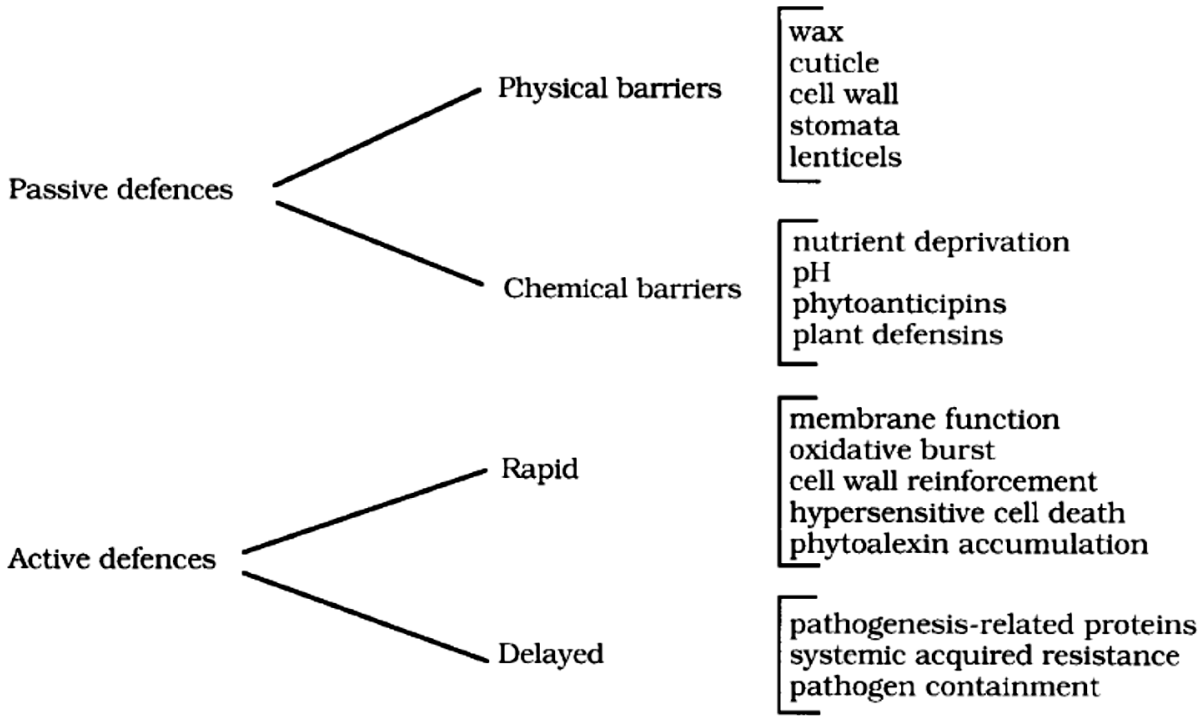
Defence Mechanism in Plants

Plant pathogens fall into two broad categories: necrotrophs (those that kill plant cells before parasitising them), and biotrophs (those that obtain nutrients from living cells). Failure of pathogens to invade suitable host cells (dead in the case of necrotrophs; alive for biotrophs) will prevent them from infecting the host and the plant will be resistant. Additionally, the establishment of a parasitic relationship is dependent on the responses of the plant under attack.



Some defence mechanisms in plants

Protection from a pathogen's initial invasion is achieved via passive defences, such as **physical** and/or **chemical** barriers.

Physical barriers largely involve properties of the plant surface, that is, the cuticle, stomata and cell walls. Pathogens produce a range of cutin-degrading enzymes, which are often crucial to the successful penetration of the plant tissue. The thickness of the cuticle, the presence of secondary cell wall, and the size of stomatal pores can all affect the success with which a pathogen invades a host. Some plants invest in very thick walls and/or cuticles, and bark (where present) can also provide a physical impediment to infection. The vertical orientation of leaves can also add to plant resistance, by preventing the formation of moisture films of the leaf surfaces, inhibiting infection by pathogens reliant on water for motility.

Chemical barriers include compounds, such as "phytoanticipins" (Any pre-existing phytoalexin that interacts with a plant pathogen), that have antimicrobial activity and compounds that affect the vectors of plant viruses. Phenols and quinones are two classes of antimicrobial compounds produced by some plants Inhibiting compounds may be excreted into the external environment, accumulate in dead cells or be sequestered into vacuoles in an inactive form. The young fruit of numerous plants (e.g. mangoes, avocado) contain antifungal or antimicrobial compounds that are gradually metabolised during fruit ripening, making unripe fruit less susceptible to disease than ripe fruit. Lactones, cyanogenic glucosides, saponins, terpenoids, stilbenes and tannins are also plant-produced compounds associated with pathogen resistance.

Rapid Active Defenses

Almost every host-parasite interaction is unique in the details of the activation, localization, timing and magnitude of the defence responses.

At the membrane

The host membrane appears to be involved in the earliest stages of pathogen recognition and signal transduction. A change in membrane permeability after exposure to a pathogen causes fluxes in ions, such as K^+, H^+ and Ca^{2+} and results in changes to gene activation and the triggering of the defence responses. Also at the membrane, the 'oxidative burst', which involves the generation of reactive oxygen species, such as hydrogen peroxide, triggers signals that affect gene expression, cross-linking in the host cell wall and initiation of later defence responses. The reactive oxygen species at the site of infection are also produced in quantities that capable of killing micro-organisms directly.

At the cell wall

Preparations for the reinforcement of the cell wall, which can improve host resistance, begin very quickly after a pathogen attempts to penetrate a host cell. This is characterized by an intensification of cytoplasmic streaming and the accumulation of host cytoplasm around the site of attempted penetration. The cytoplasmic aggregates are thought to contain cellular apparatus for the synthesis of cell wall fortifications. If the host cell can repair and reinforce its cell walls quickly enough, it might reduce the penetration efficiency of the pathogen. Several types of reinforcement are produced by host cells. A papilla (A small rounded protuberance on a part or organ of the body) is a deposit of callose (**Callose** is a plant polysaccharide), silicon, lignin and proteins between the cell wall and cell membrane, directly below the point of attempted penetration, while lignitubers are lignified callose reinforcements that ensheath invading hyphal tips.

The hypersensitive response

Hypersensitive cell death is another widespread mechanism used by hosts to prevent the spread of a pathogen. Infected cells and those surrounding them "suicide", preventing further spread, and in some cases, killing the pathogen. It is often associated with the initiation of other responses, such as lignification and the synthesis of anti-microbial compounds. The success of hypersensitive cell death as a resistance mechanism depends on the nutritional requirements of the specific pathogen and the timing, magnitude and location of the host response.

Phytoalexin

Phytoalexins are low molecular weight antibiotics produced by many (but not all) plants in response to infection. There are many biotic elicitors of phytoalexin production, such as cell wall components, as well as abiotic elicitors, such as heavy metals and ultraviolet light. Phytoalexins inhibit the growth of bacteria and fungi *in vivo* and *in vitro*, and production of these antibiotics during an infection can induce resistance to subsequent infections by that pathogen.

Characteristics of Phytoalexin

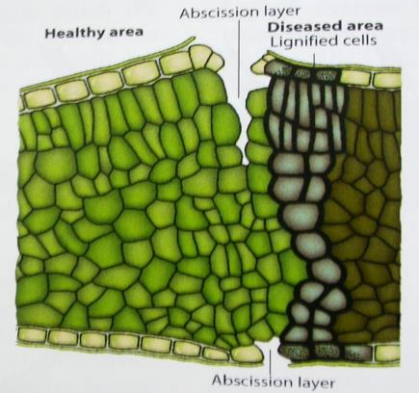
- Phytoalexins should be fungistatic and bacteristatic and active at very low conc.
- Produced by the host in response to infection or metabolic by products of micro-organisms and stimuli.
- Absent in healthy cells or present in very minute quantity.
- Usually remain close to the site of their production
- 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.

Delayed active defenses

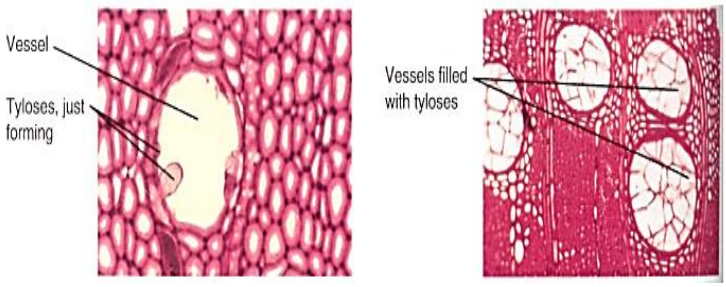
Delayed active defences include containment of the pathogen, wound repair, expression of pathogenesis-related proteins and the acquisition of systemic resistance. These mechanisms restrict the spread of the pathogen after infection is established and contain the damage to host tissues.

Physical responses:

Abscission Layer: The ability to repair wounds can help protect the plant from further infection by other, opportunistic pathogens. A secondary meristem in fleshy tissues, fruits, roots and bark, the cork cambium, can produce cork cells, which have thick, suberised walls. These cells can create a barrier to further colonization by the pathogen and, in some cases, develop an abscission layer around the site of infection, causing the infected tissue separate from the healthy tissue. Wounded tree trunks often secrete protective gums that seal the wound from further infection.



Tylose: A balloon-like extension of a parenchyma cell that protrudes into the lumen of a neighbouring xylem vessel or tracheid through a pit in the cell wall. Tyloses form most commonly in older woody tissue, possibly in response to injury; they may eventually block the vessels and thus help to prevent the spread of fungi and other pathogens within the plant. Tyloses may become filled with tannins, gums, pigments, etc., giving heartwood its dark colour, and their walls can remain thin or become lignified.



Pathogenesis-related proteins

Pathogenesis Related proteins (PR- Proteins): A group of plant coded proteins are produced under stress conditions which structurally diverse group of proteins that toxic to invading pathogens.

- They are widely distributed in plant in trace amounts but are produced in high concentration following pathogens attack or stress.
- Pr proteins exist in plant cells intracellularly (acidic in Apoplast and basic form in vacuoles) and also in the intercellular spaces.
- The Pr proteins either extremely acidic or extremely basic and therefore are high soluble and reactive.
- It's rich in aromatic amino acids and resistant to trypsin and chymotrypsin action.
- Pr proteins show very strong antifungal and other antimicrobial activity. Some of them inhibit spore release and germination also.