Lecture 3: Mechanism of Seed Infection, Factors Affecting Seed Infection, Longevity of seed-borne Pathogens, Seed Transmission and Inoculation

Introduction

Seed consists of three major components (seed coat, storage tissues and embryo) and out of these, embryos as a result of zygote play a vital role as reproductive structure. It also performs cell divisions to grow as mature plants. Storage tissues of seed are the reservoir of food mainly carbohydrates, protein and mineral nutrients. This food reservoir is important at the time of seed germination and seedling emergence. The seed coat acts as protective shield against biotic and abiotic stresses until the seed attains germination and seedling emergence. The seed coat and its major components assist seed-borne pathogens for their adherence, survival, infection and transmission to other healthy plants or seeds as a whole. However, mechanisms of infection and transmission of pathogens depend on the weather conditions and nature of crops irrespective of gymnosperm and angiosperm. The seed infection by seed-borne pathogens is entirely different from seed transmission. Seed-borne pathogens can establish successful infection and colonization in any part of seed compartment under favourable weather conditions with required inoculum potential. While seed transmission is possible by different mechanisms under favourable weather conditions. In majority of the agricultural crop diseases, true seeds or propagative materials act as a source of inoculums and, therefore, they are termed as seed-borne inoculums. The expression of disease symptoms on seedlings and adult plants by the proliferation of such inoculums and abnormal physical functions could be termed as seed-borne diseases. Different groups of microbes including fungi, bacteria and

viruses are responsible for seed-borne diseases. Seed infection is mainly due to the establishment of potential pathogenic microbes in any part of the seed and this is classified based on the nature of movement (systemic or non-systemic), structural components of seed infected (seed coat, endosperms, scutellum), etc.

The seed-borne pathogens were grouped into four classes namely:

A. The pathogens for which the seed is the main source of inoculum (the disease of such inoculum is controlled by controlling seed infection)
B. pathogens in which the seed-borne phase of the disease is of minor significance as a source of inoculum,
C. pathogens never been shown to cause disease as a result of their presence on seeds and it is the largest group of seed-borne microorganisms and
D. Pathogens that can infect the seed either in the field or in storage and reduce yield and seed quality.

These four groups of pathogens are transmitted either systemically, non-systemically or both as seed-borne or seed contaminations.

Plant pathogens use diverse life strategies. Pathogenic bacteria proliferate in intercellular spaces (the apoplast) after entering through gas or water pores (stomata and hydathodes, respectively) or gain access via wounds. Fungi can directly enter plant epidermal cells or extend hyphae on top of, between or through plant cells. Pathogens invaginate feeding structures (haustoria), into the host cell plasma membrane. Haustorial plasma membranes, the extracellular matrix and host plasma membranes form an intimate interface at which the outcome of the interaction is determined. These diverse pathogens deliver effector molecules (virulence factors) into the plant cell to enhance their fitness. This is a general phenomenon in almost

all biotrophic and necrotrophic pathogens, which are infecting seeds and adult plants.

Defence Mechanisms in Seeds to Counteract Infection by Seed-Borne Pathogens

Seeds contain major nutrients like protein, lipids and almost all forms of carbohydrates and these nutrients are meant to support the seed germination and seedling growth. However, these also serve as nutrients for the growth and development of most of the seed-borne fungi, which are necrotrophs. The infection of seed-borne pathogens will not be successful unless the natural defence mechanisms of seeds and seedlings are defeated by the pathogen. Generally, physical structures of seeds like the seed coat, waxing and maturity standards could defend the infection by acting as barriers. the thick cell walls of the endosperm act as a physical barrier which in turn can slow down the penetration of pathogenic fungal hyphae. some seeds like barley contain the defence compounds, viz. thionin, endo-chitinase, ribosomal-inactivating proteins, β-glucanase, non-specific lipid transfer protein, lectin, peroxidase, thaumatin-like protein, inhibitors and α-amylase, and proteinases. cereal seeds contain different types of proteinase inhibitors than legumes; perhaps due to this, seed-borne pathogens are also different in both the groups of seeds. Seeds also contain phenolics, lectins and many more anti-pathogenic proteins. Therefore, for attaining successful pathogenesis, the seed-borne pathogens should overcome all these physical and biochemical barriers.

Systemic Infection and Transmission of Seed-Borne Pathogens

A particular group of seed-borne pathogens are recognized based on their nature of infection and transmission. In this systemic group, infection takes place in any part of the plants but movement of pathogen will happen from one part to another

systemically. Successful systemic pathogens will move directly by injuries due to natural or artificial wounds or through vascular system or plasmodesmata connecting cells. Viable pathogens infect seeds by these mechanisms. For example, the halo blight pathogen of bean, i.e. *Xanthomonas campestris* pv. *phaseoli*, infects bean seeds through the vascular system, by natural openings (from the pod suture goes to the funiculus then to the raphe and tegument, or it can also happen through the micropyle).

Systemic Infection

The active association of fungal pathogens with seeds is the common phenomenon in systemic infection. Generally, seed infection is the establishment of a pathogen within any part of a seed, which may occur systemically, either through vascular system or plasmodesmatic connections or directly through floral infection or penetration of the ovary wall, seed coat or natural openings. Fungal pathogen infects through flower, fruits or seed stalks or penetration through stigma (*Sclerospora graminicola*, *Ustilago nuda*, *Ustilago tritici* and *Claviceps fusiformis*); infection through ovary wall or seed coat (*Colletotrichum lagenarium* in watermelon and *Cercospora kikuchii* and *Colletotrichum truncatum* in soybean); natural openings or injuries (*Cercospora sojina*) enter through pores and enter through hilum (*Alternaria sesamicola*). However, infection of any one seed may take place by more than one process. For some seed-borne pathogens, stigma acts as cushion to lodged spores and subsequently same spores germinate with available moisture over stigma. Active hyphae from germinated spores reach the ovary through style and infect the same via ovary wall and further proliferate. In some cases, pathogenic mycelia remain as dormant asexual structure and become active whenever respective seed germinates (Figure).

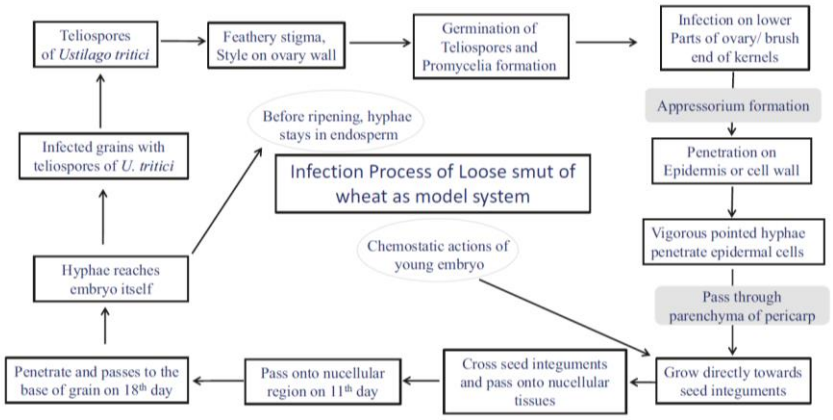

Figure illustrates Infection process of loose smut pathogen in wheat

Non-systemic Infection

This type of infection occurs through the ovary wall, pericarp and integuments of seed coat. The loose smut of wheat is the best example for this type of infection. In another type, fungal pathogen penetrates during different developmental stages of a fruit/pod. They invade maturing seeds (anthracnose of bean incited by *Colletotrichum lindemuthianum* and *Ascochyta pisi* of pea). In the case of linseed anthracnose, the pathogen (*Colletotrichum linicola*) penetrates through drying petals and maturing capsules. Pathogen *Rhizoctonia solani* penetrates through the pericarp of capsicum and enters further through funiculus into the embryo. Similarly, *Sclerotinia sclerotiorum* of crucifers penetrates in drying petals which provides foodhold for mycelia invasion of wind-borne ascospores. Most of the leaf blight pathogens which are transmitted through seeds spread and infect in non-systemic means. Atmospheric weather factors like low temperature, dew deposition and wind current are crucial factors for such transmission and infection on foliage and subsequent colonization in seeds. The leaf blight of wheat caused by *Bipolaris sorokiniana* is the best example that the seed-borne conidia germinate and colonize

on foliages of seedlings and adult plants and the environmental conditions favour further spread and infection in the field (Figure).

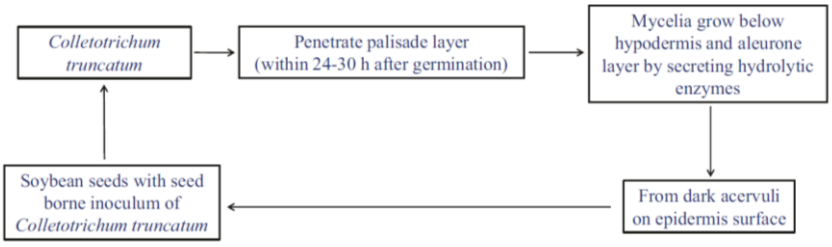

Figure illustrates Infection process of Non-systemic seed-borne pathogen.

Seed Contamination or Infestation

Seed-borne pathogens are also transmitted by infestation or contamination. This is passive association of pathogen with the seeds. Pathogens adhere to surface or are mixed with seeds at anytime during harvest, extraction, threshing or processing until packing of seeds in suitable containers. The seed concomitant contamination will take place with pathogenic structures, mixed with infected plant parts and soil as well. Fungal pathogens carried on the seed coat surface transmitted by means of seed contamination or infestation are like *Alternaria brassicae* and *A. brassicicola* (cruciferous), *Alternaria linicola* (flax), *A. longipes* (tobacco), *A. radicina* (carrot), *Ascochyta pinodella* (pea), *Ascochyta rabiei* (gram), *Drechslera sorokiniana* and *D. oryzae* (rice), *Pyricularia oryzae* (rice), *Protomyces macrosporus* (coriander), *Rhizoctonia solani* (eggplant), *Colletotrichum graminicola* (pearl millet), *Sclerospora graminicola* (finger millet), *Peronospora manshurica* (soybean), *Phytophthora phaseoli* (bean), *Neovossia indica*, *Urocystis agropyri* and *Tilletia caries* (wheat), *Ustilago hordei* (barley and oat), *U. maydis* (maize), *Sphacelotheca reiliana*, *S. cruenta* and *S. sorghi* (sorghum), *Puccinia carthami* (sunflower),

Melampsora lini (linseed) and *Uromyces betae* (beetroot). Some pathogens gain entry directly into the ovary and become seed-borne. For example, infective hyphae of covered smut of barley (*Ustilago nuda*) enter directly into the ovary but there will not be any indication about the presence of hyphae on the style. The flag smut of wheat, which was considered as minor disease has also become the major problem in some pockets of wheat-growing regions in India. The pathogen (*Urocystis tritici* Koern/*U. agropyri*) infects mostly leaf tissues. Severely infected plants form shrivelled grains. Spores of this pathogen are both seed- and soil-borne and after germination, they infect seedlings and reside in apical cells to become systemic. Infective hyphae grow both inter- and intracellularly and symptom expressions occur in any growth stage based on weather conditions. Another important seed-borne disease, i.e. hill bunt or stinking bunt, incited by *Tilletia caries* (DC) and *T. foetida* is very common in northern hills of India. This pathogen is a typical example of seed contamination in which teliospores are carried on seeds as a source of inoculum in addition to inoculums present in soil as a primary source. The smutted seeds contaminate healthy seeds and such seeds favour germination of spores in soil.