Chapter 12

Disrupted Reproduction

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I. INTRODUCTION

Reproduction is the sine qua non of life. Living things reproduce. Nonliving things do not. Therefore, diseases that strike at the reproductive system strike at the very heart of life.

To the plant, reproduction is critical to the survival of the species. Reproduction and reseeding must occur every growing season for annual plants, while for perennials this process may not be necessary for many years. How well plants are able to reproduce may play a large role in whether they can move into and occupy an ecological niche. The development of plant communities through a series of successions is usually
dependent on how well the reproductive units, e.g., seeds, can be trans­
ported into and develop within a new site. In cultivated situations, many
crops are established each year from seeds, the viability and health of
which become critical factors in establishing a productive crop. Flower­
ing also provides a mechanism for the genetic recombination of traits
which aid in the evolution of plant species, either naturally or man
guided.

Aside from their role in the maintenance of the species, the repro­
ductive portions of a plant are often the portion that man uses for food.
As such, crop productivity is often directly related to a plant’s repro­
ductive capacity.

Mangelsdorf (1966) said that over the long reaches of history man has
used about 3,000 species of plants for food, that about 150 are in world
commerce, but that only 15 really feed the people on this globe. Of
Mangelsdorf’s 15 species, the reproductive bodies of 10 are eaten by
man including rice, wheat, maize, sorghum, barley, common bean, soy­
bean, peanut (groundnut), banana, and coconut.

The reproductive disease on wheat known as bunt or stinking smut
has plagued man ever since wheat was domesticated. Rice blast is a
frightening word in whatever language used by the rice farmers of the
world who feed more people than growers of any single other crop.

Last, since “man shall not live by bread alone,” his spirit is nourished
by the beauty provided by the flowers of many plants. Who among us
has not been affected by the beauty of a rose, an orchid, or a tulip? The
classic disease of a flower is the famous and infamous virus disease of
tulips known as “breaking.” The petals show streaks of color rather than
being a solid color. A painting of it in 1576 in the Netherlands is prob­
ably the earliest published illustration of a plant disease. Moreover, it
created the famous “tulipomania” in Holland in the 1630’s. At this time,
Dutch overseas trade had increased to the point that the country was
ripe for an atmosphere of speculation. Tulips were relatively new to
Holland, having been imported from Turkey. Ordinary tulips were easy
to obtain, but the prize ones were those showing the “breaking” pattern.
By 1634, the mania for tulips had increased to the point that prices of
single broken tulips were bid up to fabulous levels. Large fortunes were
invested in them until the bubble burst, leaving individuals and the
country as a whole on the verge of bankruptcy. Today, the ornamental
plant and floral industry is a thriving one, indicating man’s need and
quest for beauty as well as for food.

Disrupted reproduction affects, then, not only plants themselves but
also man who depends on them, or is influenced by them. Much of the
art and science of plant pathology is based on the prevention of this disruption.

II. PATHOGENS AND POLLEN

A. Effects on Pollen Production

Viruses sometimes induce male sterility in normally self-fertile plants. This sterility may be due to the inhibition of pollen formation, abortion of pollen after it is formed, or dysfunction of the pollen. Tomato aspermy virus in tomato interferes with normal meiotic processes, such that when tetrads are formed in pollen development they are aborted prior to maturity as pollen grains (Caldwell, 1952). An abnormal aggregation of chromosomes at pachytene may be involved in this disruption.

Pollen abortion, for whatever reason, is also involved in the male sterility of geraniums infected with tomato ringspot virus (Murdock et al., 1976), and of barley infected with barley stripe mosaic virus (BSMV) (Inouye, 1962). The abortion process seems to involve a disintegration of the cytoplasm in the pollen grain followed by collapse of the wall of the pollen grain. In BSMV-infected barley, the number of pollen grains produced per anther is reduced 20–40% as compared to healthy controls.

B. Pollen Germination and Germ Tube Elongation

Pathogens may also decrease the vigor of pollen. A reduction in the number of viable pollen grains occurs in geraniums infected with tomato ringspot virus (Scarborough and Smith, 1977) and in BSMV-infected barley (Inouye, 1962). The decrease may be as much as three- to five-fold in the latter. In soybeans infected with tobacco ringspot virus, pollen production and germination problems are expressed as reduced numbers of pollen grains per flower, reduced germination, and reduced germ tube length. Similar effects on pollen vigor have been observed in tomatoes exposed to fluoride (Sulzbach and Pack, 1972). In controlled crossing experiments with raspberries, Lister and Murant (1967) noted that virus-free pollen greatly decreased the ability of pollen from infected plants to set seed, illustrating again the decreased vigor or competitive ability of virus-infected pollen.

The deleterious effect of BSMV infection in barley on pollen development and vigor is similar to that in barley with genetic male sterility (Roath and Hockett, 1971; Inouye, 1962). This suggests that the mech-
anisms may be similar and may involve the disruption of nucleic acid reproduction.

C. Anther Destruction

The most dramatic effect on male reproductive organs is their complete destruction. In the Netherlands, a necrosis of bud tissues in tulips has been described (deMunk and Beijer, 1971). In light cases only stamen primordia are affected, while in more severe cases the entire flower bud may be necrotic. When such bulbs are planted, some or all of the stamens are decayed but the remainder of the flower is healthy. Some fascinating detective work by deMunk (1972) has shown that this condition is caused by the invasion of mites. However, the entry site for the mites is provided by open buds. The buds open in response to increased ethylene concentrations which result from infection of the bulbs by *Fusarium oxysporum f. tulipae* in conjunction with poor ventilation. This is an example of the interaction between pests which can result in a more adverse effect on the plant than would be caused by either pest alone.

III. PATHOGENIC EFFECTS ON OVULES, OVARIES, AND FRUIT

A. Ovule Abortion

Abortion of flower buds and their contents occurs in some plants infected with a variety of pathogens. In some cases, the abortion occurs even prior to anthesis, while in others the ovary and its contents are affected at the time the flower opens or shortly thereafter. The mechanism of such adverse effects ranges from an interference in nuclear division at certain stages of meiosis to a wholesale rotting of the tissues. Tomato aspermy virus interferes with meiosis (Caldwell, 1952) by disrupting meiotic divisions of the megaspore mother cell. In geraniums infected with tobacco and tomato ringspot viruses the total number of potential florets is not affected, but virus infection does reduce the number of florets that complete their development (Scarborough and Smith, 1977). Infected florets show chlorosis which progresses to necrosis of the floret. By the time sepals and petals of a floret are chlorotic, the tissues interior to the petals are necrotic. Even though the ovules are oriented correctly within the ovary and development of the nucellus is
normal, the integument layers fail to surround the nucellus (Murdock et al., 1976).

B. Phyllody

In some diseases, sex organs are transformed into leaves, a condition known as phyllody. Smut infections are classic examples. *Sphacelotheca reiliana* in corn and sorghum often results in large, leafy structures instead of the normal smutty heads typical of this host-parasite combination. Early botanists, including Linnaeus himself, often regarded such infected plants as new species or new varieties of a species.

Smuts are not the only pathogens capable of inducing this response. In downy mildew of millet, caused by *Sclerospora graminicola*, the grain and sometimes the stamens are replaced by a short, leafy shoot. In Israel and India, Klein (1970) and Sahambi (1970) describe phyllody diseases of safflower and sesame that are vectored by leafhoppers. Both are probably caused by mycoplasmas.

Bos (1957) has discussed the possible mechanisms of phyllody. He argues that when a shoot on a plant begins to flower, the vegetative character of the shoot is suppressed and leaves become flower parts. If, therefore, a disease counteracts the suppression during the flowering process, the vegetative character reasserts itself and leaves are formed again. Presumably the whole process is hormonally regulated.

C. Tissue Substitution

In some diseases the host tissue becomes substituted for by fungus tissue as in ergot and the smuts. The logo on the jacket of this volume depicts the substitution of the grains of wheat by the tissue of the loose smut fungus.

1. Ergot

Ergot, caused by *Claviceps purpurea*, is one of the oldest diseases known to man. It is a classic case of tissue substitution. The host tissue is transformed into an elongated purplish body called an ergot or a sclerotium. Campbell (1958) indicates that the base of the unfertilized ovary is the main site of penetration, which occurs within 24 hr of inoculation. However, others have observed penetration of the stigma. The ovary wall is colonized first, but by the fourth or fifth day, the mycelium has become intracellular and has engulfed the ovule. A hymenium develops on the surface of the ovary and produces spores in a liquid called...
honeydew. Mower and Hancock (1975) determined that a 10- to 50-fold difference in water potential exists between host and parasite, thus causing an increased translocation of sucrose to the infected ovary. During parasitism, the fungus converts the sucrose to a variety of honeydew sugars.

While ergot is usually thought of as a disease of rye and other cross-pollinated grasses, recent work has shown that genetically and cytoplasmically male sterile wheat and barley are also highly susceptible to ergot (Puranik and Mathre, 1971; Stoskopf and Rai, 1972). Similarly, barley made sterile by BSMV shows an enhanced susceptibility to ergot (Darlington, et al., 1976). The flowers of male sterile wheat and barley are highly susceptible to ergot because of the open flower condition which occurs in such plants. Until the flower is fertilized, it remains open and thereby is exposed to ergot inoculum for a long time. Several studies (Cunfer et al., 1975; Darlington and Mathre, 1976; Watkins and Littlefield, 1976) have shown that the fertilized ovary becomes resistant to ergot infection shortly after it has been pollinated with some indication that the resistance mechanism is induced within 30 min after pollination (Darlington and Mathre, 1976). The exact mechanism of resistance is unknown, but pollination is known to induce large changes in hormonal levels in fertilized tissue. These physiological changes may prevent germination of ergot conidia on the stigma or prevent hyphal penetration into and development within host tissue. Inoculation of diallel crosses between spring wheat lines that develop resistance quickly after pollination and those that develop resistance slowly indicate that the resistance factor is maternally controlled. In fertile spring wheat with some resistance, Platford and Bernier (1970) indicated that the ovaries are infected but further development of the fungus is inhibited and it produces only small sclerotia and reduced honeydew. If the development and use of hybrid spring wheat and barley are to be successful, the ergot problem will have to be controlled by use of resistant cultivars, resistance triggered by fertilization, or elimination of the sources of primary inoculum.

2. Smut

In addition to ergot, the other disease most often associated with the complete substitution of the female flowering parts of the plant is smut. Usually, the presence of the smut teliospores in the heads of infected grasses indicates the destruction of the floral parts, but Cherewick (1965) reports that oats infected with *Ustilago koller* or *U. avenae* may show “blasting” or sterility of the heads even though little or no smut develops in them.

For those smuts which attack the head and reduce the ovary to a mass
of teliospores, the mycelium which produces such spores usually enters the floret as the result of a systemic infection, e.g., from mycelium which has invaded the growing point. The infective hyphae have little or no effect on the apical meristem until the plant has reached the reproductive stage when the mycelium is induced to form teliospores. According to Fischer and Holton (1957), this aspect of smut biology was one of the least understood in 1957. It still is.

**D. Flower Blights**

The least specific type of disease of floral parts is the complete decay or blight of flowers, as can be caused by bacteria or fungi. Perhaps the best known pathogen in this respect and one with a wide host range is *Botrytis cinerea* which can attack blossoms as well as fruit. In the "early" *Botrytis* rot of grapes, the fungus invades the stigma and style but then becomes latent in the necrotic stigmatal and stylar tissue that remains attached to the developing grape berry (McClellan and Hewitt, 1973). In this situation, as with others (Ogawa and English, 1960; Chou and Preece, 1968), the presence of pollen is stimulatory to the development of the pathogen, resulting in increased and more rapid germination of the conidia and more rapid germ tube elongation. The stimulation for these processes is in the form of nutrients in leachates or exudates from the host (Barash et al., 1964; Kosuge and Hewitt, 1964). In strawberries, the presence of pollen grains in the infection droplet reduces considerably the number of conidia needed for infection (Chou and Preece, 1968). For infection of developing apricot fruit, *B. cinerea* must establish itself first on dead floral parts which adhere to the fruit. Styles that fail to dehisce are the primary avenues of infection (Ogawa and English, 1960). This suggests that a saprophytic type of growth is necessary prior to infection to allow *B. cinerea* a chance to produce pectic enzymes, particularly polygalacturonase, which breaks down or alters the host, thus facilitating entry into the ovary or developing fruit.

Other factors that affect *Botrytis* blight are related to the retention of free moisture on plant surfaces. In castorbeans, those cultivars with compact inflorescences and dwarfed internodes are most susceptible (Thomas and Orellana, 1963).

Fire blight, caused by *Erwinia amylovora*, also causes a complete destruction of flowers. While stomatalike openings in the nectaries are considered to be the main avenues of entry, numerous other natural openings on flowers are also involved, including stomata on the styles and sepals (Schroth et al., 1974). In California, Miller and Schroth (1972) found *E. amylovora* existing as an epiphyte in pear flowers and
on other plant parts during the spring. Amazingly enough, during epi-
phytotics, upward of $10^4$ to $10^6$ cells were found in many flowers but only
a low percentage of such flowers actually became infected. Why the in-
fection percentage of "inhabited" flowers is not greater seems to be
somewhat of a mystery, and raises questions regarding the interaction of
inoculum density and host susceptibility. Miller and Schroth (1972)
suggest that "in nature, inoculation may often be effected by a mass, or
large number of cells and not by low dosages which some workers logi-
cally contend should be used when conducting pathogenicity tests in
greenhouse and field studies to duplicate natural processes." Low dosages
of inoculum may therefore not be the norm for bacterial diseases!

An interesting bacterial–environmental interaction involving a blossom
blight has been observed in Connecticut. In years with cold, moist con-
ditions during flowering in pears, many blossoms abort and fail to pro-
duce fruit. The usual explanation was that the abortion was due to a
physiological response of the pear tree to the adverse environment.
However, work by Sands and McIntyre (1977) has shown that the
abortion of blossoms is mainly due to infection by *Pseudomonas syringae*,
since this organism can be isolated from infected blossoms, and fruit
yields are significantly increased by use of antibiotic sprays during the
blossoming period. Such a response would not be observed if the blossom
blight were strictly a physiological response to cold temperatures.

IV. TRANSVESTISM

Webster's New Collegiate Dictionary defines transvestism as "adopting
the dress and behavior of the opposite sex." Diseases may make plants
show behavior of the opposite sex, i.e., induce stamens in the female
flower and ovaries in the male flower. For example, *Ustilago violacea*
attacks members of the Caryophyllaceae which normally are dioecious.
In species of *Lychnis*, this fungus stimulates the production of stamens on
otherwise female flowers (see Fischer and Holton, 1957). In contrast,
this same fungus in *Melandrium album* and *M. dioecum* stimulates the
production of ovaries in the male flowers. Both responses, no doubt, are
due to altered hormonal balance in infected plants.

V. POSTFERTILIZATION EFFECTS

A. Fleshy Fruit Destruction

One of the most disease-prone plant parts is the fleshy fruit of a
number of vegetables and fruits. Latency of infection is involved in some
of these diseases. In summer bunch rot of Thompson seedless grapes in
California, Strobel and Hewitt (1964) found that spores of *Diplodia viticola* germinate on and infect the stigmatic portion of the flower. However, the mycelium in the fertilized, developing grape berry remains latent until the fruit is nearly mature. The latency mechanism is believed to be due to low pH values in the developing grape berry resulting from high levels of tartaric and malic acids. As the fruit matures, these acids are metabolized to sugars, thus raising the pH level enough to allow the pathogen to resume growth and rot the berry. An even more superficial latency of infection has been reported in avocados attacked by *Colletotrichum gloeosporioides* (Binyamini and Schiffman-Nadel, 1972). In this case the fungus forms appressoria within the wax layers on the immature fruit surface. During fruit softening at maturity, the appressoria commence growth again and hyphae penetrate on into the fruit.

The value of a food base for infection is seen in *Botrytis* rot of strawberries (Jarvis, 1962; Powelson, 1960), where infection rarely occurs in the absence of previous saprophytic growth on flower parts. Only under conditions of continuous high humidity will infections of immature fruit develop to the point that the fruit is destroyed. The role of a food base as a necessity for infection can even allow some pathogens to attack fruit which they normally would not infect. The vascular pathogen *Fusarium oxysporum* is capable of causing a pod decay of snap beans when old blossoms become infected and serve as the foci of pod infection (Goth, 1966).

The resistance of immature fruit to invasion by a variety of fungal pathogens seems to be a rather common phenomenon. In oranges, resistance to *Diplodia natalensis* is related to failure of the fungus to penetrate the cuticle and wound the periderm (Brown and Wilson, 1968). Entry can occur at the time of fruit abscission via natural openings that develop in the separation layer between the button (calyx and disc) and the fruit. The resistance of immature papaya fruit to invasion by *Colletotrichum gloeosporioides* is also related to formation of a liquefied periderm which walls off the pathogen (Stanghellini and Aragaki, 1966). In apple fruit, susceptibility occurs at or just before the climactic rise in respiration (Sitterly and Shay, 1960). This increased respiration may allow the accumulation of sugar for use by pathogens as an energy source, suggesting that the lack of such energy sources in immature fruit may be part of the reason for their resistance.

### B. Nonfleshy Fruit or Seed Destruction

In those fruits where the pericarp is united to the seed coat to form a nonfleshy fruit, e.g., the caryopsis of the Graminae, or where no pericarp exists, e.g., the seeds of gymnosperms, pathogens have evolved that can
injure or destroy such organs. However, few direct pathogens of gymnosperm cones or seeds appear to exist; exceptions are the cone rusts which reduce the amount of seed produced but never destroy the entire crop (Boyce, 1961). Head, ear, and kernel rots of various cereals, however, are not uncommon and, because of the significance of cereals in world food supplies, are far from being unimportant. The scab disease of wheat and barley heads, caused by *Gibberella zeae* is particularly important in areas where corn is grown in rotation with these crops and where moisture levels at heading time tend to be high. Susceptibility to invasion of wheat heads is greatest after flowering. Strange and Smith (1971) and Strange *et al.* (1974) showed that massive fungal growth occurs on extruded anthers and the resulting spikelet infections are significantly greater in nonemasculated plants than in emasculated plants. Extracts of anthers stimulate fungal growth due to the presence of choline and betaine.

Kernel and ear infections in maize have received renewed interest because of the apparently high susceptibility of corn lines containing the opaque-2 gene for high lysine. These were developed because of their increased protein and nutritional value (Ullstrup, 1971). There was fear for awhile that this aspect of these new corn lines would greatly detract from their utility, but a report by Loesch *et al.* (1976) suggests that such high lysine types can be developed with good resistance to ear-rotting fungi.

C. Indirect Effects on Fruiting and Fruit Development

Pathogens need not directly invade flowers and/or fruit to have an effect on plant reproduction. The damage done is often measured by decreased numbers of seed or fruits, probably as a result of decreased photosynthesis or decreased water transport, or both, in infected plants. In plants with a determinant type of growth, e.g., wheat, the potential number of flowers that can develop is set early in the life of the plant when the spike is initiated. However, anything which affects the growth and vigor of the plant after the time the spike is differentiated will have an effect on the number of flowers that will be fertile. Early stem rust infections which greatly decrease the photosynthetic capacity of the plant and interrupt its water economy through increased transpiration can effectively sterilize a wheat plant, but later infections have a less detrimental effect on the number of fertile florets and the size of seed that develop. However, a generalization seems to be that in plants with a determinant growth habit, leaf-infecting pathogens that reduce photosynthetic capacity probably have their biggest effect on seed or fruit size rather than on seed or fruit number. This is likely to be less true in plants with an indeterminant growth habit.
D. Effects on Floral Esthetic Qualities

While not being a direct effect on the reproduction of a plant per se, there are diseases which affect reproductive plant parts used for esthetic purposes, i.e., flowers grown as ornamentals. Such effects can range from shortened internodes in gladiolas caused by tomato ringspot virus (Bozarth and Corbett, 1958), to distortions of flower parts caused by chrysanthemum aspermy virus (Hollings, 1955), to partial or complete necrosis of flower petals as caused by Sclerotinia sclerotiorum (Raabe, 1971).

VI. SEED-BORNE PATHOGENS

A. Biological Implications

While in many cases pathogens disrupt reproduction to the point that the reproductive units of a plant are nonfunctional, another situation exists that is important to the host plant, its pathogens, and to man who is dependent on these units for "seed" to begin a new crop. This involves the transmission of pathogens on or within reproductive units, be they seeds, tubers, corms, or seed pieces. As Baker (1972) points out, seed transmission is a method par excellence by which pathogens (1) are introduced into new geographic areas, (2) can survive periods when the host is lacking, (3) are preferentially selected and distributed as host-specific strains, and (4) are distributed throughout the plant population as foci of primary inoculum.

The long-distance dissemination of plants has certainly had a major impact on the agriculture of the world (Crosby, 1972). Most of our major crop plants are now grown in areas where they did not evolve but have been disseminated by man, particularly as a result of what is termed "the Columbian Exchange" beginning in 1492. An important aspect of this exchange was the dissemination of plant pathogens between both the Old and the New World. A recent problem may be the dissemination of seed-borne pathogens occurring as a result of the massive movements of seed, particularly wheat and rice seed, as a part of the "Green Revolution." Quarantines and pesticide seed treatments may help eliminate the problems of pathogens borne on the outside of the seed, but they rarely have any effect on internally borne pathogens, particularly the viruses.

The survival of pathogens within seed is particularly important to those that are obligate parasites. For many viruses, seed transmission may be the single (or only) important source of virus carryover from one growing season to the next (Shepherd, 1972). This is exemplified by the case with lettuce mosaic virus in lettuce whereby the use of seed lots with
no infected plants in 30,000 seeds assayed can effectively keep the crop nearly virus-free throughout the growing season (Baker, 1972).

The mechanisms by which pathogens are transferred to the seed from the mother plant are varied and include: (1) the pathogen is a passive contaminant on the exterior of the seed, (2) the pathogen spreads into the seed from the fruit, (3) the pathogen enters the seed via vascular connections, (4) the pathogen enters the embryo via infected pollen or ovarian tissue, and (5) the pathogen actively and directly penetrates the seed coat (Baker and Smith, 1966).

B. Viral Seed-Borne Diseases

1. Importance and Frequency

It is often assumed that relatively few virus diseases are seed-borne. However, Shepherd (1972) points out that at least 49 different viruses are seed-borne in at least one of their hosts. In some groups of viruses, e.g., the nematode-transmitted viruses, seed transmission is the rule rather than the exception. The degree or extent of seed transmission is highly variable and is affected by host cultivar, virus strain, stage at which the mother plant is infected, temperature after infection, and perhaps other factors. The types of viruses that are often seed transmitted include those that are readily juice transmitted, thus indicating their ability to invade parenchyma, while phloem-inhabiting, leaf-hopper vectored, and persistent-aphid vectored viruses generally are not seed transmitted (Bennett, 1969).

The importance of seed transmission in the ecology of certain viruses is illustrated by Murant and Lister's work (1967). For several of the nematode-transmitted viruses of raspberries, the persistence of such viruses through periods of fallow or fasting of the vector was found to depend on a continuing supply of infected seedlings produced by virus-containing weed seeds. Tomlinson and Carter (1970) have also documented the importance of weed seed transmission of cucumber mosaic virus in providing new sources of inoculum.

2. Mechanisms of Transmission

For many viruses, including some for which seed transmission has been reported, the seed offers a highly effective barrier to the passage of viruses from one plant generation to the next. Thus, propagation of plants through seed has therapeutic advantages to a plant species helping to ensure its survival. In view of this, the problem becomes one of determining the mechanisms by which some viruses do become seed-borne
and the factors that prevent others from being propagated in this way. Many hypotheses have been presented suggesting reasons why seed transmission is somewhat rare including: (1) the presence of inhibitory materials in embryonic tissues, (2) the lack of plasmodesmatal connections between embryo and mother plant tissue, (3) the adverse effects of virus on meiotic divisions, and (4) phosphate starvation in rapidly dividing tissues which restrains or prevents virus multiplication.

a. Seed Coat Transmission. It appears that very few viruses are transmitted to new plants from inoculum existing only on the seed coat. The major exception is tobacco mosaic virus (TMV) on tomato seeds. Even in this case, if extreme care is taken to prevent wounding of emerging seedlings, little or no transmission will occur (Taylor et al., 1961; Broadbent, 1965). In other cases, e.g., pea streak virus in peas, the virus may be transmitted only by immature seed but, because of its association only with the seed coat, it is inactivated as the seed matures (Ford, 1966).

b. Pollen Transmission. That infected pollen can serve to transmit viruses to the embryo, and hence to the seed, has been shown for a variety of viruses including grapevine fanleaf virus, cherry necrotic ring-spot virus, bean virus one, alfalfa mosaic virus in alfalfa, and barley stripe mosaic virus (BSMV). Early studies to detect pollen transmissibility were carried out using pollen from infected plants to fertilize pistils on healthy plants. The actual mechanism of pollen transmission as observed visually is well known for BSMV. This virus is able to invade the male floral meristem with subsequent invasion of the pollen mother cells (Carroll and Mayhew, 1976a), followed by invasion of the sperm. The cell-to-cell transfer of the virus is believed to occur via microtubule transfer at the time of cell division (Mayhew and Carroll, 1974b) (Figs. 1-3). Carroll (1974) proposed two explanations for the ultimate transmission of BSMV from pollen to embryo. First, the virus could be carried by the sperm cell which infects the egg cell and thus the zygote and embryo, or second, the virus could be carried in the cytoplasm of the vegetative cell as a "contaminant" which the sperm would pass on to the egg cell. Because of the visualization of virions inside of sperm nuclei, Carroll favors the first explanation (Fig. 2). Yang and Hamilton (1974) have also studied the seed transmissibility of tobacco ringspot virus in soybeans. Virus aggregates were found consistently in the intine of pollen grains, and in the cell wall and cytoplasm of the generative cell, but not in the cytoplasm of vegetative cells nor in nuclei of vegetative or generative cells. The lack of virus in such areas probably explains why they were unable to show pollen transfer of this virus.
c. Embryonic Transmission. Transmission in the embryo implies that the embryo must become infected via one or more of three possible mechanisms: (1) through the zygote via infected sperms—a situation described above for BSMV, (2) through the zygote via infected eggs, or (3) by direct invasion of the embryo from the mother plant following embryo initiation. The first two possibilities are believed to account for the majority of cases of embryonic transmission. For BSMV in barley, early in the development of the female primary floral meristem virions can be seen in the ovular tissue often associated with microtubules (see Fig. 3) (Carroll, 1969; Carroll and Mayhew, 1976b; Mayhew and Carroll, 1974a,b). Virions are also observed in the megaspore mother cell, megaspores, and then in the embryo sac including the egg cell. These results provide strong support for Bennett's (1969) suggestion that those viruses capable of being embryonically transmitted in the seed are those which can invade the primary meristem of the host early in its develop-
Fig. 2. Portion of BSMV-infected sperm cell. Note virions (arrows) of BSMV in the nucleus (N) and cytoplasm (C) of the sperm cell. Also shown are the nuclear envelope (E) and the boundary (B) of the sperm cell and the cytoplasm of the vegetative cell (VC). The bar represents 0.5 μm. (Reproduced by permission of Academic Press from Carroll, 1974.)

ment before the embryo is effectively “walled-off” from the mother plant. Because spherical viruses are morphologically similar to ribosomes, they are difficult to detect in the tissue with the electron microscope. However, Yang and Hamilton (1974), in a study of the seed transmission of tobacco ringspot virus in soybeans, were able to detect virulike particles aggregated in the sieve tubes in integuments as well as in the nucleus, embryo sac wall, and megagametophytic cells.

Whether viruses can directly invade embryos from the mother plant after the embryo has been initiated is unclear. Eslick and Afanasiev (1955) reported that a low level of seed transmission of BSMV did occur in barley inoculated long after flowering, but others could not confirm this. Schippers (1963) also reported a lack of seed transmission of bean common mosaic in beans when plants were inoculated after flowering.
Fig. 3. Integument and nucellar cells of the ovule infected by the MI-1 isolate of barley stripe mosaic virus. Portions of two cells within the outer integument (OIC). Many virions are associated with wall microtubules (arrows). No virions are evident within plasmodesmata (P). Portions of two cells of the inner integument (IIC) are also visible. Premieiotic stage. × 23,500. (Reproduced by permission of the National Research Council of Canada from Carroll and Mayhew, 1976b).

Evidently the phenomenon of direct invasion is rare, probably because of either a barrier of disintegrating nucellar tissue around the developing embryo or the lack of plasmodesmatal or vascular connection between the mother plant and the embryo.

C. Bacterial Seed-Borne Diseases

1. Importance

The importance of seed transmission in the epidemiology of several bacterial bean and pea diseases has been a major factor in the continuing practice of producing seed in the dry irrigated areas of the western United States (Baker, 1972). In humid areas, as few as 12 infected bean
seeds per acre, or 0.02% infection, can provide enough primary inoculum for the development of severe epidemics of *Pseudomonas phaseolicola*.

2. **Mechanisms of Transmission**

While seed transmission of bacterial pathogens can result from inoculum in the embryo, endosperm, or seed coat, or from contamination of the seed coat surface, it seems unlikely that embryo infections are very important. If embryos were infected, the seed would probably be non-viable. Internal infections are known, however, particularly for those diseases where the bacterium is a systemic vascular inhabitant. Zaumeyer (1932) reports that *P. phaseoli* enters bean seeds via vascular elements or the micropyle. In the latter case, the bacteria break out from systematically invaded funicular tissue or from vessel elements along the dorsal suture of the pod. The bacteria are then harbored just beneath the seed coat.

The role of the “seed contamination” in seed transmission is illustrated by the studies of Grogan and Kimble (1967) and Guthrie (1970). Development of *P. phaseolicola* in bean fields sown with western-grown seed suggested to them that such seed might be carrying low levels of inoculum. They showed that seed contamination can occur even though seed field inspections fail to detect the disease. This contamination was found to occur in small natural openings on the seed coat, including the hilum and micropyle. Similar types of contamination have also been observed for a variety of other bacterial pathogens including *Xanthomonas malvacearum*, *X. campestris*, and *X. oryzae*.

**D. Fungal Seed-Borne Diseases**

A wide variety of fungal pathogens are known to be seed-borne, carried either as infectious mycelium internally or as contaminants on the seed coat. The methods by which seeds become infected or contaminated include: (1) floral infection whereby the embryo becomes infected but remains viable, e.g., loose smut of cereals; (2) infection of the seed coat via vascular connections, e.g., Verticillium wilt of safflower; and (3) contamination with propagules produced on other plant parts, particularly leaves, e.g., anthracnose of beans.

1. **Mechanisms of Transmission**

With few exceptions, the loose smut fungi are unique in their ability to invade floral tissues and become established in the embryo without destroying it. Infection appears to occur directly through any part of the ovary wall (Batts, 1955) after which the mycelium becomes prevalent
in the scutellar portion of the embryo. Mycelium is rarely found in the endosperm. As the seed matures, the mycelium becomes dormant and further development is restricted until the seed imbibes water and germinates. Infection can take place prior to or several days after pollination as long as the floret is open to allow entry of the teliospore inoculum. With the development of hybrid wheat and barley and the use of male sterile female plants to produce such seed, this pathogen may become more serious than it has been in the past because the flower of the male sterile female must remain open to allow for cross-pollination. This allows for a longer exposure to loose smut inoculum.

While seed-borne vascular pathogens are not very common, in those cases where there is a vascular connection into the seed coat infection has occurred. In safflower (*Carthamus tinctorius*), Klisiewicz (1963, 1975) has found *Verticillium dahliae* and *Fusarium oxysporum f. carthami* capable of infecting seed. In the latter case, 42% of the seed harvested from infected seed heads carried the fungus. Mycelium and spores were evident on the seed surface while hyphae were observed in the pericarp in association with xylem and sclerenchyma tissue. No embryo infections were observed.

In those plants where seeds are produced within a protective fruit wall, e.g., beans, fungal infections of the seed can occur by direct invasion of the seed coat from mycelium in the fruit or pods. Ellis *et al.* (1976), in a study of dry bean pod invasions in South America, determined that seeds become infected by a variety of fungi only in pods in contact with soil. Because of this source of inoculum, they recommended that growers in Central and South America hand pick pods not in contact with soil and save the seeds therein for the next growing season. Seed selected in this manner was found to have an emergence potential of 97% versus 52% for seeds from pods in contact with the soil.

**VII. PATHOGENIC EFFECTS ON SEED QUALITY**

The farmer must have seed that can germinate and develop into a healthy seedling. When seed is infected with a pathogen, the seed may be rendered nonviable or it may remain viable but produce a weak seedling. In some cases, the infected seedling may not be severely weakened, but may serve as a source of primary inoculum within a community of plants.

The extreme case where the seed is rendered nonviable occurs with those pathogens which are vigorous direct invaders of plant tissue, as illustrated by *Whetzelinia sclerotiorum* on beans (Steadman, 1975). In
Illinois, the problem in viability of soybean seed lots is correlated with environmental conditions in seed production fields (Tenne et al., 1974). Seed from northern Illinois show consistently higher germination than seed from the southern part of the state. Where temperature, rainfall, and humidity are more conducive to pod infection by a variety of fungi and bacteria, there is good correlation between seed infection and lowered viability.

Corn seed pathogens are also capable of reducing seed viability. One of relatively recent importance, *Colletotrichum graminicola*, has been reported to reduce germination from 90% for healthy seed, to 37% for kernels with a few infection sites, to 6% for kernels with extensively damaged kernels (Warren and Nicholson, 1975).

### References


12. DISRUPTED REPRODUCTION


