

White Mold - A Serious

One of the most destructive diseases of beans in temperate regions is white mold (Fig. 1). Lima beans in California, navy and pinto beans in Michigan and Canada, pink beans in Washington, pinto beans in Colorado and North Dakota, green beans in Florida and New York, and great northern and pinto beans in Nebraska all suffer losses due to white mold disease. The fungus causing the disease is *Sclerotinia sclerotiorum* (Lib.) de Bary. This fungus and the closely related species *S. minor* and *S. trifoliorum* are among the world's most omnivorous and successful plant pathogens. A recent list (6) shows that 361 species of plants in 64 families have been reported as hosts of *S. sclerotiorum*. *S. sclerotiorum* does the most damage to vegetables and oilseed species; *S. minor*, to peanuts and lettuce; and *S. trifoliorum*, to forage legumes. Controversy exists as to whether the three *Sclerotinia* species are truly distinct taxonomic species and not variants of one species, *S. sclerotiorum*. Recent studies by Kohn (5) and Willetts and Wong (9), however, give strong support to the existence of three separate species and define *S. sclerotiorum* as a cosmopolitan ascomycete that produces large (2–20 mm long) sclerotia and lacks functional conidia or other asexual spores.

Life Cycle of the Fungus

The sclerotia are the perpetuating structures of *S. sclerotiorum* (Fig. 2). They may survive for 5 or more years in soil, assuring pathogen availability when a host crop is planted. These survival structures become distributed throughout the tilled levels of soil. Although some

sclerotia are destroyed by other organisms, a substantial number remain viable near the soil surface each year regardless of the host crop being grown.

In white mold disease, sclerotia germination to ultimately produce ascospores (carpogenic germination) is the key event needed for infection to occur. The soil conditions that promote carpogenic germination are not well understood, however. Sclerotia produced on an infected plant will not germinate to form apothecia until they have been "preconditioned." This preconditioning, or physiological maturation, occurs during the winter or noncropped season; freezing is not necessary, as evidenced by white mold's distribution in southern Florida and California. Sustained adequate moisture and cool temperatures (4–20 C) trigger the conversion of a dormant sclerotium into one that produces the sexual stage within a few weeks.

Sclerotia must be at or within 5 cm of the soil surface for apothecia production to occur (Fig. 3). When preconditioned sclerotia begin to germinate, stipes, or apothecial stalks, are formed. These may be formed in the light or under the soil without light, but light is necessary to stimulate formation of ascospore-containing disks at the end of the stipes. Since stipes are seldom longer than 5 cm, only sclerotia located within 5 cm of the soil surface complete spore production. Apothecia observed in the field are usually on the soil surface and are seldom raised above the surface by the stipes. This position on the soil and often under a plant is not the most advantageous for spore dispersal. When ascospores ripen or mature, however, a large number (10,000–30,000) mature simultaneously. Thus, when a sudden change in relative humidity triggers forcible discharge, many ascospores are released simultaneously, causing a "puffing" phenomenon that creates turbulence and assists aerial dispersal. Ascospores do escape above the canopy and have been detected on leaves 50–100 m from the source and in aerial samples collected above cropped fields. The importance of aerial dissemina-

tion in epidemics, however, has not been demonstrated.

Sclerotia may be conditioned anytime from fall harvest to bean flowering the next year, but in semiarid regions, carpogenic germination is usually initiated after the plant canopy has covered the soil surface. The requisite canopy development occurs in beans near the end of the first bloom and is responsible for a number of important micrometeorological events. In unshaded areas, soil temperatures near the surface could be above 30 C during daytime, and without frequent moisture from rain or irrigation, soil moisture would readily fall below field capacity. Neither the temperature nor the moisture situation would favor apothecia formation. Under the plant canopy, however, temperatures are seldom above 25 C and soil remains wetter between rains or irrigations. Optimum apothecia production occurs in 10–14 days at a soil matric potential (ψ) of -0.25 bars (20% soil moisture in Tripp fine sandy loam soil) at a soil temperature of 15–18 C. In addition, the canopy tends to trap a large percentage of the more than 2 million spores produced by each apothecium during its 5–10 day functional life. While somewhat limiting the potential for long-range spore dispersal, this tends to saturate available infection sites and promotes a high local infection potential.

The presence of white mold in most major bean production areas is evidence of its dissemination success. Honeybees efficiently distribute spores to the site of initial infection (*unpublished data*). Although functional aerial ascospore dispersal is somewhat limited in irrigated semiarid regions, spores as well as colonized plant debris and sclerotia can travel within and between bean-growing areas in water moving through irrigation canals. In high rainfall areas such as New York, apothecia production in orchards or semiopen areas would be more conducive to medium- or long-range aerial dispersal. The introduction of sclerotia with seed or more rarely as infected seed probably plays a minor role in dissemination. The most important

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Yield-Limiting Disease of Bean

dispersal factor, however, is the long-term survival of sclerotia associated with such a wide range of hosts.

Outside semiarid regions, sclerotia germination may precede crop planting. In New York, apothecia are formed in early spring just after snowmelt and long before beans are planted. Similarly, in California and some southern locations, apothecia are produced during the late winter and early spring months (January, February, and March), ie, before beans have been planted. In fact, beans and other crop hosts of *Sclerotinia* may be only incidental in its ecology; it can and often does survive on various weeds if crops are not available.

S. sclerotiorum is somewhat unique as a pathogen in that it requires an exogenous energy source for the ascospores to infect healthy or green plant leaves, pods, or stems. Senescent or injured organs on the plant or on the soil beneath the plant can provide the necessary exogenous energy. On beans, the most frequent source is the flower. After colonization of the flower, the fungal mycelium can infect adjacent

green pods (Fig. 4), leaves, or stems within 2 or 3 days. If ascospores are discharged before flowers or other

senescent tissue is available, the spores can survive on plant surfaces or on the soil surface for nearly 2 weeks. Once a

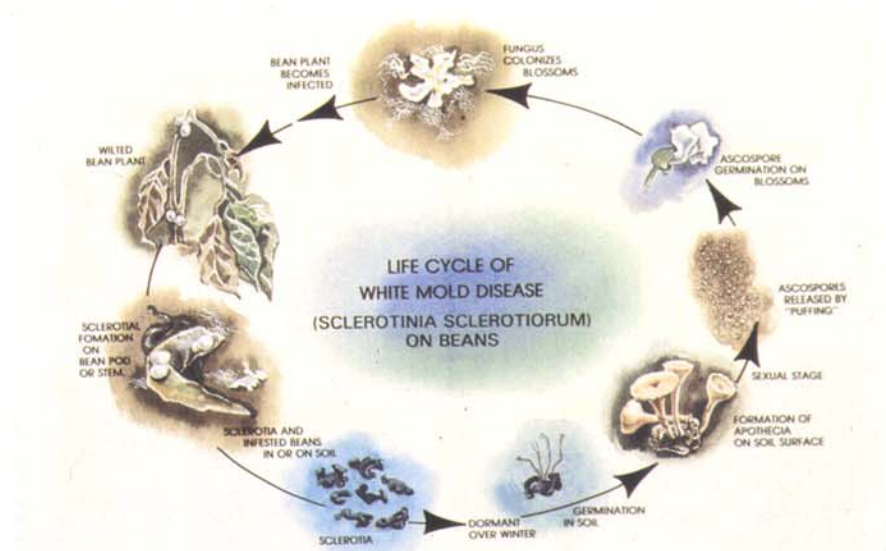


Fig. 2. Life cycle of *Sclerotinia sclerotiorum*.



Fig. 1. White mold disease on great northern bean.



Fig. 3. Apothecia of *Sclerotinia sclerotiorum* produced near soil surface.



Fig. 4. Bean blossom colonized by *Sclerotinia sclerotiorum* in contact with pod.

blossom is colonized, the mycelium remains viable for more than a month.

Mycelium from colonized senescent tissue has the capacity to initiate infection, but mycelium from a sclerotium is unlikely to infect directly. The food reserve in a sclerotium apparently does not supply the energy necessary for formation of the infection cushions or appressoria and for subsequent entry into the host. Thus, even if sclerotia germinate to produce vegetative hyphal strands, it is much less likely that one or two sclerotia would be closer to senescent tissue than 1 or 2 million spores. Also, since sclerotia are soilborne, they cannot be involved directly in initiating infection in the aboveground plant canopy.

The plant canopy influence is as great on spore germination, mycelial colonization, and subsequent infection as it is on sclerotia germination. The ambient air temperature commonly reaches 40–45 C in semiarid regions in midsummer. The plant canopy temperature 10 cm above the ground, however, is only 25 C or less. Similarly, the dew point will be exceeded longer in the plant canopy than outside the canopy. Temperatures less than 30 C (the threshold temperature above which ascospore germination ceases) and plant surface moisture for 12–16 hours recurring on a daily basis or continuous surface wetness for 42–72 hours are prerequisites for white mold development. The maintenance of greater soil moisture, lower daytime temperature, and lack of air movement that facilitates boundary layer exchange contribute to plant canopy microclimate differences.

The disease first appears as wilted leaves scattered in a field. When the vines are observed more closely, soft, watery spots on leaves, pods, or stems can be seen. Each lesion enlarges to become a rotted, watery piece of tissue covered with the white mycelial signs of the fungus.

When stems or branches are attacked, wilting occurs, and branches eventually die and take on a dry, bleached appearance. The sclerotia form in and on affected plant parts. The bleached stem symptom and sclerotia formation are diagnostic of white mold (Fig. 5). Normal senescence or stress from drought or other bean diseases causes the plant to turn yellow to tan as it dries and, of course, does not show the associated sclerotia.

Secondary infection results from green tissue coming in contact with an infected area, but no secondary infection propagules are produced. Severe initial infections tend to be self-limiting in that the dying plant no longer has the canopy to provide the necessary microclimate for further infection. Continuous germination of sclerotia and continuous spore discharge from each apothecium ensure adequate infection potential over a 3–4 week period. The limited hyphal growth emanating from newly produced sclerotia does not appear to play a role in secondary infection.

Control Measures

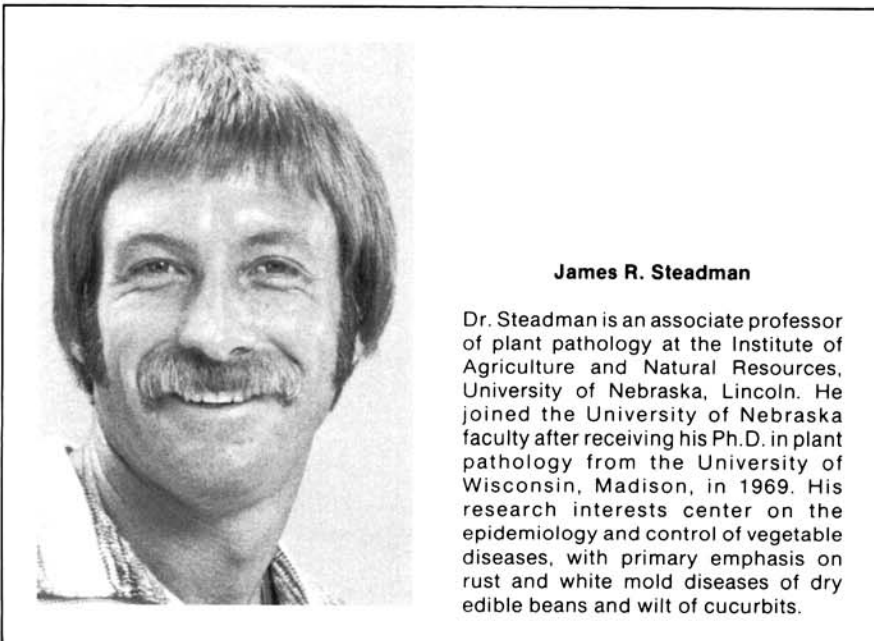
Fungicides. The importance of blossom infection and of a favorable microclimate provides keys for controlling the disease.

Fungicidal protection of bean blossoms has met with some success, more so on snap beans than on dry beans. Studies in New York (4) demonstrated that spraying the whole plant or only bean blossoms with benomyl resulted in effective control when plants were subsequently inoculated with ascospores. Conversely, no control was observed when all aboveground plant parts except blossoms were covered with benomyl. Erratic control by benomyl could result, however, from inadequate blossom coverage because of indeterminate flowering and growth habit and/or location and frequency of

inoculum production. Snap beans have a determinate growth and flowering habit and need protective fungicide coverage for about 2 weeks after flowering. Most dry edible beans are indeterminate and produce blossoms for at least 4 weeks after initial flowering. In Nebraska, when two applications of benomyl were made to dry edible beans at first bloom and 7 days later, just before canopy coverage precluded further ground applications, residues were detected on or in blossoms for only 2 weeks after the last spray. Residue results were similar after a single application when all plants had at least one open blossom (Fig. 6). Apothecia were found both within (an average of 10–34/m²) and outside (an average of 5–15/m²) irrigated bean fields from 2 weeks after first bloom to near harvest. Thus, erratic control in Nebraska compared with consistent control in New York (when fungicide treatment timing is correct) may reflect the difficulty in protecting indeterminate bean cultivars that produce flowers (potential infection sites) until maturity and during a period when inoculum production is intense. In addition, when disease severity is high (60% or more), a single fungicide application is not effective.

Lack of fungicide control has been observed in viny bean types in California, Washington, and Colorado as well as in Nebraska. In all instances, aerial application has been the method of fungicide treatment. Benomyl residue data from Nebraska indicate that aerial application can be as effective as ground application in covering the first set of blossoms. Improper timing and inefficient application procedures often result in poor control, however. In Idaho, sprinkler application of benomyl has been shown to give effective control of white mold. In Florida, aerial application of benomyl combined with an earlier ground spray and an in-furrow treatment at planting gave excellent control of white mold on the upright open canopy of pole beans. A bean plant that is less viny and has a more open canopy would be easier to protect with a fungicide. Where chemical control has been effective, blossom coverage probably was timely.

In most crops, one application of a fungicide such as benomyl, 2,6-dichloro-4-nitroaniline (DCNA), or thiophanate-methyl can be economical if disease reduction is satisfactory. For example, in Nebraska bean fields, losses due to *S. sclerotiorum* averaged 13% over 4 years. This would result in a \$200/ha loss at the present price of beans and would be slightly more than twice the estimated cost of a fungicide application. Thus, only a single aerial application would be cost-effective. In New York, chemical control may be critical, because in addition to direct losses in the field, detection of more than 2% snap bean pod infection can result in rejection of the entire truckload at the processing plant.



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In all cases, chemical applications must precede the onset of disease, and if epidemics could be predicted, the expense of routine fungicide applications could be eliminated or reduced. In one attempt at forecasting, numbers of sclerotia (inoculum potential) in bean fields were not correlated with epidemic potential. Hunter (3) recently developed a forecast system for New York snap bean growers based on soil moisture and plant canopy size to enable more selective applications of fungicides. More work on forecasting in semiarid irrigated areas is needed.

Culture modifications. Crop rotation is a common disease control recommendation and often has been advocated for control of *Sclerotinia* diseases. Sclerotia survive in the soil at least 3 years, however, and tillage operations generally assure the presence of sclerotia at or near the soil surface. In Nebraska, sclerotia populations were comparable in all field samples in various corn, sugar beet, bean crop rotations despite differences in occurrence of the bean host in the previous crop history. In addition, apothecia were found in fields of nonhost crops. This would assure that airborne or irrigation-waterborne ascospore dissemination could occur. Deep plowing also has been recommended for control of white mold of bean, but plowing to a depth of 25 cm did not affect disease severity in Nebraska and thus may not be a valid general recommendation.

Reduction in the number of irrigations, especially those at the end of the season, can lower disease incidence in the absence of rainfall, but reducing irrigation often results in decreased yield of dry edible beans. Thus, the final irrigation should not be eliminated unless the disease is present in the field or disease potential is great. Studies conducted in Nebraska over the past 3 years on irrigation frequency and white mold disease development showed that both apothecia production and disease severity were reduced by decreasing irrigation frequency. Yield increases at the lower water rates were correlated with lower disease severity. Watering plants thoroughly until a continuous canopy forms, then reducing irrigation amount and frequency later in the season will result in less white mold and a stable yield. Not reusing surface irrigation runoff water could reduce the chances of spreading sclerotia, mycelia, or ascospores from one field to another, but recent Environmental Protection Agency regulations require reuse of irrigation runoff water. Research on the treatment of reuse water to eliminate plant-pathogenic microorganism contamination indicates that when combined with filtration and sedimentation, chlorine (as hypochlorite) can eliminate ascospores (8). Sclerotia are not killed, however, and chlorine treatment would have limited success in

reducing *S. sclerotiorum* dissemination in water. Application of contaminated reuse water to nonhost crops is the best way to minimize pathogen dissemination in irrigation water.

Disease avoidance and/or resistance. The common bean (*Phaseolus vulgaris*) has been shown to be susceptible, or semiresistant at best, to *S. sclerotiorum* in inoculated greenhouse or controlled environment tests. Only in a related species, the scarlet runner bean (*P. coccineus*), has a stronger type of resistance to *S. sclerotiorum* been demonstrated in these tests. In Nebraska field tests under natural infection conditions, a few *P. vulgaris* cultivars

have consistently shown less white mold disease than the majority of pinto, great northern, small white, or other bean types. Whether the reduced disease severity is due to the host plant actively resisting the pathogen infection process or to disease avoidance resulting from canopy architecture has not been established in all instances.

An association between plant canopy development and *Sclerotinia* disease incidence and severity has been observed (Fig. 7). Vigorous, viny cultivars produce the densest canopy and, when irrigated heavily, are the coolest and wettest and have the highest disease severity. Canopy structure and, more specifically, distribu-



Fig. 5. Pinto bean infected with *Sclerotinia sclerotiorum* showing characteristic bleached stem (center) and sclerotia formation. (Courtesy H. F. Schwartz)



Fig. 7. (Left) Closed canopy of vigorous, viny great northern bean cultivar Tara resulted in 60% white mold severity. (Right) Open (between-row) canopy of semiviny small white bean cultivar Aurora resulted in 4% white mold severity.



tion of leaf area near the ground in terms of leaf area \times dry weight/height affect white mold incidence and severity (7). The growth habit, ie, determinate or indeterminate, does not exclusively influence infection. Upright indeterminate and open bush types both can result in reduced white mold severity compared with that found in dense, compact bush or vine types.

A number of institutions and companies are attempting to incorporate resistance to *S. sclerotiorum* into snap beans (2), dry beans (1), and other types. The combination of physiological resistance and canopy architectural avoidance

theoretically is the best approach to improved, high-yielding, white-mold-resistant cultivars. Unfortunately, high yields and high white mold disease severity are both correlated with vigorous, viny plant types, and the identification of *S. sclerotiorum* resistance has been slow and difficult. In the past, improved navy cultivars of a bush or short vine type were selected principally because of less white mold than the older viny cultivars. Yields in nondisease years were not higher, however. Similarly, where bush kidney cultivars were substituted for pinto cultivars, white mold disease was less and yields were

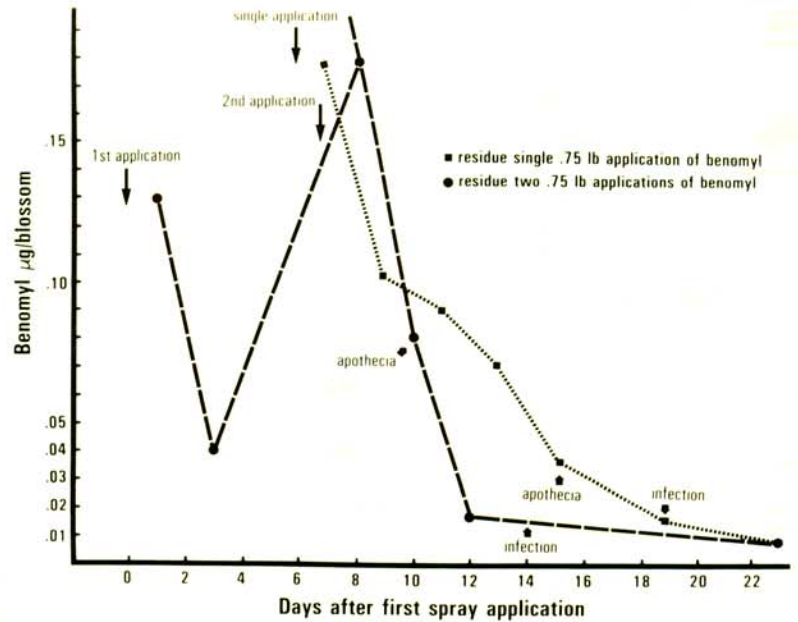


Fig. 6. Quantities of benomyl detected by bioassay in great northern bean blossoms until 23 days after single or double spray applications.

lower. Unless we gain a better understanding of the relationship of canopy architecture and microclimate and/or are able to use physiologic resistance to *S. sclerotiorum*, yield potential of bean cultivars may have to be compromised to minimize white mold losses.

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