriction of bacterial multiplication within intercellular spaces.

***Pseudomonads* in French bean and soyabeans.**

- The most detailed studies on the involvement of phytoalexins in bacterial infections concern the resistance of leaves of French bean and certain cultivars to halo blight caused by *Pseudomonas phaseolicola* and the resistance of pods to avirulent isolates of *P. syringae.*
- Propagation of compatible and incompatible races of *P. phaseolicola* in bean cv. Red Mexico and the time of onset of symptoms were confirmed (Schmelz *et al*., 2014).
- The compatible strain is expected to multiply rapidly, causing water-soaked lesions within two to four days of infection; these lesions turn brown and dry up after five days.
- The incompatible strain multiplies more slowly and causes a hypersensitivity reaction, causing the inoculation sites to collapse, forming localized desiccated brown lesions within two days.
- Tissue collapse occurring during a hypersensitivity reaction is closely related to bacterial growth arrest.

Resistance to Viruses

- Antifungal phytoalexins accumulate when viruses cause localized necrotic lesions in legumes and *Nicotiana* spp. but they are not systematically found in infected plants (Jeandet *et al*., 2002).
- Few attempts have been made to determine whether phytoalexins inhibit virus replication and thus limit the size of the lesion.
- Hammerschmidt (1999) found that incubation of Tobacco Necrosis Virus (TNV) in low glyceollin soybean leaf extract did not affect viral infectivity. However, they suggested that the presence of phytoalexin in the tissues immediately surrounding the lesions may indirectly make them unsuitable for further virus propagation. Glyceollin was not

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transferred and did not participate in the systemic protection against TNV provided by previous infection of soybeans with the virus.

Conclusion

Since phytoalexins accumulate in both susceptible and resistant plants, the real question is whether they are contributors to defense or simply the end result of pathogen- or stress-induced metabolism. phytoalexins are only one part of the complex mechanisms of plant disease resistance. The study of phytoalexins alone has greatly influenced plant biochemistry and molecular biology. Phytoalexin production and accumulation occurs in healthy plant cells surrounding injured or infected cells and is stimulated by perturbing agents produced and released from injured cells and spread into adjacent healthy cells. Phytoalexins are not produced during compatible biotrophic infections. Most phytoalexin inducers are usually high molecular weight substances that are components of the fungal cell wall, such as glucans, chitosan, glycoproteins and polysaccharides.

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