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Cheatgrass (*Bromus tectorum*) Biocontrol Using Indigenous Fungal Pathogens

Susan E. Meyer, David L. Nelson, Suzette Clement, and Julie Beckstead

Abstract—Cheatgrass (*Bromus tectorum*) is an exotic winter annual grass weed that has invaded millions of hectares in the Intermountain West. Restoration of cheatgrass-invaded wildlands is generally impractical without some form of cheatgrass control. We are investigating the possibility of manipulating indigenous fungal pathogens that already occur on cheatgrass for short-term biocontrol in conjunction with restoration seedings. Three potential biocontrol organisms have been identified. The head smut pathogen (*Ustilago bullata*) and the chestnut bunt pathogen (*Tilletia fusca*) infect at the seedling stage and prevent seed set, while the black-fingers-of-death pathogen (*Pyrenophora semeniperda*) kills seeds in the seed bank. Both head smut and chestnut bunt pathogen races on cheatgrass are host-specific, whereas black-fingers-of-death is a generalist grass seed pathogen that does not appear to form host-specific races. Inoculation trials with the head smut pathogen yielded high levels of disease only when seedlings emerged at moderate temperatures in fall, whereas the chestnut bunt pathogen infects at near-freezing winter temperatures but requires persistent snow cover for successful infection. The black-fingers-of-death pathogen is most effective at destroying seeds in the carryover seed bank. A combined approach using all three pathogens shows some promise for biocontrol of this troublesome weed.

Introduction

The invasion of cheatgrass (*Bromus tectorum*) into the Intermountain West has been called the most significant plant invasion in the modern history of North America (D'Antonio and Vitousek 1992). Cheatgrass creates the disturbance it needs to perpetuate itself by producing a continuous, fine fuel that is associated with increases in the frequency and size of wildfires (Whisenant 1990). With repeated burning, vast areas are converted to cheatgrass monocultures that are extremely difficult to restore to native plant communities or even to rehabilitate with introduced forage grasses. For a seeding to be successful, some form of cheatgrass control is necessary, especially in the arid and semiarid habitats where this plant is most problematic (Monsen 1994).

Traditional methods of cheatgrass control include early season burning to prevent seed dispersal, tillage after emergence in the fall, and the use of various herbicides. These methods tend to be risky, expensive, or damaging to remnant perennial vegetation, as well as resulting in unpredictable levels of control. We have initiated research on possible biocontrol organisms for cheatgrass because of the need for targeted, environmentally benign, and effective control methods to be used in conjunction with restoration seeding. At least one biocontrol organism has already been patented for use against cheatgrass in winter cereal crops (Kennedy and others 1991, 2001). This organism is a specific rhizobacterial strain that targets the cheatgrass root system and greatly reduces its growth, thereby reducing grain yield losses to this weed. There is a good possibility that this biocontrol agent could also be used on wildlands, possibly in conjunction with the biocontrol agents that are currently under study at our laboratory (Ann Kennedy, personal communication).

In our cheatgrass biocontrol studies, we are investigating three fungal pathogens that target different stages of the life history of cheatgrass, but each of which has the net effect of reducing the size of the seed bank (fig. 1). Two of these pathogens (*Ustilago bullata* and *Tilletia fusca*) are smut fungi that cause systemic diseases resulting in prevention of seed production in infected plants, while the third (*Pyrenophora semeniperda*) is an ascomycete fungus that kills seeds directly in the seed bank.

The Head Smut Pathogen (*Ustilago Bullata*)

We began our biocontrol investigations with work on the head smut pathogen *U. bullata*. This organism has a wide host range, infecting several genera of cool season grasses, but Fischer (1940) determined that this pathogen is characterized by a high degree of host specificity, with different races infecting different grass host species. This has been confirmed in our own studies; races of the pathogen from cheatgrass are unable to cause significant levels of disease on any of the native perennial grass or introduced forage grass species that commonly co-occur with cheatgrass (Meyer and others, in review).

Almost every cheatgrass population contains endemic levels of head smut disease, and the disease can sometimes reach epidemic levels. The focus of our research has been

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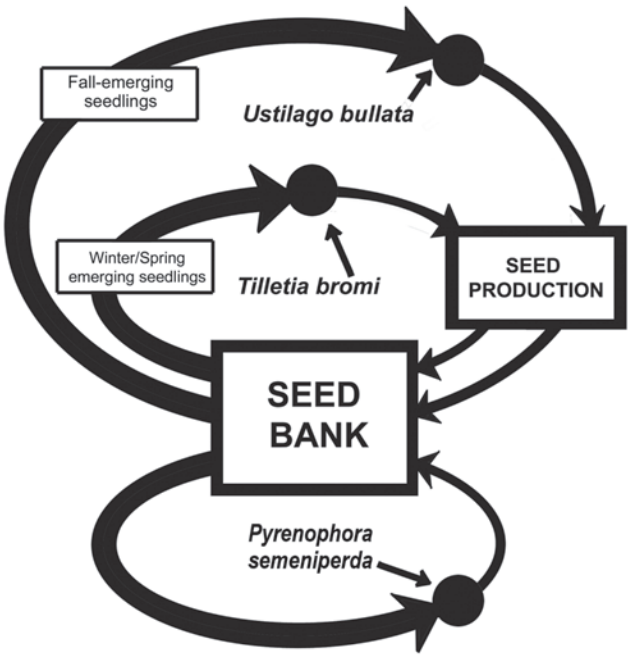

Figure 1—Schematic diagram showing how three proposed cheatgrass biocontrol organisms affect the target host at different stages of its life cycle to negatively impact the seed bank. *Ustilago bullata* infects fall-germinating cheatgrass seedlings at relatively warm temperatures, grows systemically in the plant and prevents seed set. *Tilletia fusca* infects cheatgrass seedlings that emerge at cold temperatures in winter and early spring. It also grows systemically in the plant and prevents seed set. *Pyrenophora semeniperda* attacks and kills ungerminated cheatgrass seeds in the seed bank.

to study the genetic and environmental factors that control disease levels in natural populations. We wanted to know how to cause head smut epidemics through artificial inoculation, so that seed production would be drastically reduced, facilitating the success of a restoration seeding. Our studies on the genetics of the cheatgrass-head smut pathosystem have revealed a complex array of resistance phenotypes in cheatgrass populations and corresponding virulence races in co-occurring populations of the pathogen (Meyer and others 2001, 2005). But these patterns of co-evolutionary response control disease levels only when environmental factors are highly conducive to disease development (Meyer and others, in review). Otherwise, many susceptible host individuals fail to develop the disease even in the presence of pathogen races that can successfully attack them. In order to create epidemics artificially, we have to make sure that our inoculum includes all the necessary pathogen races, but we also have to understand how environmental factors influence disease development.

The life cycle of the head smut pathogen starts with germination of diploid teliospores to produce haploid gametes through meiosis. These gametes, called sporidia, are capable of saprophytic proliferation in the yeast-like haploid state, in effect making many hundreds of copies of themselves, increasing the chances of encountering an infection site. When sporidia of opposite mating types fuse, they form a dikaryotic infection hypha. This hypha is not saprophytic; it has only a short time to encounter an infection site before it spends its limited resources. If penetration at an infection site on a young cheatgrass seedling coleoptile successfully occurs, then the fungus grows systemically inside its host, overwintering in vegetative tissues and growing upward during bolting in spring to take over the floral meristems for teliospore production.

Because the head smut pathogen infects at the seedling stage, conditions during seed germination and seedling emergence mediate infection levels. As with many biological processes, temperature is an important factor in infection success. We made a detailed study of the effect of temperature on different phases of the infection process, including eight different pathogen populations (Boguena and others 2007). Teliospore germination rate increased with temperature, as did cheatgrass seed germination rate. At temperatures of 10–25 °C, teliospore germination was faster than seed germination, while at a cold temperature, below 5 °C, teliospore germination lagged behind seed germination (fig. 2a). Sporidial proliferation rate was also strongly influenced by temperature, with an exponential increase in rate as a function of temperature over the range 2–25 °C (fig. 2b). One outcome of these temperature relationships is that disease incidence was drastically reduced at low temperatures in growth chamber inoculation trials (fig. 2c). Not only did teliospores germinate very slowly at 2 °C, they germinated directly to the dikaryotic state, greatly reducing chances of successful infection and precluding survival as sporidia until temperatures became more favorable (Boguena and others 2007).

Under field conditions, these temperature effects translate to successful infection by this pathogen only when cheatgrass seeds germinate at moderate temperatures in the fall. When inoculated seeds are planted in early fall, disease levels can be very high, but when inoculated seeds are prevented from fall-emerging by sowing on a late planting date, disease levels are very low (fig. 3). This is also the pattern we have observed with naturally occurring levels of this disease. Epidemic levels (>50 percent disease incidence) are rarely seen except in mesic environments with predictable autumn precipitation (Meyer and Smith, unpublished report on file at the USFS Shrub Sciences Laboratory, Provo, Utah). This will limit effective use of the head smut pathogen as a biocontrol agent for cheatgrass to environments with reliable fall precipitation and near-complete fall germination of cheatgrass seeds.

For commercial development of a biocontrol organism, it is necessary to find a way to produce inoculum in bulk, preferably *in vitro*, for example, as yeast is produced in industrial facilities. For an obligate biotroph like the head smut pathogen, this presents problems, because direct production of teliospores would be possible only through 'farming' cheatgrass for the teliospore crop. Fortunately, the saprophytic sporidial stage of the life cycle is more amenable to mass production techniques. We have developed the technology to produce sporidial inoculum and to dehydrate it onto a carrier that can be used in a field setting, though

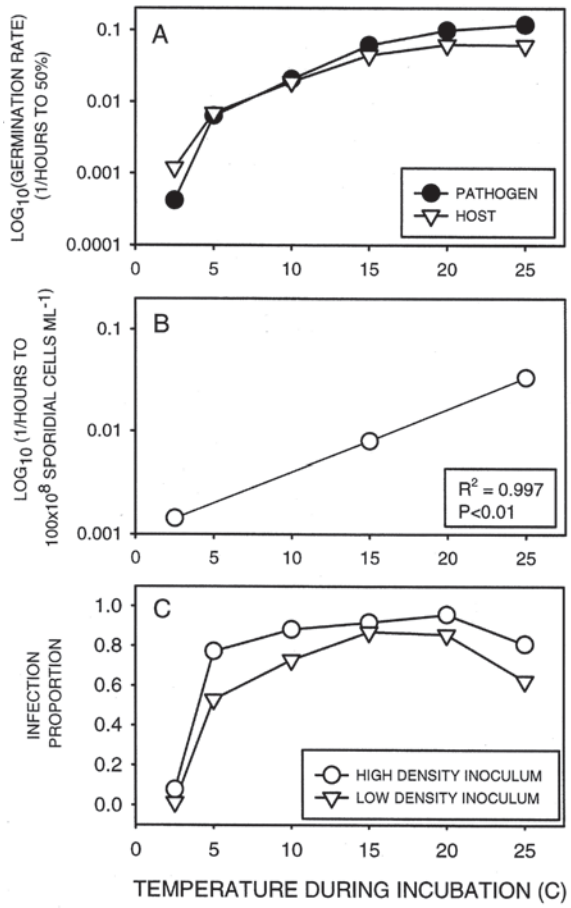

Figure 2—The effect of temperature on: (A) Germination rate of *Bromus tectorum* (host) seeds and *Ustilago bullata* (pathogen) teliospores, (B) Proliferation rate of *Ustilago bullata* sporidial cells in liquid culture, and (C) *Ustilago bullata* disease incidence on *Bromus tectorum* plants after inoculation at two densities at a range of temperatures. Data represent the means of 8 pathogen populations (from Boguena and others 2007).

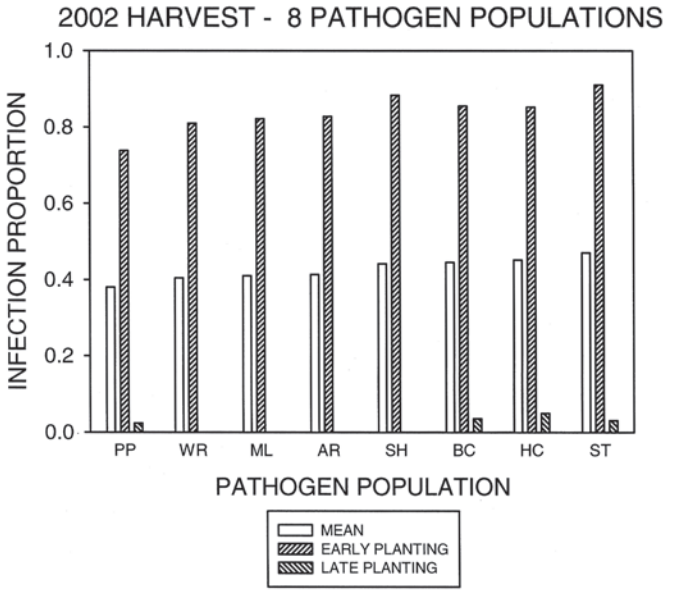

Figure 3—Disease incidence on cheatgrass plants after seeds were inoculated with teliospores from eight different populations of the head smut pathogen and planted early in the fall (mid-September) or late in the fall (mid-November). Within the early planting, bars headed by the same letter are not significantly different at $P < 0.05$; there were no significant differences among pathogen populations within the late fall planting (from Boguena and others 2007).

our studies on inoculum production are still in the preliminary stages. We have obtained high infection using liquid sporidial inoculum, but we still need to optimize dehydration and storage protocols to obtain these high infection levels with dehydrated sporidial inoculum.

The Chestnut Bunt Pathogen (*Tilletia Fusca*)

The discovery of environmental limitations on the use of the head smut pathogen for biocontrol of cheatgrass motivated us to consider other pathogens that might act in complementary roles. The chestnut bunt pathogen is related to the head smut pathogen and has a similar life history, infecting at the seedling stage, growing systemically, and preempting the floral structures for teliospore production. It also forms host-specific pathogen races (Hoffman and Meiners 1971). There are some important differences, however (table 1). Teliospores of the chestnut bunt pathogen germinate only at cold temperatures (fig. 4) (Meiners and Waldher 1959). Teliospores of both pathogens are dormant at dispersal and lose dormancy under dry conditions as a function of temperature in a manner parallel to dormancy loss in seeds of the host (Bauer and others 1998; Meyer and Clement, unpublished data). But nondormant teliospores of the head smut pathogen germinate very slowly if at all in the cold, whereas nondormant teliospores of the chestnut bunt pathogen germinate only in the cold, with no germination at all at temperatures of 10 °C or higher. This pathogen is adapted for infection of emerged cheatgrass coleoptiles under snow cover (Meiners 1958).

Another difference between the two organisms is teliospore longevity. We have data to suggest that head smut teliospores rarely if ever live for more than a year in the field; recolonization through teliospore dispersal from existing populations reestablishes populations after local extinction events. Teliospores are dispersed by wind after the bullae are ruptured by swelling when wet. Chestnut bunt teliospores, on the other hand, are adapted for long term persistence as a soil spore bank. They are produced in 'bunts,' modified cheatgrass florets that have no dehiscence mechanism. The spikelets containing the bunts are bent to the ground with the first winter snows, and the teliospores are released slowly from these bunts by weathering over time. We have some data to indicate that chestnut bunt teliospores undergo

Table 1—A comparison of life history attributes of the head smut pathogen (*Ustilago bullata*) and the chestnut bunt pathogen (*Tilletia fusca*).

Ustilago bullata	*Tilletia fusca*
Infects at warm temperatures in autumn.	Infects at cold temperatures under the snow.
Spores short-lived in soil.	Spores form persistent banks in soil.
Infects barely-emerging *coleoptile*; seed inoculation effective.	Infects *coleoptile* after emergence from soil; seed inoculation not effective.
Easy to grow in culture.	Hard to grow in culture.
Easy to work with in greenhouse.	Hard to work with in greenhouse.
Common, occurs at some level in almost every cheatgrass population.	Less common, restricted to populations in places with frequent heavy snow cover.

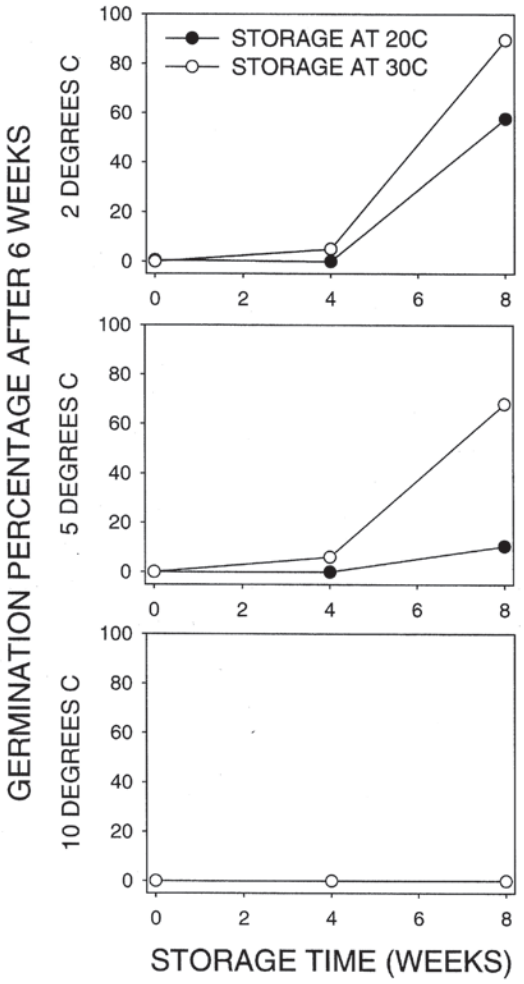

Figure 4—The effect of storage time, storage temperature, and incubation temperature on germination of *Tilletia fusca* teliospores. Data represent the means of teliospore collections from six populations.

cyclic dormancy changes, becoming less dormant as winter approaches and more dormant with the onset of warmer weather (Meyer and Clement, unpublished data). This would also be an adaptation for long term persistence.

Our inoculation trials with the chestnut bunt organism have met with limited success. This organism is not as tractable as the head smut pathogen in culture or in inoculation trials (table 1). Direct inoculation onto the seeds prior to planting does not result in infection; the teliospores must be placed on the soil surface in order for infection to occur, but conditions at the surface must be carefully controlled, for example, through the use of snow-simulating insulating materials. In a survey of levels of head smut and chestnut bunt disease in cheatgrass populations over a range of habitats, we determined that chestnut bunt disease incidence was closely tied to the presence of persistent snow cover in winter (Meyer and Smith, unpublished report on file at the USFS Shrub Sciences Laboratory, Provo, Utah). Use of this organism for cheatgrass biocontrol would thus present many of the same environmental constraints as use of the head smut pathogen, even though they infect under contrasting conditions. This is because mesic habitats, where reliable autumn precipitation occurs, are also the habitats that are most likely to have persistent snow cover in winter. Xeric sites with low autumn precipitation probability also have low probability of persistent winter snow cover and negligible levels of chestnut bunt disease. Higher elevation sites sometimes have epidemic levels of this disease. But these sites are in habitats where cheatgrass is usually not a major problem and where natural succession to native perennial communities can readily occur. This new information has led us to place reduced emphasis on developing the chestnut bunt pathogen as a cheatgrass biocontrol organism.

The Black-Fingers-of-Death Pathogen (*Pyrenophora Semeniperda*)

The third organism that we have investigated as a possible biocontrol agent for cheatgrass is the black-fingers-of-death pathogen, *Pyrenophora semeniperda,* which infects mature grass seeds in a wide range of genera (Medd 1992; Medd and others 2005). This ascomycete fungus is usually seen as its

anamorph (asexual state), *Drechslera campanulata*, which forms characteristic stromatal fruiting bodies that resemble black fingers, hence the common name. These fruiting bodies are readily visible and very distinctive, making this seed bank pathogen relatively easy to detect and to study. Most of the work with this pathogen has centered on nondormant cereal crop seeds (Medd and Campbell 2003). Direct inoculation of mature seeds that are nondormant and that germinate quickly rarely results in seed mortality, though the fungus can sporulate as a weak pathogen on germinated seeds. These infected germinated seeds are usually little impacted by the pathogen. This led most early workers to assume that *Pyrenophora semeniperda* would always act as a weak pathogen, although Kreitlow and Bleak (1964) showed conclusively that this pathogen could cause major mortality in the transient seed banks of native perennial and introduced forage grasses. We have shown that the ability of this pathogen to cause cheatgrass seed mortality is directly related to seed germination rate (Beckstead and others, in press). Nondormant cheatgrass seeds in the transient seed bank in autumn can germinate very quickly and are rarely killed at naturally occurring inoculum levels, while seeds in secondary dormancy in the carryover seed bank can suffer high mortality. We demonstrated this clearly in laboratory inoculation experiments with cheatgrass seeds that differed in dormancy status (fig. 5). Seeds in primary dormancy germinated very slowly if at all, with low percentages even after 28 days. Seeds that were fully after-ripened germinated to high percentages in less than two days, with partially after-ripened seeds germinating somewhat more slowly than fully after-ripened seeds. When the seeds were inoculated with conidia (spores) of the pathogen and incubated at laboratory temperature, 100 percent of the dormant seeds eventually succumbed, while only 8 percent of the fully after-ripened seeds and 13 percent of the partially after-ripened seeds were killed by the pathogen. Uninoculated controls showed a similar pattern but at much reduced absolute levels, with 6 percent of the dormant seeds, 2 percent of the partially after-ripened seeds, and 0.5 percent of the fully after-ripened seeds killed by the pathogen. This mortality was caused by conidia that dispersed to the seeds prior to collection in the field. All seeds inoculated with pathogen conidia eventually exhibited the characteristic black stromata, showing that all seeds were infected. But only seeds that exhibited black fingers prior to germinating were killed by the pathogen.

The black-fingers-of-death pathogen has been demonstrated to cause high mortality in cheatgrass seed banks under natural conditions (Meyer and others 2007). In order to evaluate temporal patterns of seed mortality caused by this pathogen, we collected monthly seed bank samples during winter and spring 2006 at the Whiterocks study site in Skull Valley, Utah. The 2004 to 2005 growing season was favorable for seed production, with over 50,000 seeds m^{-2} on the ground at the end of summer. The autumn of 2005 was dry at the site, and germination did not occur until late December, when a relatively warm winter storm triggered germination of 52 percent of the seed bank. Most of the remaining seeds entered secondary dormancy by early February. Each month, we determined the density of field-killed seeds, the density of ungerminated seeds that exhibited stromata during the first 7 days of incubation and were assumed to be infected prior to collection, and the total density of ungerminated seeds that exhibited stromata during the 28-day incubation period (fig. 6). In January, the density of field-killed plus field-infected seeds was low, but each month during the spring these numbers increased, showing that mortality was taking place on dormant seeds during the long, wet spring. By the end of spring (mid-May), 86 percent of the

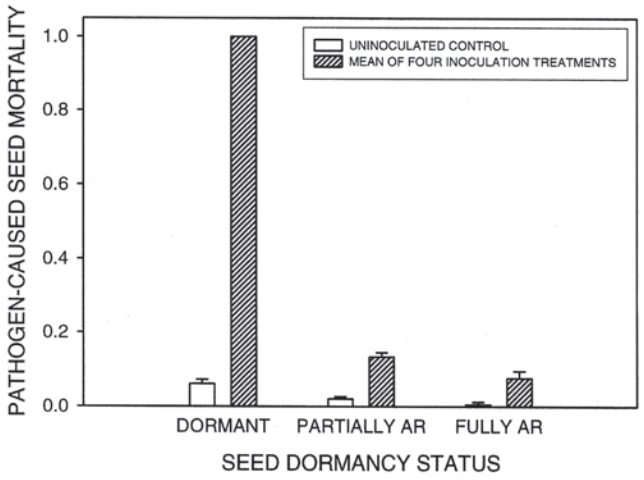

Figure 5—Mortality caused by the pathogen *Pyrenophora semeniperda* in laboratory inoculation experiments with *Bromus tectorum* seeds of differing dormancy status (seeds dormant, partially after-ripened, or fully after-ripened). Data represent means of two seed collections. Error bars shown are standard errors of the mean.

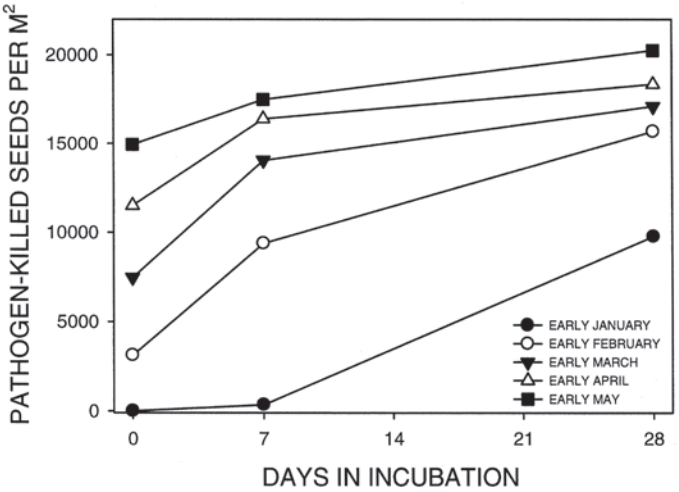

Figure 6—Number of seeds per square meter killed by *Pyrenophora semeniperda* at natural inoculum levels in the field: prior to sample collection (day 0), in the first 7 days of laboratory incubation, and after 28 days of laboratory incubation. Plotted values represent means for twenty samples collected at monthly intervals during spring 2006 at the Whiterocks study site in Skull Valley, Utah.

potential carryover seed bank had been infected and killed by the pathogen. The carryover seed bank was reduced from a potential 48 percent of total seeds present in the autumn to only 7 percent. This study shows that this pathogen can have a major negative impact on cheatgrass seed banks even at naturally occurring inoculum loads.

There are two principal problems associated with the use of the black-fingers-of-death pathogen as a cheatgrass biocontrol agent. First, in the field its impact seems to be limited to causing mortality in the carryover seed bank. Because most cheatgrass plants that successfully produce seed establish from the transient (current-year) seed bank, negative impacts to the carryover seed bank, even if large, may have little effect on population dynamics. It is only in years when establishment and seed production from the transient seed bank fails, due to fire, grasshopper herbivory, a head smut epidemic, or some other catastrophe, that the fate of the carryover seed bank becomes pivotal. But if other forms of control can target the plants produced from the transient seed bank, this pathogen could be useful in eliminating the carryover seed bank that remains. Very few control methods impact ungerminated seeds, and this is a potential strength of this particular biocontrol agent. Cheatgrass establishment from the carryover seed bank can be a substantial impediment to seeding success even when control of actively growing plants or of seed production is successful.

Another approach to overcoming the obstacle of low impact of this pathogen on the transient cheatgrass seed bank would be to select for pathogen strains that can kill rapidly germinating cheatgrass seeds. There is evidence that different strains of this pathogen possess different degrees of virulence (Capio and others 2004; Campbell and others 2003), so the potential for artificial selection for increased virulence probably exists. We are currently developing a protocol for screening multiple isolates of the pathogen for ability to kill nondormant cheatgrass seeds.

A second problem with use of the black-fingers-of -death pathogen for cheatgrass biocontrol is the fact that it is a generalist pathogen that apparently lacks host-specific races (Beckstead, unpublished data). If this organism were successfully used for biocontrol of cheatgrass by eliminating the seed bank, the inoculum produced on these killed seeds could pose a threat to the seeds of planted species. The host range of this pathogen is not completely known, but it seems to attack mainly cool season grasses of the Hordeae and Festuceae. It may be possible to seed with species that have low susceptibility, or to develop fungicidal seed dressings to protect seeded species. The risk to seeded species depends on several factors, including the density of target seeds and resulting inoculum production as well as the ability of inoculum to persist in the absence of host seeds. We have data to suggest that inoculum does not persist for more than a year without a host seed. One strategy would be to carry out biocontrol and seeding on burns, where the cheatgrass seed density is already much reduced, so that inoculum production from these seeds would be low. This would also increase the chances of achieving complete or near-complete cheatgrass control.

Conclusions

In summary, our work on indigenous fungal pathogens as potential biocontrol organisms for cheatgrass has given us a sense of guarded optimism. These organisms pose none of the threats posed by classical biocontrol organisms imported from the Old World range of cheatgrass, and we know that there are scenarios where each of them has resulted in local extinction or near-extinction of the cheatgrass host population. By investigating the ecological requirements of these organisms and understanding how they interact with cheatgrass seeds to cause either endemic or epidemic levels of disease, we may be able to combine them with each other and with other methods of control to achieve the near-complete control required to permit successful seeding of native species into cheatgrass monocultures on arid and semiarid sites. As more and more plant communities in the Intermountain West are invaded and supplanted by this highly successful weed, the imperative to find ways to effectively restore cheatgrass monocultures can only become stronger.

Acknowledgments

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