

Smut fungus infection in the perennial grass *Andropogon virginicus* (broomsedge): individual and population level consequences

Bojdani, E., B. Graf, R.P. Shupak, A. Romanchuk, J.J. Decker, and J.A. Morrison

Department of Biology, The College of New Jersey, P.O. Box 7718, Ewing, NJ, 08628

Abstract

Pathogens have important effects on individual plants and their populations, but have not been well studied. We investigated a plant-pathogen system in which the perennial old-field grass *Andropogon virginicus* (broomsedge) is attacked by the parasitic smut fungus *Sporisorium ellisii*. This smut replaces its host plant's reproductive structures with its own teliospores. In a three-year field study conducted in New Jersey from 2004-2006, we investigated the relationship between host population density and disease frequency by counting infected and healthy plants within 480 permanent 1 m² plots each year. The effect of disease upon individual plants was measured on focal plants. Data from a 2002 pilot study of 192 plots provided a population baseline, with mean plant density 5.47 / m² and, on average, 29.8% plants infected per plot. By 2006 mean density had decreased to 3.26 and mean disease to 5.9%. Higher disease rates were correlated with lower plant density in plots where disease was present, suggesting that smut fungus infection has a negative effect on the broomsedge population. Fully infected plants were smaller than healthy plants, were at significantly higher risk for mortality, and produced no seeds. Although 21.2% of infected individuals recovered from disease, 70% of infected plants died after two years compared to 43% mortality in healthy plants. Therefore *S. ellisii* has a negative effect upon *A. virginicus* and may act both in population regulation and, potentially, as an agent of natural selection that drives the evolution of *A. virginicus* populations towards increased resistance.

Introduction

Plant pathogens can play an important role in the evolutionary ecology of host populations, yet little is known about patterns of disease, mortality, and resistance within natural populations. Understanding these patterns relies on an exploration of the interaction between plant and pathogen across space at geographical scales which are heterogeneous in environmental factors and biotic factors, including the patchy growth regimes of the host plant population. Determination of the distribution and spatial dynamics at play within plant-pathogen populations is essential for further investigation into the coevolution and regulatory mechanisms of coexisting species.

By monitoring a plant-pathogen system with marked focal plants in a mapped local population over the course of three years, we were able to characterize the spatial and temporal patterns of disease and begin to determine the role it plays in the regulation of host population dynamics. We conducted our research in a New Jersey field dominated by the perennial C₄ bunch grass *Andropogon virginicus* (broomsedge). Broomsedge is an early successional species that is native to eastern North United States, but has also been introduced to California and to Hawaii, where it is highly invasive. It flowers between September and November, and disperses tiny seeds throughout the winter. Infection by the smut fungus *Sporisorium ellisii* prevents flower development, instead replacing seeds with its own black teliospores. By reducing or eliminating reproduction, this pathogen may play an important ecological role in the host population. Our population study analyzed disease rates and host plant density over time and space, and included detailed observations of marked focal plants throughout the population to study the relationship between plant size, mortality, and disease history.

Methods

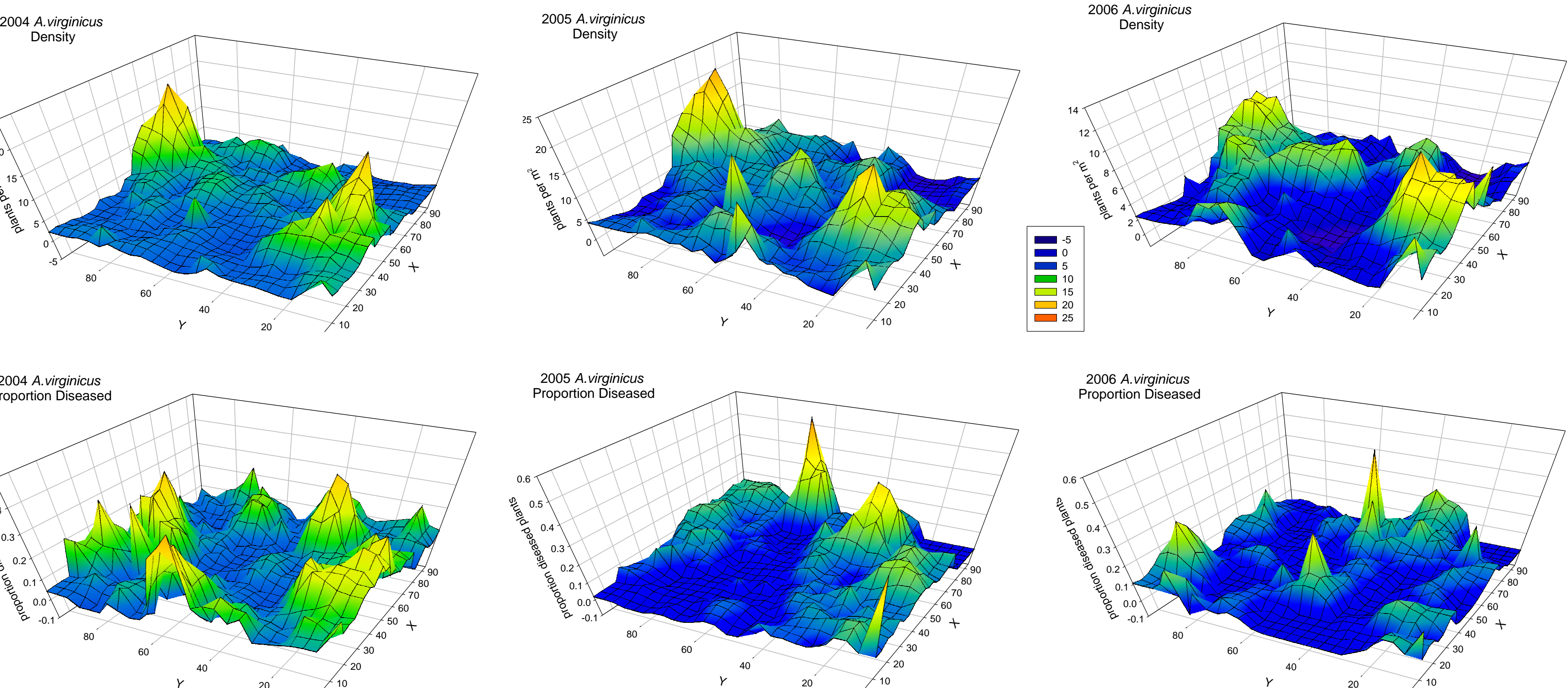
- Gridded a 100m x 100m portion of large old-field community where broomsedge is the dominant species
- Designated 480 permanent regularly spaced 1 m² plots.
- Tagged a focal plant in each plot (infected or uninfected) if broomsedge was present.
- Measured broomsedge density and disease frequency within each plot yearly from 2004-2006.
- Measured focal plant height; counted number of shoots with no infection, partial infection, and total infection.

Acknowledgements This work was supported by grant USDA-NRI # NJR-2004-01992 to J.A. Morrison, an American Society of Plant Biologists Summer Undergraduate Research Fellowship to A. Romanchuk, the Support of Scholarly Activity (SOSA) Committee of The College of New Jersey (TCNJ) for sabbatical leave and reassigned time to J.A. Morrison, and TCNJ's Biology Summer Research Program (BSRP). Thanks to other TCNJ students who contributed field data: Brian Policastro, Tony Mazzarella, Theo Sabelnik, Sara Smith, Kara Horner, and Mirjana Jojic.

Results

Figure 1. Spatial and temporal patterns of plant density and disease, 2004-2006.

Mean plants/m² ranged from 5.47 in a 2002 pilot study to 3.59 in 2004, 5.52 in 2005, and 3.26 in 2006. Density was positively correlated across all years (all $r > 0.64$, all $P < 0.0001$, correlation with CRH correction for spatial autocorrelation).



Mean disease frequency in 1m² plots ranged from 29.8% in the 2002 pilot study to 10.6% in 2004, 8.9% in 2005, and 5.9% in 2006. Disease frequency was positively correlated across adjacent years only (2004-2005, $P < 0.0001$; 2005-2006, $P = 0.0005$). In plots with disease, disease frequency was negatively correlated with plant density, in each year ($r = -0.50$ in 2004, 0.45 in 2005, -0.63 in 2006; all $P < 0.001$, with CRH correction). There was no evident relationship between disease and density across adjacent years.

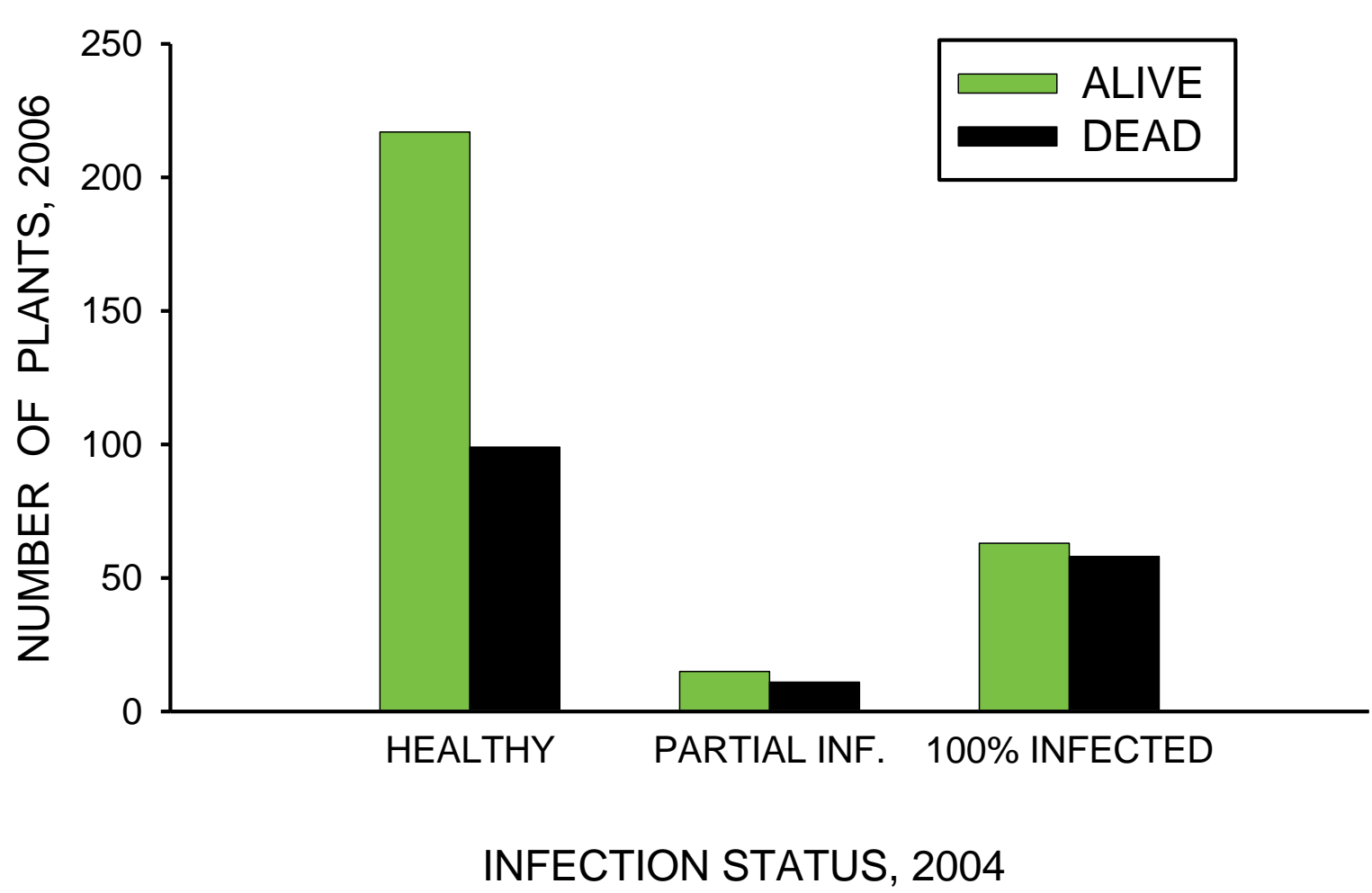


Figure 2. Mortality of focal plants with different infection status. Infected plants were more likely to die between 2004 and 2006 than were healthy plants (χ^2 for heterogeneity = 10.87, $P < 0.005$, $df = 2$).

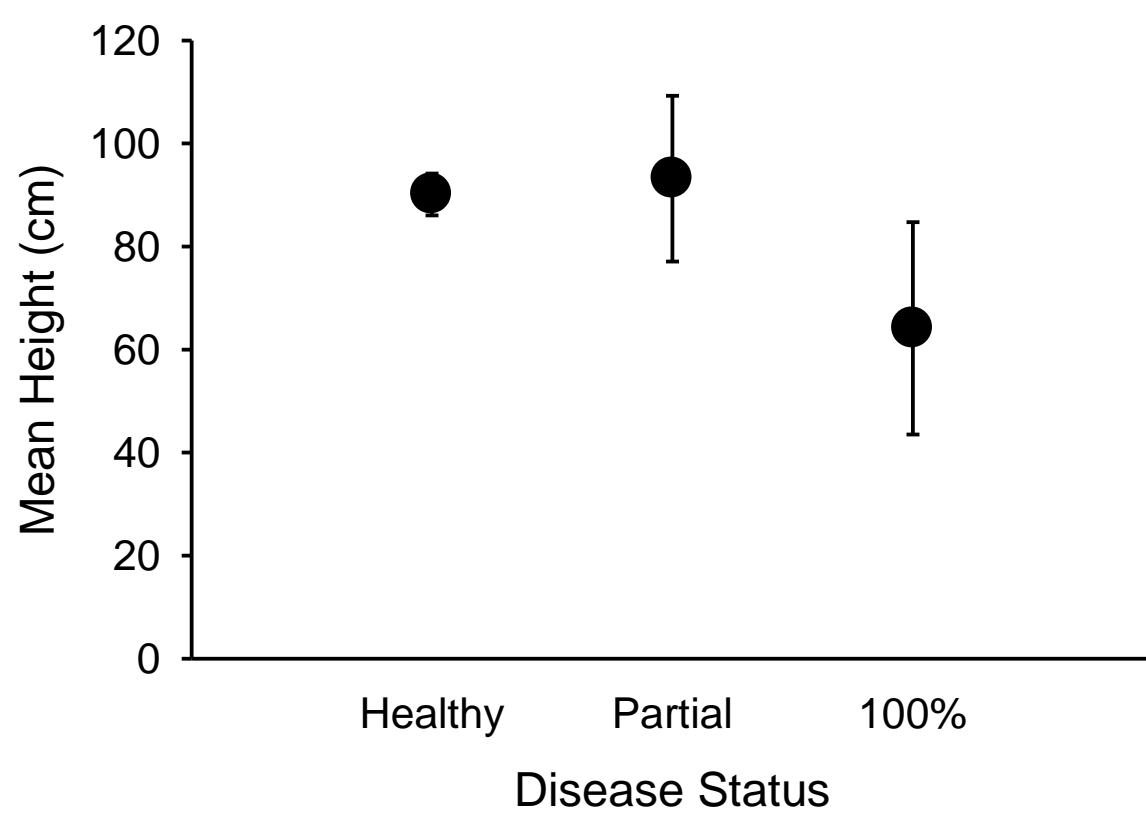


Figure 3. Height of tallest shoot on focal plants with different infection status in 2006. Healthy plants and plants with just some infected shoots grew to the same height. Completely infected plants were shorter (ANOVA: $F = 4.25$; $df = 2,166$; $P = 0.016$).

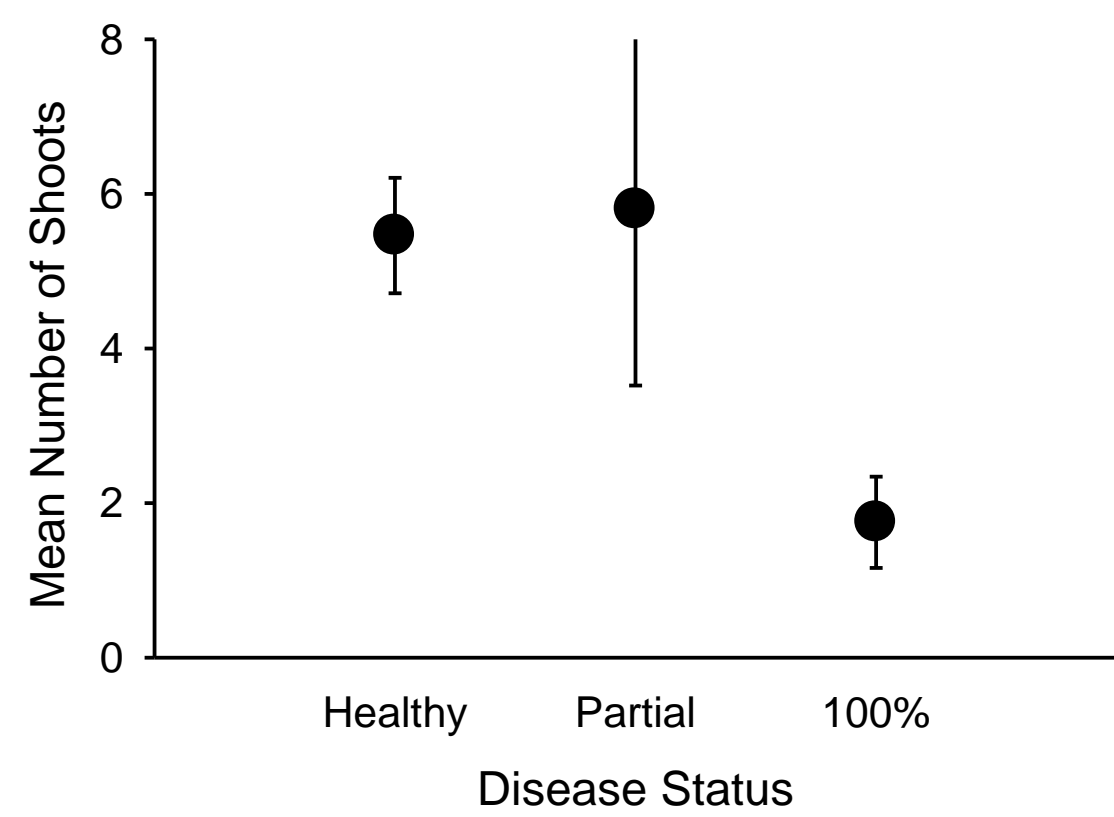


Figure 4. Number of shoots on focal plants with different infection status in 2006. Healthy plants and plants with just some infected shoots produced a similar number of shoots. Completely infected plants produced fewer shoots (ANOVA: $F = 3.47$; $df = 2,166$; $P = 0.030$).



Clockwise from top left: study site broomsedge population, healthy grass, non-native population in Hawai'i, smutted grass.

Discussion

Completely infected plants were smaller, and more partially and fully infected plants died, suggesting that this disease may negatively affect host population size. An infected plant may allocate resources that would otherwise be used for growth and reproduction toward fighting off disease. The disease may also act as an agent of natural selection, selecting for increased resistance in the host. The negative relationship between host density and disease within years also suggests a regulatory role for the pathogen; where disease was high host density was low, within any one year. However, the correlations did not show a relationship between disease in one year and density in the next year; we may need more time to detect the effect of current disease on future host dynamics.

Disease and density were very dynamic, as seen in the surface plots. Mean disease decreased dramatically over the years, and density fluctuated. Both disease and density may be influenced by disturbance. Mowing, which last occurred in 2003, wounds plants and may spread the pathogen, and it also halts succession, thereby promoting broomsedge density. Deer beds also disrupt the vegetation in this site.

The remaining challenge, in this study as in other plant-pathogen studies, is to determine how important disease is for host regulation in the context of the many other environmental factors that can influence population dynamics. Does disease matter?