Mechanisms of Seed Infection and Pathogenesis

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Seeds, from the time of their inception at flowering of the parent plants until they germinate and develop into seedlings, are prone to microbial attack. They are storehouses of food and energy, and many microorganisms have evolved abilities to invade and use them. Infection frequently damages the seed, and may provide a means for pathogens to survive from one generation of host plants to the next, and travel from one geographical area to another.

There have been several reviews on seed-attacking microorganisms. Neergaard's book (51) provides a great deal of information on seedborne pathogens and on storage diseases. Christensen (9) edited a valuable monograph on grain storage, and Richardson (56) has compiled a list of seedborne pathogens.

General Seed Environments

Seeds must survive three very different environments. In this symposium, Mills (47) also considers these environments (see his Fig. 1). These environments are: the seed production field, where seeds form, develop, and mature; the storage area, where seeds are kept before planting; and the soil environment, in which planted seeds must either produce a new generation of plants, or perish.

Organisms associated with these habitats, and their mechanisms of infection and pathogensis are considered in this paper.

The Seed Production Field

The environment. The developing seed is an actively-growing structure, and water is not a limiting factor for pathogen growth until seeds are mature. Developing seeds are frequently enclosed within protective parental tissues, which form a barrier to invasion by the pathogen. J. M. Halloin (23) will discuss these and other structures limiting infection. Seeds of commercially-grown cultivars usually are produced in relatively large plantings, which allows adapted microorganisms to increase on a favorable host. Seeds of many crops are grown in isolated geographical areas where very severe pathogen pressure is lessened.

Seeds infected by pathogens in the seed production field may survive and be sources of primary inoculum in the next generation. Losses from these pathogens can be very large. For example, seedborne inoculum of *Phoma lingam* and *Xanthomonas campestris* initiated severe nationwide outbreaks of black leg and black rot (respectively) of crucifers during the early 1970s. The initial inoculum in these epiphytotics probably arose from seedborne microorganisms.

Viruses. Viruses are frequently seedborne, and this inoculum source can result in severe losses in the daughter crop, particularly if an efficient vector is present in large numbers.

Generally, viruses invade developing seeds from systemically-infected parent plants. Infection usually occurs in embryo tissues (3); however, tobacco mosaic virus may be transmitted to germinating seedlings from infested seed coat tissue (3,67). Most frequently, gametes (particularly the ovules) become infected and the virus persists during seed development. Infection that results in seedborne transmission of viruses is largely restricted to this very early stage of development since developing embryos and seeds are not connected to the parent plant via plasmodesmata (3,53). Intact cell walls are formidable barriers to viral passage (3).

There is direct evidence (3) for pollen transmission of viral particles that cause lettuce mosaic (58), Lychnis ringspot virus (2), false barley stripe mosaic (21), necrotic ringspot, prune dwarf mosaic of sour cherry (20), and bean common mosaic (46). In most of these cases, investigators have shown that pollination of virus-free plants with pollen from infected plants results in seed transmission of the virus. Gold et al (21) found viruslike rods of false barley stripe in pistils, pollen, and seeds of diseased plants but not in those of healthy plants.

Although direct evidence for infection of ovules is more difficult to obtain, Bennett (3) presented support for several arguments that it does occur and that it is critical for seed transmission of many viruses. First, only the mother plant needs to be infected for the resulting seeds to be infected. Second, with some viruses, seed transmission occurs only from plants that were systemically infected before flowering. Third, environmental conditions must be favorable for transmission only until flowering; after flowering, conditions may be unfavorable without influencing seed transmission (3).

There are also a few viruses that directly infect the embryo rather than the gametes. Barley plants inoculated at the hard dough stage transmitted barley stripe mosaic virus (17). Bean southern mosaic virus may infect embryos at 4 days, but not at 7 or 10 days, after flowering (14).

Seedborne viruses may have direct detrimental effects on seeds. Infection of the parent plant by soybean mosaic virus induces mottling of the seed coat at low temperatures (eg, 20 C), but not at high (eg, 30 C) temperatures (57). Mottled seeds may or may not be viruliferous (53). Seed coats of soybean seeds infected by soybean stunt virus are mottled with concentric rings (38), and broad bean seeds with broad bean stain virus may have brown necrotic bands (42). Seed infection by many viruses results in small, shrunken seeds (51).

Bacteria. Seedborne bacteria, like viruses, can cause severe losses in fields planted with infected seeds. The pattern of seed infection, however, differs. Whereas viruses commonly infect gametes and survive with the developing seed until it matures, seedborne bacteria more commonly infect the developing embryo. However, the causal agent of Stewart's wilt of corn can be isolated from pollen of infected plants (35).

A typical example of seed infection by bacteria was provided by Skoric (63) in studies with bacterial blight of pea (*Pseudomonas syringae* pv. *pisi*). He inoculated flowers or developing pods with aqueous bacterial suspensions. Flowers and very young pods died, but older pods developed lesions, and bacteria accumulated on the inner surface of pods, on the funiculus, and on, but not in, seeds. If the dorsal suture of the pod was infected, bacteria moved from this site into the funiculus, and from there digested their way toward the micropylar opening. If the seeds were sufficiently developed to survive bacterial infection, bacteria moved into the seed coat where cavities formed that contained large numbers of bacteria. The bacteria were on the surface of the embryo, but did not penetrate it.

The organisms causing common and halo blights of bean, angular leafspot of cucumber, bacterial blight of cotton, black rot of cabbage, and Stewart's wilt of corn infect seeds and are trans-

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mitted similarly (4,5,11,35,48,72,73). Bacteria enter the vascular system from either a localized or a systemic infection, and then enter the seed through the funiculus or, in corn, through spiral vascular vessels (35). The bacteria then reside in the seed coat or in the very outer layers of the embryo or endosperm.

Upon seed germination, bacteria do enter embryo tissue. Zaumeyer (73) observed that rods of *Xanthomonas campestris* pv. *phaseoli* (common blight of bean) were carried by water flow into spaces created by imbibitional stress. Some of these bacteria entered the vascular system of the seed, and were then carried to the vascular system of the shoot-root axes. Other bacteria presumably enter embryo tissue similarly and cause systemic or localized disease according to the host-pathogen interaction of the disease in question.

Seeds, however, do not have to be internally infected to transmit bacterial diseases. Grogan and Kimble (22) demonstrated that dust from debris of bean plants infected with *Pseudomonas syringae* pv. *phaseolicola* is highly infective, that it can coat seeds and become lodged in seed coat cracks and in natural openings (eg, the hilum and micropyle). Surface sterilization will not eradicate the pathogen from seeds so contaminated. Several other bacteria are carried on the seed coat surface, including *X. campestris* pv. *vesicatoria* on tomato, *Corynebacterium flaccumfaciens* on bean, *P. syringae* pv. *lacrymans* on cucumber, *X. campestris* pv. *incanae* on stock, and *X. campestris* pv. *campestris* on cabbage (51).

Bacterial infection of seeds frequently results in smaller, shrivelled seeds (51). Additionally, *Bacillus subtilus*, which is seedborne in a fashion similar to *P. syringae* pv. *pisi*, causes a reduction in seed germination and emergence, but causes little or no serious disease on the growing plant (68).

Fungi. Fungi also infect developing seeds and have a wide range of mechanisms for becoming seedborne and for being transmitted to germinating seedlings. Neergaard (51) listed eight combinations of infection sites (intraembryal, extraembryal, seed contamination, and organspecific infection) and infection types (systemic, local infection and saprophytic, followed by systemic, local, or organspecific infection). Fungi are the only group of pathogens with representatives in all eight classes (51), demonstrating the wide range of seed-infecting adaptabilities in this group.

Fungi may be seed transmitted in the embryo, and this infection may have an essential place in the life cycle of the fungus. The loose smuts of wheat and barley (*Ustilago nuda* and *U. tritici*) are examples. Teliospores are produced in masses on inflorescences of infected plants. Spores are scattered by winds and land on florets of healthy plants. The spores then germinate and form promycelia, which fuse and form binucleate infective hyphae. These infect through the wall of the ovule or perhaps infect the style and then grow to the ovule. The invading hyphae increase in the developing seed, and after 3–4 wk reach the embryo. During maturation of the grain the hyphae are located in the pericarp, testae, the aleurone, and scutellum. With the germination of the grain, the hyphae again resume growth, although obvious symptoms are not present. When the host flowers, the fungal growth erupts and replaces the inflorescence with spore masses (18,51). The ergots also possess highly specialized adaptations to parasitize seeds; these are discussed by Mills (47).

Fungi Imperfecti are frequently seedborne as a consequence of infection of seed coats or pericarps. Neergaard (51) listed seed transmission of this type for many fungal species representing the genera *Alternaria, Cercospora, Colletotrichum, Drechslera, Acroconidiella, Aureobasidium, Septoria, Botrytis,* and *Fusarium*. *Phoma lingam* infects the outer epidermis and subepidermal parenchyma of *Brassica* seed coats, and also (occasionally) the tissues of the embryo (36). *Fusarium moniliforme* and *F. oxysporum* apparently infect corn kernels by invasion through the pedicel (39); other *Fusarium* spp., *Curvularia* spp., *Alternaria* spp., and *Ascochyta* spp. also may be embryoborne (51).

Most of the seedborne organisms discussed so far are economically important because they can cause severe disease in fields planted with infested seeds. Other organisms, however, have severe effects on seed quality. For seed quality to be a serious concern, a high percentage of the seeds must contain the pathogen; something that rarely occurs with organisms capable of causing severe epiphytotics in the planted field.

Alternaria brassicicola, which infects *Brassica* seeds, and *Phomopsis sojae* (*Diaporthe* spp.), which infects soybean seeds, are two typical examples. Both are weak pathogens of the growing plant but cause major damage on dying or senescing tissue (44,50). Both invade through the pod and infect seeds (1,50), although *Phomopsis*, at least may exist in actively growing soybean plants (62) and *A. brassicicola* causes a leaf spot on growing plants (50). If harvest is delayed, these organisms can proliferate; *P. sojae*, in particular, can cause severe reductions in seed germinability (49,71). Neither pathogen usually kills seeds outright, since chemical eradication of these fungi, particularly *Phomopsis*, frequently results in increased germination levels (6,49). However, seed vigor may not be fully restored to that of noninfected seeds even after eradication (7), and biochemical changes indicative of seed deterioration occur as a consequence of infection (33).

Seed Storage

The environment. After harvest, seeds are stored under dry conditions. Therefore, no free moisture is available. Generally, masses of seeds are stored together, so soil and vegetative material are, or at least should be, absent.

Healthy and freshly-harvested seeds have maximum germination potential. During storage they gradually lose vigor and viability under even the best of conditions; the higher the moisture and temperature, the more rapid is this physiological deterioration. If storage fungi invade seeds, quality loss may occur very quickly and seeds may become unfit for planting.

Microorganisms involved. If seeds are dried and then stored in equilibrium with a relative humidity (RH) of 68% or less, no microbial growth will occur, but various microorganisms, including seedborne pathogens described above, may remain quiescent (9,10). As seeds equilibrate with RHs above 68%, xerophytic microorganisms can grow. In seed storage, *Aspergillus* spp. are the dominant organisms, although *Penicillium* spp. may also be present (9,10). *A. halophilicus* is able to grow in seed at moisture contents too low for growth of other fungi. As moisture increases *A. restrictus,* the *A. glaucus* group, *A. candidus*, *A. ochraceus, Penicillium* spp., and *A. flavus* also may grow in stored grains (9).

Christensen and his co-workers (9) tested many grain samples over many years, and have found storage fungi in only a few kernels in production fields. Shortly after arrival at the grain elevator, however, seeds are likely to become infected unless their moisture contents are low enough to inhibit growth of *Aspergillus* spp. Insects can transport inoculum to seed and while feeding, provide favorable sites for infection. These aspects are discussed by Mills (47).

Sites of infections. In the infection of stored pepper seeds by *A. halophilicus*, conidia germinate on the seed coat and form hyphae with appressoria. Penetration pegs breach the seed coat, and subsequently hyphae ramify over the seed coat surface (60).

Embryos of cereal grains may become discolored as a consequence of infection by storage fungi (8,9,29,52). Conversely, embryos of peas and squash are rarely infected, although the seed coats are readily invaded by members of the *A. glaucus* group, *A. restrictus*, or *A. flavus* (29). Maturing peanut hulls and seed coats are invaded by *A. flavus*, but embryo infection seldom occurs (40). Storage fungi capable of infecting and killing other seeds seldom infect tomato seeds (29). The reasons for differences in susceptibility of seeds or embryos of different species are not understood in most cases. Pea embryos contain no phytoalexins or other inhibitors of fungal growth (55), but immature peanut embryos do contain substances inhibitory to *A. flavus* (41).

Mechanisms of seed damage. Infection of pea seed coats and wheat embryos result in reduced viability of the seeds (29). In stored wheat, this loss is probably due directly to damage caused by invasion of the embryo. In stored peas, even though the same fungi are involved, this cannot be the case, since the embryos are not invaded (27). This suggests that involvement of a toxin, which was

found in peas infected with *A. ruber* (24,25). No toxin was found in wheat seeds infected with *A. ruber* (G. E. Harman, *unpublished*) or in barley seeds infected with *A. repens* (30). The toxin from peas was not toxic to wheat, tomato, lettuce, lima beans, or squash seeds (25).

Squash seed coats are invaded by most storage fungi tested, but only one isolate of *A. flavus* reduced germination. This isolate also damaged wheat seeds, but did not invade peas. The damage by *A. flavus* occurred during germination; removal of the seed coat allowed seeds to germinate normally (29).

Thus, storage fungi have different mechanisms whereby they damage different kinds of seeds. There is some degree of host specificity in these fungi, and they induce damage through an array of biochemical and physiological changes in the seed. These changes will be discussed by other speakers in this symposium.

The Soil

The environment. Conditions and practices in both the seed production field and storage, as well as in the planting field, have their ultimate test when seeds are planted in the complex environment of the soil. Sources of food and fiber largely arise from sexually propagated plants, so seeds must perform well when planted.

Moisture levels range from levels insufficient for seed germination to waterlogged soils in which oxygen deficits may occur. Temperatures vary from 0 to >30 C. The pH in agricultural soils commonly varies from 5 or less to 8 or more, and levels of organic matter, amounts of various ions, and soil texture differ.

Microorganisms involved. Soils are infested by microorganisms, some of which cause seed rots and subsequent diseases of growing plants, while others that colonize seeds cause no direct ill effects. Still others, such as *Rhizobium*, are beneficial to the plant. The remainder of this paper will deal primarily with the seed-rotting fungi (*Pythium* spp., *Rhizoctonia solani*, and *Fusarium* spp.) and with seed-colonizing *Pseudomonas* spp.

Resting structures of soil-inhabiting microorganisms are in a state of stasis (15,43), ie, they are in a dormant state which is imposed by the environment. Oospores and sporangia, chlamydospores, and sclerotia are the primary resting structures of *Pythium, Fusarium,* and *Rhizoctonia solani* in soil (31,32,69). These structures germinate when provided with proper stimuli.

Seeds in the soil stimulate germination of microbial propagules. Poorer quality seeds are more stimulatory than better quality seeds, and a greater percentage of them are rotted. The greater stimulation of microorganisms by poor quality seeds has been related to their greater exudation of water-soluble exudates during the early stages of germination. Carbohydrates and amino acids seem to be particularly effective in enhancing spore germination (12,19,37,45,54,61), although other compounds also leak from seeds (70).

Volatile exudates from seeds, as well as water-soluble exudates, can stimulate spores in soil-induced stasis to germinate (28) and can support growth of several microorganisms (59). More volatiles are produced by aged (low vigor) than by unaged (high vigor) seeds. As little as 200 ppb of known compounds in aerial solution stimulate germination of conidia of *F. solani* and *Alternaria alternata*, while chlamydospores of *F. solani*, which require nutrients to germinate, need approximately 100-fold more stimulatory material (26). Volatiles of seeds also stimulate growth of naturally-occurring *Pythium* and *Fusarium* spp. in field soils (G. E. Harman, *unpublished*).

At temperatures between 18 and 25 C, sporangia of *P. ultimum* begin to germinate near seeds of beans or peas within 1.5 hr of planting (64), while chlamydospores of *F. solani* begin to germinate 4–5 hr after planting (13). Seed coats are infected by *P. ultimum* within 6–10 hr of planting, and colonies of hyphae can be observed on the outside of the seed coat 20 hr after planting. Embryos are infected 40 hr after planting (66). Apparently *P. ultimum* requires approximately 30 hr to penetrate the seed coat. Seeds with damaged seed coats are more readily infected than those with intact coats. As few as one or two infections are sufficient to cause rot of the entire seed (65).

In addition to seed-rotting fungi, other organisms readily colonize planted seeds. Seeds of squash, snap bean, or pea are colonized rapidly by fluorescent pseudomonads from soil. Populations go from 0 to 10^6 propagules per seed within 48 hr (34). These bacteria have no direct effect on seeds, but volatile metabolites produced by them are detected by seed maggot adults (16,34) that oviposit near planted seeds. The maggots that emerge from the eggs can seriously damage the planted crop. Whether these volatiles have any effect on germination of fungal spores is unknown.

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