

TOWARDS IMPROVING CONTROL OF *BOTRYTIS* DRY ROT OF GARLIC (*BOTRYTIS PORRI*)

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Abstract

A review of the limited understanding of this disease is presented. Attempts were made to induce an epidemic of *Botrytis porri* in a 1-acre field of garlic grown in 2000-2001. While some *B. porri* dry rot did occur, the attempt to induce a general epidemic failed; possible reasons why are discussed. A fungicide spray trial was conducted, but too little disease occurred to evaluate relative control by various products included. Cloves from this crop are being grown in the greenhouse in an attempt to find sporulation of *B. porri* on young plants, and to verify that this fungus may be carried in seedlots. Seed from a damaged commercial crop was planted in the fall of 2001, both with and without Rovral fungicide treatments, in a further attempt to discern whether this disease is expressed early from seedlot infection, and whether seed treatment or fall season control efforts may suppress disease development in late winter or early spring.

Introduction and Literature Review

In central Oregon, neck rot of garlic, caused by *Botrytis porri*, has been an erratic problem in the garlic industry in the western United States since about 1980 (Sommerville et al. 1984). Because this disease is termed "dry rot" elsewhere, dry rot will be used in this discussion. While it also occurs in the central regions of California, it is more frequently a problem in the cooler seed production regions of Oregon, Nevada, and northern California. In Oregon's Willamette Valley, *Botrytis allii* (the common onion neck rot pathogen) may incite a garlic disease similar to that caused by *B. porri*, and both fungal species may be present (P. Koepsell, personal communication, Oregon State University).

B. porri has been widely reported in garlic (Harvey 1981, Kovachevsky 1958, Stoikova 1984) with reported crop losses up to 20 percent, but there is essentially no literature on epidemiology of this pathogen or control of this disease. It also is reported in leeks (Asiedu et al. 1986, Cronshey 1947) and wild garlic (Cronshey 1947). The western United States is the only garlic-raising region that reports a chronic problem with *B. porri* on garlic, but this may be a reporting difficulty rather than a true measure of higher or more frequent incidence.

In Northern Europe, *B. porri* causes a storage rot of leeks following field infection in the summer and fall (Hoftun 1978, Tahvonen 1980). That the fungus may continue to grow in cold storage underscores observations that *B. porri* is a cool and cold weather disease in garlic. Curiously, some leek seed is produced in central Oregon, and no *B. porri* problems have been reported, but perhaps too few observations have been made to recognize its presence.

Field symptoms on garlic start with lesions that form on the neck at or near the soil line. Lesions expand somewhat upward, but especially downward and inward into the neck from leaf to leaf. On smaller and younger plants, necks may be totally killed, leading to death of the plant or stunted, unproductive plants. If bulbs are developing at the time lesions appear, bulb size may be reduced by loss of some or all leaves. If plants are large and if bulbs are already fully formed at the time of lesion appearance, ingress into the neck is delayed and there may be little effect on yield. As the fungus grows downward on lesions, the covering bulb leaf sheaths and cloves may be decayed, bulbs may be small, and surviving cloves may shatter on harvest.

As lesions expand, *B. porri* produces mats of grayish-brown asexual spores (conidia). It also produces large, irregular, convoluted sclerotia on lesions and rotted areas on leaves and bulbs. Such sclerotia initially are somewhat soft and gray, but quickly become black and firm as they mature. Late infected bulbs and covering leaf sheaths may remain intact and may or may not be encrusted with sclerotia. Seed lots may contain late-infected cloves, and may be infested with sclerotia and conidia, which might conceivably contribute to disease occurrence in the next planting. Once plants die and/or the crop is harvested, most sclerotia remain shallowly buried in the soil or on the soil surface, along with rotted garlic tissues.

In the laboratory, sclerotia may give rise to both conidial sporulation over the surface of sclerotia, and also to sexual structures (apothecia) from which sexual spores (ascospores) may be forcibly ejected (Van Beyma Thoe Kingma 1927, Kovachevsky 1958, Stoilova 1984, Summerville et al. 1984). Apothecia may have stalks up to several centimeters long. On related fungi, stalked apothecia allow ascospore release into the air from shallowly buried sclerotia, but such production from buried sclerotia in the field has not been confirmed for *B. porri*. I sometimes have observed apothecia in the field in central Oregon in the late winter/early spring that formed on sclerotia lying on the soil surface. I have very commonly observed abundant conidial formation during the winter and spring on sclerotia lying on the soil surface.

The relative contribution to current season epidemics of spores arising from infected or infested seed, from infected volunteer garlic or leeks, from sclerotia on or in soil, or from the previous season's garlic debris is unknown--presumably all may contribute to primary infection. Also unknown is the relative contribution of ascospores and conidia. However, based on what is known about this and related pathogens, it is presumed that most secondary or epidemic infection arises from conidial cycling on the current season's lesions just above the soil line.

Information that may be useful towards designing an efficient disease control program include:

1. Sources of epidemic:
 - a. What is the relative occurrence/importance of spore production from dead and decayed garlic, sclerotia on soil surface, volunteer garlic,

- infected/infested garlic seed in current field, leek field sources, etc.? And when are these spores produced?
- b. How are conidial and ascospore production influenced by temperature, wetness, depth in soil, etc.?
 - c. Under what conditions and time of day are ascospores discharged?
2. What are the infection requirements (wetness, temperature, light quality)? Are these different for conidia and ascospores?
 3. Are there positional effects that affect epidemics? What distances can spores travel, and is this different for conidia and ascospores? This could be important with respect to volunteer management, soil management to deeply bury garlic residue and sclerotia, etc., and understanding the relative importance of within-field sources of inoculum (e.g., infected/infested seed) vs. outside sources of spores.
1. What is the lag period between infection and symptoms. Is the appearance of noticeable neck lesions a useful tool for timing of spray applications, or do spores spread and infect well in advance of symptom development? Is the lag period shortened as the season warms and daylength increases?
 2. Preventative control program:
 - a. Is seed treatment or better grading of seed lots beneficial if infected/infested seed is a source of initial infection?
 - b. Does fall infection on emerged garlic occur?
 - c. When should a spray program be initiated and discontinued?
 - d. Can sprays be modeled based on wetness and temperature and cloudiness, etc.?
 - e. Should sprays be coordinated with irrigation cycles, soil type,?
 - f. Where to direct sprays (probably neck and soil line)? How effective is aerial application?

What are the best products to use, and how should concerns about resistance development, cost, etc. be addressed?

In general, foliar diseases incited by species of *Botrytis* are encouraged by extended periods of leaf wetness, high humidity, cloudiness, poor aeration, etc.--conditions that extend the time that spores are likely to infect leaves. For some *Botrytis* diseases, there can be significant lag times between spore infection and appearance of lesions or other decay. We have found no information concerning these aspects for *B. porri*. Possibly useful observations I made in 1984-2000 include the following, but no such observations except "c" and "f" have been verified by experimentation or formal survey:

- a. Conidia may be present throughout the winter on sclerotia (and possibly on decaying garlic tissue) located at the soil surface. Apothecia probably only form in late winter and/or early spring, primarily from sclerotia on or very close to the soil surface, and may not form in some or many years. Apothecia seem to take a long time (weeks) to develop.

- b. Dry rot has been observed on volunteer garlic in fields where neck rot occurred the previous year. In those same fields, sclerotia with active conidial sporulation also can usually be found.
- c. Dry rot is more chronic on heavier soil types. Seemingly, soil dampness at the soil line encourages neck infection at that location.
- d. Infection and epidemic development is greater when cool, damp, cloudy weather persists.
- e. Irrigation management strongly influences occurrence of dry rot, especially on heavier soils. It is especially important to keep sprinkler irrigation from extending soil and foliage wetness generated by rainfall and cloudy periods. Dry rot can be abundant even in sandy soil when excessive irrigation aggravates already-conducive weather conditions. Whether furrow or drip irrigation reduces neck infection has not been determined.
- f. At least for central Oregon, infection may be abundant well before the irrigation season begins about mid-April, especially if there have been extended periods of wet, cloudy weather. Neck lesions have been seen as early as February in some years, and are common in March and early April.
- g. Preventative spray applications commonly are initiated well after early epidemic disease development has begun.
- h. Early loss of very small garlic plants may go unrecognized.
- i. Epidemics may ebb during extended dry and sunny periods, then re-activate if conducive weather resumes.
- j. Preventative spray applications commonly are extended too late in the season, when new neck infections likely will not damage larger plants, and when earlier infections cannot be stopped.
- k. Choice of control products and timing of applications usually is determined by convenience, cost of products, cost and difficulty of repeated applications, perceived crop yield, and current disease status. Too often, there is elevated interest in preventative fungicide applications *after* epidemic development is well advanced.
- l. Disease severity may be region-wide under highly adverse weather conditions, but may vary greatly from field to field in less conducive periods. This suggests a strong influence of local management.
- m. Incidence commonly is worse in fields of the varieties 'Chinese' and 'California Late' than in fields of 'California Early', although it can be severe in the latter, too. We are uncertain whether this a varietal effect or seedlot management.
- n. Some in the industry believe that fungicide applications to fall-emerged foliage are beneficial.

In the mid- to late-1980's, P. Koepsell and I conducted fungicide research for control of *B. porri* at Madras, Oregon. While somewhat inconclusive, some things were learned. Several products effective against other *Botrytis* species were applied to foliage in randomized, replicated field trials. During one season, no dry rot occurred from within the trial area, including the untreated check plots, but abundant dry rot occurred in the surrounding garlic field. This suggested that the products worked but that the untreated check plots also became protected. This may have occurred by overspraying the plots or by drift of products onto those plots. Alternatively, infection may have spread in the

surrounding field without spores effectively bridging the sprayed plots surrounding the untreated check plots. In a second season, no products worked very well, and it was surmised that the spray program may have begun too late.

Materials and Methods

A 1-acre field of garlic ('California Early') was commercially planted on October 5, 2000 at the Central Oregon Agricultural Research Center (COARC)-Madras station. The crop was commercially managed with respect to fertilizer and weed control. Beginning in 2001, this field was irrigated at about twice the frequency as required, in an attempt to keep the soil line and neck areas damp, toward inducing an epidemic of *B. porri*.

Once in late April, and twice in May, fungicide applications were directed at the neck/soil line area on 3-bed x 30-ft plots, with four replications. Fungicides included Benlate[®], Folicur[®], Quadris[®], and Botran[®], all at specific rates useful for control of *Botrytis* diseases on various crops, and several rates and timing combinations of Rovral[®], as material commonly used in central Oregon. Specific rates are not listed here.

Garlic seed was obtained from a commercial field of garlic in the Willamette Valley where abundant *B. porri* dry rot occurred during 2001. This seed was planted in the field on October 10, 2001, and in-furrow sprayed with 4.3 lb/acre Rovral as per the white rot (*Sclerotium cepivorum*) label for this product. There were three, 200-ft-long x 2-bed plots of both Rovral-treated and untreated garlic in this small trial, which will be evaluated for disease in the 2002 season.

Seed from the COARC field was planted at various times in the greenhouse during the winter of 2000-2001 to force garlic growth and observe for early signs or symptoms of *B. porri* on small plants.

Results

Symptomatic plants did appear in late April, 2001, and by mid-May, a scattering of infected necks had occurred throughout the field. In mid-May, sporulating cultures of *B. porri* were blended and mixed with water and sprayed over the tops of plots. Nevertheless, no widespread or field-wide damaging development of *B. porri* occurred prior to water cut-off in early July. No fungicide comparisons were made after initial comparisons between untreated checks and selected sprayed plots indicated that there was no difference in garlic performance. In contrast, abundant *B. porri* dry rot occurred in various commercial fields in central Oregon where water management was less conducive than in our 1-acre trial.

At the time of this report, we have no data from the 2001 field planting, nor from greenhouse testing.

Discussion

We suggest that our failure to induce an epidemic of *B. porri* in the 1-acre planting can be explained by lack of suitable humidity and shade within the garlic canopy. The garlic was a thin stand and undoubtedly there were strong drying edge effects in such a small planting. We had hoped to overcome these effects by over-irrigating, but did not succeed. Furthermore, immediately following the application of inoculum of *B. porri*, a major rainfall occurred, which may have washed the inoculum from the foliage rather than allowing it to remain in contact. Finally, we may have under-fertilized (in combination with over-irrigating), as we suspect that *B. porri* incidence may be favored by high fertility rates in combination with neck-wetness, high canopy foliage density, and cloudy, cool weather.

Our experiments in progress will be reported later in 2002.

Acknowledgments

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