Phenomenon of infection – pre-penetration, penetration and post penetration

The "infection process" can be divided into three phases: **pre-entry**, **entry** and **colonisation**. It encompasses the germination or multiplication of an infective propagule in or on a potential host through to the establishment of a parasitic relationship between the pathogen and the host. The process of infection is influenced by properties of the pathogen, the host and the external environment. If any of the stages of the infection process is inhibited by any of these factors, the pathogen will not cause disease in the host.

While some parasites colonise the outside of the plant (ectoparasites), pathogens may also enter the host plant by **penetration**, through a natural opening (like a stomatal pore) or via a wound. The symptoms of the diseases produced by these pathogens result from the disruption of respiration, photosynthesis, translocation of nutrients, transpiration, and other aspects of growth and development.

Pre Entry

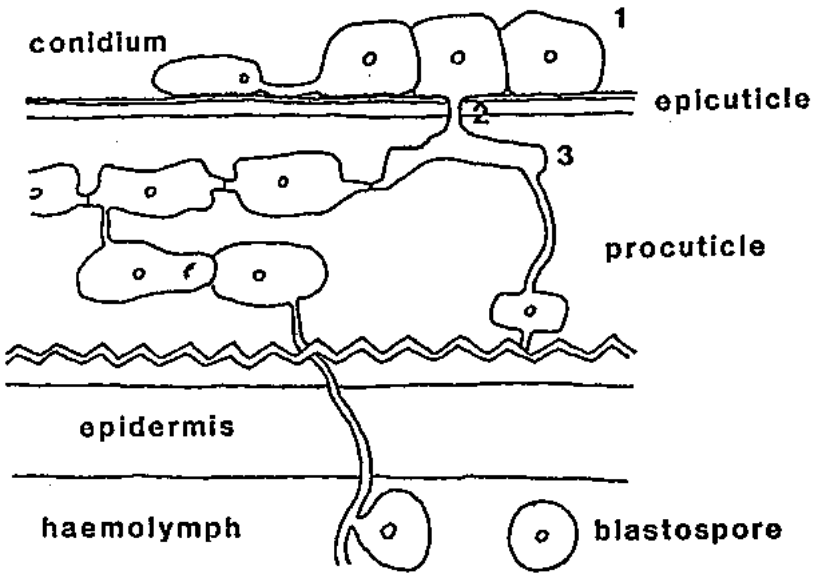
Before a pathogen can penetrate a host tissue, a spore must germinate and grow on the surface of the plant. In the case of motile pathogens, they must find the host and negotiate its surface before entering the host. Some pathogens develop specialised penetration structures, such as appressoria, while others utilise pre-existing openings in the plant's surface, such as wounds or stomatal pores. Plant viruses are often transported and introduced into the plant via vectors such as fungi or insects.

The initial contact between infective propagules of a parasite and a potential host plant is called **inoculation**. Pathogens use a variety of stimuli to identify a suitable entry point. Several fungi use topographical cues on the plant surface to guide them towards a likely stomatal site. Once the hypha reaches a stoma, volatile compounds escaping from the pore appear to provide a signal for the formation of a specialised penetration structure, the **appressorium**. Sugars, amino acids and minerals secreted by plants at the leaf surface can non-specifically trigger spore germination or provide nutrition for the pathogen. Some pathogenic spores will not germinate in the absence of these substances.

Pathogen development is influenced by temperature, moisture, light, aeration, nutrient availability and pH. The conditions necessary for survival and successful infection differ between pathogens.

Entry

Pathogens exploit every possible pathway to enter their host, although individual species of pathogen tend to have a preferred method. Fungal pathogens often use **direct penetration** of the plant surface to enter the host. This requires adhesion to the plant surface, followed by the application of *pressure* and then *enzymatic degradation* of the cuticle and cell wall, in order to overcome the physical barriers presented by the plant's surface. During the degradation of the cuticle and wall, a succession of genes are switched on and off in the pathogen, so that cutinase, followed by cellulase, then pectinase and protease are produced, attacking the cuticle, cell wall, and middle lamella in the order that they are encountered. The pressure needed for the hypha to penetrate the cell wall is achieved by first firmly attaching the appressorium to the plant surface with a proteinaceous glue. The cell wall of the apressorium then becomes impregnated with melanin, making it watertight, and capable of containing the high turgor pressure that builds up within the appressorium. The point of the appresorium that is in contact with the cuticle is called the penetration pore, and the wall is thinnest at this point. The increasing turgor pressure causes the pore to herniate, forming a **penetration peg**, which applies huge pressure to the host cuticle and cell wall.



Penetration of host cuticle by a Deuteromycetes entomopathogens
1 = appressorial complex, 2 = penetration peg, 3 = penetration plate

The alternative pathway for pathogen entry is via a **pre-existing opening** in the plant surface. This can be a natural opening or a wound. Pathogenic bacteria and nematodes often enter through stomatal pores when there is a film of moisture on the leaf surface. Fungi can also penetrate open stomata without the formation of any specialised structures. Some fungi form a swollen appressorium over the stomatal aperture and a fine penetration hypha enters the airspace inside the leaf, where it forms a sub-stomatal vesicle, from which infection hyphae emerge and form **haustoria** in surrounding cells. Also vulnerable to pathogen invasion are **hydathodes**, pores at the leaf margin that are continuous with the xylem. Under particularly humid conditions, droplets of xylem fluid (guttation droplets) can emerge at the surface of the leaf where they can be exposed to pathogenic bacteria, which then enter the plant when the droplet retreats back into the hydathode as the humidity decreases. Lenticels are raised pores that allow gas exchange across the bark of woody plants. They exclude most pathogens, but some are able to enter the plant via this route. Some specialised pathogens can also use more unusual openings, such as nectaries, styles and **ectodesmata**. Entry through a wound does not require the formation of specialised structures, and many of the pathogens that utilise wounds to enter the plant are unable to penetrate the plant surface otherwise. Most plant viruses entrer through wounds, such as those made by their insect vectors.

Colonisation

A successful infection requires the establishment of a parasitic relationship between the pathogen and the host, once the host has gained entry to the plant. There are two broad categories of pathogens are **biotrophs** (those that establish an infection in living tissue) and **necrotrophs** (those that kill cells before colonising them, by secreting toxins that diffuse ahead of the advancing pathogen). These two kinds of pathogens are also sometimes known as 'sneaks' and 'thugs', because of the tactics they use to acquire nutrients from their hosts. The toxins produced by necrotrophs can be specific to the host or non-specific. Non-specific toxins are involved in a broad range of plant-fungus or plant-bacterial interactions, and will therefore not usually determine the host range of the pathogen producing them. Necrotrophs often enter the plant through wounds and cause immediate and severe symptoms. An intermediate category of parasite is the **hemibiotrophs**, which start off as biotrophs and eventually become necrotrophic, employing tactics from both classes of pathogen.

Pathogens that colonise the surface of plants, extracting nutrients through haustoria in epidermal or mesophyll cells are termed **ectoparasites**. The haustoria are the only structures that penetrate the host cells. Some parasites colonise the area between the cuticle and the outer wall of the epidermal cells, penetrating host epidermal and mesophyll cells with haustoria. These are called **sub-cuticular infections**. Pathogens can also form colonies deeper in the plant tissues. These are **mesophyll and parenchyma infections,** and can be necrotrophic, hemibiotrophicor biotrophic relationships. Necrotrophs do not produce specialised penetration structures. Instead, they kill host cells by secreting toxins, then degrade the cell wall and middle lamella, allowing their hyphae to penetrate the plant cell walls and the cells themselves. In hemibiotrophic infections, intercellular hyphae can form haustoria in living mesophyll cells, but as the lesion expands under favourable conditions, those heavily parasitised cells at the inner, older part of the colony collapse and die. A similar sequence of events can take place in plants infected by burrowing nematodes. Viruses, mildews and rusts develop specialised biotrophic relationships with their hosts. Intercellular hyphae of downy mildew colonise host mesophyll cells and form haustoria. The mildew sporulates and the infected cells eventually die, although necrosis is delayed and contained, compared to that caused by necrotrophic pathogens. Rust fungi can also delay senescence in infected cells while they sporulate. **Vascular infections** usually cause wilting and discoloration as a result of the physical blockage of infected xylem vessels. True vascular wilt pathogens colonise the vascular tissue exclusively, although other pathogens can cause the same symptoms if they infect the vascular system as well as other tissues. There are a few pathogens that manage to achieve **systemic infection** of their host. For example, many viruses can spread to most parts of the plant, although not necessarily all tissues. Some downy mildews can also systemically infect their host by invading the vascular tissue and growing throughout the host, causing deformation, rather than necrosis. Finally, there are some pathogens that complete their entire life cycle within the cells of their host, and may spread from cell to cell during cytokinesis. These are **endobiotic infections**.

Disease Physiology

While necrotrophs have little effect on plant physiology, since they kill host cells before colonising them, biotrophic pathogens become incorporated into and subtly modify various aspects of host physiology, such as respiration, photosynthesis, translocation, transpiration and growth and development. The **respiration** rate of plants invariably increases following infection

by fungi, bacteria or viruses. The higher rate of glucose catabolism causes a measurable increase in the temperature of infected leaves. An early step in the plant's response to infection is an oxidative burst, which is manifested as a rapid increase in oxygen consumption, and the release of reactive oxygen species, such as hydrogen peroxide (H_2O_2) and the superoxide anion (O_2^-). The oxidative burst is involved in a range of disease resistance and wound repair mechanisms.

LINK to Rapid Active Defense

In resistant plants, the increase in respiration and glucose catabolism is used to produce defence-related metabolites via the pentose phosphate pathway. In susceptible plants, the extra energy produced is used by the growing pathogen.

Pathogens also affect **photosynthesis**, both directly and indirectly. Pathogens that cause defoliation rob the plant of photosynthetic tissue, while necrotrophs decrease the photosynthetic rate by damaging chloroplasts and killing cells. Biotrophs affect photosynthesis in varying degrees, depending on the severity of the infection. A biotrophic infection site becomes a strong metabolic sink, changing the pattern of nutrient **translocation** within the plant, and causing net influx of nutrients into infected leaves to satisfy the demands of the pathogen. The depletion, diversion and retention of photosynthetic products by the pathogen stunts plant growth, and further reduced the plant's photosynthetic efficiency. In addition, pathogens affect water relations in the plants they infect. Biotrophs have little effect on **transpiration** rate until sporulation ruptures the cuticle, at which point the plant wilts rapidly. Pathogens that infect the roots directly affect the plant's ability to absorb water by killing the root system, thus producing secondary symptoms such as wilting and defoliation. Pathogens of the vascular system similarly affect water movement by blocking xylem vessels. **Growth and development** in general are affected by pathogen infection, as a result of the changes in source-sink patterns in the plant. Many pathogens disturb the hormone balance in plants by either releasing plant hormones themselves, or by triggering an increase or a decrease in synthesis or degradation of hormones in the plant. This can cause a variety of symptoms, such as the formation of adventitious roots, gall development, and epinasty (the down-turning of petioles).