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LETTER

Disease and fire interact to influence transitions between savanna–forest ecosystems over a multi-decadal experiment

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Abstract

Global change is shifting disturbance regimes that may rapidly change ecosystems, sometimes causing ecosystems to shift between states. Interactions between disturbances such as fire and disease could have especially severe effects, but experimental tests of multi-decadal changes in disturbance regimes are rare. Here, we surveyed vegetation for 35 years in a 54-year fire frequency experiment in a temperate oak savanna–forest ecotone that experienced a recent outbreak of oak wilt. Different fire regimes determined whether plots were savanna or forest by regulating tree abundance ($r^2 = 0.70$), but disease rapidly reversed the effect of fire exclusion, increasing mortality by 765% in unburned forests, but causing relatively minor changes in frequently burned savannas. Model simulations demonstrated that disease caused unburned forests to transition towards a unique woodland that was prone to transition to savanna if fire was reintroduced. Consequently, disease–fire interactions could shift ecosystem resilience and biome boundaries as pathogen distributions change.

Keywords

Alternative states, fire, forest, forest model, fungal pathogen, plant disease, savanna, tree rings.

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INTRODUCTION

Rapid changes in disturbance regimes are having an increasingly large effect on ecosystem dynamics (McDowell *et al.* 2020). Yet disturbance events cannot be considered in isolation, as their effects are often compounding, such that past disturbances can either amplify or dampen the sensitivity of ecosystems to future disturbances (Cavender-Bares & Reich 2012; Seidl *et al.* 2017). The effects of one disturbance can change in the presence of another disturbance and depend on their sequence of occurrence (Fukami 2001). Despite the importance of interacting disturbance regimes, experimental tests of interactions are rare, especially in the context of repeated disturbances that can cause novel responses not evident after a single disturbance event.

Fire and disease are two disturbances that are spatially extensive and impactful on ecosystem processes both in isolation and in concert (Hicke *et al.* 2012; Boyd *et al.* 2013; Pellegrini *et al.* 2018; McDowell *et al.* 2020). Independently, fire and disease can have large effects on ecosystems by killing susceptible trees (Bond *et al.* 2005; Boyd *et al.* 2013; Seidl *et al.* 2017), leading to a cascade of changes in the ecosystem (Seidl *et al.* 2017). When fire and disease occur together, their interactions can either dampen or amplify the severity of either disturbance (Tepley *et al.* 2018). For example, disease can change fuel loads and fire can change disease host density, both of which alter the severity of future disturbances (Dale *et al.* 2001).

Here, we use a savanna–forest gradient maintained by variation in controlled burning, which is being invaded by a novel pathogen, oak wilt, as a case study to evaluate how repeated fire and disease independently and interactively affect ecosystems. It is well established that repeated fire can determine the distribution of savanna and forest ecosystems (Bond *et al.* 2005; Staver *et al.* 2011), but the effects of plant pathogens and pathogen–fire interactions have received little study. This may be a key knowledge gap given the empirical evidence for large effects of diseases on trees in other systems (Hicke *et al.* 2012; Boyd *et al.* 2013) and the shifting distributions of disease.

The combination of fire and disease may have unique consequences in savanna–forest ecotones because each can impact plant growth and mortality through different pathways. Surface fires that characterise savannas kill small trees that cannot tolerate heating and/or are unable to re-sprout following fire (Higgins *et al.* 2000; Hoffmann *et al.* 2012). Large trees can have much higher survival in surface fires, but tree population growth is inhibited by frequent burning that top-kills fire-sensitive saplings (Higgins *et al.* 2000; Peterson & Reich 2001). In these ecotones, grasses are the primary fuel for

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surface fires, and thus fire intensity can decline as adult trees grow and form a closed canopy that shades out flammable grasses (Peterson *et al.* 2007; Hoffmann *et al.* 2012), structuring a negative feedback between tree growth and fire. Concurrent changes in tree species composition further modify this relationship as fire exclusion results in the replacement of fire-tolerant savanna species by fast growing but more fire-sensitive forest species (Hoffmann *et al.* 2012).

On the other hand, diseases that exhibit increasing transmission with increasing susceptible host density spread most rapidly in areas with high tree densities (Meentemeyer et al. 2011; Hicke *et al.* 2012). Similar to fire, the tree composition is important for regulating the effect of many diseases because diseases can be species or lineage specific (Dobson & Crawley 1994). If the tree species increasing in abundance in low fire frequency environments are more sensitive to disease, then fire exclusion could predispose an ecosystem to disease. Consequently, a fire-retardant closed canopy forest may be more prone to tree disease because of higher susceptible host density; contrastingly, a fire-promoting savanna could be resistant to disease because of lower susceptible host density.

To test the interactive effects of fire and disease, we sampled a large-scale replicated fire-frequency manipulation experiment ongoing for 54 years at the Cedar Creek Ecosystem Science Reserve in central Minnesota, USA – the Cedar Creek Savanna Fire Experiment, established in 1964 (CCSFE, Table S1, Supporting Information) (Peterson & Reich 2001). Repeated surveys of vegetation from 1984 to 2018 allowed us to track temporal changes in the tree communities both in response to fire as well as an outbreak of a tree pathogen, oak wilt, across the experimental plots.

METHODS

Study site and experimental design

CCSFE is in the Cedar Creek Ecosystem Science Reserve in Minnesota, USA (latitude: 45.40°, longitude: −93.19°; established in 1942). CCSFE experiences a temperate mesic climate (780-mm mean annual precipitation and 6.72°C mean annual temperature) and is a mosaic of savanna and forest ecosystems. Prior to the establishment of the reserve, fires in the grassland and savanna occurred every 1–3 years until *c.* 1920, after which fire was excluded for *c.* 40 years until the CCSFE was established (Pellegrini *et al.* 2020). The CCSFE was established on plots that were savanna with 17–39% tree cover. In 1964, plots 2.4–18.4 ha in size were distributed across a *c.* 300 ha landscape, delineated by fire breaks and began receiving a range of burn regimes. Monitoring of trees began in 1984, with permanent monitoring on all plots by 1995. The plots are burned in the spring (April–May) with low-intensity surface fires (<1 m flame height). Here, we focused on 12 plots that span unburned ($n = 3$), burning around once every decade ($n = 2$), once every 3 years ($n = 2$), once every other year ($n = 1$), twice every 3 years ($n = 2$) and three times every 4 years ($n = 3$), with slight variation from the target frequency over the years, generating a frequency gradient from 0 to 0.77 fires per year. The permanent monitoring plots are on well-drained and sandy soils with similar underlying textures and depths.

The savannas include two main tree species, *Quercus ellipsoidalis* (red oak group; northern pin oak) and *Q. macrocarpa* (white oak group; bur oak). Unburned plots include a low abundance of other angiosperm mesic forest species (e.g. *Acer rubrum, Betula papyrifera*, *Q. alba*, and *Q. rubra*) but are dominated by Q. *ellipsoidalis* and Q. *macrocarpa*. The two dominant oaks have different sensitivities to both fire and oak wilt, with *Q. ellipsoidalis* more sensitive to both.

At CCSFE, the fungal pathogen oak wilt (*Bretziella fagacearum*) has spread rapidly in the last decade, leading to exponential increases in the number of standing dead trees as a result of recent mortality (Figure 1a). Oak wilt kills susceptible oak trees within the red oak lineage (*Quercus* subgenus Lobatae) (Jacobi & MacDonald 1980) through inefficient tylose formation that blocks the xylem, usually causing death within 1 year of infection (Jacobi & MacDonald 1980; Menges & Loucks 1984). White oaks (*Quercus* subgenus Quercus) have more efficient immune responses and are much less susceptible (Juzwik *et al.* 2011). The disease is primarily spread locally via root networks (Kuntz & Riker 1956) but also is dispersed long distance over land by *Nitidulid* beetles that are attracted to fungal mats that form once the disease is at an advanced stage (Jagemann *et al.* 2018); thus, local transmission is rapid when susceptible tree density is high (Menges & Loucks 1984). Because local disease spread is via roots and soil heating during fire is minimal in the CCSFE burns, it is unlikely that fire has direct sterilising effects by killing the disease. Rapid disease progression in the red oak species (*Q. ellipsoidalis* and *Q. rubra,* where mortality is 100%), which also tend to grow rapidly when fire is excluded, was apparent in the CCSFE plots (Figure 1a–c).

Vegetation surveys

We surveyed the tree and herbaceous community repeatedly in twelve 3750 m^2 permanent plots. Surveys began in 1984, 20 years after the establishment of the experiment, and occurred every 3–6 years, which encompassed >3,000 trees (Table S1). Adults were classified as trees $\geq$ 20 cm diameter at breast height (Peterson & Reich 2001), based on their ability to survive a fire (e.g. oaks $\geq$ 20 cm have a >90% chance of survival).

Tree-ring ages

We tested how fire affected tree recruitment by reconstructing tree age distributions with tree rings. In 2000, we cored and dated tree rings for 301 trees in four of the burn plots with two replicates in the fire frequency treatments of one fire every 3 years and one fire every 10 years (Supporting Information). These fire frequency treatments were chosen for this purpose because they were thought to straddle the hypothetical threshold fire return interval of 7–10 years we hypothesized would be required for trees to recruit in between fires.

Statistics

To test for temporal trends in tree population sizes, we fit generalised additive models with a maximum of three degrees

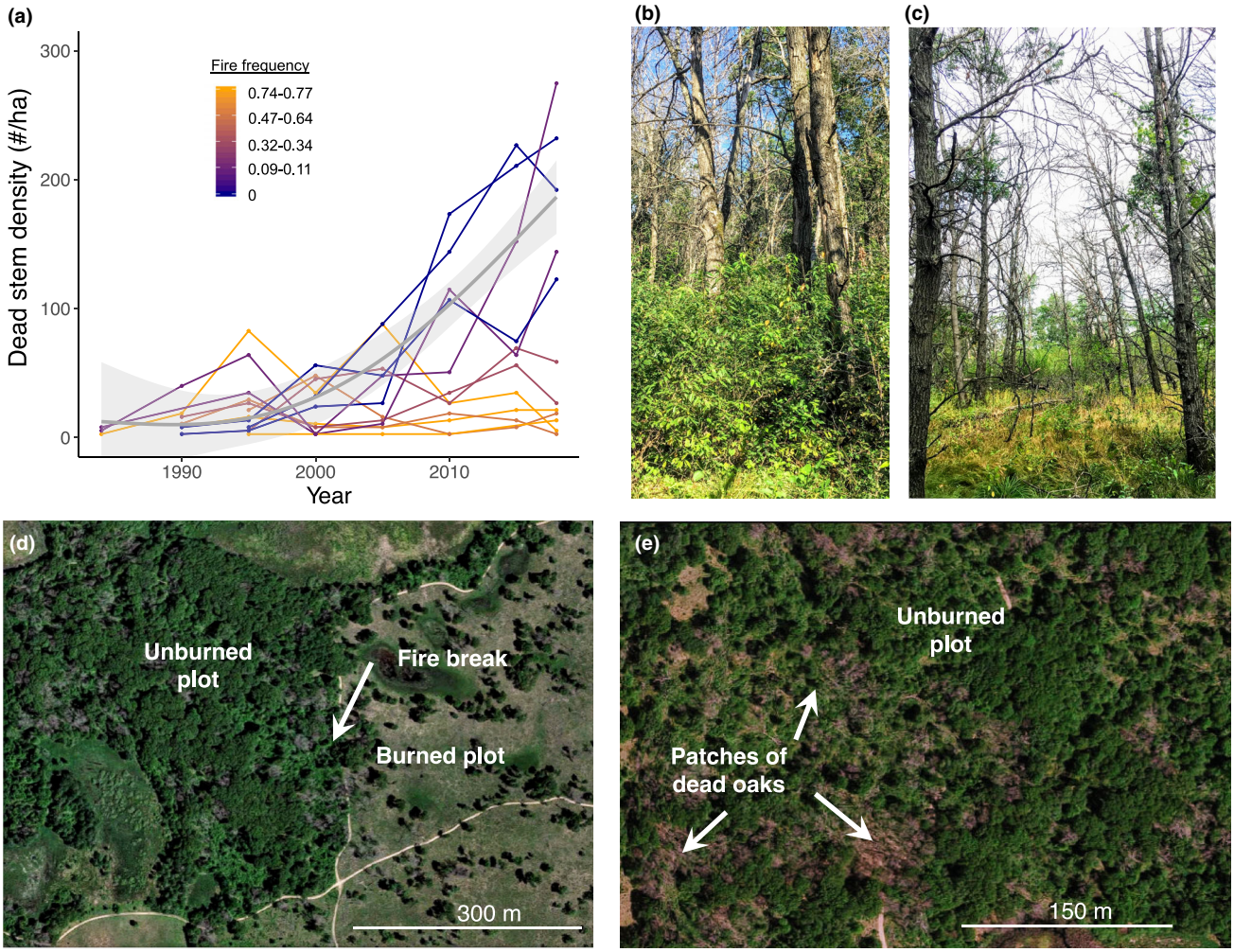

Figure 1 Local trends in the extent of oak wilt and examples of vegetation types across the fire manipulation plots. (a) Survey of standing dead trees in monitoring plots. Each line is a plot and the color of the lines indicates the prescribed fire frequency. The grey line is an exponential model fit ± standard error to the plots with no fire or one fire every decade. (b and c) Examples of unburned plots that have experienced large amounts of tree mortality. (d and e) Examples of the impact of fire and oak wilt on tree cover. (d) Two contrasting plots receiving different burning frequencies adjacent to one another, separated by a fire break. This unburned plot has not yet been affected by oak wilt. (e) An unburned plot that has experienced large declines in oaks due to oak wilt.

of freedom using the package *mgcv* v. 1.8-33 (Wood 2001). We performed two sets of tests: (1) trends in tree population sizes through time, which was conducted in each plot, (2) effects of fire through time, which was conducted every survey year. To assess fit, we report percent deviance explained based on the likelihood of the model fit.

Demographic rates were calculated using pairs of surveys that followed one another. Pre-disease rates integrate data from 1995 to 2010; post-disease is 2010–2018, which is based on the observation that oak wilt began spreading exponentially in CCSFE around 2010. Mortality rates were analysed using a binomial generalised linear model with a logit link function on burned plots. Recruitment rates were calculated by the appearance of a stem (size threshold of $\geq$ 5 cm). Growth rates were calculated by fitting linear models between basal area and time for each individual and extracting the slope coefficient from models with fits of $P < 0.10$ and dividing by the starting basal area to relativise growth rates. For fire effects on individual-stem data, we fit a mixed-effects model with replicate plot as a random intercept using package *lme4* v. 1.1-26 (Bates *et al.* 2015), with model significance evaluated using Satterthwaite's estimation of degrees of freedom. The relationship between grass cover and tree cover was tested using a mixed-effects model with year as a random intercept. All analyses performed in R v. 4.0.1 (R Development Core Team 2010).

Ecosystem model description

To test how fire–disease interactions influence the dynamics of savanna and forest ecosystems, we used a non-spatial stage-structured demographic model that tracks densities of saplings and adult trees (see Supporting Information, Figure S7). The equations were constructed to facilitate direct measurements of parameters to minimise the number of fitted parameters.

The model tracks demography of pin oaks because bur oak population sizes change relatively little and as a result our estimates of demographic parameters for bur oaks are not well constrained; however, we evaluate a two-species model in the Supporting Information, which exhibits qualitatively similar dynamics (Supporting Information). Pin oaks have three classes: fire-sensitive saplings, fire-resistant adults and infected trees.

$$\frac{dA}{dt} = \gamma S - \mu_A A - \left(\frac{\beta}{I+\alpha}\right) AI \quad (1)$$

$$\frac{dS}{dt} = re^{(-bA)} A - \gamma S - \theta\sigma\left(\frac{1}{\omega(A+S)+1}\right) S - \mu_S S - \left(\frac{\beta}{I+\alpha}\right) SI \quad (2)$$

$$\frac{dI}{dt} = \left(\frac{\beta}{I+\alpha}\right) AI + \left(\frac{\beta}{I+\alpha}\right) SI - \mu_I I \quad (3)$$

Adults (A), trees ≥ 20 cm diameter, form as a function of the proportion of saplings (γ) and die according to background mortality (μ_A). Adults are assumed to be fire resistant (see Results). Pin oaks can be killed by disease, which is a saturating Michaelis–Menten transmission function that assumes spread increases with density but saturates at high values. Infected trees (I) die at rate μ_I, which is assumed to be equal to 0.5 year^{-1} (Jacobi & MacDonald 1980; Menges & Loucks 1984). Saplings (S) recruit as a function of adult reproduction, r, which is multiplied by a self-thinning function of the absolute number of adults $e^{(-b*A)}$ to integrate reproduction and shading (functional form and parameters based on empirical data, Supporting Information); saplings are lost when they grow to adults (γ), background mortality (μ_S) and fire.

We model fire effects as killing saplings (Supporting Information). Fire-driven mortality is a function of the frequency of fire (θ, an experimentally controlled parameter) and potential intensity governed by grass cover, which is implicitly modelled as a function of the number of trees in the landscape because they shade the grass $\left(\frac{1}{\omega(A+S)+1}\right)$. Both frequency and intensity are then related to mortality via σ (Supporting Information).

We fit three unmeasured parameters via optimisation using the time series data from the plots, while all the other parameters are based on direct measurements. These three parameters were fit using the *optim* function in R using the Nelder–Mead simplex algorithm (Nelder & Mead 1965) to minimise the sum of squared errors of the model's predictions. Specifically, within the optimiser, predictions for each parameter set were generated by numerically integrating differential equations to compute predicted adult, sapling and diseased tree abundances (Supporting Information). Sum of squared errors was computed by comparing these predicted abundances to data.

RESULTS

Fire and disease jointly regulate the persistence of savannas

In the absence of disease, tree population sizes were strongly influenced by fire frequency. After 46 years of fire treatments (1964–2010), unburned plots had 246% higher stem density, 43% more basal area and 36% more biomass than plots burned at intermediate frequencies (one fire every 3 years), and 696% higher stem density, 286% more basal area and 276% more biomass than plots burned most frequently (three fires every 4 years) (Figure 2a, Figure S1, Tables S2 and S3). These differences arose because tree density, basal area and biomass increased over time in unburned plots (Figure 2b); in more frequently burned plots, tree density declined, but basal area and biomass were relatively stable over the 46 years (Figure 2b, Figure S1, Tables S2,S4). Consequently, unburned plots transitioned from savanna to forest, while intermediately and frequently burned plots remained savanna.

Trends in tree populations changed strikingly from 2010 to 2018, with rapid declines in tree biomass in several plots, coinciding with the outbreak of oak wilt (Figure 1b and 2b, Tables S2 and S4). The sharpest declines occurred in the plots with the most tree biomass in 2010. For example, from 2010 to 2018, biomass declined 10.2 ± 6.7 Mg ha^{-1} year^{-1} in unburned plots, with the losses more than twice as rapid as the gains during pre-disease fire exclusion (Figure 2c, Table S5). Not all plots lost trees, and disease spread was spatially heterogeneous across plots (Figure 2b-c); the rate of biomass decline was positively related to biomass at the start of the outbreak in 2010 ($r^2 = 0.65$, Figure 2d). A comprehensive survey in 2019 illustrated that the infected *Q. ellipsoidalis* trees occurred in either the unburned or least frequently burned plots and were all large (average diameter at breast height of 32.6 cm, the 88th percentile of all pin oak tree sizes).

As a result of disease-induced losses of trees, the biomass difference between unburned and high fire frequency plots declined from 154 Mg ha^{-1} in 2010 to only 99 Mg ha^{-1} in 2018, but the effect of fire history on tree biomass persisted (Figure 2a).

Species composition mediates effects of fire and disease

Trends in the sensitivity of trees to fire and disease were strongly linked with the relative dominance of two oak species: northern pin oak (*Q. ellipsoidalis*) or bur oak (*Q. macrocarpa*) (accounting for >95% of tree biomass across plots). Tracking individual stems demonstrated that individual pin oaks grew significantly faster than bur oaks (relative basal area gain of 0.12 ± 0.01 year^{-1} vs. 0.05 ± 0.01 year^{-1}, ± standard error; $F_{1,1334} = 78.8$, $P < 0.001$), allowing them to rapidly grow when fire was excluded, but the growth advantage diminished in the more frequently burned plots (fire–-species interaction: $F_{8,1325} = 4.5$, $P < 0.001$, Figure 3a). However, the probability of survival as a function of starting stem diameter was lower for pin oaks (Figure 3b). Consequently, pin oak saplings needed to grow to a 13% larger diameter to become as resistant to fire as bur oaks (17 cm vs. 15.1 cm respectively, to attain 50% probability of mortality, Figure 3b, bur oaks: $z = 7.42$, $P < 0.001$ and pin oaks: $z = 10.6$, $P < 0.001$; saplings have < 20 cm stem diameters). The integration of these individual-level dynamics resulted in pin oak population biomass to decline by 10 Mg ha^{-1} in the most frequently burned plots from 1995 to 2010 (Figure 3c, Table S6). Bur oak population biomass, on the other hand, significantly increased by 12 Mg ha^{-1} in the same burn plots

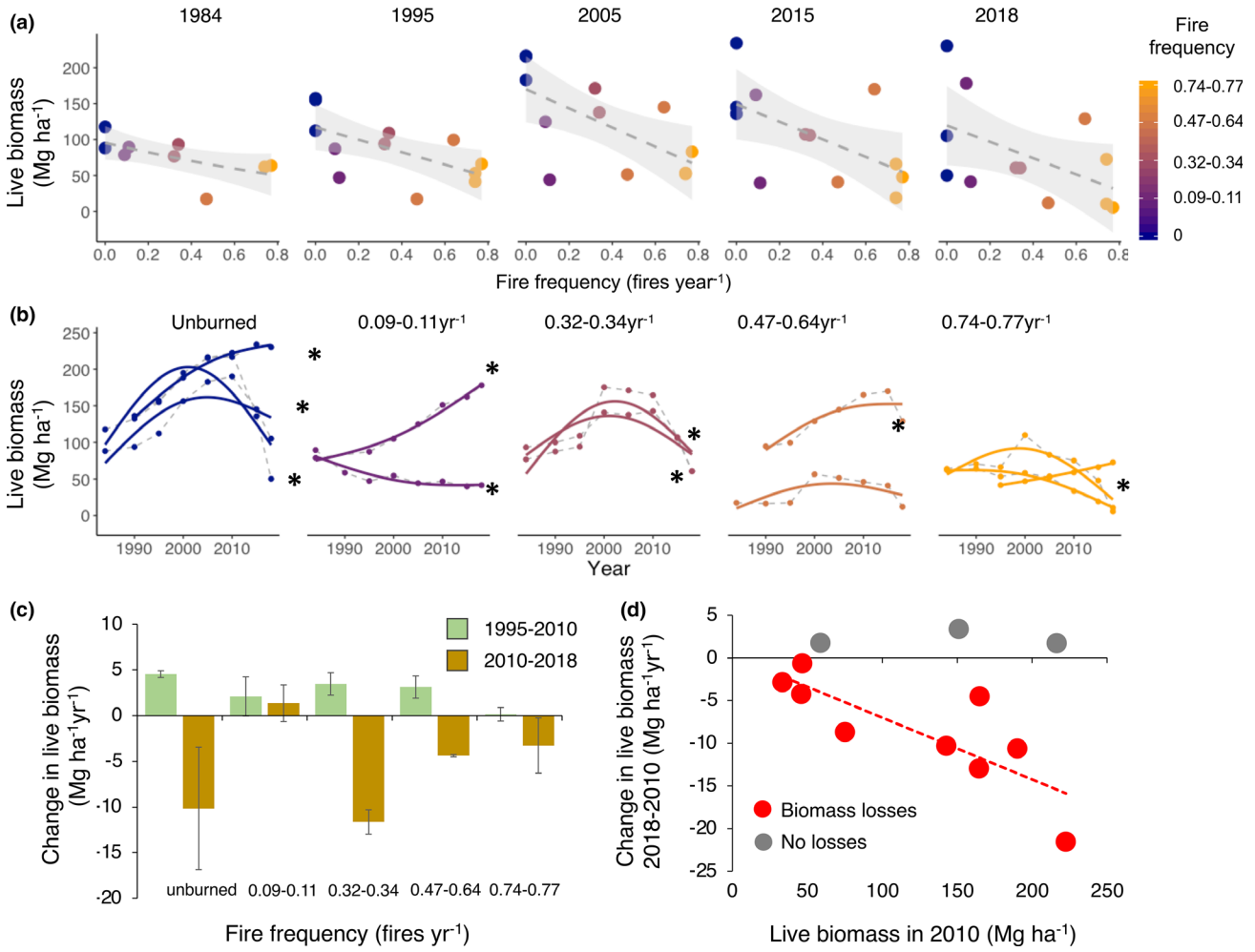

Figure 2 Fire and disease interact to determine trends in tree biomass. (a) Trends in live aboveground tree biomass across fire frequency treatments through time (x-axis units are number of fires per year). The grey lines are linear regressions performed within each survey year ± the standard error of the model fit. Colors represent fire frequency groupings. (b) Longitudinal trends within plots in the different fire frequency treatments. Asterisks indicate significance of the trend over the entire time period. Raw data expressed as points and the solid smoothed line is a generalised additive model fit within the plot. (c) Rates of change of living tree biomass across the two time periods from 1995 to 2010 (green bars) and 2010 to 2018 (brown bars), with the difference calculated between the latter – earlier years and the error bars indicating the standard deviation among plots. The plots are aggregated into different fire frequency bins to display means. (d) Across all plots, regression between the standing biomass in 2010 and the rate of change in biomass between 2010 and 2018 for plots that lost biomass by disease (red dots). Grey dots are plots that did not lose biomass.

(Table S6, Figure 3d). The survival–growth tradeoffs resulted in bur oaks being more abundant in frequently burned plots and pin oaks being more abundant in infrequently or unburned plots (fire–species interaction for biomass in 2010: $F_{1,20} = 11.6$, $P = 0.003$).

When the disease spread, the high abundance and rapid death following infection of pin oaks contributed to rapid biomass losses. For example, from 2010 to 2018 in the unburned plots, pin oak biomass declined by 87 Mg ha^{-1} (48% drop), while bur oak biomass increased by 0.8 Mg ha^{-1} (Figure 3c and d, Table S6).

Because bur and pin oaks comprised the majority of individuals and biomass in the tree community, the entire tree community responded in similar ways such that frequent burning regulated tree populations primarily by inhibiting the survival of saplings ($F_{8,23} = 4.4$, $P = 0.002$) and not adults ($P > 0.40$). Average growth depended on species type in saplings and was unchanged in adults ($F_{1,11} = 3.5$, $P = 0.089$ and $P > 0.50$ respectively (Figure 3e–f, S2–S4, Tables S7–S8). In turn, frequent fire reduced adult biomass by only 10% and sapling biomass by 55% from 1995 to 2010 (Figures S2, Table S8). Consequently, frequent burning regulated tree populations by inhibiting the survival and growth of saplings via a demographic bottleneck.

Tree-ring ages illustrated that the demographic bottleneck occurred at a threshold fire frequency of between one fire every 10 years and one fire every 3 years (Figure S5a–b). In the intermediate frequency plots, 98% of the trees were older than the establishment of the experiment, demonstrating fire eliminated the recruitment of trees to ≥ 5 cm size (Figure S5c–d). In the low frequency plots, 76% of trees established after 1964, demonstrating recruitment throughout the experiment. The mean age of 5-cm stems in the low frequency plots was 14.6 ± 4 years, which illustrates that decadal fires

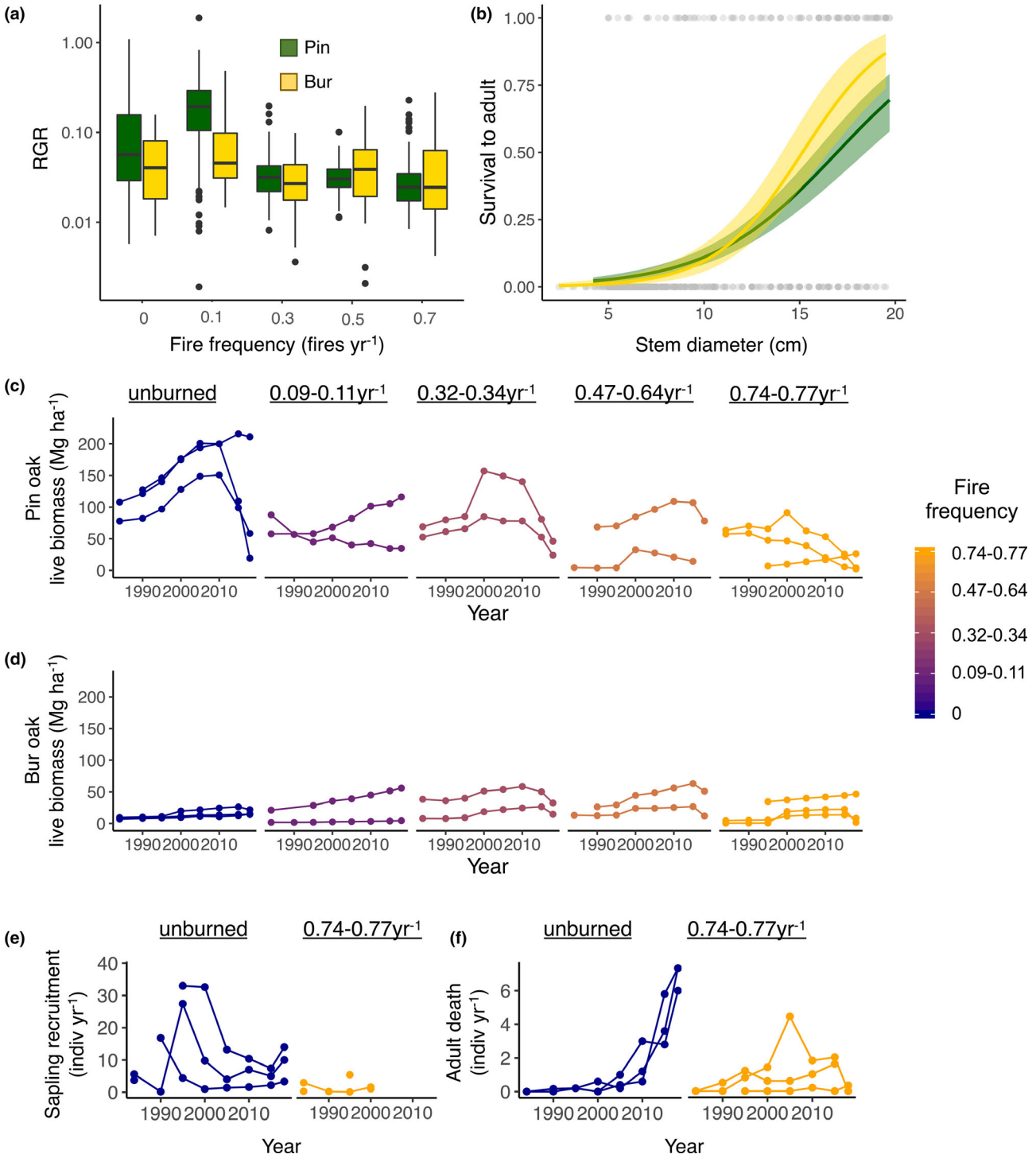

Figure 3 Fire and disease effects emerged through different demographic processes and depended on species composition. (a) Box and whiskers plot of relative growth rates (RGR) for all trees in the different fire frequency treatment categories expressed as relative basal area increment, in year^{-1}. (b) Probability that a sapling grows to adulthood as a function of starting stem diameter in the burned plots fit with a binomial generalised linear model. Each point is an individual tree tracked to adulthood/mortality. Pin oak: intercept=−5.1, coefficient = 0.3, n = 669; bur oak: intercept= −6.6, coefficient = 0.4, n = 246. (c and d) Changes in the biomass of pin and bur oaks through time in the different fire frequency treatments. (e and f) Demography of saplings (e) and adults (f) for all tree species calculated from pairs of surveys that followed one another (e.g. 2005 vs. 2010). (e) Sapling recruitment is the appearance of a new tree in a survey that was not previously documented. (f) Death of adult trees is the disappearance of the individual in a subsequent survey.

allow for some, albeit inhibited, recruitment while any higher frequency practically eliminates recruitment.

Disease caused tree populations to decline (particularly in pin oaks) (Figure 3f, Tables S7,S8). When all tree species were combined, the mortality of adults in unburned plots, which were mostly pin oaks, increased by 765% from 2 to 15 individuals ha^{-1} $year^{-1}$ pre- and post-disease respectively; resulting in mortality becoming roughly equal to sapling recruitment in unburned plots (adult mortality: 15 ± 5 individuals ha^{-1} $year^{-1}$ and sapling recruitment: 19 ± 12 individuals ha^{-1} $year^{-1}$) (Figure 3e, Table S7). Thus, adult mortality of susceptible species declined precipitously following disease spread.

Disease changed the efficacy of fire's control on ecosystem structure

Several observations suggest fire–disease interactions influence the potential for savannas and forests to persist. First, the outbreak of disease decreased the fire frequency required to maintain an open canopy typical of a savanna. In the presence of disease, plots burned at intermediate frequencies (one fire every 3 years) had tree cover (31% in 2018) similar to that of the most frequently burned plots (three fires every 4 years) before the disease (28% in 2010).

Second, disease changed several properties of the forests relevant to their fire resistance. Before disease, tree cover decreased light penetration to the herbaceous layer ($t = -4.8$, $P = 0.001$), nearly excluding grasses, which fuel fires in this system (Wragg *et al.* 2018) ($t = -4.0$, $P < 0.001$; Figure S6). Congruently, in the fire plots, fire intensity declined with tree cover, evidenced by a negative correlation between fire temperature and tree canopy leaf area ($F_{1,12} = 6.4$, $P = 0.027$, Supporting Information). Thus, fire suppression allowed for the transition from a savanna with an extensive and flammable grassy fuel layer to a forest with little grass biomass. Disease-driven tree mortality began to reverse this transition, evidenced by the increase in grass cover in two unburned plots ($t = 3.2$, $P = 0.033$). The rapid death of adult trees in these plots resulted in a greater proportion of trees being fire-sensitive saplings (84% of individuals). Consequently, disease shifted several properties of forests that point to greater potential for fire spread and tree mortality in fires.

Model simulations revealed a strong, nonlinear dependence of disease spread on historical fire frequency. In low frequency and unburned plots, the high initial tree densities allowed disease to spread more quickly than infected individuals died (see Supporting Information for equilibrium conditions and Figures S7–S8 for parameterisation). In intermediate to high frequency plots, the low host density kept disease levels low (Figure S9). Thus, adult stem density peaked at intermediate fire frequencies that limited the rate of disease spread but where fires were infrequent enough to allow saplings to grow into adults (Figure S10). Populations with and without disease exhibited long transient dynamics (> 100 years) during which sapling and adult tree densities continued to change (Figure S9, S11–S12). Both during transient dynamics and at equilibrium, the unburned plots in the presence of disease exhibited a novel size structure in which saplings had a high relative abundance and overall tree cover was lower than in disease-free forests (Figure S9). We term this state a 'diseased woodland' (Figure S10).

We next evaluated how disease determined the potential severity of fire and how this also depended on the historical fire regime. To do so, we introduced a common fire frequency of one fire every 2 years to plots burned less frequently and tracked the resulting tree mortality over 15 years at the new frequency. We conducted these simulations with two different disease conditions: one that allowed disease to invade dynamically and the other that kept disease at a low density. In the presence of disease, the vulnerability of tree density to a new fire frequency was lowest in systems that had historically burned at an intermediate frequency, with large mortality of adult trees in plots that historically had been unburned (Figure 4c and d).

These simulations suggest that the formation of the diseased woodland is likely transient if fire is reintroduced into the system. To evaluate this, we calculated the simulated time required for the system to reach sufficient grass cover for fire to percolate (*c.* 35%), and found that diseased woodlands shifted at a 10 times faster rate than forests without disease (Figure 4e) exposed to the same fire frequency. Plots with an intermediate historical fire frequency had the slowest transition rate to savanna (Figure 4e).

DISCUSSION

Our empirical analysis demonstrated that while fire alone determined whether a plot stayed a savanna or transitioned into a forest, disease invasion decreased tree biomass, increased light penetration and grass cover, and shifted tree populations from being dominated by fire-resistant adults to fire-sensitive saplings, especially in the unburned plots (Figure 2b, 5). Our model predicted that disease increased an ecosystem's vulnerability to hypothetical future fire by moving them into a 'diseased woodland' structure that rapidly transitioned to savanna when fires were reintroduced (Figure 4e, 5). Taken together, shifting pathogens can alter the effect of fire frequency in ways that may influence the distribution of savanna and forest ecosystems.

Fire–disease interactions and density dependence

In savannas, fire is mostly propagated by grasses, which tend to decline with increasing tree cover in savannas and be abruptly excluded at forest edges (Hoffmann *et al.* 2012). This negative tree density–fire relationship contrasts with the positive density dependence of pathogen spread, especially of conspecific trees (Menges & Loucks 1984; Packer & Clay 2003). Thus, the introduction of disease promotes fire because the low tree cover state, created after disease spread, burns more readily.

In systems that become more flammable as stands become denser and older (Parker *et al.* 2006), disease may interact with fire differently. For example, disease outbreaks could potentially decrease fire risk. However, disease-killed trees often become important fuel for fire, which is compounded by the open tree canopy increasing surface fuel flammability

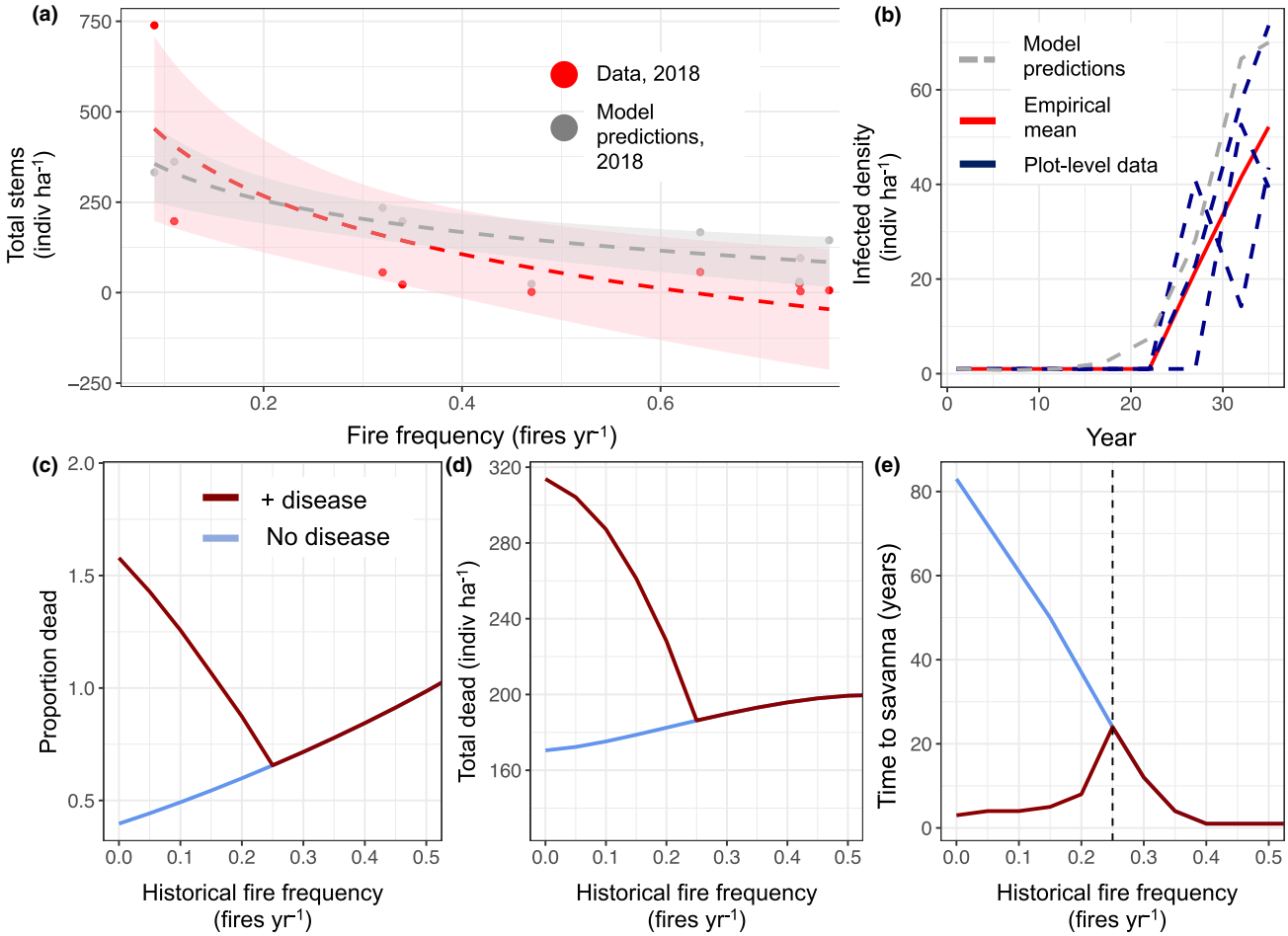

Figure 4 Disease increases the sensitivity of ecosystems to fire, especially in historically fire suppressed systems. (a) Comparison between model predictions and measurements of total stem density in the plots at the end of the time series. (b) Measured and modelled trends in the unburned plots for densities of infected trees. (c–e) Simulations of populations to a steady state under different historical fire frequencies and then changing fire frequency to a constant value (here 0.50 fires year^{-1}). (c and d) Tracking the dynamics of dead trees summed through the first 15 years, expressed as the proportion of all initial trees (c) and total number of trees per hectare (d). (e) Calculating the number of years that it takes plots to transition to a savanna (grass cover > 35%) based on the historical fire frequency when disease is either absent or introduced. Dashed line illustrates the threshold fire frequency of the plots where disease was unable to become abundant.

(Tepley *et al.* 2018), both of which contribute to disease promoting fire susceptibility. Other biotic disturbances such as herbivory by insects can also increase following fire suppression (Kurz *et al.* 2008), resulting in more severe outbreaks in areas that have experienced long periods of fire suppression (Parker *et al.* 2006; Jenkins *et al.* 2008). More severe fires generally follow these outbreaks (Parker *et al.* 2006; Jenkins *et al.* 2008). Consequently, the phenomenon that disease primes the system to fire by promoting fuel accumulation is likely applicable to other ecosystems with plant pathogen–fire interactions.

Whether disease and fire either positively or negatively covary to promote low tree cover ecosystems is likely explained by the relationship between successional state and fire susceptibility. When early-successional vegetation is more flammable, positive fire–vegetation feedbacks can better maintain a non-forested landscape (Tepley *et al.* 2018); in turn, disease outbreaks in these systems may promote the existence of the non-forested landscape. In other ecosystems, disturbed landscapes are less susceptible to fire because disturbance favours less flammable plant species or lower fuel loads (Kulakowski *et al.* 2013), resulting in disease outbreaks reducing fire occurrence and spread.

Although our results are taken from a single site, tree pathogen outbreaks are common worldwide and may be increasing with pathogen invasion and climate change. For instance, South African savannas have recently been exposed to the fungal pathogen, *Fusarium euwallacea*, which spreads by an invasive beetle and kills trees (Paap *et al.* 2018). Pathogen pressure may be further altered as climate change shifts the spread of vectors. Consequently, the relevance of plant pathogens to savanna–forest biome distributions may be more important than previously considered.

Role of species composition

Disease had a large effect on forests in our system largely because *Q. ellipsoidalis* was responsible for the rapid growth

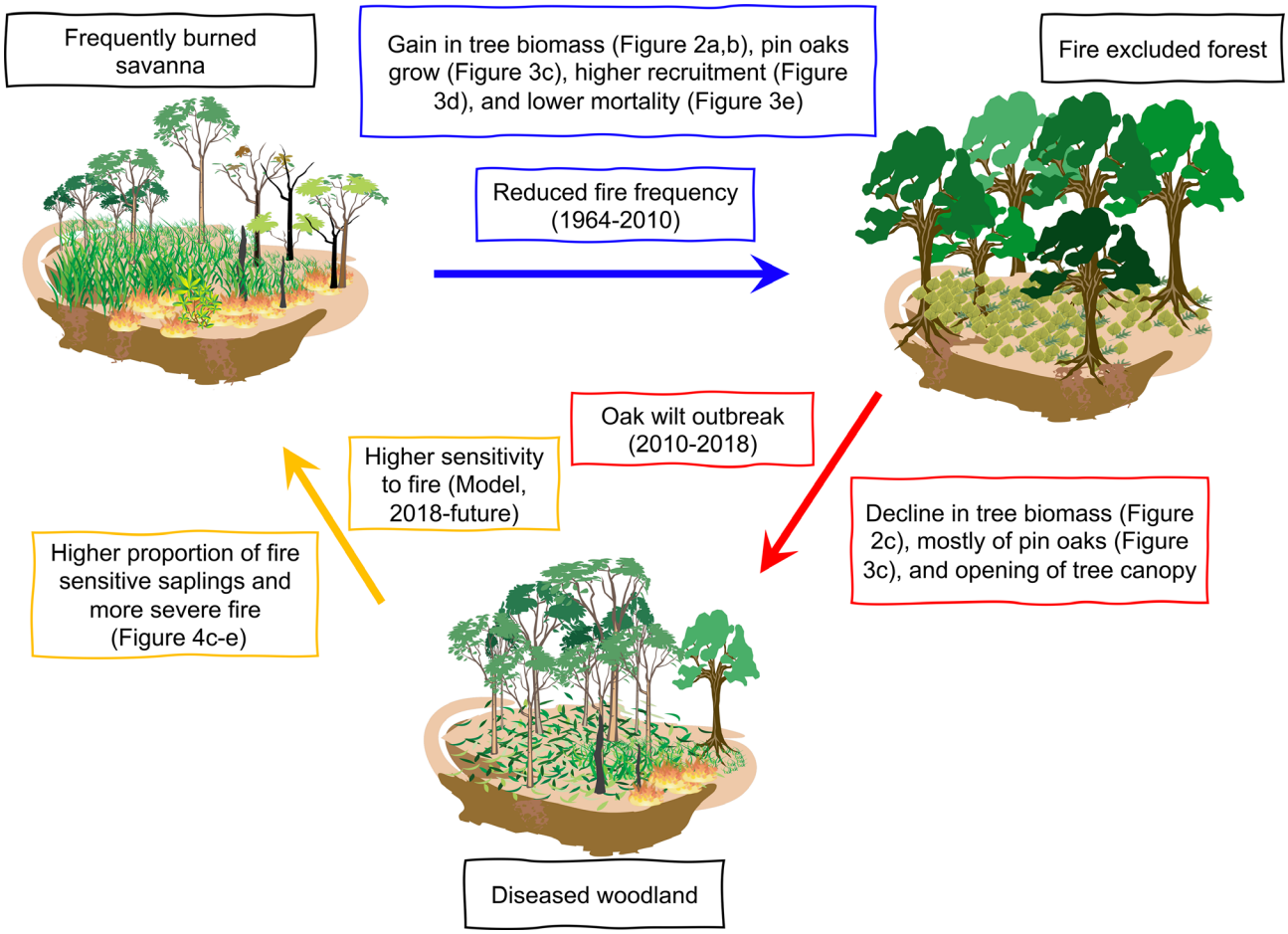

Figure 5 Conceptual schematic illustrating the effects of fire and disease on ecosystems which integrates model and data results. In the absence of disease (blue arrow), fire determines whether a landscape is a savanna or a forest, which have distinct properties. But when disease is present, it kills the dominant tree species in forests, opens the canopy and results in saplings dominating the tree community, creating a diseased woodland. Our model predicted that this diseased woodland is more sensitive to the re-introduction of fire than forests, creating a pathway that can promote biome transitions from forests into savannas (orange arrow).

and forest formation. Many plant pathogens are genus specific or species specific (e.g. Beech bark disease, *Nectria spp*., targets *Fagus grandifolia*) or clade specific (e.g. the root rots *Heterobasidion annosum* and *Armillaria* spp. target multiple conifer species, e.g. (Gerlach *et al*. 1997)). Many diseases can have large effects on tree mortality, such as sudden oak death killing > 3 million trees in California (Goheen *et al*. 2006).

In systems with specialised diseases but a diverse tree community, over longer timescales the colonisation and growth of disease-resistant tree species could compensate for the mortality of susceptible species. Despite the existence of several tree species in other forests in Cedar Creek, the forests that formed under fire suppression in the CCSFE were dominated by pin oaks. The other tree species are much more sensitive to fire (e.g. maple) and are almost completely absent in the plots that have remained savanna, likely limiting their ability to rapidly recruit when fire was excluded (Peterson & Reich 2001). As forest succession continues, pin and bur oak forests are replaced by red oaks and maples (Frelich et al. 2015), but this has not yet occurred in CCSFE. While oak forests in eastern Minnesota can still burn (but fire is heavily suppressed), they would do so less frequently even under a natural fire regime than the savanna (fire return intervals of 15–50 vs. 3–6 years respectively). We were not confident in the ability of our two-species model to predict the long-term dynamics of bur oaks because of limited demographic events, and thus we did not explore species turnover in the model.

Our model does not explicitly consider the spatial dynamics of fire or disease spread, although both fire (Schertzer & Staver 2018) and disease spread (Levin & Durrett 1996) are spatial processes. Our modelling of disease implicitly invoke spatial processes, however. In plants, the idea that individuals need to be connected for a disease to spread readily through a population – intrinsic to the well-known concept of R_0, for instance (Anderson & May 1992) – simply translates into the idea that more densely packed plants may be more disease susceptible. This idea is well supported empirically in the agricultural literature (Burdon & Chilvers 1982) and in a diverse literature exploring Janzen–Connell effects in natural ecological systems (Comita *et al*. 2014). Further spatially explicit modeling work may be of interest in this system. By the same token, we assume a constant fire frequency and not stochastic ignitions, which may influence dynamics (Higgins *et al*. 2000). However, we do not expect these factors to alter the

conceptual conclusion that fire–disease interactions affect the dynamics of savanna–forest ecosystems.

Other modifying factors

The long-term stability of savanna–forest ecotones will depend ultimately on fire–vegetation feedbacks at the landscape scale such as fires being rapidly extinguished at forest edges (Hoffmann *et al.* 2012) and how ecotones are influenced by rising CO_2 and climate change (e.g. (Bond & Midgley 2000; Reich *et al.* 2015)). Across the savanna–forest boundary in CCSFE, there are threshold changes in herbaceous biomass and cover (Peterson *et al.* 2007; Pellegrini *et al.* 2020), where herbaceous biomass remains relatively unchanged across savanna plots but drops sharply by 63% in forests. Lower herbaceous biomass likely diminishes fire spread in forests because there is a threshold response between grass biomass and fire spread (Wragg *et al.* 2018). Consequently, we expect that fires originating in savannas diminish at the forest edge; these edge effects likely contribute to the persistence of forests in the region, but further experiments are needed.

Future measurements of how the forests continue to change in response to the disease outbreak are required to validate our model's forecasts. The occurrence of oak wilt is relatively recent at Cedar Creek, with little evidence it was an endemic disease prior to this recent outbreak. Fire, on the other hand, has periodically occurred throughout the past *c.* 500 years, although forests always existed in the landscape (Grimm 1984; Leys *et al.* 2019). Whether the persistence of forests is indicative of fire–vegetation feedbacks or the presence of fire refugia such as in riparian areas (Danz *et al.* 2011) is unclear.

Herbivory also plays an important role in savanna dynamics because browsers can limit the growth of tree saplings and in some cases the survival of adult trees. At Cedar Creek, deer can have significant effects of tree growth in the open savanna, although the magnitude of the effect has received mixed interpretations (Inouye *et al.* 1994; Ritchie *et al.* 1998). It is more likely that herbivory, severe droughts and competition with herbaceous species all contribute to limiting tree encroachment (Inouye *et al.* 1994).

CONCLUSION

In conclusion, our analyses illustrate that increasing fire frequencies can dampen the effect of disease, and our model predicts that the presence of disease amplifies the efficiency of fire at maintaining savannas. The relevance of disease–fire interactions for savanna–forest ecotones will depend on the geographic scope of tree diseases and pathogen spread, but they present an under-explored mechanism that could contribute to the existence of savannas and forests as alternative ecosystem states.

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CODE AVAILABILITY

The statistical and model analyses are conducted using code described in the statistical software packages cited within our manuscript.

AUTHOR CONTRIBUTION

AFAP, AH, PBR and SEH conceived this specific study. PBR, SEH and JCB coordinated and implemented the long-term fire frequency experiment. AFAP and AH developed the model with feedback from ACS. JCB and RAM help to lead the monitoring of oak wilt. FS performed tree-ring analyses. AFAP ran statistics with input from AH. AFAP wrote first draft of the manuscript and all authors provided feedback.

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DATA AVAILABILITY STATEMENT

All data will be made accessible at Figshare, https://doi.org/10.6084/m9.figshare.13729258.v1, No custom code was created.

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