RAMA UNIVERSITY

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FACULTY OF AGRICULTURAL SCIENCES & ALLIED INDUSTRIES

LECTURE 5

SURVIVAL AND DISPERSAL OF IMPORTANT PLANT PATHOGENS

Survival of Pathogen

The survival of a pathogen between cropping seasons and its effective dispersal to healthy plants are crucial aspects of the plant disease cycle. If either of these is prevented, the disease will not occur. Most pathogens possess mechanisms to survive intercrop periods or periods of unfavourable environmental conditions. The spread of inoculum can be airborne, soil borne, water borne, seed or clone borne, or vector borne. Airborne inoculum can travel for great distances, even across oceans, while soil-borne inoculum is rarely spread any great distance. Many pathogens are dispersed by more than one mechanism. These fungi are able to form resting structures that enable them to survive long periods without a suitable host or when environmental conditions are unfavourable.

Mycelium

It is believed that in perennial hosts such as horseradish, the mycelium is capable of overwintering in the infected crowns and lateral roots. Remaining dormant during the winter, the mycelium resumes its activity and grows into new shoots that the host produces in spring.

Sporangia

Sporangia of *Albugo bliti* attached to host tissues remain viable for 15 days at 3–10 °C, whereas detached sporangia remain viable only for 24 h within this temperature range. At 30 °C, viability is lost after 4 h when attached and within 2 h when detached from host tissues. Sporangia of *Albugo candida* can survive for 4.5 days at 15 °C on detached infected *Brassica juncea* leaves, but lose their viability within 18 h if separated and incubated without host tissues. However, sporangia can be stored for 105 days at −40°C as a dry powdered mass (Table 4.1 and 4.2).

Zoosporangia of *A. candida* (Indian isolate) on *B. juncea* infected leaves can survive up to 4 days at 20 °C and for 2 days at 25 °C while Canadian race 2 up to 3 days at 20°C and 8 h at 25°C.

Oospores

Oospores are formed in the hypertrophied tissues (leaves, stems, inflorescences, pods, roots) of infected host plants. Overwintered oospores in infected plant debris in soil function as the source of primary inoculum of the pathogen. Oospores have also been observed in naturally infected senesced leaves of *B. juncea* and *Brassica rapa* var. *Toria*. 8.75×10^5 oospores in 1 g of hypertrophied cup-shaped leaves and 21.85×105 oospores in 1 g of hypertrophied staghead portions have been reported. The high percentage of oospore production has been recorded in detached cotyledon culture (>73%) and naturally senescing cotyledons (>92%). Uunder delayed sowing conditions, higher disease severity on leaves (32%), and incidence (43%) of stagheads can be attributed to higher germination of overwintered oospores in infected plant debris in soil.

Viability of *A. candida* under different storage conditions

Storage conditions	Survival period (days)
Dry sporangial mass in lab conditions[a]	0.75
Dry sporangial mass at 10 °C	0.92
Sporangia on detached leaf in lab conditions	4.50
Dry sporangial mass at 0 °C	21.00
Dry sporangial mass at −20 °C	85.00
Dry sporangial mass at −40 °C (defreeze dry)	105.00

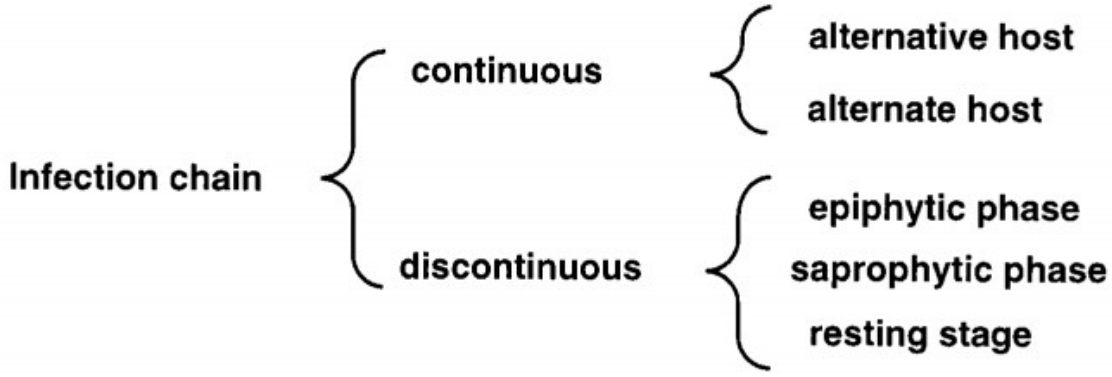
[a] Mean laboratory temperature was 15 °C

Sporangial thermal death point of *A. candida*

Temperature (°C)	Sporangial germination	
	When attached with leaf	When detached from leaf
20	++	++
22	++	++
24	++	++
26	++	++
28	++	+
30	+	+
32	+	–
34	–	–

++ more than 50% sporangia germinated, + less than 50% sporangia germinated, – no germination

Most plant pathogens can survive only by repeatedly reinfecting host plants. This process of repeated infection is known as an infection chain. If the infection chain is broken the pathogen dies out. The Swiss plant pathologist Ernst Gaumann (1950) in his book Principles of Plant Infection divided infection chains into two types depending on how the inoculum of the pathogen survives between leaving the infected host (the distributor host) and arriving on the host to be infected (the receiver host). In continuous infection chains the pathogen survives by continually infecting susceptible plants in an uninterrupted fashion. Discontinuous infection chains occur when the disease cycle is interrupted by an epiphytic phase, a saprophytic phase or a resting stage.



Five types of infection chains available to plant pathogens

Continuous infection chains involving the same or alternative hosts

Continuous infection chains involving the same or alternative hosts occur when the parasite survives by continually infecting plants of the same host species or plant species other than the main crop species. Such plants are called alternative hosts and are often related to the crop species. If the alternative host, which is often a weed species, does not show symptoms of disease, it is called a disease carrier. The parasite does not form resting structures nor does it survive saprophytically or epiphytically on plant surfaces. Survival therefore is dependent upon the presence of susceptible host plants. Such infection chains are common in all major groups of plant parasites.

Continuous infection chains involving alternate host

Some plant pathogens cannot be directly transmitted from an infected plant to another plant of the same species. They must alternate between two completely unrelated biological species. Even with the modern technologies available today, scientists cannot transmit most plant viruses and phytoplasmas directly from one plant to another. They can only be transmitted via a vector, usually but not always, an insect. In many cases viruses enter into a biological relationship with their vector and in some instances multiply in the vector. The vector therefore acts as an alternate or intermediate host and the infection chain alternates between the two unrelated species. Thus, the infection chain consists of plant-insect vector-plant. If the alternate host {the vector) is absent the infection chain is broken. Some heteroecious rust fungi also require an alternate host in the infection chain. They cannot complete their disease cycle in the absence of the alternate host. An example is the European pear or cluster rust fungus (*Gymnosporangium fuscum*) which is present in Europe, Asia Minor and North Africa. It has been introduced into North America but is currently absent from the Southern Hemisphere. The fungus forms clusters of aecia opposite spermagonia on the underside of pear leaves (*Pyrus communis*. The aeciospores cannot infect pear. They can only infect the alternate host, the gymnosperm Juniper (*Juniperus* spp.). Teliospores formed on the alternate host cannot re-infect juniper plants, they can only infect pear. The fungus does not form urediniospores and therefore cannot reproduce asexually on Junipents.

Discontinuous infection chains involving an epiphytic phase

Several fungal and bacterial pathogens survive on the surfaces of their hosts and other plants in a non-parasitic relationship as epiphytes. For example, the peach leaf curl fungus (*Taphrina dejormans*) causes leaves to develop yellow to reddish areas which progressively thicken and pucker, causing the leaf to curl and drop prematurely. Naked asci containing ascospores are produced on the upper surface of the curled leaves. Ascospores germinate to form budding, yeast-like conidia that can only infect very young leaves which are unavailable at the time when the budding colonies are formed. The fungus survives during the summer months (when only mature leaves are present on trees) and through the winter months (when no leaves are present on the deciduous peach trees) as an epiphyte on the bark and on the scales of the following year's leaf buds. In the following spring, when the leaf buds swell and young leaves begin to emerge, the budding cells infect the young leaves and form a mycelium in young, undifferentiated host tissue. The fungus produces asci in the early summer and the life cycle is completed. Satisfactory control of peach leaf curl can be achieved by a single application of a protectant fungicide (e.g. copper based sprays) just before bud-burst in the spring. Bacterial canker of stone fruit caused by *Pseudomonas syringae* pv. *syringae* causes sunken cankers, which frequently exude gum, on the woody tissue of affected trees. Infection can lead to premature death of whole trees or infected branches. The bacterium overwinters in cankers and in spring it multiplies and spreads by rain splash to leaves where large epiphytic populations develop. These epiphytic populations provide the main source of inoculum for wound and leaf scar infections in the following autumn. The same bacterium can also cause bacterial blight of peas. Again, the bacterium is present as part of the normal leaf-surface microflora of healthy peas. Infection occurs following wounding of plants.

Discontinuous infection chains involving a saprophytic phase

Many plant pathogenic bacteria and fungi survive during intercrop periods as saprophytes on diseased plant debris or on organic material present in or on soil. For example, the apple scab fungus (*Venturia enaequalls*) infects apple leaves and fruits during spring and summer. The fungus

overwinters as pseudothecial initials in infected leaves and fruit that fall to the ground during autumn. Following the union of mycelium of compatible mating types in autumn, black pseudothecia develop in the fallen leaves. The fungus then overwinters in the fallen leaves. In the following spring, the pseudothecia produce ascospores which serve as the primary inoculum which starts the disease cycle in the new season. The primary infections resulting from ascospore infection produce conidia which serve as the secondary inoculum that contributes towards the development of epidemics. Disease levels can be reduced by preventing the fungus from overwintering in fallen leaves. This can be achieved by spraying with a systemic fungicide before leaves fall during autumn so that pseudothecial development is prevented. The application of nitrogen (e.g. urea) to the leaf litter accelerates leaf decomposition under mild winter conditions reducing the survival rate in leaves. Many root-infecting pathogens of annual crops have a saprophytic phase in their life cycle. When infected plants die, either as a result of disease or natural senescence, the pathogen survives saprophytically on the infected debris. For example the take-all fungus of wheat (*Gaeumannomyces graminis* var. *tritici*) persists as a saprophyte in infected host debris during the intercrop period. Some pathogens that surrive in diseased plant debris compete poorly as saprophytes with the normal microbial inhabitants of soil. Consequently, their numbers decline during the intercrop period. Such pathogens have been called root inhabitants or soil lnvaders.

Other plant pathogenic bacteria {e.g. *Agrobacterium rhizogenes* which causes crown gall, *Ralstonta solanacear*um which causes bacterial wilt and Streptomgces scabies which causes common scab of potato) and fungi (e.g. damping-off fungi and several species of Frlsarium, Pgthium and Rhizoctonia) can compete very effectively as saprophytes with the normal soil microflora. These parasites are often referred to as soil inhabitants because they are part of the normal soil microflora.

Discontinuous infection chains involving a resting stage

Plant pathogenic bacteria, phytoplasmas and viruses do not form resting structures and can survive only by means of continuous infection chains or in the case of bacteria as saprophytes or epiphytes. Plant pathogenic bacteria do not form endospores. Some viruses retain their infectiousness in infected plant debris for long periods of time. For example, leaves infected with tobacco mosaic virus retain their infectiousness when kept dry under laboratory conditions for over 5O years. However, most viruses lose their infectiousness soon after their host plants die. Fungi and nematodes

on the other hand often form resting structures that enable them to survive, often for long periods, during the absence of suitable host plants or when the environment is unfavourable. The downy mildews produce oospores, the rusts and smuts form teliospores and many fungi produce chlamydospores. In some instances, resting spores can survive for periods of up to twenty years or more. The resting spores of *Plasmodiophora brassicae* (the cause of club root of brassicas) and *Spongospora subterranea* (the cause of powdery scab of potato) can survive in soil for many years. Moreover, the spores of both species are stimulated to germinate only by exudates secreted from the roots of certain plant species reducing the probability of spores germinating in a 'no win' situation when potential hosts are absent. Many fungi produce sclerotia (e.g. *Sclerotium* rolfsii, *Sclerotinta sclerotiorum*, *Verticillium dahliae*) which can survive in soil or in infected plant debris for periods ranging from months to many years, depending on the species.

Other resting structures formed by fungi include sexual fruiting structures such as cleistothecia, perithecia and pseudothecia. Nematodes also have adaptations that enable survival for long periods in soil in the absence of host plants. Eggs of the cyst nematodes (*Globodera* and *Heterodera* spp.) survive in egg cysts while those of root-knot nematodes (Meloidogyne spp.) survive in gelatinous egg masses. Both structures reduce the rate of egg desiccation. Some species such as *Angutna funesta* and *Ditglenchus dipsaci* can survive for several years in an anhydrobiotic state (metabolically 200 JohnBrotun inactive with loss of water from body fluids). Some cyst nematodes (e.g. the potato cyst nematode, *Globodera rostochiensrs*) will not hatch in the absence of host root exudates. Some fungal pathogens infect their host, enter into a dormant state and resume activity at a later stage of host development. For example, the loose smut fungus of wheat (*Ustilago tritici*) infects developing wheat embryos during flowering. Within a week of flowering, the embryo becomes resistant to infection. The fungus survives as dormant mycelium within the embryo of the seed. When infected seed germinates, the pathogen is activated. As plants approach maturity, the inflorescence is replaced by masses of smut teliospores. Some of the ripe-rots or postharvest rots of fruits are caused by fungi that infect the young developing fruit and then enter into a quiescent or latent state. They later resume activity when fruits commence to ripen. The black spot fungus of citrus is an example of such a fungus. Ascospores produced in pseudothecia on leaves that fell to the ground 6 weeks to 6 months previously infect very immature citrus fruits (between fruit set and 4 months development). From the appressorium, an infection peg penetrates the cuticle and forms a small mass of subcuticular (between

the cuticle and epidermis) mycelium. The fungus then remains in a quiescent state for about six months until the fruits are fully grown and mature. It resumes growth and grows into the rind tissue to produce black spot symptoms. Thus, the disease cycle involves a saprophytic state in fallen leaves and a resting state in immature fruits.