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An integrated control of white mold (*Sclerotinia sclerotiorum*) of beans, with emphasis on recent advances in biological control

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Abstract. This article states the importance of white mold disease in bean production worldwide; briefly discusses the etiology and epidemiology of the fungus and the disease; and reviews an integrated control measure that includes a) site selection, b) no-till and rotation, c) seed treatment, d) resistant cultivar, e) plant type, f) row width and plant density, g) chemical control and h) biological control for which various bioagents are summarized and the recent advances in two high profile bioagents *Gliocladium virens* and *Coniothyrium minitans* are discussed further.

Keywords: Bean; Biological control; Integrated control; *Sclerotinaia sclerotiorum*; White mold.

Contents

Introduction

White mold, also known as sclerotinia rot and sclerotinia wilt, is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary. This fungus attacks a wide range of hosts and has a worldwide distribution on numerous field crops and vegetables (Purdy, 1979). White mold frequently causes serious and unpredictable yield losses in beans (dry and green) (*Phaseolus vulgaris* L.). In Ontario, a recent study placed the average incidence of white mold on bean crops at about 25% (Tu, 1986). The incidence of disease in different fields ranged from a trace to 100% (Tu, 1989a). Differences in incidence among fields were attributable to variations in precipitation, soil drainage, cultural practices, and sclerotial density in different localities.

Etiology and Epidemiology

This disease and its causal organisms have been subjects of intensive investigation. The etiology, biology, and epidemiology of white mold in beans have been studied extensively and summarized in several reviews and symposia (Abawi and Grogan, 1979; Adams and Ayers, 1979; Kohn, 1979; Le Tourneau, 1979; Lumsden, 1979; Phillips, 1987; Purdy, 1979; Roberts et al., 1982). Briefly, primary infections are initiated chiefly by ascospores, which germinate with the help of free moisture and exogenous energy sources of fallen flower petals and necrotic tissues (Steadman, 1979). Other means of primary infection are associated with ascospore contaminated leaves in contact with moist soil and leaves coming in contact with sclerotia

on the soil surface (Tu, 1989b). Secondary infections are achieved by natural contact of healthy plant parts with diseased ones. Later, many black resting bodies (sclerotia) of the fungus are formed on or in the infected stem tissues.

The sclerotia eventually fall from the diseased plants or are incorporated into the soil with the infected plant debris, where they can survive for several years. Each year, sclerotia that are shallowly buried at a depth of less than 3 cm will germinate to produce apothecia, each containing millions of ascospores.

The epidemiology of white mold is dependent on several factors: (a) soil inoculum, (b) soil moisture, (c) rainfall, (d) cultivar susceptibility, (e) row width, and (f) plant density. Fields with high inoculum density and high soil moisture have high disease incidence because disease initiation was favoured by cool and damp soil conditions. Secondary spread occurs through plant-to-plant contact. The ratio of within-row spread to between-row spread was approximately 6, 4, 3, and 1.5 for row widths of 80, 60, 40, and 20 cm, respectively. Disease incidence increased with reduction in row width and increase in plant density. Cultivar susceptibility was an important factor in disease epidemiology. In a susceptible cultivar (Fleetwood), the percentage of infected plants increased from 0 to 100% in 4 weeks in 80 cm row planting while in a tolerant cultivar (ExRico 23) it progressed from 0 to 35%. The frequency of rainfall was a more important factor in disease epidemiology than total rainfall (Tu, 1987).

Integrated Control

There are no simple ways of achieving total control of this disease. However, a well executed integrated control can reduce the disease significantly and keep the yield loss to a minimum. The integrated control measure includes the following items:

1) Site Selection

The degree of field infestation by *S. sclerotiorum* varies greatly. Such variation contributes in part to the difference in disease incidence in the fields ranging from 0 to 85% (Tu, 1986). Farmers should know their fields. Fields with a previous history of severe white mold should be planted with resistant crops.

2) No-Till and Rotation

As mentioned previously, most of the sclerotia near the top 2–3 cm of soil deteriorate within a year (Davis, 1925; Tu, 1986), and sclerotia buried deeper in the soil have a higher rate of survival. Should it become necessary to plant beans in a field with a history of white mold, a combination of proper crop rotation and no-till operation can reduce the risk.

3) Seed Treatment

Sclerotinia sclerotiorum are able to survive in infected seeds as dormant mycelia in testae and cotyledons for 3 years or longer (Tu, 1988). When infected seeds in a recent study were sown, 88–100% failed to germinate. Seedlings from infected seeds subsequently died from white mold at an early stage. Seeds that failed to germinate were rotted by *S. sclerotiorum*, and three to six sclerotia were formed in place of each seed (Tu, 1988). These sclerotia could become a source of inoculum. Captan and thiophanate-methyl used in seed treatment were 100% effective in eradicating the fungus from the infected seeds (Tu, 1989a).

4) Resistant Cultivar

Sclerotinia sclerotiorum has a wide host range without known strain specificity in pathogenicity (Steadman, 1979). Many researchers formerly believed that resistance to *S. sclerotiorum* did not exist. More recently, however, field resistance to this fungus was observed in several crops (Steadman, 1979). In beans, some Great Northern and Black Turtle Soup varieties were reported to have intrinsic disease resistance that appeared to be quantitatively inherited (Roberts et al., 1982). Unfortunately, because commercial white beans have many specific traits that must be maintained, attempts to use these materials in breeding commercial white bean cultivars for white mold resistance have met with limited success. More recently, tolerance to white mold was discovered in the white bean ExRico 23 in Ontario (Tu and Beversdorf, 1982). The mechanism of resistance appears to be associated with its tolerance to oxalic acid secreted by the white mold fungus (Tu, 1985). ExRico 23 was registered for commercial planting in Ontario and has since gained worldwide acceptance as a cultivar and as a main source for genetical resistance in white bean breeding.

5) Plant Type

A positive correlation between plant canopy and white mold incidence and severity has been established (Steadman, 1979). In general, upright types of plants have less severe white mold than bush (or viny) types (Saindon and Huang, 1992). Compact bush types have more mold than open bush types. Bean plants with indeterminate growth habits often have more severe white mold disease than those with determinate ones (Coyne et al., 1974; Schwartz et al., 1978). Plants that have a large and dense canopy with leaves close to the soil maintain a wet microclimate longer than plants that have a small and thin canopy with upright characteristics. Consequently, the former type of plants have a higher incidence of and more severe white mold than the latter. To avoid white mold, selection of plant types with upright characteristics and thin canopy that allow adequate penetration of sunlight and under-the-canopy aeration is important.

6) Row Width and Plant Density

The effects of row spacing and plant density of bean plants in relation to white mold incidence and severity have been reported (Coyne et al., 1974; Steadman, 1979). Narrow row and high plant density reduce air circulation and trap moisture in the canopy. Thus, they contribute to higher incidence and more severe white mold than conventional

row width and reduced plant density. Narrow row and high plant density not only increase early senescence but also increase contact of plant parts. Increase in senescence and in contact of plant parts promote infection and spread of white mold, respectively.

Air circulation between rows of beans can be improved by planting the rows parallel to the prevailing winds, by reducing the seeding rate, and by practising stringent weed control. However, reducing the seeding rate often reduces bean yield as well.

7) Chemical Control

If the disease is developed, fungicide sprays can provide effective control. Proper timing of spray and method of application have a great impact on results. For example, aerial application by aircraft is relatively ineffective as compared with ground application by boom sprayer. The latter gives better penetration of the spray into the crop canopy.

Several registered chemicals, such as benomyl, chlorothalonil, thiophanate methyl, iprodione and dicloran are available. They are effective but costly.

8) Biological Control

Recently, scientists have researched various areas of biological control in an attempt to control this disease while reducing chemical contamination of the environment. Biocontrol measures using microorganisms against the white mold fungus have been researched extensively. Many of these results have yet to be put into practical application at present because bioagents are subjected to strict registration guidelines. Registered bioagents for disease control are few and far between. Nevertheless, some scientific advances have been made in this area which will be discussed in the next section.

Advances in Biocontrol Research

Since no single method can effectively control white mold disease, the best approach to controlling the disease is by the integration of various control measures, including biological control. The time to implement biological control is in the resting (or sclerotium) stage of *S. sclerotiorum,* during which the pathogen has little mobility, or at the germinating stage, during which the pathogen is most vulnerable to attack. Adams and Ayers (1979) stated that the most significant component of soil affecting the survival of sclerotia appears to be the biota.

Any biological measures should target the dormant sclerotia in the field. Contrary to the traditional operation of ploughing the sclerotia deep into the soil where it will remain dormant for several years, it has been found that by adopting a rotation with a non-suscept, such as corn, and using chiselling for ground preparation, most of the sclerotia will remain on the surface or in the top 3 inches of soil where the weathering and biodegradation are maximal. Sclerotia that remain near the soil surface are subjected to frequent dry-wet cycles. The sclerotia can develop cracks in the rinds when dried and rewetted in soil. Cracked sclerotia leak nutrients and are easily colonized by other microorganisms and rotted (Adams, 1975). Also, sclerotia survive poorly in flooded soil (Moore, 1949). Increasing soil moisture accelerates their degeneration (Williams and Western, 1965). The sclerotia that survive the winter are conditioned to germinate in the spring. With rotation to non-suscept, the ascospores liberated from the apothecia of the germinated sclerotia eventually die without consequence to the crop.

Near the soil surface, biological activities are at their highest. Animals, birds, soil fauna, and insects can consume and injure the sclerotia. Numerous antagonistic and mycoparasitic fungi and bacteria are also present and frequently isolated from parasitised sclerotia. They are *Coniothyrium minitans, Trichoderma* spp., *Gliocladium* spp. and *Sporidesmium sclerotivorum* (Adams and Ayers, 1979). Other mycoparasites that belong to the genera *Acrostalagmus, Fusarium, Hormodendrum, Mucor, Penicillium, Aspergillus, Stachybotrys,* and *Verticillium* (Bedi, 1961; Makkonen and Pohjakallio, 1960) have been described as parasites on sclerotia. Among them, *C. minitans* and *Gliocladium virens* have shown practical potential for biological control of *S. sclerotiorum* (Budge et al., 1995). Although *G. virens* shows the potential to infect sclerotia of *Sclerotinia* and *Sclerotium* spp., it has not been evaluated on a large scale against *S. sclerotiorum*. In contrast, *C. minitans* applied to soil as a solid-substrate inoculum can infect sclerotia of *S. sclerotiorum* year-round and effectively reduce their number and viability in the soil (Budge et al., 1995; Gerlagh et al., 1994; Huang, 1977). Foliar application of spore suspension of *C. minitans* has been shown to reduce disease severity (Trutmann et al., 1982). Most recently, others (Budge and Whipps, 1991; Whipps and Gerlagh, 1992) have shown that applications of spore suspensions of *C. minitans* to crop residues occupied by *S. sclerotiorum* may reduce disease carryover and could therefore be used in combination with soil incorporation treatments. Growing the fungus in the plant debris and reintroducing it into the soil could conceivably hasten the destruction of sclerotia in the field.

Little information is available about the effect of bacteria on the survival of sclerotia. However, damaged sclerotia appear more susceptible to bacterial invasion. Bacteria are consistently found in rotted sclerotia as in many other saprophytic fungi. Thus, many bacteria, such as *P. fluorescence, P. putida,* and *B. subtilis*, may have a direct or indirect impact on the survival of the sclerotia, particularly those which produce antibiotics. More research in this area is needed.

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