Defense Mechanism in Plants

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P.N. Sharma

Department of Plant Pathology,
CSK HPKV, Palampur (H.P.)
Defense Mechanism in Plants

- constitutive or Induced
- Structural (Morphological) defense
- Biochemical defense

*Both the defenses are affected by:*

  - Age of the plant
  - Type of organ infected
  - Nutritional status of the host
  - Environmental conditions
Fungal pathogen:

Preinfection:
- Germination
- Germ tube search
- Appressorium formation
- Penetration peg

Postinfection:
- Haustorium formation (biotroph)
- Toxin formation (necrotroph)
- Detoxification of phytoalexins
- Reproduction

Host:

General induced defenses:
- Papillae formation

Cork & lignin layers

Specific recognition:
- Hypersensitivity
- Phytoalexins

Systemic acquired resistance

CONSTITUTIVE DEFENSES

structural general induced defenses

chemical specific recognition
Defense or Resistance

• Always controlled by genes
100,000 Fungi
500 Bacteria
1,000 Viruses

2,500 PHP

500 Nematodes

Non-host resistance. Plant healthy.

Quantitative (polygenic) resistance. Some infections and symptoms possible. Plants generally survive and produce.

Monogenic (R gene) resistance. Plants either are resistant and remain healthy or are susceptible and become severely diseased.

Healthy

Severely infected
Non-host Resistance

- When a plant resist the attack of pathogenic organism which is otherwise not the host of that pathogen is termed as non-host resistance
  - e.g. potato late blight pathogen do not infect apple or wheat plant and similarly apple pathogen like *V. inaequalis* do not attack potato

- Similarly, the fungus that causes powdery mildew on wheat (*Blumeria (Erysiphe) graminis f. sp. tritici*) does not infect barley and vice versa, the fungus that causes powdery mildew on barley (*B. graminis f. sp. hordei*) does not infect wheat, and so on.
Host defenses

**Constitutive**
- Structural:
  - wax
  - cuticle
  - thickness
- Biochemical:
  - pre-formed inhibitors
  - phenolics
- Hairs
- Shape and activity of stomata

**Induced**
- Structural:
  - cork layer
  - abscission layer
- Biochemical:
  - hypersensitivity
  - phytoalexins
  - gums
  - papillae
  - system acquired resistance (SAR)
  - tyloses
  - anti-microbial enzymes
Plant Defense

- **Constitutive**
  - Physical barriers (Structural)
  - Phytoanticipins (Biochemical)

- **Inducible**
  - Structural
    - Corky, absiccion layers, tylose etc.
  - Biochemical
  - Phytoalexins
  - Hypersensitive Response
  - Systemic Acquired Response (SAR)
Structural defense

Pre existing/ pre-infectional structural barriers

- **Surface barrier**
  - Presence of wax layers
  - Hairs
  - Lignin
  - Thickness of cuticle e.g. in resistant flax *M. lini*, *Barberry- P. graminis tritici*

- The thickness and toughness of the outer wall of epidermal cells are apparently important factors in the resistance of some plants to certain pathogens.
  - **Size of stomata** (Resistant citrus vars. have small stomata- *X. campestris*),
  - **location and shape of stomata**, opening & closing (resistant wheat vars. – Rusts).
Post infectious defense structures

- **Histological defense**
  - Tylose formation e.g. wilts
  - Corky layers e.g. in potato- *R. solani*
    - Cellular defense – altered walls of cells (changes in cell wall-thickening, deposition of callose papillae)
    - Cytoplasmic defense
    - Hypersensitive response
Hypersensitive response

- Result in sudden death of the host cells in the vicinity of the pathogen
  - Highest degree of resistance
- Both structural & biochemical in nature
- Common in obligate pathogens like funji, viruses and nematodes, also found in other fungal & bacterial infection
- Due to HR
  - The necrotic tissues isolate the obligate pathogen from living cells.
  - Devoid the pathogen of nutrition, thus starved and die.
Induced structural defenses

Abscission layer
*X. pruni*- shot hole

Cork layer
*R. solani*- canker in potato

The formation of cork or abscission layers can limit the size of lesions, and consequently the extent of damage that can be caused by a single infection.

Provide protection by
- inhibiting the further spread of pathogen
- Block the spread of toxic substances of the pathogen,
- stop the flow of nutrients to infection point
Induced structural defenses

**Tyloses**

are overgrowths of living cells that protrude via pits into xylem vessels blocking the vascular system.

If they form abundantly and quickly, they can stop the spread of vascular wilt pathogens.

formation is triggered by a ‘stress condition’.
Biochemical defense
Pre-existing biochemical defense

– Inhibitors released by the plants in its environment

• Fungitoxic exudates – of tomato and sugarbeet inhibit germination of *Botrytis* and *Cercospora*;

• oil capric acid on elm seed toxic to *Ceratocystis ulmi* spores

• Phenolics: red scale onion against smudge- *Colletotrichum circinans*
Inhibitors present in plant cells

- Stored in vacuoles, lysogenous glands, heart wood periderm of plants
  - Phenolics – onion (catechol & portocatechoic acid)
  - Saponins (tomatin in tomato, avenacin in oats (most of these compounds inhibits hydrolytic enzymes produced by pathogen e.g. pectolytic enzymes etc.)
  - Some plants also contains hydrolytic enzymes e.g. glucanases, chitinases etc.
Constitutive biochemical defense

In contrast to red and yellow onions, white onions do not contain significant quantities of certain phenolic chemicals (one is catechol). If present, these phenolics confer resistance to onion smudge disease.
Pre-existing biochemical defense

- **Defense through lack of essential factors**
  - Lack of recognitions
  - Lack of host receptor and sensitive sites for toxins in the host.
  - Lack of essential nutrients for the pathogen e.g. high sugar pathogen like *H. oryzae*; *E. carotovora* var. *atroseptica*
Post infectional biochemical defense

- **HR- hypersensitive response**
- Biochemical inhibitors produced in response to injury by pathogen
  - Phenolics (Chlorogenic acid, caffeic acid)
  - Oxidation products of plants (floretin, hydroquinonesm hydroxytyromine)
  - Phytoalexins
Induced biochemical defenses

**hypersensitive response**

The **hypersensitive response (HR)** is a localized death of host cells at the site of infection. It is the result of a specific recognition of a pathogen attack by the host.

The HR is considered to be a type of programmed cell death.

The HR act by:
- Isolation of the pathogen
- Stop flow of nutrients—thus starvation
• The hypersensitive response is the culmination of the plant defense responses initiated by:
  – the recognition by the plant of specific pathogen-produced signal molecules, known as elicitors. Recognition of the elicitors by the host plant activates a cascade of biochemical reactions in the attacked and surrounding plant cells and
  – leads to new or altered cell functions and to new or greatly activated defense-related compounds

• The most common new cell functions and compounds include:
  – a rapid burst of reactive oxygen species, leading to a dramatic increase of oxidative reactions;
  – increased ion movement, especially of K+ and H+ through the cell membrane;
  – disruption of membranes and loss of cellular compartmentalization

• production of antimicrobial substances such as phenolics (phytoalexins); and formation of antimicrobial so-called pathogenesis-related proteins such as
Defense through increased level of phenolics

- Plants contain aromatic acids that act as precursors of phenolic compounds
  - Simplest compound is phenol: having a single OH group on benzene ring
  - When benzene ring has more than one OH group – polyphenols (lignins, phenyl glucoside, flavonoids, anthocyanin etc.)
  - Quinone: displacement of H
In plants, phenolic synthesis takes place through shikimic acid pathway and acetic acid pathway-PPP (PPP - this operates in diseased plants),

Shikimic acid pathway is more important.

These products formed in Glycolysis and pentose phosphate pathway (PPP) enzymes participating in these pathways- phenol oxidising enzymes: phenolases, phenol oxidases (PO- laccase & tyrosinase). PPO,

Precursors of phenolic compounds e.g. caffeic acid, cinnamic acid, chlorogenic acid
Phenolic compounds produced by plants

- Caffeic acid: sweet potato
- Chlorogenic acid: sweet potato, potato, carrot
- Phloretin: Apple
- Hydroqunonones: sweet potato
Phytoalexins

• Concept given by Borger & Muller in Potato- lateblight interactions
• Defined as antibiotics produced in plant-pathogen interaction or as a response to injury or physiological stimuli (Kuc, 1972)
• Paxton (1981) defined phytoalexins as low mol. Wt. antimicrobial compounds which are synthesized by and accumulates in plant cells after microbial infection.
• Involves the role of elicitors in their production
• Not produced during biotrophic infection
• Many phytoalexins have been isolated from plants (>20) families.
  – E.g. leguminosae, solanaceae, malvaceae, graminae, compositae, umbelliferae, chenopodiaceae.
Characteristics of phytoalexins

- Should be fungistatic & bacteristatic and active at very low conc.
- **Produced by the host in response to infection or metabolic byproducts of micro-organisms and stimuli.**
- Absent in healthy cells or present in very minute quantity.
- **Usually remain close to the site of their production**
- Produced in quantities proportionate to the size of inoculum
- **Produced in large quantity in response to weak pathogen or non pathogen than virulent one.**
- Produced relatively quickly in cells after infection
- Host specific rather than pathogen specific.
Best studied phytoalexins are:

- **Legumes**
  - Phaseollin; Common beans
  - Vestitol: Red clover
  - Phaseollidin: Common bean
  - Medicarpin; alfalfa, lucern, beans, chickpea
  - Pisatin: Pea

- **Solanaceae**
  - Rishitin: potato, tomato
  - Capsidol: chilli/ pepper
  - Phytubrin: potato

- **Graminae**
  - Avenalumin-I,II, III : Oats

- **6-mehtoxymellin: carrot**

- **Orchidaceae**
  - Orchinol: Orchid
Phytoalexin molecules

Several hundred have been isolated and characterized from at least 15 plant families.
Fungitoxic phenolics released from non-toxic phenolic compounds

• Some plants have non-toxic glycosidic compounds consisting of sugars attached to phenols.
• Some enzyme like glycosidase act on them an release the phenols that affect the pathogen
Role of phenol oxidizing enzymes

- Enzymes like polyphenol oxidases oxidise phenols to quinones.
- Peroxidases increase the polymerization of the phenols into lignins, the complex phenols.
Role of induced synthesis of enzymes

- PAL (phenyl ammonia lyase)
  - A key enzyme for the synthesis of phenols, phytoalexins and other defense related chemicals

**Defense through inactivation of pathogen enzymes**

**Defense through detoxification of pathogen toxin**
Example of attack and counterattack

Phytoalexins are synthesized in response to pathogen attack.

Some pathogens counterattack producing enzymes that degrade the phytoalexin.
Induced biochemical defenses

- **Systemic Acquired Resistance** is the activation of defenses in distal, non-infected parts of the plant.

The plant becomes more resistant to a secondary, challenge inoculation there.

After a primary inoculation here.
Systemic Acquired Resistance

• SAR confers broad-based resistance to different pathogens. For example, primary inoculation with a fungal leaf spot pathogen reduces susceptibility of the host plant to other fungi as well as to bacterial and viral pathogens.

• Salicylic acid (chemical related to aspirin) is part of signaling pathway involved in transmission of the defense response throughout the plant to produce SAR. This has lead to the development of synthetic chemicals that mimic the role of salicylic acid.
Induction of Systemic Acquired Resistance

1- Production of H$_2$O$_2$ (plus antioxidants)
   Hydrogen peroxide has been associated with secondary induction of SAR and direct toxic activity to invading pathogens.

2- Thickening of plant cell wall
   Production of phenolics (lignin, tannic acid) that strengthen walls and inhibit pathogen enzymes.

3- Accumulation of pathogenesis related proteins
   “PR-proteins” chitinases, β-1,3 Glucanases
   These enzymes accumulate in vacuole of plant cell. Upon attack, they directly degrade fungal cell walls. Indirectly, their action results in the release of fungal wall components that elicit additional defense reactions.
The signal compounds responsible for induction of PR proteins include:

- salicylic acid,
- ethylene,
- xylanase,
- the polypeptide systemin,
- jasmonic acid and
- others such as poly acrylic acid, 2-chloroethylphosphonic acid, acetyl- salicylic acid, benzoic acid, indole-3-acetic acid, 2, 4-dichlorophenoxyacetic acid and b-benzyl aminopurine (Bozarth and Ford, 1988).
Plants can have more than one type of acquired resistance.

Strains of bacteria living on roots or synthetic chemicals can induce resistance in plants.

- Induced Resistance
- Systemic Acquired Resistance (SAR)
- Induced Systemic Resistance (ISR)
- Pathogens or biotic elicitor
- Salicylate
- Jasmone & Ethylene
Pathogenesis-Related Proteins (PR-proteins)

- Pathogenesis related proteins, called PR-proteins: A group of plant coded proteins
- Are structurally diverse group toxic to invading pathogens.
- Produced under stress
- Pathogenesis related proteins of Antomiw et al. (1980) are also known as “B-proteins” by Gianinazzi and Ahl (1983).
• They are widely distributed in plants in trace amounts but are produced in high concentration following pathogen attack or stress.

• The PR proteins exist in plant cells intracellularly (acidic in Apoplast & basic form in vacuoles) and also in the intercellular spaces.

• Varying types of PR-proteins have been isolated from several crop plants.
• The several groups of PR-proteins have been classified according to their function, serological relationship, amino acid sequence, molecular weight and certain other properties.

• The PR proteins are either extremely acidic or extremely basic and therefore are highly soluble and reactive.
• PR-proteins are low molecular weight (approximately 14000 - 30000) compounds, extractable at low pH (2-3),

• rich in aromatic amino acids and resistant to trypsin and chemo trypsin action.
• The better known PR proteins are:

- PR 1 proteins, B-1, 3-glucanases, chitinases, lysozymes,
- PR 4 proteins, thaumatinelike proteins, osmotinlike proteins, cysteine-rich proteins, glycine-rich proteins, proteinase inhibitors, proteinases, chitosanases and peroxidases. There are often numerous isoforms of each PR-protein in various host plants.
Although healthy plants may contain trace amounts of several PR proteins, attack by pathogens, treatment with elicitors, wounding, or stress induces transcription of a battery of genes that code for PR-proteins.

This occurs as a part of a massive switch in the overall pattern of gene expression during which normal protein production nearly ceases.
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Significance of PR-proteins

• The significance of PR-proteins lies in the fact that they show strong antifungal and other antimicrobial activity.

• Some of them inhibit spore release and germination, whereas others are associated with strengthening of the host cell wall and its outgrowths and papillae.
Some of the PR-proteins, for example, B-1, 3-glucanase and chitinase, diffuse towards and affect (break down) the chitin-supported structure of the cell walls of several plant pathogenic fungi, whereas lysozymes degrade the glucosamine and muramic acid components of bacterial cell walls.
• Plants genetically engineered to express chitinase genes show good resistance against the soil-borne pathogen *Rhizoctonia solani*.

• Signal molecules that induce PR-proteins synthesis seem to be transported systemically to other parts of the plant and to reduce disease initiation and intensity in those parts for several days or even weeks.
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