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Niche specialization in *Bromus tectorum* seed bank pathogens

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Abstract

Niche theory predicts that when two species exhibit major niche overlap, one will eventually be eliminated through competitive exclusion. Thus, some degree of niche specialization is required to facilitate coexistence. We examined whether two important seed bank pathogens on the invasive winter annual grass *Bromus tectorum* (cheatgrass, downy brome) exhibit niche specialization. These pathogens utilize seed resources in complementary ways. *Pyrenophora semeniperda* is specialized to attack dormant seeds. It penetrates directly through the seed coverings. Hyphae ramify first through the endosperm and then throughout the seed. Seed death results as the embryo is consumed. In contrast, the *Fusarium* seed rot pathogen (*Fusarium* sp.) is specialized to attack non-dormant seeds in the early stages of germination. It cannot penetrate seed coverings directly. Instead, it responds to a cue emanating from the radicle end with directional hyphal growth and subsequent penetration at the point of radicle emergence, causing seed death. Non-dormant seeds usually escape *P. semeniperda* through germination even if infected because it develops more slowly than *Fusarium*. When water stress slows non-dormant seed germination, both *P. semeniperda* and *Fusarium* can attack and cause seed mortality more effectively. The *Fusarium* seed rot pathogen can sometimes reach epidemic levels and may result in *B. tectorum* stand failure ('die-off'). Stands usually re-establish from the persistent seed bank, but if *P. semeniperda* has also reached high levels and eliminated the seed bank, a die-off can persist indefinitely.

Introduction

An ecological niche is the sum of all the interactions of a species with the biotic and abiotic factors of its environment (Polechová and Storch, 2008). Niche theory predicts that two species with major niche overlap cannot coexist in the same environment, or in the case of plant pathogens, on the same host, indefinitely. Eventually one will be eliminated through competitive exclusion. In order to persist together, two species must exhibit some degree of niche specialization relative to each other. Many studies of niche specialization in fungi focus on closely related species, with the emerging generalization that related species often occupy divergent niches (Taylor *et al.*, 2014). In some cases differences in niche dimensions can be difficult to ascertain, especially for sibling species on the same host, but close examination has revealed niche differentiation based on separation in time, in space, or in resource use even in these cases (Fitt *et al.*, 2006). In other cases, where two plant pathogens are not closely related and have markedly different temporal or spatial patterns of resource use, the unique dimensions of species niche space are more easily determined.

In this paper we use the framework of niche theory to explore how two of the most important fungal pathogens in seed banks of the invasive annual grass *Bromus tectorum* can coexist at high abundance while apparently exploiting a common resource. *Bromus tectorum* is an invasive annual grass that forms monocultures over very large areas in the semi-arid western United States (Fig. 1A; Chambers *et al.*, 2014). It provides an abundant resource for a diverse community of fungal pathogens, including the two pathogens discussed here (Meyer *et al.*, 2016). *Pyrenophora semeniperda* (the 'black fingers of death' pathogen) is a well-understood seed pathogen that is abundant in *B. tectorum* seed banks but rarely causes direct population-level effects (Meyer *et al.*, 2007, 2016). The Fusarium seed rot pathogen (*Fusarium* sp.) also occurs regularly in *B. tectorum* seed banks and is the likely causal agent of *B. tectorum* stand failure or 'die-off' (Fig. 1B; Meyer *et al.* 2014a). We first briefly describe *B. tectorum* seed ecology and seed bank dynamics as a framework for the discussion of these two seed pathogens. We then examine pathogen life history and infection modality, focusing on contrasts that could indicate niche differentiation. Lastly we consider the interacting effects of these two pathogens on host population ecology in order to begin to elucidate the phenomenon of *B. tectorum* stand failure and recovery.

Figure 1. (A) *Bromus tectorum* monoculture near Dugway in Skull Valley, western Utah, USA. (B) *Bromus tectorum* die-off area (foreground) in Buena Vista Valley, central Nevada, USA.

Host seed ecology

Bromus tectorum monocultures present a relatively simple ecological setting for seed bank studies. The species has large seeds (mean floret mass *ca* 3 mg; Meyer, 2010) that are easily retrieved from the seed bank by screening and hand selection, and there are few, if any, other grass seeds in the samples that could confound identification. There is no regular mechanism for deep burial in wildland settings, and most viable seeds are found in the litter layer or in the top 2 cm of mineral soil (J. Beckstead, unpublished data). The seeds form a short-persistent seed bank, with an exponential decline in numbers of viable seeds in a seed cohort through time (Meyer *et al.*, 2007). Very few seeds survive in the soil seed bank for more than two years (Smith *et al.*, 2008).

The seed ecology of *B. tectorum* has been well studied by our group, including ecophysiological modelling of seed dormancy loss, germination, and secondary dormancy induction (Christensen *et al.*, 1996; Bauer *et al.*, 1998; Bair *et al.*, 2006; Meyer and Allen, 2009; Hawkins *et al.*, 2017) and examination of patterns of seed bank persistence in the field (Beckstead *et al.*, 2007; Meyer *et al.*, 2007; Smith *et al.*, 2008). These investigations have shown that seeds of this species are at least conditionally dormant at dispersal in early summer, lose dormancy through dry after-ripening under summer conditions, and are then poised to germinate rapidly in response to autumn rains. If autumn precipitation is insufficient to trigger full germination, the remaining viable seeds can enter secondary dormancy under partially hydrated conditions in late autumn or early winter. These seeds generally remain dormant through spring, lose dormancy over the following summer, and become germinable in time for autumn rains the following year. In exceptionally dry years, seeds may remain ungerminated but non-dormant through the winter, then germinate in response to spring precipitation. This phenological cycling of seeds between dormant and non-dormant states has important implications for seed pathogen attack.

Pyrenophora semeniperda

The Ascomycete genus *Pyrenophora* belongs to the family Pleosporaceae (order Pleosporales, class Dothidiomycetes). *Pyrenophora semeniperda* is the only known seed pathogen in the genus, and is only distantly related to species that are important foliar pathogens on cereal crops (Zhang and Berbee, 2001). *Pyrenophora semeniperda* is a common pathogen of cosmopolitan distribution (Yonow *et al.*, 2004; Stewart *et al.*, 2009) and has a very wide host range, particularly among cool-season grasses (Beckstead *et al.*, 2014, 2016). This fungus is an obligate pathogen known to reproduce only on seeds.

Pyrenophora semeniperda is an excellent candidate for study as a model system for understanding pathogenesis in soil seed banks. It forms macroscopic fruiting structures on seeds that make quantification in field seed bank samples a straightforward process (Fig. 2). It is most effective at attacking dormant *B. tectorum* seeds, either in summer before primary dormancy is lost (Hawkins, 2013) or after secondary dormancy is induced in late autumn in the carry-over seed bank (Meyer *et al.*, 2007; Hawkins *et al.*, 2017). *Pyrenophora semeniperda* reproduces asexually through the production of septate conidial spores that are produced on the tips of branched conidiophores formed along the margins of elongate, black stromatal structures (Fig. 2 F). These conidial spores are produced under dry conditions in spring or early summer, and remain in the seed bank until rains stimulate their germination. The spores can germinate anywhere on the surface of the floret coverings or the naked caryopsis wall (Fig. 2A; Finch-Boekweg *et al.*, 2016). They apparently do not require an immediate stimulus from the host seed in order to initiate growth. The mycelial network ramifies on the surface, forming club-shaped appressorial structures at each point of penetration into the seed (Fig. 2B). These structures enable the hyphae to penetrate directly through the caryopsis wall and proliferate in the testa layer. The fungus then sends hyphae directly into the heart of the endosperm, which is digested first, resulting in a hollowing out of the interior of the seed and dense ramification of melanized hyphae throughout the testa, making killed seeds rigid (Fig. 2C). The fungus then produces the characteristic 'black fingers' or stromata that protrude through the coverings at any location on the surface (Fig. 2D–F). The pathogen does not target the embryo directly, and embryo dissolution is slower than the digestion of the endosperm, possibly explaining how a non-dormant seed that is infected and shows disease signs can often produce a healthy seedling (Beckstead *et al.*, 2007). Eventually, however, the embryo of a dormant seed is consumed, resulting in seed death and abundant stromatal production, which typically occurs *ca* 14 days following inoculation.

The propensity for *P. semeniperda* to attack mostly dormant seeds in the seed bank can be explained by two features of this life cycle. First, the pathogen is able to effectively penetrate the seed coverings and does not require any signal from the germinating seed to initiate growth. This means that it can attack and kill dormant seeds. On the other hand, the fungus is relatively slow to develop once inside the seed, and primarily targets the endosperm rather than the embryo. This makes it possible for most non-dormant seeds under optimum germination conditions to escape

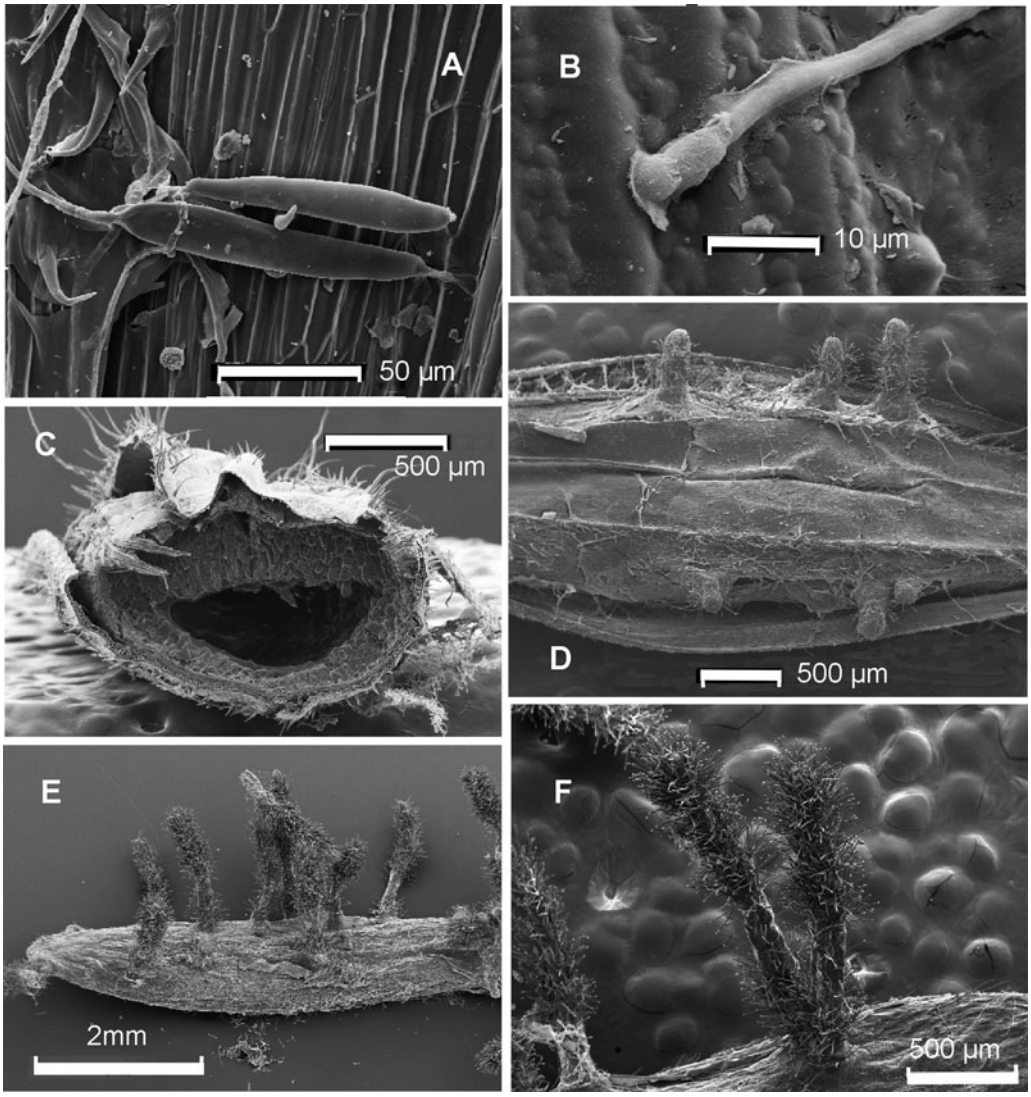

Figure 2. Stages in the life cycle of *Pyrenophora semeniperda*. (A) Germination of club-shaped conidia on the surface of a *B. tectorum* floret. (B) Formation of an appressorium for penetration into the floret tissue. (C) Floret cross-section at 10 days post-inoculation showing the hollowing out of the endosperm by the pathogen. (D) Pathogen stromata beginning to protrude from the killed seed. (E) Mature stromata protruding from the surface of a killed host seed. (F) Close-up of stromata with bristle-like conidiophores that bear the conidial spores. Photographs by Heather Finch-Boekweg; see Finch-Boekweg *et al.* (2016) for details.

mortality and develop into seedlings, which deprives the pathogen of resources that it can easily obtain from a dormant seed. Even dormant seeds can apparently defend themselves from attack, however, as disease development time is much reduced when killed seeds rather than living dormant seeds are inoculated, with stromata appearing within 5–6 days post-inoculation (Finch-Boekweg *et al.*, 2016).

Beckstead *et al.* (2007) coined the phrase 'race for survival' to describe the importance of dormancy status and thus seed germination rate in determining outcomes of seed infection by *P. semeniperda*. Fully after-ripened seeds germinated rapidly (<2 days) and escaped mortality, even though they could be infected and show disease signs on seeds that produced healthy seedlings. In contrast, dormant or slow-germinating seeds were almost always killed.

In spite of its adaptations for attacking dormant seeds, there is evidence that *P. semeniperda* can cause mortality of non-dormant seeds under some scenarios. One possible explanation is that there is genetic variation among strains in the ability to kill non-dormant seeds at high inoculum loads (Meyer *et al.*, 2010). Interestingly, the slowest-growing strains were better able to kill non-dormant seeds than faster-growing strains, in seeming contradiction to the 'race for survival' hypothesis. It was already known that this pathogen can produce large quantities of the cytotoxin cytochalasin B (Evidente *et al.*, 2002). This fungal secondary metabolite is well known for its ability to disrupt mitosis through its effect on actin spindle fibre formation (MacClean-Fletcher and Pollard, 1980). We determined that slow-growing strains of *P. semeniperda* make significantly more cytochalasin B than fast-growing strains, probably because the metabolic cost of producing this complex molecule limits resources for growth (Masi *et al.*, 2014; Meyer *et al.*, 2015). Increased cytochalasin B production might then be associated with increased ability to disable and kill germinating seeds, explaining why slow-growing strains caused higher mortality.

We also determined that cytochalasin B production by the fungus was induced only in solid culture on seeds or in liquid culture that contained seed constituents, further evidence of its role in pathogenesis (Meyer *et al.*, 2015). In addition, inoculation

into living dormant host seeds reduced cytochalasin B production by a factor of three relative to inoculation into killed host seeds, showing that living host seeds were able to suppress the production of this powerful toxin (Meyer *et al.*, 2015).

In contrast to the results for non-dormant seeds, we showed that fast-growing strains of *P. semeniperda* were more likely than slow-growing strains to cause mortality on dormant *B. tectorum* seeds at low inoculum loads (Meyer *et al.*, 2015), whereas at moderate to high inoculum loads mortality is very high regardless of strain (e.g. Beckstead *et al.*, 2007, 2016; Finch *et al.*, 2013). We reasoned that faster mycelial proliferation might be similar to increased inoculum load in its effect on seed mortality. We also used molecular-genetic techniques to show that a faster-growing strain had a competitive advantage over a slow-growing strain in terms of number of stromata produced in co-inoculations on dormant seeds, whereas the slow-growing strain produced more stromata than the fast-growing strain in competition on non-dormant seeds (Meyer *et al.*, 2015). Based on these results we proposed that temporally varying selection on different seed dormancy phenotypes could maintain the growth rate polymorphisms observed in natural populations of the fungus by favouring strains with slow growth rates in the transient autumn seed bank but strains with fast growth rates in the dormant seed bank.

Another mechanism that could explain how *P. semeniperda* causes non-dormant seed mortality in spite of the 'race for survival' is related to the intermittent autumn precipitation regime and resulting seed bed water stress characteristic of semi-arid habitats where this pathogen is most successful. Hydrothermal time models developed for *B. tectorum* seed germination indicate that few non-dormant seeds are germinable at water potentials <2 MPa (Christensen *et al.*, 1996). This fungus, on the other hand, can germinate and grow at water potentials as low as −10 MPa (Barth *et al.*, 2015). This suggests that the pathogen may have an advantage under conditions of intermittent water stress. Experiments with pre-incubation for 7 days at germination-suppressive water potentials resulted in seed mortality at optimum temperature (20°C) of 22% in water after the −2 MPa treatment and 47% after the −1.5 MPa treatment (Finch *et al.*, 2013). Seeds incubated in free water without a water stress pre-incubation treatment suffered no mortality. In further experiments, we showed that pathogen inoculum within seeds incubated in water for 24 h post-inoculation prior to transfer to much lower water potentials could complete most steps of pathogenesis over a 3-week period and then produce stromata almost immediately upon transfer to water, causing seed mortality (Allen *et al.*, in press). This process could take place at water potentials as low as −40 MPa.

Fusarium seed rot pathogen

The Ascomycete genus *Fusarium* belongs to the family Nectriaceae (order Hypocreales, class Sordariomycetes). The genus is extremely diverse, including plant pathogens that produce diseases with widely varying signs and symptoms on a broad range of hosts, including many important crop diseases. Based on molecular genetic analysis, the *Fusarium* seed rot pathogen found in *B. tectorum* seed banks belongs to one or more undescribed species within the *Fusarium tricinctum* species group (O'Donnell *et al.*, 2013; Franke *et al.*, 2014; S.E. Meyer, unpublished data). This group consists mainly of grass pathogens such as *F. tricinctum*, *F. avenaceum* and *F. acuminatum* that cause various head blight, seed rot and pre- and post-emergence seedling diseases. The process of naming one or more new *Fusarium* species from *B. tectorum* seed banks is underway; they will subsequently be referenced in this review as the *Fusarium* seed rot pathogen or simply as *Fusarium*.

The *Fusarium* seed rot pathogen was first identified in 2009 from greenhouse studies aimed at determining the causal organisms involved in *B. tectorum* stand failure or 'die-off' at two field sites (Meyer *et al.*, 2014a). This phenomenon manifests itself as total emergence failure in an area that supported a full stand of *B. tectorum* in the previous year. We hypothesized that this emergence failure might be caused by a fungal pathogen that could kill germinating seeds or germinants that had not yet emerged from the soil. In isolations from killed seeds in these experiments, 80% of the isolates obtained from the unsterilized soil treatment belonged to the genus *Fusarium* and were later shown to be members of the *F. tricinctum* species group. This strongly suggested that *Fusarium* might be implicated as a key die-off organism.

In pathogenicity trials with 16 of the isolated *Fusarium* strains, we found wide variation in the ability to kill non-dormant *B. tectorum* seeds, while there was low mortality on dormant seeds regardless of strain (Meyer *et al.*, 2014a; S.E. Meyer, unpublished data). Mortality on non-dormant seeds inoculated and incubated in free water averaged 24%, whereas on dormant seeds mortality averaged less than 5%. As with *P. semeniperda*, adding a 7-day water stress pretreatment that imitated intermittent water availability in the field greatly increased mortality, especially of non-dormant seeds. Mean mortality was increased to 55%, and the most aggressive strains caused mortality as high as 83%. Dormant seed mortality was also slightly increased with water stress (mean 12% mortality). These results showed that some of the *Fusarium* isolates were strongly pathogenic on *B. tectorum* seeds and could potentially play a role in stand failure in the field. The results also demonstrated that this pathogen targets non-dormant *B. tectorum* seeds, in direct contrast to *P. semeniperda*.

We used scanning electron microscopy to examine in detail the process of seed infection by the *Fusarium* seed rot pathogen (Franke *et al.*, 2014). As with *P. semeniperda*, fungal spores could germinate at any location on the surface of the floret or naked caryopsis (Fig. 3B), but instead of penetrating the seed coverings directly, the hyphae exhibited clear directional surface growth towards the floret attachment scar that would later be the point of radicle emergence (Fig. 3A). The mechanism driving this directional growth has not been determined with certainty, but the most likely explanation is that the germinating seeds produce a chemical cue that emanates from the tip of the embryo through the porous tissue of the attachment scar (Fig. 3A; Nelson, 1991). The fungus could detect this cue and follow the gradient to the point where the radicle will emerge. It then formed a conspicuous infection cushion over the attachment scar, visible macroscopically in inoculation trials as a 'white tuft' of mycelium (Fig. 3C,D; Franke *et al.*, 2014). With incubation under water stress, this infection cushion remained at a relatively constant size indefinitely, apparently unable to penetrate the embryo under these conditions. However, once the seeds were transferred to free water, the fungus could rapidly invade the embryo through the weakened endosperm cap, killing the seed while undergoing massive mycelial proliferation. In later experiments, we determined that a water stress treatment as short as 24 h was sufficient to give *Fusarium* the head start it needs to cause high mortality of germinating seeds (J. Beckstead, unpublished data).

The *Fusarium* seed rot pathogen does not develop a specialized appressorial structure for penetrating host tissue, relying instead

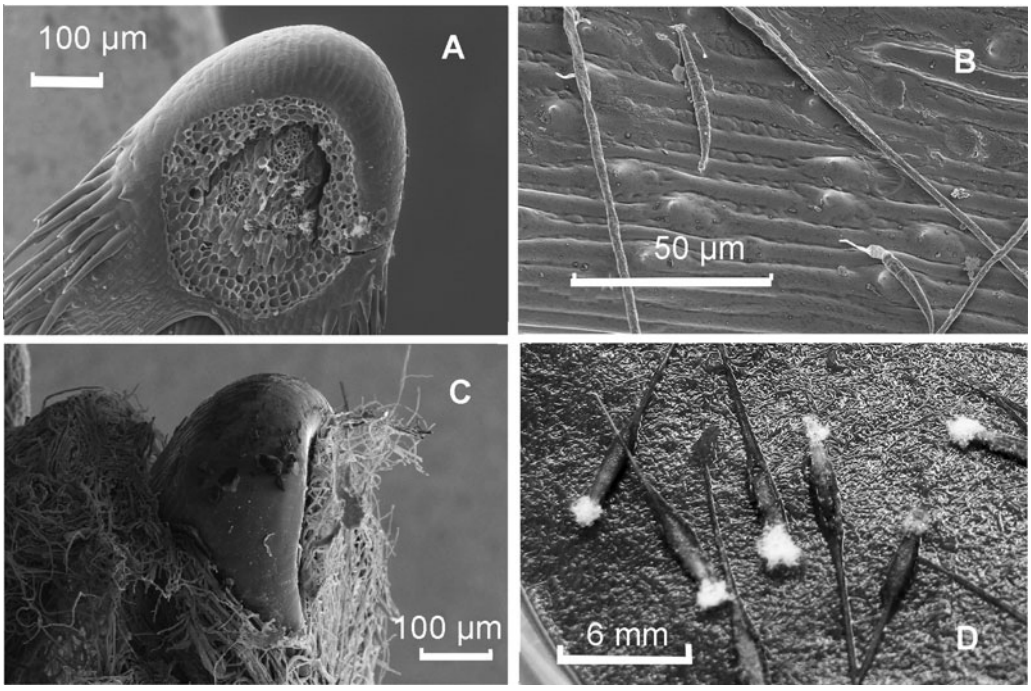

Figure 3. Stages in the life cycle of the *Fusarium* seed rot pathogen. (A) The floret attachment scar where pathogen attack will occur. (B) Adhesion and germination of sickle-shaped macroconidia on the surface of a *B. tectorum* floret. (C) Formation of the pathogen infection cushion over the floret attachment scar. (D) Infected florets in a Petri dish, showing the macroscopic infection cushions visible as 'white tufts'. Photographs by JanaLynn Franke Pearce; see Franke *et al.* (2014) for details.

on an infection cushion (Franke *et al.*, 2014). This limits its ability to infect directly through more resistant host tissues, including seed coverings. Adaptations for locating zones of weakness where this penetration method can be effective are thus under strong selection, including a sensing mechanism for assuring that the infection cushion forms over the most vulnerable tissue. Because this sensing mechanism apparently depends on exudates from the radicle end of a seed that has initiated germination, it largely restricts attack by this pathogen to germinable, i.e. non-dormant, seeds. Occasionally a 'white tuft' can form at a point on the surface where a seed has suffered mechanical damage; this may be one way that dormant seeds can be attacked.

The *Fusarium* seed rot pathogen also appears to be a generalist on seeds of many grasses Host range testing has not been systematically performed for these strains, but some strains are known to attack seeds of at least two native cool-season grass species (Meyer *et al.*, 2014b; J. Beckstead, unpublished data).

Pathogen niche specialization

When the adaptations exhibited by the two major pathogens on *B. tectorum* seeds in wildland seed banks are examined together, it is apparent that they occupy distinct niches in the *B. tectorum* seed bank pathogen community and probably experience limited competitive interaction, in spite of exploiting the same seed resource. The *Fusarium* seed rot pathogen targets *B. tectorum* seeds in the autumn when they are non-dormant and ready to germinate. It is a relatively fast-growing fungus that can achieve success even in environments with reliable autumn rains that elicit fast seed germination. The pathogen is likely to experience even greater success in drier areas with less reliable precipitation, given the advantage it enjoys under intermittent precipitation scenarios. It is clearly specialized to detect and respond to germinating seeds by targeting its attack to the point of radicle emergence where it can quickly destroy the embryo.

In contrast, the slower-growing pathogen *P. semeniperda* targets dormant seeds in the seed bank through its ability to form powerful penetration structures that can puncture seed coverings and reach endosperm reserves directly from any position on the exterior of the floret. By attacking dormant seeds, it ensures that all seed resources will be available for its use in spite of its relatively slow growth rate and the fact that it does not directly target the embryo. Intermittent precipitation scenarios in autumn also potentially increase *P. semeniperda* success, but because of its slow development, it requires much longer periods of water stress to achieve high seed mortality, whereas *Fusarium* can cause high mortality after a single relatively short water stress period. This would appear to give *Fusarium* a competitive advantage under intermittent autumn precipitation scenarios, which would in turn tend to select for even more specialization on dormant seeds in *P. semeniperda*. The exact environmental conditions for successful exploitation of water stress to increase the infection window may be sufficiently different for the two pathogens that interspecific competition even in this narrowly defined part of the niche space is minimized.

Die-off causes and consequences

Our current hypothesis for how these two pathogens interact to influence *B. tectorum* stand failure and recovery is that the *Fusarium* seed rot pathogen is the primary organism responsible for the large-scale seed death that results in stand failure, while *P. semeniperda* plays a role in the rate of post-stand failure recovery through its impacts on the persistent seed bank.

We first tested the hypothesis that *P. semeniperda* was directly implicated in stand failure by examining pathogen loads in both

die-off areas and adjacent areas with current-year stands (Baughman and Meyer, 2013). We found no evidence that this pathogen could cause stand failure, even though it can potentially reduce stand density through its impact on non-dormant seeds. Seed densities in the *B. tectorum* autumn seed bank can number in the tens of thousands per square metre (Meyer *et al.*, 2007; Smith *et al.*, 2008), so that even significant mortality can leave ample densities of germinating seeds for stand establishment. This makes it even more remarkable that any pathogen can cause complete stand failure.

The case for *Fusarium* as the primary die-off pathogen is also still not conclusive (Meyer *et al.*, 2014a), but its proven ability to kill germinating *B. tectorum* seeds in pathogenicity trials coupled with its ability to reduce stand density in field inoculum addition experiments (S.E. Meyer, unpublished data) is strong evidence that it is one of the primary players. Another line of evidence that *Fusarium* is implicated in die-offs comes from Baughman and Meyer (2013). Post-die-off viable seed densities in carry-over seed banks were not statistically different in paired die-off and control sites, indicating that the pathogen killed germinating seeds but did not attack dormant seeds.

Stand recovery the year following *B. tectorum* stand failure is the most common pattern observed in the field (Weisberg *et al.*, 2017). The presence of a persistent seed bank not affected by the die-off causal agent, as mentioned above, is the most likely explanation for this stand recovery. Most die-offs are therefore transient phenomena. However, sometimes stand recovery does not occur so quickly. Two alternative hypotheses have been proposed to explain this. First, the organism that caused the die-off may still be active the following year. Precision seeding experiments (using toothpick-mounted seeds that could be followed individually) in die-off areas and adjacent areas with intact stands at two sites showed no significant difference in *B. tectorum* emergence the year following a die-off (Nicholson, 2014). This makes residual effects of the pathogen an unlikely explanation for a persistent die-off. A second hypothesis is that there was insufficient carry-over seed to establish a post-die-off stand. We examined this possibility with a precision seeding experiment into an unrecovered die-off, and demonstrated that seed addition was the only requirement for stand establishment (Nicholson, 2014). This supported the idea that lack of a persistent seed bank following the die-off was the cause of slow stand recovery. While we have no direct evidence that the carry-over seed bank was eliminated due to *P. semeniperda* attack, this remains the most likely explanation.

Conclusions

Mechanistic or even descriptive studies of natural pathosystems involving seeds as hosts are uncommon, in spite of their potential importance as mediators of host population dynamics (Chambers and MacMahon, 1994; Gilbert, 2002). The seed bank pathogen community is more often treated as a 'black box', that is, an assemblage of multiple pathogens that act in concert to affect seeds. These are manipulated as a group, for example, through fungicide applications (Blaney and Kotanen, 2001; Orrock and Damschen, 2005) or through the measurement of soil legacy effects (Bever, 1994; Klironomos, 2002). Our investigations have made a unique contribution to the study of seed bank pathosystems in natural settings by focusing on the biology and ecology of specific pathogens, enabling us to more fully understand their effects on host seed ecology.

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