Forecasting the limiting effects of *Phytophthora* root rot on restoration of the American chestnut in the eastern United States

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1. Abstract

American chestnut (*Castanea dentata*) was functionally extirpated from eastern U.S. forests by a chestnut blight from Asia. As efforts to produce blight-resistant American chestnut genomes advance, approaches to reintroduce chestnut throughout its former range are being developed. However, chestnut is quite susceptible to a root disease in the southern half of its former range, and the pathogen that causes the disease is expected to move northward as climate warms. Because restoration of a self-sustaining chestnut population is a landscape-scale problem, we used a process-based forest landscape model (LANDIS-II) to conduct experiments to quantify the effects of root rot on the effectiveness of chestnut population restoration efforts in the center of the former range of chestnut under various climate scenarios. We found that…

Key words: climate change, elevated CO2, American chestnut restoration, root rot disease, *Phytophthora cinnamomi,* *Castanea dentata*, LANDIS-II, PnET-Succession

1. Introduction
   1. Background of problem studied

American chestnut (*Castanea dentata*) was an abundant species in many eastern U.S. forests (Ellison et al. 2005) prior to its functional extinction by two invasive fungal pathogens. Mortality of chestnut in the southern U.S. was first reported in the mid-19th century and is now attributed to infection by the root pathogen *Phytophthora cinnamomi* (Anagnostakis 2002). Chestnut blight, caused by *Cryphonectria parasitica*, was likely introduced to the US in the late 18th century, and killed most large American chestnuts throughout the species’ range by the 1950s. Efforts to develop bight-resistant American chestnut populations using backcross hybridization (Steiner et al. 2017) and transgenic techniques (inserting an oxalate oxidase encoding gene from wheat, e.g., Zhang et al. 2013) are underway. More recently, capturing resistance to both root rot and blight in American chestnut breeding populations has become a goal of American chestnut restoration (Westbrook et al. 2019). Once genetically diverse populations of blight-resistant American chestnuts are produced, offspring of these trees will be reintroduced throughout its former range, with the hope of restoring the ecological, economic, and social benefits the species once provided.

There are many uncertainties associated with such an undertaking. Chestnut must be capable of successfully competing with established cohorts of other species in order to achieve a self-sustaining population. Chestnut must also be able to adapt to the novel abiotic (e.g., climate, air chemistry) and biotic (e.g., insect pests, exotic species) conditions that are becoming quite different than they were when the species was dominant. Gustafson et al (2018) used a forest landscape model (LANDIS-II) to project the efficacy of various climate and chestnut restoration scenarios in western Maryland (USA) by mechanistically accounting for temperature and elevated CO2 effects on growth and for natural and anthropogenic disturbances. They found that with aggressive restoration efforts, chestnut can again become an important component of forested ecosystems in the Appalachian Mountains. However, one critique of that study was the omission of the effects of Phytophthora root rot.

American chestnut is quite susceptible to the root disease (root rot) caused by *Phytophthora cinnamomi* Rands and it is thought to have suffered extensive mortality caused by the root pathogen prior to the arrival of chestnut blight in the southern half of its former range (Anagnostakis 2012). The pathogen infects a wide range of hosts with pathogenic activity increased by warm wet soils, but is limited in soils that freeze deeply in winter (Sinclair and Lyon 2005). Because the pathogen currently only occurs below 40⁰N latitude due to cold-limitation, it is expected to move northward (and upward) as conditions warm (McConnell and Balci 2014, Burgess et al 2017). Restoration plantings north of 40⁰N latitude currently do not need resistance to *P. cinnamomi*,but as climate warms, resistance will became increasingly important throughout the former range of American chestnut. It is currently unknown what level of mortality will occur in restoration plantings or what level of resistance will be needed to reach restoration goals, but resistance to the root pathogen is believed to be important for survival on some sites (Clark et al 2019 and references within).

* 1. Rationale for study

Restoration of a self-sustaining American chestnut population is a landscape-scale problem, requiring landscape-scale implementation to produce landscape-wide outcomes. Furthermore, because the occurrence of an American chestnut population within its former range under the climate, pests and disturbance regimes expected in the future is a distinctly novel combination, it is not advisable to use the past to attempt to predict the outcome of restoration efforts (Gustafson 2013). Mechanistic forest landscape models based on first principles provide a robust tool for such a study because they are process-based (Cuddington et al. 2013) and incorporate most of the major factors that structure forested landscapes in time and space at landscape scale. Forest dynamics in such models are an emergent property of the interaction of the processes (including growth and competition) and the inputs (including abiotic soils and climate conditions), and produce the most reliable projections of expected future forest dynamics.

* 1. Approach used

Following Gustafson et al (2017a, 2018), we used the physiologically mechanistic PnET-Succession forest growth simulation extension linked to process-based disturbance extensions within the LANDIS-II forest landscape model (Scheller et al 2007) to conduct a simulation experiment to assess the outcome of American chestnut restoration efforts in the presence of the root rot pathogen. We created a new disturbance extension that simulates cohort damage and mortality caused by the root rot pathogen that accounts for the presence of suitable hosts, soil moisture, temperature and the development of resistance to the pathogen by natural selection. We focused on the center of the former chestnut range, which coincides with the northern edge of the range of the root rot pathogen(*Phytophthora cinnamomi*). Using this one location, we experimentally modified climate inputs to produce 1) a no root rot scenario representing the cold-protected northern part of chestnut range, 2) a current root rot scenario for the study area, and 3) a warmer, elevated root rot scenario representing both the southern part of chestnut range today and the potential future of the study area.

* 1. Objectives

Our objectives were to 1) quantify the impact of root rot on chestnut biomass as restoration activities proceed, 2) assess whether root rot has the potential to completely thwart chestnut restoration efforts, and 3) explore whether the results suggest management strategies that might help mitigate the negative effect of root rot on restoration efforts. Any hypotheses to test?

1. Methods
   1. Study area

Our study was centered on the Savage River State Forest (SRSF) in western Maryland (USA), located near the center of the former range of American chestnut (Figure study area). The SRSF is located on the Appalachian Plateau, and receives abundant rainfall (114-140 cm/yr; Brown and Brown 1984). Topography of the plateau consists of steep and dissected ravines or undulating terrain on broad ridgetops underlain by sandstone and shale, with elevation ranging from 375–900 m (Stone and Mathews 1977). The study area is dominated by northern red oak (*Quercus rubra*), with sugar maple (*Acer saccharum*) codominant on mesic slope positions, chestnut oak (*Q. prinus*) codominant on drier slope positions, and red maple common in the understory.

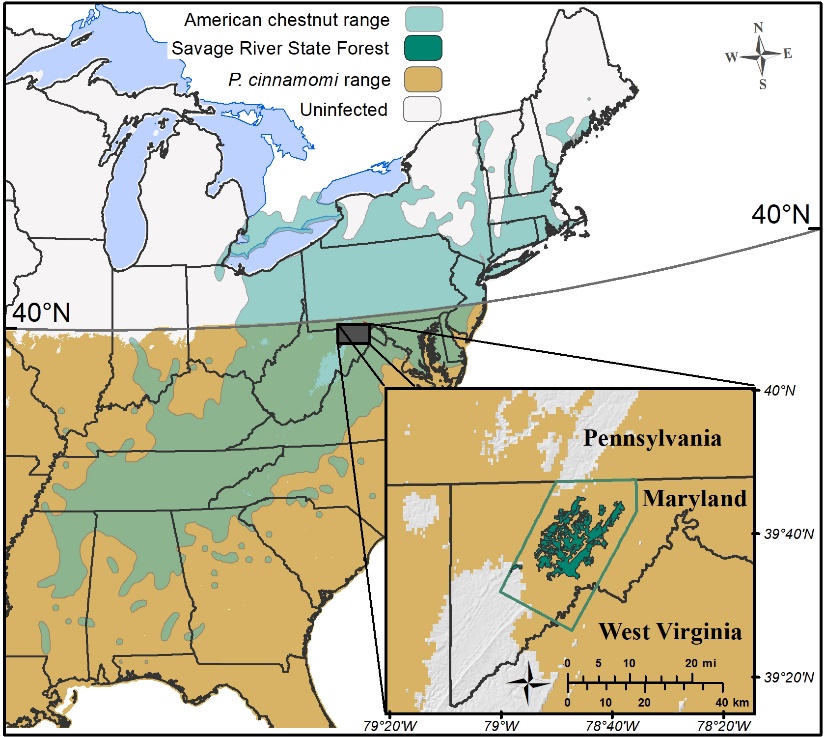


Figure study area. Location of study area relative to the former range of American chestnut and the approximate range of *P. cinnamomi*, estimated using USDA plant hardiness zones and a northern limit of 40o N latitude.

* 1. Experimental design
     1. Restoration scenario

The simulation experiment was conducted as a 2 x2 factorial, with the climate factor having two levels (historical and RCP 8.5) and the root rot factor having two levels (with and without root rot simulated). We used a clearcut-and-plant chestnut restoration strategy in all simulations, which was the most aggressive strategy implemented by Gustafson et al (2018). This strategy plants chestnut in all planned silvicultural treatments that use clearcutting. Each factorial combination was simulated for 200 years with three replicates.

* + 1. Climate factor – describe GCM scenarios

The historical climate scenario used weather data (including photosynthetically active radiation, i.e., light) for an area surrounding the study area that was subset from the Daymet Daily surface weather 1-km grid for North America, 1980-2015 (Thornton et al. 2014). We used monthly averages prior to 1980 (for “spin-up” of the biomass of existing cohorts), and actual records through 2014, repeating the observations of the period 1980-2014 for 200 years to create a “historical” weather scenario into the future. CO2 was set at 335 ppm prior to 1980, gradually increasing to 390 ppm by 2010, and held constant after that. For the climate change scenario, we used projections from the GFDL-CM3 GCM (RCP 8.5 emissions scenario, run=r1i1p1) centered on the study area for the period 2006-2100, repeating the last 30 years of the projections through 2216. This scenario produced an average temperature 4.6 oC higher than the historical scenario, and 7% more precipitation. We used the extended RCP8.5 CO2 concentrations of Meinshausen et al. (2011), with CO2 concentration reaching 1902 ppm by 2216. The GCM data did not include photosynthetically active radiation, so we repeatedly applied the historical light data from 1980-2014.

* 1. Model used
     1. LANDIS-II overview

The simulation experiment was conducted using LANDIS-II (Scheller et al. 2007), a forest landscape modeling platform using extensions (plug-ins) to mechanistically simulate forest growth and disturbance (e.g., insect outbreaks and timber harvesting). LANDIS-II models species cohorts rather than individual trees, representing space as a grid of cells (30m resolution in this study). Each ecological process is encapsulated by an independent extension that modifies cohort biomass conditionally based on abiotic and vegetation conditions on the cell and input parameters. Interactions among climate, growth, succession and disturbance are not specified *a priori*, but emerge out of the cumulative effects of the independently simulated processes.

We used the PnET-Succession extension (v4.0, De Bruijn et al. 2014b) to simulate growth processes (establishment, growth, competition, senescence) because its mechanistic use of physiological first principles is best suited to model novel situations such as climate change and the introduction of new species. Note that this is a newer version than used by Gustafson et al (2017a, 2018), having modified algorithms for cohort establishment and temperature effects on photosynthesis. PnET-Succession models growth as a competition of cohorts for light and water, and cohorts die when their respiration exceeds net photosynthesis sufficiently to exhaust their carbon reserves. As soil water availability decreases, photosynthesis decreases. Available soil water is determined by precipitation, loss to evaporation and runoff, soil porosity, and consumption by cohorts. When water is adequate, the rate of photosynthesis for a given species cohort increases with light available to the cohort (dependent on canopy position and leaf area), atmospheric CO2 concentration and foliar N, and decreases with age and departure from optimal temperature. Temperature also affects vapor pressure deficit, respiration and evapotranspiration rates. Thus, growth rates vary monthly by species and cohort as a function of precipitation and temperature (including extreme events), directly affecting competition and ultimately successional outcomes.

We used the PnET-Succession tree species life history and physiological parameters found in Gustafson (2018), modifying the revised establishment and temperature parameters to produce behavior similar to the prior study, making our results generally comparable with those studies. Chestnut restoration activities were held constant for all factor combinations, and were simulated using the Biomass Harvest extension (v4.3, Gustafson et al 2000). Disturbance by three exotic insect pests either recently introduced or imminent to the study area [emerald ash borer (*Agrilus planipennis*) hemlock woolly adelgid (*Adelges tsugae*), and the Asian Longhorned Beetle (*Anoplophora glabripennis*)] was also simulated for all factor combinations using the Biological Disturbance Agent (BDA) extension (v3.0, Sturtevant et al. 2004; Sturtevant et al. 2017).

* + 1. Details of RR extension

For this study, we developed a new LANDIS-II disturbance extension (Root Rot v1.0) to simulate biomass loss (representing individuals killed) and complete cohort mortality caused by infestations of the root rot pathogen (*Phytophthora cinnamomi*). The extension does not simulate dispersal of the pathogen, instead assuming the pathogen can reach any site in the simulation landscape (e.g., SRSF) at any time. Each site has a mutually exclusive status of Susceptible, Infected (non-symptomatic) or Diseased (symptomatic). Cells that are Infected or Diseased can revert to a status of Susceptible only when the pathogen is absent (Presence = 0). Cells that are Diseased can stochastically revert to a status of Infected, and will always revert to Infected if all susceptible tree hosts are eliminated. The user can optionally provide an input map giving the initial status of each cell; otherwise all active cells are initially assumed to be Susceptible. Extension inputs (including the time step of the extension) are provided to the model in a text file (Table RR inputs). In the time steps the extension is executed, each site is evaluated for transitions between states, with the probability of each transition determined by the presence of the pathogen (controlled by a temperature variable (dTemp)) and a conducive environment variable (controlled by a soil water variable (dWater)).

dTemp = (AnnTmin - LethalTemp) / ABS(LethalTemp) (1)

where AnnTmin is the average minimum monthly temperature across years in the time step (Figure dTemp). Presence is then computed as a binary value, being 1 if a uniform random number is greater than dTemp, or 0 otherwise. If Presence = = 0, the site transitions to Susceptible (S) regardless of current state. If Presence = = 1, other transitions are possible based on the conducive environment variable.



Figure dTemp. Graphical depiction of the behavior of equation 1. Probability of pathogen presence is proportional to dTemp.

Table RR inputs. Root rot extension inputs.

|  |  |  |
| --- | --- | --- |
| Input parameter | Description | Notes 1 |
| Species susceptibility table | Index of species susceptibility to damage when disease occurs, ranging from 0.0 – 1.0 | 1.0 is completely susceptible and 0.0 is unsusceptible |
| LethalTemp | The minimum temperature (oC) below which *P. cinnamomi* cannot survive | e.g., -24 indicates *P. cinnamomi* unable to survive in USDA hardiness zone 5 or colder |
| phWet 2 | The pressurehead threshold below which the soil is considered wet | Under wet conditions it is possible for a site to progress from S to I and from I to D |
| phDry 2 | The pressurehead threshold above which the soil is considered dry | Under dry conditions it is possible for a site to progress from I to D. |
| phMax 2 | The pressurehead threshold above which the soil is considered optimal for pathogen | These are optimal conditions for a site to progress from I to D |
| minProbID | The minimum probability of infected sites converting to diseased | At moderate pressurehead, the probability of D will be greater than this value |
| maxProbDI | The maximum probability of diseased converting to infected | At moderate pressurehead, the probability of D will be less than this value |

1 Site status is one of Susceptible (S), Infected non-symptomatic (I) or Diseased symptomatic (D)

2 ph= m of pressure head, a unit of soil water potential. In PnET-Succession, ph is tracked using absolute values. Pressurehead equals 0 when soil is saturated and increases as water is reduced. A pressurehead of approximately 33 equates to soil field capacity, and 150 equates to soil wilting point.

A conducive (to pathogen) environment is a function of the wetness of soil (dWater) and the presence of susceptible hosts. A currently Susceptible (S) Site can transition to Infected (I) or Diseased (D). The probability of an S site converting to I [p(S:I)] (Figure pSI) is:

p(S:I) = dWater = IF(ph < phWet) THEN (-1/phWet \* ph + 1) OTHERWISE (0) (2)

where ph=pressure head.



Figure pSI. Graphical depiction of the behavior of equation 2.

The probability of an I site converting to D [p(I:D)] is bimodal (Figure pID). Probability decreases from 1 when saturated (ph = 0) to 0 at phWet, and increases from 0 at phDry to 1 at phMax according to:

p(I:D) = dWater = IF(ph < phWet) THEN (minProbID – 1)/phWet \* ph + 1; ELSEIF(ph > phDry) THENIF(ph > phMax) THEN (1); ELSE (m1 \* ph + b1); ELSE (minProbID)) (3)

where m1 = (1-minProbID)/(phMax - phDry), and b1 = minProbID – (phDry \* m1).



Figure pID. Graphical depiction of the behavior of equation 3.

The probability of an S site converting to D [p(S:D)] is the product of the probabilities p(S:I) and p(I:D), that is, it must make both transitions.

p(S:D) = p(S:I) \* p(I:D)

A currently diseased site (D) can transition to Susceptible (S) or Infected (I). The probability of D converting to S [p(D:S)] is binary depending on the presence of the pathogen. If Presence = = 0, then p(D:S) = 1; if Presence = =1, then p(D:S) = 0. A D site converts to I if no cohorts with susceptibility >0 are present, or with probability [p(D:I)] when pressurehead is between phWet and phDry. Maximum probability occurs at the midpoint between phWet and phDry:

p(D:I) = MIN(maxProbDI, IF(ph < phWet) THEN 0, ELSEIF(ph > phDry) THEN (0), ELSEIF(ph <= (phDry – phWet)/2) THEN (m2 \* ph + b2), THEN m3 \* ph + b3)))) (4)

where m2 = 1/((phDry – phWet)/2 - phWet); b2 = -1\*phWet \* m2; m3 = 1/((phDry – phWet)/2 - phDry); b3 = -1\*phDry \* m3.



Figure pDI. Graphical depiction of the behavior of equation 4.

For any site with a status of Diseased (D), the extension removes a proportion of cohort biomass equal to the susceptibility of the species (regardless of cohort age or biomass), representing the death of that proportion of individual trees. The extension writes a record of its activity at each time step to both an event log and a summary log. If requested, the extension will output maps at each time step of the biomass removed from each site and TOLP.

* 1. Analytical methods
     1. Line graphs (with 95% CI envelopes) of chestnut biomass under each factorial combination
     2. MANOVA of factorial experiment?
     3. See if root rot scenarios include 0 chestnut biomass within 95% CI.

1. Results



Figure chestnut. Effect of treatment combinations on the mean biomass of chestnut across the landscape through simulated time. Color indicates climate treatment, shape indicates root rot treatment, and line indicates ALB treatment. Error bars show 95% confidence intervals of replicate variability.

* 1. Baseline – no root rot (proxy for cold-limited landscapes)
  2. Historical w/ root rot (will inform how much our prior projections may have been off)
  3. CC (south of study area, and possible future of study area)
  4. Hypothesis or statistical tests

1. Discussion
   1. Major insights
      1. How much root rot might be expected to hamper chestnut restoration efforts at various latitudes and elevations.
      2. How does root rot affect other species? Does it substantially modify forest composition?
      3. How current results modify interpretation of our prior studies in this study area
      4. Do the results suggest any management or restoration options?
   2. Assumptions
      1. Pathogen can disperse everywhere. This assumption is reasonable for landscapes that do not span the northern range limit of *P. cinnamomi*, but may be problematic for large landscapes that include that range limit.
      2. Each active cell has a mutually exclusive status of Susceptible (S), Infected non-symptomatic (I) or Diseased symptomatic (D).
      3. Cells that are Infected (I) or Diseased (D) only revert to a status of Susceptible (S) when pathogen is absent (Presence == 0).
      4. Cells that are Diseased (D) can revert to a status of Infected (I), and will always revert to I if all susceptible tree hosts are eliminated.
   3. Caveats
      1. Susceptibility does not account for cohort age or size
      2. Relative abundance or host biomass do not contribute to the calculations.
      3. The pathogen reproduces very quickly and saturated soil for 24hrs may be enough for symptom development in some inoculation systems. For example, single heavy rain events could be enough for conversion from a state of S to I. Our model would be fairly insensitive to such events because the extension time step can never be less than 1 year, and temperature and precipitation inputs have a monthly resolution. However, p(S:I) would be much lower in the case of a single rain event compared to periods of prolonged wetness, so we believe that our approach produces valid projections regardless of the temporal variability of rainfall within a month.
      4. Variability among replicates is low, resulting from stochasticity in access of cohorts to light and water, cohort establishment, and disturbances. Uncertainty related to model and parameter specification and future climate are certainly much higher, but are controlled to increase the signal from the experimental treatments.
   4. Conclusions
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