A Predictive Model for Soil Seed Bank Outcomes in the *Pyrenophora semeniperda–Bromus tectorum* Pathosystem

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Abstract

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Pyrenophora semeniperda is abundant in soil seed banks of *Bromus tectorum*, where it kills a fraction of seeds throughout the year. The pathogen engages in a race with host seeds for endosperm resources; the pathogen success is negatively correlated with seed germination speed. We developed a deterministic model to predict pathosystem outcomes (seed death versus seed escape), using seed bank data from 80 sites collected over a 13-year period. The response variable (killed seeds in the spring seed bank) was regressed on multiple predictor variables (pathogen and host densities at seed dispersal, amount and timing of precipitation). Increased mortality was associated with high seed rain, high pathogen density, and low autumn precipitation. On xeric sites, a positive feedback loop between pathogen and host is created by a large carryover seed bank containing secondarily dormant seeds vulnerable to fungal attack and results in higher inoculum loads at seed dispersal the following year.

Keywords: *Pyrenophora semeniperda*; *Drechslera campanulata*; *Bromus tectorum*; deterministic model; seed pathogen

Bromus tectorum is an exotic winter annual native to Eurasia. This grass was introduced into western North America during the late 1800s, probably as a contaminant in grain seed (Mack 1981). It is highly invasive in cold deserts of western North America and has become the most common vascular plant in the region (Meyer *et al.* 2007). The generalist pathogen *Pyrenophora semeniperda* attacks seeds of cool-season grasses, and is abundant in seed banks of *B. tectorum.* Its impact depends upon seed germination rate; seeds that germinate quickly escape mortality, while those that germinate slowly are often killed. Fast-germinating *B. tectorum* seeds in the autumn seed bank are rarely killed, while dormant seeds in the potential carryover seed bank can suffer high mortality.

MATERIAL AND METHODS

Using seed bank data (i.e. seeds retrieved from the top 4 cm of soil in late spring before current-year seed dispersal or in early autumn before germination-inducing precipitation) from 80 *in situ* populations across a range of habitats throughout the western United States collected over a 13-year period (e.g. Beckstead *et al.* 2007, 2010) we developed a deterministic model to predict pathosystem outcomes (seed death versus seed escape). A subset of seed bank collections was chosen for model development, and a separate subset for model validation. The response variable (killed seeds present in the spring seed bank) was regressed on multiple predictor variables (pathogen and host densities at seed dispersal, amount and timing of precipitation) to obtain equations for model predictions.

Model development required the knowledge of key life cycle stages for both host and pathogen. *B. tectorum* is a facultative winter annual. Seed populations produce variably dormant seeds in early summer, but this primary dormancy is lost through dry after-ripening at warm temperatures (Meyer *et al.* 1997; Meyer & Allen 2009). Seeds germinate in response to autumn rains if precipita-

tion is adequate (Bauer *et al.* 1998). In years and habitats with insufficient autumn precipitation, seeds may postpone germination until winter or spring. However, a fraction of the seed population is induced into dormancy by winter conditions (i.e. secondary dormancy, Hawkins *et al.* 2013). These dormant seeds have the potential to carry over across years as components of the soil seed bank. The persistent seed bank of *B. tectorum* (i.e. the fraction that carries over for one year at least) is highly variable, but it is unlikely that seeds persist in the soil beyond 3 years (Meyer *et al.* 2007).

P. semeniperda is commonly observed on grass seeds as stromata of the asexual (anamorph) state of the fungus, *Drechslera campanulata* (Finch *et al.* 2013). These stromata are macroscopically visible as black finger-like protrusions (up to 1 mm in diameter and up to a centimetre or more in length). They bear conidiophores that produce conidia (spores), which are then released into the seed bank. Spores on the surface of seeds germinate within 8 h following wetting, penetrate seed coverings, and grow as mycelium inside the seeds (Finch-Boekweg *et al.* 2013). Under ideal conditions (e.g. 20°C in free water), stromata begin to appear about 11 days following infection. With continuous hydration in field and laboratory studies, most seeds killed by the fungus are dormant; non-dormant seeds escape death by germinating quickly. However, because the fungus can grow at water potentials below those that allow for seed germination, intermittent hydration resulting in delayed seed germination favours the fungus. Seed dormancy as well as timing and amount of precipitation inputs are critical variables in determining the level of the pathogen success due to their influence on germination speed.

RESULTS AND DISCUSSION

Important triggers in our model are summarised in Figure 1. Increased seed mortality was associated with high seed rain, high pathogen density and low autumn precipitation (which resulted in high seed bank carryover). Predicted and observed mortality was generally greatest on xeric sites due to lower autumn germination and subsequently greater carryover seed bank. We were able to observe and predict specific scenarios that produced very high mortality of *B. tectorum* seeds (i.e. repeated high pathogen and host densities coupled with high seed bank carryover) on xeric sites (where *P. semeniperda* killed up to 20 000/m^2, or > 90% of potential carryover seeds). On mesic sites, rapid germination of non-dormant seeds in autumn resulted in lower pathogen-caused mortality. Year-to-year variation in pathogen inoculum loads, host seed rain, and weather patterns resulted in wide variation in this general pattern. These results

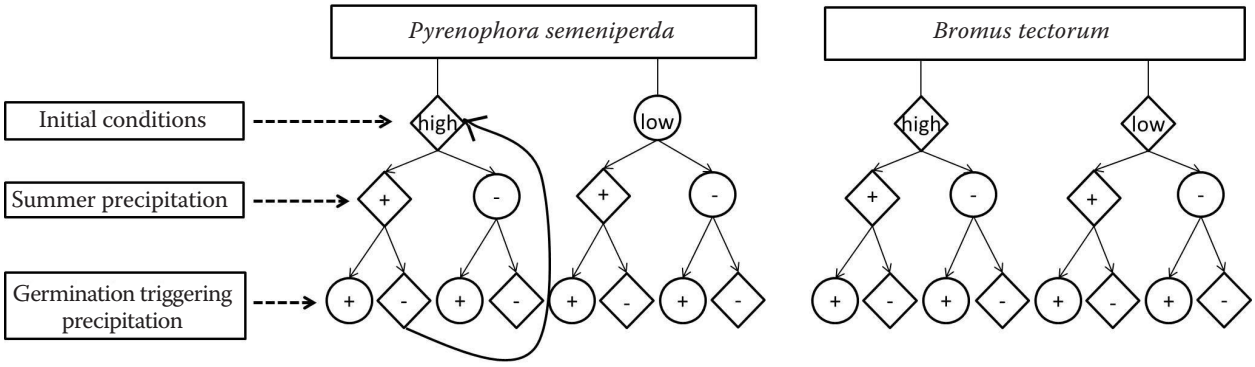

Figure 1. Prediction triggers for *Pyrenophora semeniperda*-caused seed death to *Bromus tectorum*. Initial conditions represent high or low levels of the pathogen and host, respectively. For initial conditions and precipitation events, a ◇ indicates prediction for a higher proportion of killed seeds while a ○ indicates prediction for a lower proportion of killed seeds. Note that summer precipitation, when seeds are still dormant, predicts increased mortality, while germination-triggering precipitation (i.e. sufficient precipitation for seeds to rapidly complete radicle emergence) at any other time (autumn, winter, or spring) predicts reduced mortality. The absence of germination triggering precipitation results in secondary dormancy induced by winter conditions, resulting in increased predicted mortality. Predictions can be made using initial conditions alone, or in combination with precipitation. The positive feedback loop shown is created by a large carryover seed bank (i.e. secondarily dormant seeds highly vulnerable to attack by the fungus) and results in higher inoculum loads at seed dispersal the following year

support our hypothesis that the *P. semeniperda* –*B. tectorum* pathosystem is more significant on xeric sites, where a positive feedback loop between pathogen and host is created by a large carryover seed bank (i.e. containing a level of secondarily dormant seeds that are highly vulnerable to attack by the fungus) and results in higher inoculum loads at seed dispersal the following year.

The highest *in situ* conidia production of *P. semeniperda* has been observed on previous-year *B. tectorum* caryopses in late spring (Meyer *et al.* 2007), shortly before current-year seeds mature. Most of these spores germinate during the first significant precipitation event after production. Seeds may therefore become infected any time during the year, resulting in tremendous variation in pathosystem outcomes (Finch *et al.* 2013).

While our modelling efforts resulted in successful qualitative predictions, quantitative predictions of seed death versus seed escape have been less accurate. One difficulty associated with quantitative predictions is our limited understanding of mycelium growth and survival in seeds subjected to wide fluctuations in soil water. For example, *B. tectorum* seed populations are characterised by a high degree of primary dormancy upon maturity. These seeds fail to germinate under field conditions, and are highly vulnerable to infection if summer precipitation occurs (Finch *et al.* 2013). However, a single summer precipitation event in semi-arid habitats is likely to wet soil for only a few hours to a few days (Meyer & Allen 2009), which is insufficient for the completion of conidial production. Success of the fungus during summer is therefore dependent on the ability for disease development to continue across successive episodes of hydration followed by desiccation. Furthermore, the fungus must survive a prolonged exposure to high soil temperatures between precipitation events. If the fungus is unable to survive in seeds during summer conditions, seed dormancy loss through after-ripening and associated rapid germination in the autumn may result in very low seed mortality. In contrast, if disease development continues across hydration-desiccation episodes, then autumn precipitation may result in high seed mortality. In conclusion, efforts to more accurately predict pathosystem outcomes will result from an improved understanding of the fundamental biology of the pathogen, including the knowledge of disease development under conditions where conidial production is inhibited.

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