Spatial and temporal dynamics of a smut fungus – grass interaction

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Abstract

Disease dynamics and host population dynamics in natural plant pathosystems remain poorly understood, with only a handful of well-studied systems. We investigated an interaction in which the common perennial old-field grass *Andropogon virginicus* (broomsedge) is attacked by the parasitic smut fungus *Sporisorium ellisii*, which replaces the plant's reproductive structures with its own teliospores. From 2004-2006, in a 100 x 100 m population, we investigated the relationship between host population density and disease frequency by counting infected and healthy plants within 480 permanent 1 m^2 plots each year. The effect of disease on individuals was measured on focal plants. Mean plants/m^2 ranged from 5.47 in a 2002 pilot study to 3.59 in 2004, 5.52 in 2005, and 3.26 in 2006. Plant density in plots was positively correlated across all pairs of years (all r > 0.64, all P< 0.0001, correlation with CRH correction for spatial autocorrelation). Mean disease frequency in plots ranged from 29.8% in the 2002 pilot study to 10.6% in 2004, 8.9% in 2005, and 5.9% in 2006, and was positively correlated across adjacent years only (2004-2005, P<0.0001; 2005-2006, P = 0.0005). In plots with disease present, 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). 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. *Sporisorium ellisii* has a negative effect on *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.

Old-field population of *Andropogon virginicus*.

Introduction

Andropogon virginicus (broomsedge) is a warm-season C_4 perennial grass that dominates the vegetation during early old field succession in eastern North America, particularly on poor soils. It has been introduced to California, where it is naturalizing, and to Hawaii, where it is invasive. In its native range populations are often found infected with a specialist pathogen, the smut fungus *Sporisorium ellisii*. This pathogen sporulates in the grass spikelets, eliminating seed production on infected tillers. Infection may occur in all tillers (total infection) or in some (partial infection). In order to understand the role this pathogen may have in host population regulation and evolution, and its potential as an agent of control in the invasive range, an infected population in New Jersey was followed for three years.

Smut fungi generally are important pathogens of graminoids, both in agricultural grain crops and in natural populations. They have the potential to be especially influential in their host's population biology because of their direct negative effect on reproduction and therefore plant fitness. However, only a handful of plant-pathogen systems have been studied in detail in natural populations.

Methods

A pilot study was started in 2002 in an old-field plant community developing on fallow agricultural land located in Mercer County Park Northwest, New Jersey, U.S.A. At that time the community was dominated by *A. virginicus,* and infection by *S. ellisii* was very apparent. 192 1-m^2 plots were established in a systematic repeating pattern of grouped plots. In 2003 this field was mowed, so new plots were established in 2004. The sampling pattern was expanded to 480 plots, consisting of groupings that provided a complete range of distances between plots, to enable spatially anchored analysis. Overall, the plots were arrayed across a 100 m x 100 m space.

In 2002, 2004, 2005 and 2006 the plots were assessed for number of healthy plants and number of infected plants. The 2004 - 2006 plots were identical across years, but the 2002 plots were located only in one section of the space and each plot's exact location was slightly different from the plot positions in later years in that section (because of the mowing). Also, in each plot a focal plant was tagged, and in 2004, 2005, and 2006 these individuals were measured for mortality, disease status, and size.

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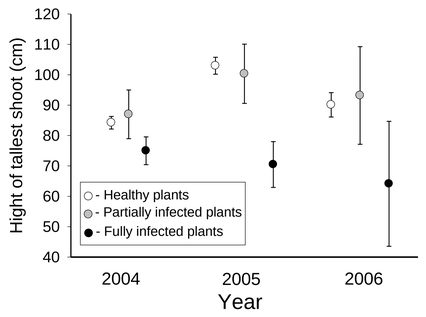
Results

Focal Plants

Smut fungus infection . . .

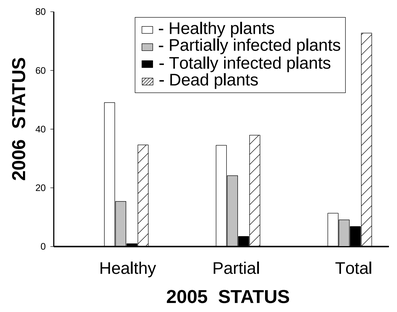
. . . eliminated seeds in infected tillers

. . . reduced plant size



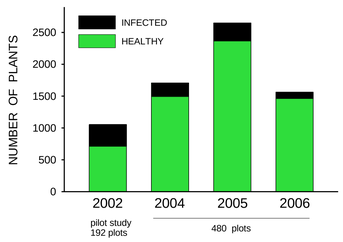
Backtransformed means ± 95% CL. ANOVA: 2004 df=2,329; F=10.98; P=0.0001. 2005 df=2,273; F=41.72; P=0.0001. 2006 df=2,166; F=4.25; P=0.0159

. . . increased plant mortality

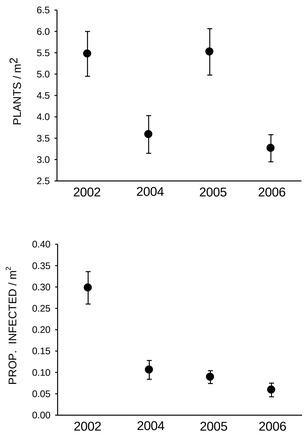


Plots

Disease frequency among all of the plants included in the study declined from 0.32 in 2002 to 0.13 in 2004, 0.11 in 2005, and 0.06 in 2006.



Mean plant density per plot varied widely among years, and mean disease frequency per plot declined.



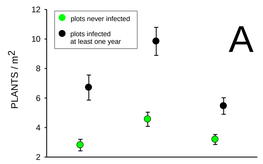
Means ± 95% CL. Top: n = 192 in 2002, 480 in other years. Bottom includes only plots where *A. virginicus* was present: n= 175 in 2002, 333 in 2004, 352 in 2005, 342 in 2006.

In 1 m^2 plots, across years, plant density was positively correlated, as was disease frequency in adjacent years (Table 1).

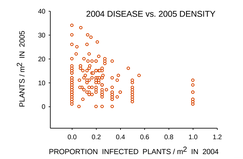
In all plots with the host plant, there were some weak positive disease-density correlations (2005 density and disease, 2004 disease and 2006 density, 2004 density and 2005 disease; Table 1).

Plots with the host plant that had disease present in at least one year had much greater plant density than plots where disease never appeared (Figure A).

Including plots only where disease was present, disease and density were strongly negatively correlated (Figures B and C include data only from plots with disease present in at least one year).



Means ± 95% CL. T-tests for all years had P<0.0001 (n, left to right = 149, 192, 167, 192, 164, 192)



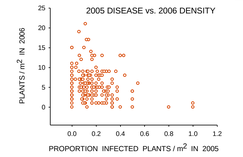
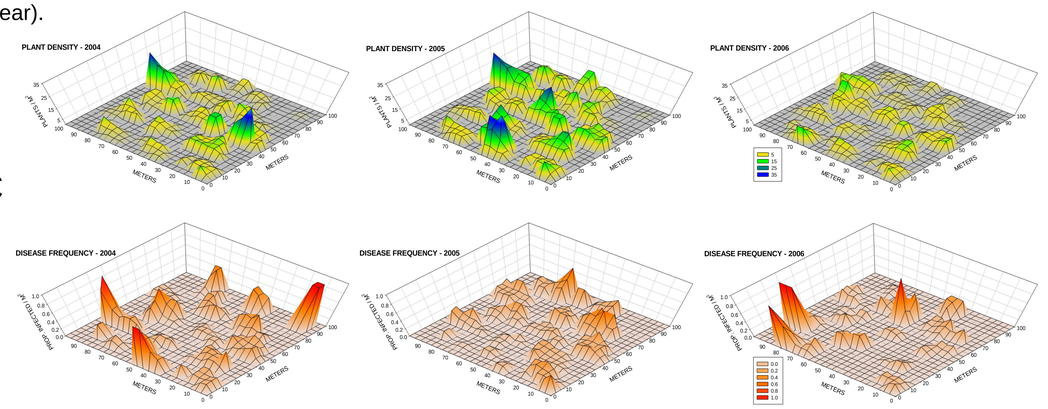


Table 1. Correlations with CRH correction for spatial autocorrelation

VARIABLES	CORRELATION	CORRECTED P	EFFECTIVE SAMPLE SIZE	NOTES
2004 DENSITY – 2005 DENSITY	0.76	<0.0001	172	ALL PLOTS
2005 DENSITY – 2006 DENSITY	0.72	<0.0001	181	
2004 DENSITY – 2006 DENSITY	0.64	<0.0001	169	
2004 DISEASE – 2005 DISEASE	0.50	<0.0001	310	ALL PLOTS WITH PLANT PRESENT IN BOTH YEARS
2005 DISEASE – 2006 DISEASE	0.20	0.0003	332	
2004 DISEASE – 2006 DISEASE	0.10	0.08	316	
2004 DISEASE – 2004 DENSITY	0.10	0.08	318	
2005 DISEASE – 2005 DENSITY	0.16	0.02	202	
2006 DISEASE – 2006 DENSITY	0.02	NS	346	
2004 DISEASE – 2005 DENSITY	0.06	NS	303	
2005 DISEASE – 2006 DENSITY	0.13	0.06	197	
2004 DISEASE – 2006 DENSITY	0.16	0.006	283	
2005 DISEASE – 2004 DENSITY	0.24	0.0008	190	
2006 DISEASE – 2004 DENSITY	0.05	NS	302	
2006 DISEASE – 2005 DENSITY	0.01	NS	321	
2004 DISEASE – 2005 DISEASE	0.25	0.002	148	ALL PLOTS WITH DISEASE PRESENT IN AT LEAST ONE OF THE YEARS
2005 DISEASE – 2006 DISEASE	-0.12	NS	139	
2004 DISEASE – 2006 DISEASE	-0.26	0.003	130	
2004 DENSITY – 2004 DENSITY	-0.50	<0.0001	103	
2005 DENSITY – 2005 DENSITY	-0.22	0.01	140	
2006 DENSITY – 2006 DENSITY	-0.63	<0.0001	64	
2004 DISEASE – 2005 DENSITY	-0.45	<0.0001	103	ALL PLOTS WITH DISEASE PRESENT IN THE DISEASE YEAR
2005 DISEASE – 2006 DENSITY	-0.32	0.0003	130	
2005 DISEASE – 2004 DENSITY	-0.22	0.01	140	
2006 DISEASE – 2004 DENSITY	-0.32	0.01	63	
2006 DISEASE – 2005 DENSITY	-0.40	0.0008	67	

C



Discussion

The consequences of infection by *S. ellisii* were dire for individual *A. virginicus* plants. Mortality of infected plants was much higher, they tended to be smaller, and reproduction was eliminated or reduced. Consequently, this pathogen has potential as a powerful force of natural selection, assuming that host suscetibility has a genetic basis and is not overwhelmed by environmental factors that influence either or both the pathogen's virulence and the host's response.

At the population level the effect of *S. ellisii* on *A. virginicus* is complex. Disease appeared in plots of much higher density than plots where disease never appeared, which suggests a threshold density for disease spread, as predicted by epidemiology. However, once disease was present, those plots with higher disease levels had lower plant density, strongly suggesting a regulatory role for the disease in the host population. If disease reduces plant density but high density is required for disease to establish or be maintained, then a decline in disease over time is expected, which is in fact what was observed here. However, the plant population fluctuated over time in a manner inconsistent with complete explanation by disease regulation; it increased in 2005 then decreased in 2006, even in only those plots with disease present in at least one year. Therefore, the strength of regulation by disease must be evaluated in the context of other environmental factors that may influence the host plant population. In this community a mowing event knocked back the plant population between 2002 and 2004, it then recovered in 2005 and declined in 2006 as succession proceeded.