REPORT TO THE OREGON PROCESSED VEGETABLE COMMISSION: 2010

Title: Impact of biological control on white mold sclerotial survival and disease severity in subsequent resistant and susceptible bean crops.

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SUMMARY:

- Six months after a November Contans (*Coniothyrium minitans*, *Cm*) application to diseased bean residues, mean *Cm* colonization of sclerotia was 47% in *Cm*-treated fields, compared to 3% in control fields. Mean sclerotial viability in *Cm* and control fields was 67% and 98%, respectively.
- Ten months after the Contans application, 8.5% of sclerotia (buried or on the surface) were alive in *Cm* treatment fields compared to 74% in the control fields (almost 9 times more living sclerotia in the control than in the *Cm* treatment fields).
- Beans were planted in the fields in July 2010. At bean harvest in September, percent foliar white mold necrosis for 91G (white mold susceptible variety) and OR-6230 (moderately white mold-resistant) was approximately 23 and 7.5%, respectively, in the control fields, and approximately 7 and 1%, respectively, in the *Cm* treatment fields. The lowest foliar disease severity was observed in the moderately resistant plants grown in *Cm*-treated fields.
- At harvest, pod white mold incidence in 91G and 6230 was approximately 17 and 11%, respectively, in the control fields, and 7 and 3% in the *Cm* treatment fields. The lowest pod disease incidence was observed in the 6230 plants grown in *Cm* treated fields.
- Plant aboveground biomass (canopy weight) was lower in the *Cm* treated fields than in the control fields, and lower in OR-6230 than in 91G.
- The lower disease severity in the beans grown in the *Cm*-treated than in the control fields was likely due to some combination of lower white mold inoculum levels and lower aboveground biomass. Lower disease severity in OR-6230 compared to 91G was likely due to a combination of higher genetic resistance and lower aboveground biomass.

INTRODUCTION:

White mold (WM, caused by *Sclerotinia sclerotiorum, Ss*) is a serious foliar and pod disease of snap beans grown for processing in western Oregon. Fields with > 6% infected bean pods are rejected by the processor, resulting in a complete crop failure. Ronilan (vinclozolin), a highly effective fungicide used through 2005 for the control of both white and gray mold (*Botrytis cinerea*), is no longer available to bean growers.

Topsin/Rovral or Topsin/Endura two-spray tank mix programs effectively control both diseases and growers shifted over to these fungicides in 2006. These fungicide programs are considerably more expensive than single Ronilan applications. Topsin must be applied 14 or more days before harvest (a problem mid-summer when bean development is rapid) and there are concerns about its sole use as *Ss* could develop resistance. Farmers are now seeking lower cost and biologically- and culturally-based WM management strategies that are practical and appropriate in the Willamette Valley.

Biology of white mold

Nearly 400 plant species can be attacked by *Ss*. This pathogen causes a single cycle disease initiated by soilborne survival structures (sclerotia). Sclerotia, which are formed on diseased host tissues, become resistant to decay as they mature and survive in soil for up to 8 years. In beans, new infections occur when sclerotia near the soil surface germinate, typically in response to the moist environment under a dense canopy, to form apothecia. Each apothecium produces millions of ascospores which infect bean flowers; the disease then spreads to stems and pods.

Biological control

Coniothyrium minitans (Cm) is a mycoparasite of Ss under natural conditions and was recently developed as a commercial product for WM suppression (Contans, <u>www.prophyta.com</u>). Cm can penetrate sclerotial tissue in less than 14 days. Infection of a sclerotium can be achieved by a single spore. Cm also can infect Ss mycelia when colonizing leaf tissue. Cm parasitizes sclerotia optimally over a temperature range of 50 - 68 °F (Turner & Tribe, 1976), with little activity occurring at > 80° or < 41 °F.

In this work, *Cm* inoculum was applied to the soil surface after flailing of diseased crop residues. Residues were then left on the soil surface. *Cm* in this scenario has 6 months or more to colonize, infect, and destroy sclerotia. Colonized sclerotia produce pycnidia. If left on the surface, these pycnidia produce conidial droplets under warm, wet conditions. During rain events, these droplets splash and disperse, generating new sclerotial infections. We have shown in previous work that the mild, wet, winter conditions in western Oregon generate a series of *Cm* colonization cycles - a "biological control epidemic" - after low rate Contans applications to surface residues containing white mold sclerotia, and this reduces sclerotial survival.

Integrated management of white mold with biological control and resistant cultivars:

Integrated pest management requires the integration of all available and cost effective control strategies into a 'systems plan' for disease management; overall disease risk is reduced as no single strategy is relied on for disease management. Jim Myers, OSU vegetable breeder, has developed snap bean varieties with moderate resistance to white mold. These varieties may not provide adequate control as a stand-alone white mold management strategy, but could be incorporated into a multi-tactic integrated approach. In this project we investigated the efficacy of integrating biological control and moderate genetic disease resistance for white mold management in snap bean production.

OBJECTIVES:

To evaluate the impact of Contans applications and reduced tillage on:

- 1) sclerotial survival,
- 2) sclerotial colonization by Coniothyrium minitans and other fungi,
- 3) apothecia production in the field in subsequent years, and
- 4) disease incidence in subsequent susceptible and moderately resistant bean crops.

METHODS:

Field preparation:

Four bean fields at the OSU Vegetable Research Farm and 4 bean fields at the OSU Lewis Brown farm were planted to 91G snap beans in July 2009, inoculated with white mold using the straw inoculation method, irrigated regularly to develop disease, and flailed at maturity in September, leaving many white mold sclerotia evenly distributed across the soil surface.

Sclerotial management:

Contans (1.5 lbs per acre) was applied to the 4 fields at the vegetable research farm (*Cm* treatment) on November 4 2009; the 4 Lewis Brown fields (control) remained untreated. In May 2010 sclerotia were collected from the field surface from all fields, bagged in the laboratory, and placed in the Lewis Brown (LB) and OSU Veg Farm (OS) fields on July 8 and July 12, respectively. Bags were collected from fields on Sept 21 2010.

At both sampling dates (May and September), sclerotia or bags were collected from the field and sclerotia were surface-sterilized in 20% bleach for 10 minutes and rinsed with DI water. All sclerotia were plated on PDA w/streptomycin and observed for development of *Ss* sclerotia, *Cm* pycnidia, and mycelia of other fungi.

Field snap bean experimental design and management:

Snap beans 91G and OR-6230 were planted in a randomized complete block design (6 plots of each variety in each of the 8 fields) on half of each field on June 29 and July 1 at OS and LB, respectively (the other half of each field will be planted to the same experiment in 2011).

Evaluation of apothecia production:

We attempted to count apothecia in the field at bean bloom but were unable to find sufficient apothecia to get a count.

Bean foliage and pod evaluation at harvest:

Beans were evaluated and harvested at LB and OS on Sept. 8/9 and Sept. 9/10, respectively. Bean aboveground biomass was harvested from two 4 ft row sections per plot and weighed. Of the plants in the 8 ft of row, twenty randomly selected plants were evaluated for white mold disease severity on a scale of 0-5 (0=healthy; 1=1-10% foliage necrotic; 2=11-30% foliage necrotic; 3=31-60% foliage necrotic; 4=61-80% foliage necrotic; and 5=91-100% foliage necrotic). Pods were stripped from all 20 bean plants and weighed. Two hundred beans from each plot were weighed and evaluated for pod white mold incidence. Diseased beans were also weighed.

RESULTS:

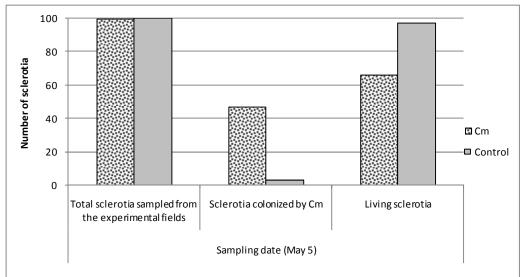


Fig. 1. Viability and *Cm* infection of sclerotia sampled from the soil surface in May 2010.

In May 2010, sclerotia generated on the previous year's bean crop were collected from the soil surface in all fields and evaluated for viability and Cm colonization. In fields in which Cm was applied, mean Cm colonization of sclerotia was 47%. In control fields, mean Cm colonization was 3% (Fig 1). Mean sclerotial viability in Cm and control fields was 67% and 98%, respectively (Fig. 1).

Sclerotia collected in May were bagged in the laboratory and the bags were placed in the field after bean planting. Bags were collected in September for evaluation. At this time, approximately 7% of buried and 10% of surface-incubated sclerotia were alive in *Cm* treatment fields. In contrast, approximately 74% of buried and surface-incubated sclerotia were alive in the control fields (with no significant effect of burial; p = 0.13)(Fig. 2). Viability of sclerotia in the *Cm* treatment fields declined from 67% in May to 7 and 10% (for surface and buried sclerotia, respectively) in September. In contrast, viability of sclerotia in control fields declined during that same period from 95 to 74%. (Figs.1 and 2) There were almost 9 times more living sclerotia in the control fields than in the *Cm* treatment fields 10 months after *Cm* application (Fig.2). Interestingly, in both *Cm* and control fields, sclerotia incubated on the soil surface were much more likely to be colonized by other fungi than sclerotia from any other treatment; this is in contrast with past years' data in which during the summer, buried sclerotia were more likely to be colonized by other fungi than surface sclerotia.

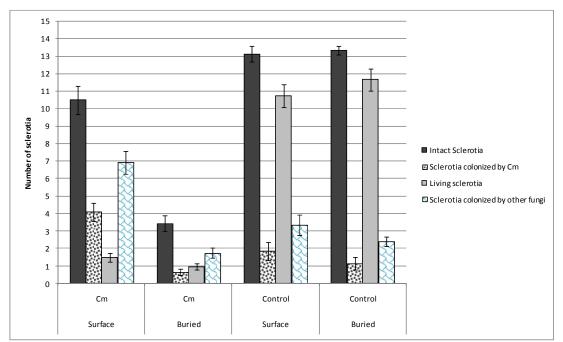


Fig. 2. Viability and colonization of sclerotia bagged in May and evaluated in September.

At harvest, foliar white mold severity was approximately 23 and 7.5%, respectively, for 91G and OR-6230 in the control fields, and 7 and 1%, respectively, in the *Cm* treatment fields. The lowest disease severity was observed in OR-6230 plants grown in *Cm* -treated fields (Fig. 3).

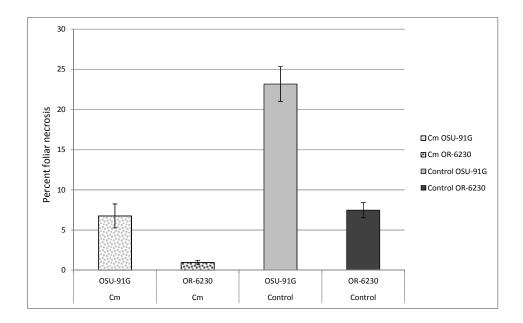
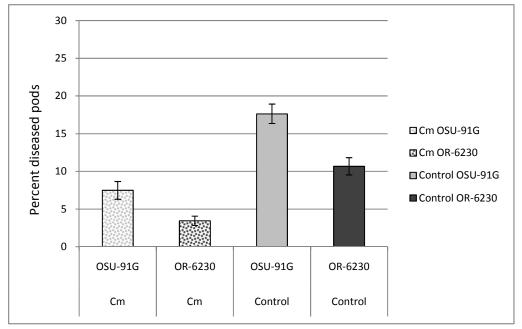
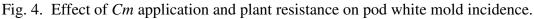


Fig. 3. Effect of *Cm* application and plant resistance on foliar white mold severity.





At harvest, pod white mold incidence in the control fields was approximately 17 and 11% in 91G and OR-6230, respectively, and 7 and 3%, respectively, in the *Cm* treatment fields. Pod disease incidence was 37% lower in 6230 than in 91G in the control fields and 60% lower in OR-6230 than in 91G in the *Cm*-treated fields. The lowest pod disease incidence was observed in the OR-6230 plants grown in *Cm*-treated fields (Fig. 4).

Bean marketable yields were not significantly different in the *Cm* and the control treatments (p = 0.16), but OR-6230 yielded less than 91G (p < 0.001) in both *Cm* and control fields (Fig.5).

There were significantly more diseased beans in the control than in the *Cm* treatment fields, and in the 91G than in the OR-6230 treatments.

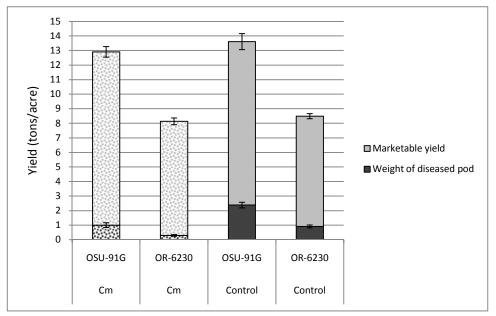


Fig.5. Marketable and diseased bean yields (tons/acre).

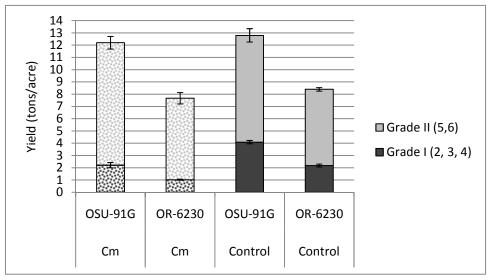


Fig. 6. Bean grade at harvest.

Beans were harvested late, resulting in a large number of large pods; this contributed to the high total yields (Figs. 5, 6). Days to harvest for the beans grown in the OS fields (*Cm* treatment) was 5 days longer than in the LB fields, as they were planted several days earlier and harvested several days later; this likely contributed to the higher proportion of Grade II beans in the OS (*Cm* treatment) fields (Fig. 6).

Mean aboveground biomass of 91G and OR-6230 bean plants was 18% higher in the *Cm* treatments than in the control treatments, and mean 91G biomass was 19% higher than mean 6230 biomass (data not shown).