1   # Effects of disease emergence on invasive grass impacts

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12   **Abstract**

13   Invasive species impact ecosystems through their large abundances and strong per capita

14   effects. Enemies can regulate abundances and per capita effects, but are notably absent for

15   many new invaders. However, invaders acquire enemies over time and as they spread;

16   processes hypothesized to mitigate negative invader impacts by reducing abundance or per

17   capita effects. Alternatively, properties of invaders or acquired enemies, such as an enemy's

18   ability to attack multiple species, may hinder enemy mitigation of invader impacts. We used field

19   experiments to evaluate disease mitigation of invader impacts using the invasive grass

20   *Microstegium vimineum*, which hosts an emerging fungal disease, and a native grass competitor,

21   *Elymus virginicus*. We manipulated competition through density gradients of each plant species,

22   and we reduced ambient foliar diseases with fungicide and autoclaving. We then modeled long-

23   term population dynamics with field-estimated parameters. In the field, disease did not reduce

24   invader abundance or per capita effects. The invader amplified disease on itself and the

25   competitor, and disease reduced invader and competitor fitness components (e.g., germination).

26   The dynamical model predicted that disease impacts on the competitor are greater than on the

27   invader, such that disease will reduce invader abundance by 18%, and competitor abundance by

28   88%, over time. Our study suggests that enemies acquired by invaders will not necessarily

29   mitigate invader impacts if the invader amplifies the enemy and the enemy attacks and

30   suppresses competitor species.

Introduction

Non-native species are frequently introduced to new locations through human-assisted pathways (1). A subset of these species become invasive and cause negative ecological impacts (2, 3). Substantial ecological impacts occur when invaders are abundant and have strong per capita effects on other species (Fig. 1A) (4, 5). Theory predicts that natural enemies can mitigate invader impact (Fig. 1B–C). For example, the enemy release hypothesis posits that invasive species can establish and outcompete native species because they lack co-evolved specialist enemies, which would otherwise reduce their abundance or per capita effects (6). Biological control is based on the premise that introducing specialist enemies reduces invader impacts (7). Most recently, the pathogen accumulation and invasive decline hypothesis suggests that enemies acquired during invasion (i.e., accumulated enemies) also mitigate invader impacts (8). Invaders accumulate enemies the longer they are established and the farther they spread (9, 10). Yet, there are few tests of whether and how accumulated enemies mitigate invader impacts (11).

An important impact of species invasions is competitive suppression of native species (2, 3). Successful biological control suggests that competitors can recover with enemy-driven declines in invader abundance (12, 13). However, a saturating abundance–impact curve (Fig. 1A) may lead to sustained impact despite reductions in invader abundance (Fig. 1B) (14). Further, invasive species may tolerate enemy attacks, either at the scale of an individual (e.g., a weak effect of enemy damage on fitness) (15) or population (e.g., compensatory population growth following attacks) (16), limiting enemy effects on invader abundance altogether.

In contrast to biological control agents and specialist enemies featured in the enemy release hypothesis, generalist enemies acquired by invasive species may attack co-occurring species, potentially leading to apparent competition (17). Generalist enemies that spill over from co-occurring species may effectively reduce invader abundance and impact (Fig. 1B) (18, 19). However, spillback of enemies from the invader to other species may lead to sustained or increased invader impact despite reductions in invader abundance (Fig. 1D) (20, 21). Additionally, enemies that depend on invader abundance for persistence or dispersal (e.g., density-dependent disease transmission) may exert less pressure as an invader declines (Fig. 1D). Therefore,

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59 characterizing how much enemies depend on and affect invaders and competitors is crucial to

60 understanding enemy effects on the invader abundance–impact relationship.

61   In addition to reducing invader abundance, enemies may also mediate invader impacts

62 by altering invader per capita effects on competitors (Fig. 1C). To evaluate the net per capita

63 effects of invaders, all types of competition, including apparent, resource, and interference, must

64 be considered together (22). Theory predicts that if the net intraspecific effects of competing

65 species exceed their net interspecific effects (i.e., they limit themselves more than others), they

66 can potentially coexist. Otherwise, a species with greater interspecific than intraspecific effects

67 may exclude its competitor, or priority effects may occur (22, 23). Intra- and interspecific effects of

68 the invader and competitors provide insights into invader impacts because they can be linked to

69 the abundance–impact relationship; intraspecific effects influence invader abundance and

70 interspecific effects are synonymous to per capita effects.

71   We hypothesized that enemy accumulation mitigates invader impacts through reduced

72 abundance or per capita effects, unless properties of the invader or enemies (e.g., saturating

73 abundance–impact curve, density dependence, spillback) hinder this process. To test this

74 hypothesis, we conducted field experiments with the invasive annual grass *Microstegium*

75 *vimineum* (stiltgrass). The recent emergence of foliar fungal pathogens on *M. vimineum* in its

76 introduced range (eastern and midwestern U.S.) may alter impacts of this invader on native

77 plants (24, 25). "Emergence" here refers to pathogens that have been recently recognized on a

78 host and have increased in incidence over time; in this case, fungi in the genus *Bipolaris* that

79 were first detected on *M. vimineum* in the past 20 years (25). In the absence of *Bipolaris*, *M.*

80 *vimineum* suppresses other plant species through resource competition and litter build-up (26,

81 27). The competitor in our study is a native perennial bunchgrass (*Elymus virginicus*) that

82 commonly co-occurs with *M. vimineum* (28) and is susceptible to *Bipolaris* infection (29, 30). As a

83 perennial, *E. virginicus* occurs as first-year seedlings and older adults, which may differ in their

84 interactions with *M. vimineum*.

85   Here, we present the results from field experiments in forest in Indiana, USA. We

86 manipulated invader and competitor abundances and disease (i.e., we reduced disease by

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87   spraying plants with a fungicide that suppresses *Bipolaris,* and by autoclaving litter). First, we

88   measured short-term (within growing season) effects of disease on invader abundance and per

89   capita effects. Then, we assessed how much disease depends on and affects fitness components

90   of the invader and competitor. We conducted two repetitions of the field experiments over two

91   years and results presented here are from second repetitions, unless otherwise noted (Methods

92   S1). Finally, we modelled long-term population dynamics to evaluate how disease-induced

93   changes in intraspecific and interspecific effects influence competitor abundance (i.e., invader

94   impacts).

95

96   **Results**

97   We found limited (non-statistically significant) evidence that disease reduced invader

98   abundance over one growing season. Reducing disease increased invader biomass by 36% [-

99   51%, 186%] (model-estimated median, 95% credible intervals in brackets, applies throughout) at

100   high invader density (Fig. 2A, Table S1). We did not find evidence that reducing disease affected

101   invader seed production (Fig. 2A, Table S2).

102   There was limited evidence that disease increased per capita effects of the invader.

103   Reducing disease reduced the per capita effect of the invader on adult competitor growth by 79%

104   [-456%, 338%] and seed production by 121% [-626%, 334%] (Fig. 2B, Tables S3–S6). Reducing

105   disease did not alter per capita effects of the invader on its own or first-year competitor biomass

106   or seed production (Fig. 2B, Tables S3–S6). Similarly, there was limited evidence that disease

107   increased per capita effects of the invader in the first repetition of the experiment (Fig. S1, Table

108   S7). There was strong evidence that invader litter reduced invader and competitor emergence

109   (Fig. 2C, Tables S8–S9). Autoclaving litter did not alter the effect of litter on invader emergence

110   (Table S8).

111   There was strong evidence that disease transmission depended on both the invader and

112   competitor. Earlier in the growing season (8–12 weeks post planting), competitor plants

113   transmitted disease to invaders in control plots and to first-year competitors in both treatments

114   (Fig. 3, Table S10). Additionally, invader plants surrounding plots transmitted disease to invaders

115   in fungicide-treated plots and first-year competitors in control plots (Fig. 3). Later in the growing

116   season (16 weeks post planting), invader plants surrounding plots transmitted disease to invaders

117   in both treatments, and invader plants within plots transmitted disease to competitors in control

118   plots (Fig. 3). In the first repetition of the experiment, we only observed intraspecific transmission

119   (Fig. S2, Table S11). Disease severity was somewhat surprisingly not related to environmental

120   variation among plots (including dew intensity, soil moisture, and canopy cover), except that

121   disease severity of first-year competitors declined with more dew intensity and soil moisture

122   (Tables S12–S13). We identified *Bipolaris* fungi on leaves of both species (Table S14).

123   Disease negatively affected key aspects of invader and competitor fitness. Relative to

124   control plots, fungicide reduced disease severity on invaders by 41% [-60%, -18%], first-year

125   competitors by 53% [-68%, -33%], and adult competitors by 21% [-55%, 23%] (Fig. 4A, Table

126   S10). Reducing disease decreased competitor germination by 25% [-41%, -7%], but did not affect

127   invader germination (Fig. 4B, Tables S15–S16). However, seed fungal infections, which were

128   more common in control plots (Fig. S3A, Table S17), decreased invader germination by up to

129   44% [-80%, -11%] (Fig. S3B, Table S18). Reducing disease increased competitor establishment

130   by 5% [0.4%, 12%] and did not change invader establishment, which was already 99% (Fig. 4C,

131   Table S19). Unexpectedly, reducing disease decreased adult competitor biomass and seed

132   production by 35% [-65%, -0.6%] and 26% [-47%, -2%] (Fig. 4D–E, Tables S4, S6), respectively,

133   despite absence of a direct fungicide effect in the greenhouse (Methods S2, Tables S20–S22).

134   When interactions were measured across biomass gradients (in contrast to density, as in Fig.

135   2A), reducing disease consistently increased adult competitor tolerance of competition (Fig. 4F–

136   H, Fig. S4–S5), although the changes were not significantly different from zero (Table S23).

137   These processes, when put together in a model of long-term dynamics, predict that

138   disease fails to mitigate invader impact, and instead enhances invader impact. Invasion reduced

139   competitor biomass by 8% in the simulation (Fig. 5A), indicating limited competitive impacts of the

140   invader in the absence of disease (Fig. S6). Disease emergence further decreased competitor

141   biomass by 88%, and reduced invader biomass by 18%. Community-wide disease reached 50%

142   (Fig. 5A). In contrast, the competitor did not maintain disease without the invader and its biomass

143   remained unchanged (Fig. S7). When invader germination was reduced in the model due to seed

144   infections (-44%), community-wide disease severity periodically fell below the threshold needed

145   for disease effects (15%), leading to spikes in invader and competitor abundances. However,

146   impacts on competitor biomass remained otherwise consistent (Fig. S8). Overall, the effects of

147   disease on the invader measured in our experiment did not suppress it enough to allow the

148   competitor to recover to its pre-invasion biomass.

149   We evaluated the influence of invader abundance and per capita effects on impact (i.e.,

150   change in competitor biomass relative to its pre-invasion level) by varying competition

151   coefficients. Because competition coefficients were based on biomass (Fig. 4F–H), with some

152   modifications (see Methods), "per capita" refers to per g/m^2. Disease decreased invader

153   abundance, but increased invader effects, leading to greater impact (Fig. 5B). Similarly, disease

154   increased the competitor's per capita effect on itself, leading to greater impact (Fig. 5C).

155   However, decreasing the competitor's effect allowed the competitor to recover more than

156   decreasing the invader's effect (Fig. 5B–C). Other parameter settings that reduced invader

157   abundance also allowed the competitor to recover (Fig. S9).

158

159   **Discussion**

160   Enemy accumulation on invasive species is widespread (9, 10), but consequences for

161   invader impacts are poorly understood (11). We demonstrated that recently emerged disease on

162   the invasive annual grass *M. vimineum* suppressed both the invader and a perennial native

163   competitor, *E. virginicus*, but the competitor was suppressed more. It is unclear whether the

164   fungal pathogens (species in the genus *Bipolaris*) established in *M. vimineum*-invaded habitats

165   before or after invasion. The first description of *Bipolaris* on *M. vimineum* in the U.S. was in 2001

166   (31), nearly a century after the first U.S. records of *M. vimineum* (32) and *Bipolaris gigantea* (33),

167   suggesting that *Bipolaris* was maintained by resident species before aquiring the necessary tools

168   to infect the invader. Invader amplification of *Bipolaris* is therefore likely a case of "spillback." We

169   found that enemy accumulation by an invader enhanced invader impacts through spillback and

170   apparent competition.

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171   Host-pathogen interactions can vary substantially over time and space. Plot-level

172   environmental conditions did not explain variation in disease severity in our experiments, which

173   may be due to limited environmental variation at this scale compared to larger spatial and

174   temporal scales. For example, prior research on *M. vimineum* has found more intense impacts of

175   disease in other locations (24, 25) and at the same location in previous years (29). Even when we

176   assumed that disease had stronger effects on the invader in our model, the competitor did not

177   recover to its pre-invasion biomass because disease increased its own intraspecific competition

178   coefficient. This result depends on the pathogen's ability to infect both species, whereas

179   specialist pathogens accumulated by invaders may be expected to lessen invader impacts (12,

180   13). We estimated small impacts of the invader on the competitor in the absence of disease.

181   Because plants were grown separately prior to planting in the field, we may have missed

182   important competitive interactions among seedlings (34). However, the result that invader-

183   amplified disease negatively impacted the competitor holds regardless of pre-disease

184   competition.

185   Our results suggest that the invader is a more competent host than the competitor,

186   sustaining disease transmission later in the growing season and throughout the simulation.

187   However, the competitor suffered greater negative effects of disease. One possible explanation is

188   asymmetric and inverse relationships between susceptibility (disease impact on biomass) and

189   competency (inoculum production) of the hosts, as has been described for hosts of sudden oak

190   death in California forest ecosystems (35). In addition, other native grasses outside the purview of

191   this study may serve as reservoir hosts on which *Bipolaris* has historically perennated without the

192   invader. Interestingly, fungicide decreased biomass, seed production, and germination of

193   competitors without affecting its growth in the greenhouse, suggesting a potential negative effect

194   of fungicide on beneficial fungi in the field.

195   The effects of enemy accumulation on invader impacts have been evaluated in few

196   systems, with a range of outcomes. For example, the invasive grass *Ammophila arenaria*

197   increased seed predation of a native legume by a native rodent, which reduced the predicted

198   growth rate of the legume (36). In contrast, a vascular wilt disease acquired by *Ailanthus altissima*

199   in its invaded range seemed to have limited effects on most co-occurring species (37, 38). In a

200   greenhouse study, we found that *Bipolaris* inoculation decreased biomass of *E. virginicus*, but not

201   two other native species (34). A general rule for how enemy accumulation by invaders affects

202   competitors is therefore difficult. Instead, host screening, as in biological control development

203   (38), paired with field studies and population modeling (39), can increase understanding of how

204   enemy accumulation will affect native competitors and inform management strategies.

205   Mitigating invader impacts is important for maintaining and restoring ecosystems.

206   Increasing documentation of new enemies associated with invaders (9, 10), and lessons from

207   research on biocontrol (12, 13), the enemy release hypothesis (6), and biotic resistance (40) have

208   led to the hypothesis that enemies may mitigate invader impacts over time (8). This hypothesis is

209   relevant to a broad range of invasive taxa that share enemies with co-occurring species (20, 41,

210   42). By pairing field experiments manipulating plant density and disease with a dynamical model,

211   we demonstrated that although accumulated enemies may cause invader declines, they can also

212   increase invader impacts because of apparent competition. We expect this result to apply broadly

213   because most enemies accumulated by invaders are generalists (to be present pre-invasion,

214   enemies must be sustained by alternative hosts, and so are unlikely to be specialists) and

215   invaders often reach high abundances (4, 5), which can amplify enemies. Continued research

216   can illuminate whether the effects of spillback persist over evolutionary timescales as enemies

217   adapt to invaders.

218

219   **Materials and Methods**

220   *Density experiment*

221   To evaluate the effects of disease on the invader abundance–impact relationship, we

222   carried out a two-year field experiment at Big Oaks National Wildlife Refuge (BONWR) in

223   Madison, IN, USA from April 2018 to October 2019. Invader (*M. vimineum*) populations are

224   established in approximately 810 ha of BONWR's 20,647 ha property, and some are infected with

225   *Bipolaris* fungi (25, 29, 30). The experiment was a full factorial combination of two disease

226   treatments (fungicide, control) and ten density treatments, replicated four times each—once at

227   each of four sites with the invader and *Bipolaris* present (Fig. S10).

228   In spring 2018, we planted focal plants— three invader seedlings, three first-year

229   competitors, and one adult competitor—in the center 1-m^2 of each 2 m × 2 m plot (Fig. S11). We

230   also planted density treatments in the center of plots, including: no additional plants or low,

231   medium, or high densities of the invader (4, 16, or 64 plants), first-year competitors (4, 8, or 16

232   plants), or adult competitors (2, 4, or 8 plants). Invaders, first-year competitors, and dead adult

233   competitors were replaced in spring 2019. Every four weeks during the growing season, we

234   sprayed fungicide-treated plots with 1.5% fungicide (Ipro 2SE, 23.8% iprodione) and control plots

235   with tap water, which did not promote disease relative to ambient levels (Table S24).

236   We monitored focal plants for survival and foliar fungal infection throughout the growing

237   season and measured growth and seed production at the end of the growing season. We

238   measured seed germination and, for invaders, seed fungal infection, in the lab. We also

239   measured environmental variables, including soil moisture, canopy cover, temperature, and

240   relative humidity in each plot. Due to changes in data collection methods between years, results

241   are presented from the second year unless otherwise noted. Additional details are described in

242   Methods S1 and Table S25.

243

244   *Litter experiments*

245   We conducted two field experiments to evaluate effects of disease and invader litter on

246   invader and competitor emergence. In spring 2018, we established seven treatments, replicated

247   once at each of four sites at BONWR (Fig. S10, Methods S3). The invader was established at

248   these sites and showed no evidence of foliar fungal infections. We collected invader litter with

249   evidence of *Bipolaris* infections from elsewhere at BONWR. We autoclaved half of this litter for 60

250   minutes and brought all back to the field. We cleared 2 m × 2 m plots and sowed 200 invader

251   seeds and 50 competitor seeds in the center 1-m^2. Plots received either no litter, autoclaved litter,

252   or control litter. The autoclaved and control litter were each applied at low (50 g/m^2), medium (100

253   g/m^2), and high (200 g/m^2) levels. We measured seedling emergence in July. We were unable,

254   however, to identify competitor seedlings amongst other species in the seedbank.

255   Because we could not estimate competitor emergence from the first litter experiment, and

256   because there was no significant effect of autoclaving on invader emergence (Fig. 1C), we

257   performed a second litter experiment to estimate competitor germination with non-autoclaved

258   litter. In April 2019, we established three litter treatments—removal, addition, and

259   unmanipulated—replicated four times (two replicates each near sites D1 and D3; Fig. S10). We

260   raked and weighed litter from each 2 m × 2 m plot, adding litter from removal plots to addition

261   plots. Prior to returning litter to plots, we planted 50 competitor seeds in removal plots and 26

262   competitor seeds in addition and control plots. Seeds were purchased from Prairie Moon Nursery

263   and glued to toothpicks to aid seedling identification. We measured tiller emergence in June.

264

265   *Statistical analyses*

266   We fit Bayesian linear regressions using the *brms* package (43) in R (version 4.0.2) (44).

267   Tables S1–S24 describe model formulae, prior and posterior distributions, and model estimates.

268   We used the concept of constant final yield (i.e., that plant biomass saturates with increasing

269   density) to estimate relationships between invader abundance and density (Fig. 2A) and a Ricker

270   model to estimate competition coefficients (Fig. 2B, 4F–H). Models were run with three chains of

271   6000 iterations each with a discarded warm-up period of 1000 iterations. We evaluated

272   convergence and fit by plotting simulated data from the posterior predictive distributions against

273   observed data, ensuring the three chains mixed well, and checking that r-hat values equaled one.

274   We used the hypothesis (43) and emtrends (for effects of continuous variables on disease

275   severity) (45) functions to extract estimates and 95% credible intervals of values of interest.

276

277   *Dynamical model*

278   We simulated the long-term population dynamics of the invader and competitor using a

279   continuous-time growing season model linked to a discrete-time yearly model (Methods S7). In

280   the continuous-time model, we tracked the dynamics of plant biomass, which changes as plants

281   grow, compete, and transmit disease during the growing season. The discrete-time model

282   projects the dynamics of adult competitors and seeds of both species at the beginning of the

11

283   growing season each year, which are determined at the end of the continuous-time model by

284   survival and seed production, respectively. Seed number is determined by seed production the

285   prior year and carry-over of seeds in the seed bank. Seed germination and establishment occur

286   quickly at the start of the growing season, and so are included as part of the discrete-time yearly

287   model. The density of newly established plants then initiate biomass at the beginning of the

288   continuous-time growing season model. Parameters derived from the density experiment were

289   set to values estimated from control plots when community-wide disease severity exceeded 15%

290   and fungicide-treated plots otherwise.

291   We performed simulations in R using the *deSolve* package (46). We began simulations

292   with ten competitor seeds. After 100 simulated years, we introduced ten invader seeds. After 100

293   more years, we introduced fungal inoculum. We assumed the growing season lasted 160 days.

294   Competition coefficients from Fig. 4F–H were used, except that the effect of fungicide on the

295   invader's intraspecific coefficient was estimated from plot-scale biomass (Fig. 2A) and positive

296   coefficients (i.e., facilitation) were set to zero. However, because adult competitors then exerted

297   no intraspecific competition on adults in fungicide plots and first-year competitors in control plots,

298   we replaced those estimates with estimates from adult competitor effects on first-year competitors

299   in fungicide plots and adults in control plots, respectively.

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Acknowledgments

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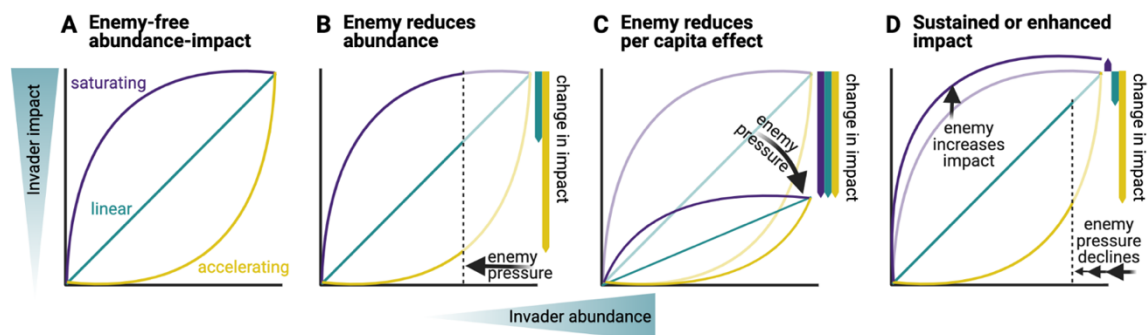
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Figures

Figure 1. Potential enemy effects on invader abundance–impact relationships. (A) Larger invader abundance generally leads to greater impact, but the shape of the relationship may vary (shown here: linear (blue), saturating (purple), accelerating (yellow)). The per capita effect determines the change in impact with each unit increase in abundance. (B) Enemy pressure may reduce invader abundance, as represented by dark lines relative to light lines. Reduced abundance may reduce impact (linear or accelerating) or have no effect on impact (saturating). (C) Enemies may reduce per capita effects of invaders, as represented by dark lines relative to light lines. Reduced per capita effects may reduce impact (all three). (D) The predictions in B and C may not be realized because the enemy increases the impact (saturating) or because enemy pressure declines with invader abundance (linear or accelerating). Note: change in impact at maximum abundance relative to the enemy-free scenario in A is depicted on the right sides of panels B–D.
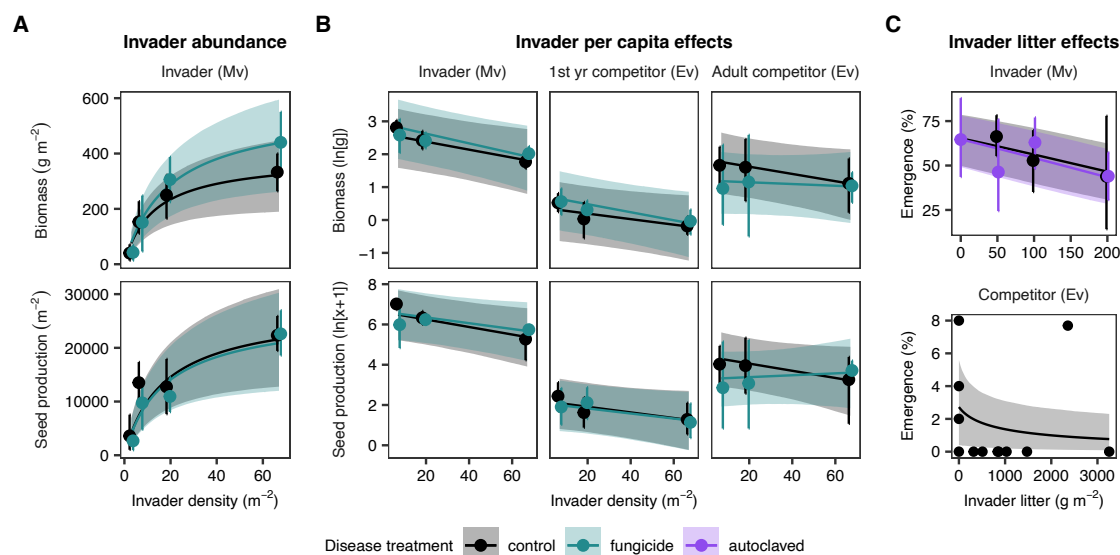
Figure 2. The effect of foliar fungal disease suppression on invader (*M. vimineum*, abbreviated as Mv) abundance, per capita effects on the invader and competitor (*E. virginicus*, abbreviated as Ev), and litter effects on both species. (A) Invader abundance was estimated with plot-scale biomass and seed production across a gradient of invader density. (B) Per capita effects were measured as change in biomass and seed production (both natural log-transformed) across the invader density gradient. (C) Litter effects were measured as change in percentage seeds emerged (emergence) across litter abundance gradients. Invader and competitor panels in C differ in invader litter gradients and disease treatments because they are two different experiments (see Methods for details). Points and error bars represent mean ± 95% bootstrapped confidence intervals of raw data, except the competitor panel of C where points represent raw data. Lines and shading represent model-estimated mean ± 95% credible intervals.
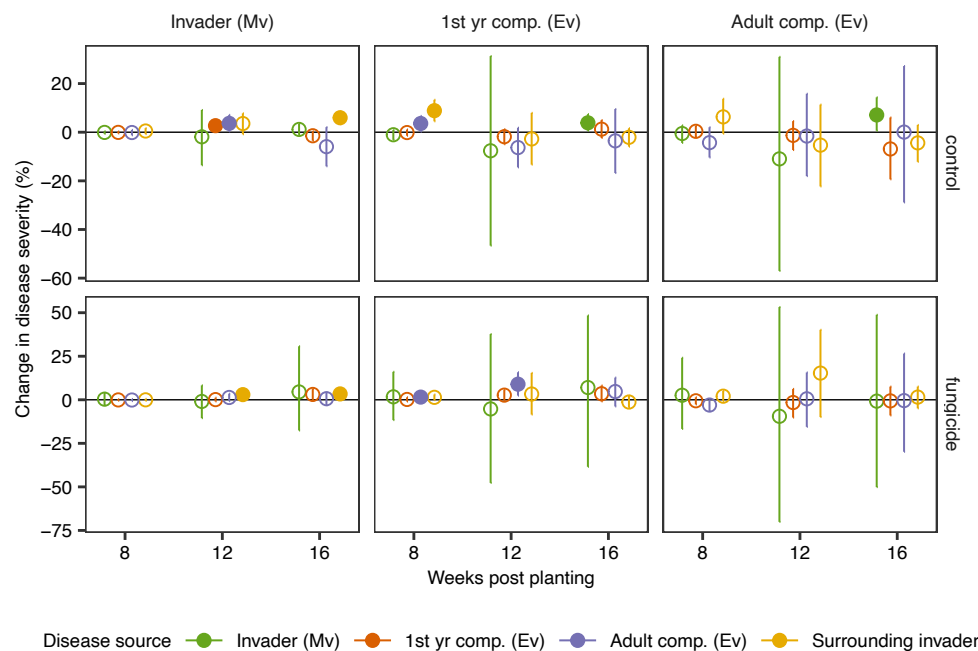
Figure 3. Disease transmission throughout the growing season, measured as change in disease severity (percentage of leaf area covered by lesions) on the invader, adult competitor, and first-year competitor (panel columns) with one unit increase in disease source four weeks prior. Disease sources included the density-scaled disease severity of invader, first-year competitor, and adult competitor plants, as well as disease severity of invader plants surrounding plots. One unit of density-scaled disease severity is one plant with 100% disease severity or 100 plants with 1% disease severity and one unit of surrounding invader (which occurred at high density) is an average disease severity of 1%. Model-estimated mean and 95% credible intervals are shown. Filled points have 95% credible intervals that omit zero.
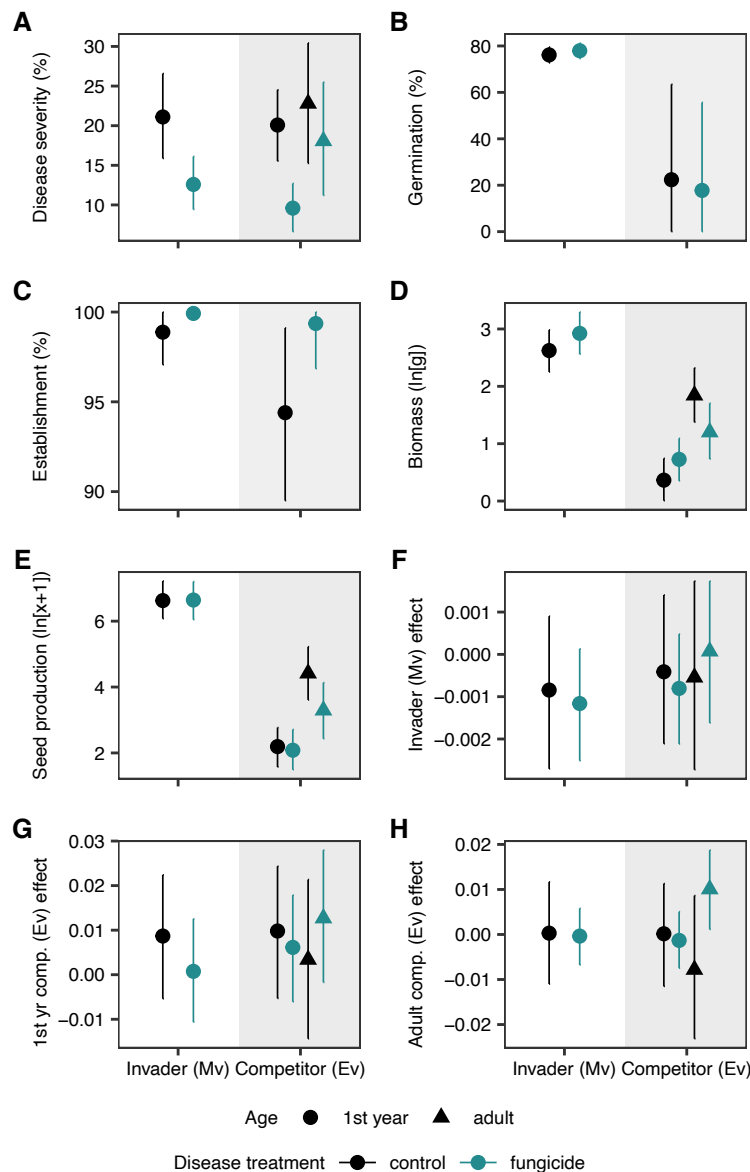
Figure 4. Effects of foliar fungal disease suppression on the invader (Mv) and competitor (Ev),

measured as (A) disease severity, (B) germination (i.e., percentage of seeds that germinated in

lab-based trials), (C) establishment (i.e., percentage of seedlings that established and survived

through the growing season), (D) biomass (natural log-transformed), (E) seed production (natural

log-transformed), and effects of (F) invaders, (G) first-year competitors, and (H) adult competitors

(natural log-transformed change in focal plant biomass per unit background biomass). Model-

estimated mean and 95% credible intervals are shown. Background shading delineates invader

(white) and competitor (gray) values.
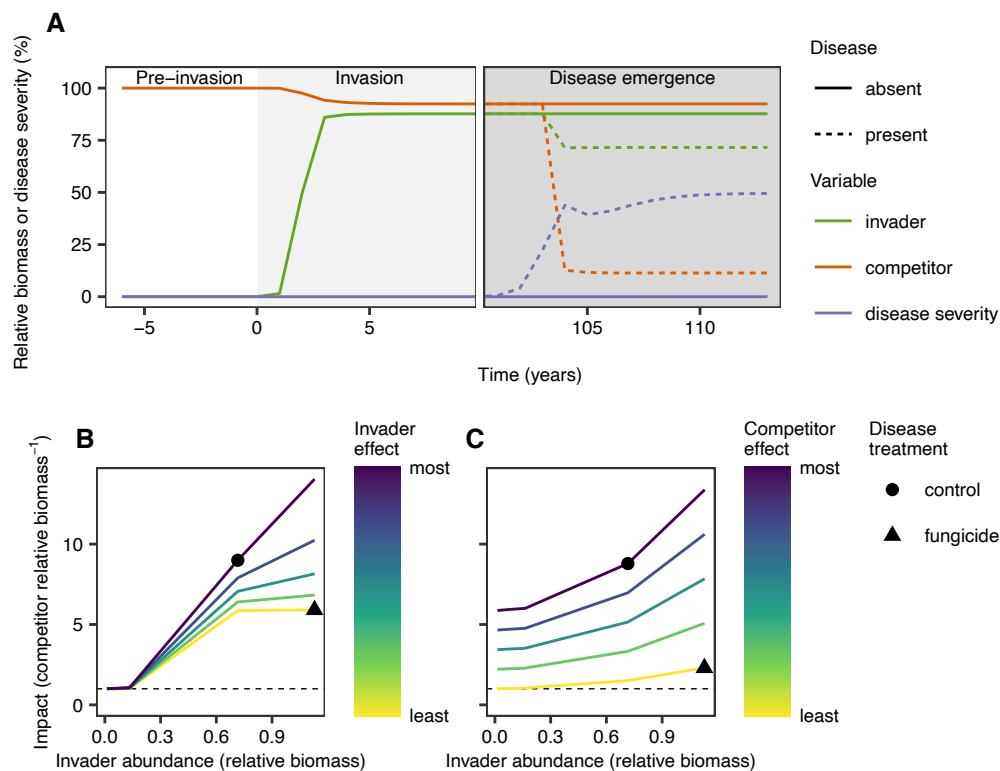
Figure 5. Simulations of population dynamics of the invader and competitor. (A) In a time series with parameter values based on field measurements, the invader reduced competitor abundance upon invasion and further reduced competitor abundance when disease emerged (years between 10 and 100 are omitted for visualization). Relative biomass is biomass divided by the biomass of the competitor pre-invasion. (B) Decreasing invader abundance and effects reduced the impact on the competitor, allowing it to recover to pre-invasion biomass at low invader abundance (dashed line). Parameter ranges: invader intraspecific effect = 0.001–0.09, invader interspecific effect = 0–5.4 × 10⁻⁴. (C) Reducing the competitor's intraspecific effect led to competitor recovery. Parameter ranges: invader intraspecific effect = 0.001–0.09, adult competitor intraspecific effect = 0.001–0.008. In B and C, points are parameter values estimated from field measurements.