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Examining the Effect of Beech Bark Disease on Radial Growth in American Beech

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Abstract: Beech bark disease (BBD) is a significant threat to forests of North America and the impact of BBD on radial growth in the American beech is substantial. We developed a novel hierarchical Bayesian (HB) model to simultaneously model disease dynamics, tree growth, and the interaction of the two. Our model can be adapted to both emerging and more mature forest–pathogen systems to aid in ecosystem loss predictions. Long-term data from a single site minimized potential confounding variables such as climate change, precipitation, land use history, and soil conditions that may influence radial growth. Here, 206 beech trees were monitored over 15 years at an 85-acre site in southwestern Vermont, measuring diameter at breast height (DBH) and progression of BBD. Our model allows us to accurately estimate error rates in disease severity estimation and DBH measurements, and estimate the true state based on environmental variables. As disease poses significant threats to many tree species around the world, researchers can obtain more value and information from their datasets utilizing an adapted HB model.

Keywords: Bayesian analysis; observer error; *Fagus grandifolia*; beech bark disease; radial growth; diameter at breast height; disease ecology; tree growth modeling

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1. Introduction

The economic and ecological impacts due to the introduction of non-native, invasive pathogens is a concern and focus for foresters worldwide [1–4]. The decline in trees has resulted in loss of habitat, flora, fauna, and ecosystem services, including carbon sequestration and ecological memory [1,5–7]. North America has been struck by multiple tree diseases, including butternut canker *Sirococcus clavigigneti-juglandacearum* (Nair, Kostichka, and Kuntz), chestnut blight *Cryphonectria parasitica* (Murr. Barr), Dutch elm disease *Ophiostoma ulmi* ((Buisman) Melin and Nannf.), *Ophiostoma. novo-ulmi* (Brasier), and many others that have killed millions of trees [6–8]. Santini et al. [3] showed that in the past two hundred years, pathogen introduction to Europe has increased exponentially. Between climate change-induced susceptibility and shifts in natural ranges and the documentation of new pathogens, these trends show no sign of decline.

Beech bark disease (BBD), *Cryptococcus fagisuga* (Lindinger) and *Nectria* spp. (Woolenweber), is considered one of the most significant forest diseases in North America [8] and is the most active and relevant pathogen in our region in the Northeastern United States. In North America, BBD is caused by the pathogenic complex of an insect, the beech scale *Cryptococcus fagisuga*, or the more recently recognized *Xylococculus betulae*, with different neonectria fungi *Neonectria ditissima* and *Neonectria faginata* [9]. The scale is thought to have been accidentally brought to North America by way of Canada with a shipment of ornamental beech in the late 1800s [10]. In less than a century the disease managed to spread to nearly the entire range of the American Beech (*Fagus grandifolia*, with projections that the full range will be infected within the next couple of years [9,11,12].

The American beech is considered foundational to the forests of Eastern North America. This abundant, deciduous, late-successional species can live upwards of 400 years and grow to more than 100 feet, contributing significantly to the canopy, while being notably shade tolerant [13]. As one of a handful of prominent masting tree species in the region, American beech helps to support small and large herbivores [14,15], while providing essential habitat throughout its lifecycle. Despite BBD, climate change models support both the persistence and prominence of American beech in the landscape [16] although the current range, from northern Florida to Nova Scotia, is expected to shift northward [12]. Beech have been shown to be resilient and adaptable to potential acid rain-induced changes both in soil pH [12,17] and elevated aluminum [18]. Despite the hardiness of this hardwood, it has succumbed quite seriously to BBD, one of the "ten most unwanted alien forest pathogens" [8].

The effects of BBD have been shown to be both acute and chronic. BBD has significant ecological impacts, critical enough that work is being done to optimize the mast output despite BBD [19]. In addition to the ecological impacts, the economic impact of BBD is notable as it "stunts American beech . . . to unmerchantable size" [20] with some Canadian sawmills harvesting beech ahead of BBD onset [21]. BBD initially compromises the bark and cambium layers, but over time, the phloem, xylem, and heartwood are also impacted, damaging the value of the resulting lumber. Resistance to BBD is at the level of the beech scale. This insect bores a tiny hole into the outer bark of the beech tree and lays its eggs. The scale life cycle typically includes an overwintering in which hatched nymphs secrete a waxy protective layer that protects it from weather and predation. Once the nymphs molt, they depend on wind to travel to other trees to begin the cycle again. The tiny holes left behind by this process provides the ideal habitat for the *Neonectria* fungi ascospores to propagate and eventually kill the tree. The potential molecular mechanism for resistance to *Neonectria ditissima* remains elusive [22]. Proteomic and genomic studies in American beech have provided some promising insights [23,24], but it is generally accepted that less than 5% of beech are truly resistant to the beech scale, with some sites having fewer than 1% of trees being resistant [25,26].

Management is critical since BBD can also negatively impact other highly valued trees. Unruly young beech thickets have taken over large forested areas that have highly infected trees, out-competing both sugar maples (*Acer saccharum* Marsh.) and yellow birch (*Betula alleghaniensis* Britt.) [20,27]. Although the role of BBD in this phenomenon is debated, it is agreed that disease impact on propagation by seed and self sprouting may be regional and hard to predict [28]. The U.S. Forest Service and Canadian Ministries of Natural Resources provide management suggestions [28,29], and selective breeding for resistance continues [30,31].

Many studies have examined the detrimental effects of BBD on forest systems and structures [14,32–35], as well as economic consequences, but with less focus on the influence of the disease on the growth of the tree itself. A recent article, "Not Dead Yet" captures the phenomenon that BBD, in many cases, kills trees quite slowly, with beech hanging on for decades after infection [36]. Some trees infected as early as the 1930s in Downeast Maine were still alive 50 years later [37].

Long and short-term monitoring of the impacts of disease on tree populations is taken on by governmental agencies and academic research laboratories in conjunction with ecological management and restoration efforts. Tree health monitoring efforts regularly record the radial size or diameter at breast height (DBH) through tape measurement or calipers. Observational errors can occur, independent of tree species and regardless of whether breast height has been estimated or measured, with a root mean squared error of up to 10% [38,39]. The rate of radial growth, in relation to tree fitness, has far-reaching implications since "many ecosystem functions and services are depending on the course of tree growth" [40].

To explore the impact of beech bark disease (BBD) on radial growth, an earlier study [37] analyzed potential differences in the annual ring measurements of paired resistant and susceptible American beech at two different study sites in Maine. Growth decline differences were noted between the two sites but conclusions were limited as the climate varied and there were differences in the onset of the decline. The study revealed the

complexity of trying to evaluate the potential effect of BBD on radial growth and ultimately showed no statistically significant negative impact due to disease susceptibility, with some years even showing a relative increase. For our study, we decided to stay within one smaller forest system to minimize variation in climate, and in spatial and temporal disease spread.

Measuring DBH can become more complicated in trees inflicted with BBD, as bark cankering increases as the trees become sicker. To be able to ascertain the impact of BBD on radial growth, we developed a hierarchical Bayesian model that incorporates DBH measurement error. We also used this model to account for observer error in determining the level of BBD infection. The current ranking system for BBD requires direct observer assessments of both qualitative and quantitative traits relating to the presence of disease and the overall health of the tree [41]. The use of this ranking system by multiple observers over multiple years can lead to significant inconsistencies [42]. The new model seeks to address documented observer error both in DBH recording as well as disease ranking to best ascertain the potential impact of BBD on radial growth in beech trees.

With its flexibility and ability to handle interdependent forms of uncertainty and variability, hierarchical Bayesian modeling offers many advantages in applied ecology [43,44]. Bayesian modeling has been used to describe the spatial dynamics of various ecological processes [45], including disease transmission and progression [46]. Ellison [47]) provides a detailed overview of the advantages and uses of Bayesian analyses as a tool for ecological modeling. In our situation, traditional statistical methods face several challenges. Due to the extended nature of the study and the inherent difficulties in studying 206 trees scattered through a forest, we have missing data for some years. Furthermore, observer error merits extra consideration in ecological fieldwork involving multiple researchers as crew turnover is expected [48].

Although we hypothesize that BBD, being pervasive and ultimately fatal, will negatively impact the radial growth of infected trees in comparison to trees showing no sign of infection, this study also aims to acknowledge and address observer error and other sources of variability as well as their interactions. This will provide a model that can be applied specifically to BBD and also to the many forest pathogens found in ecological systems worldwide, with greater power and sensitivity than existing models.

2. Methods

2.1. Site Description

The 85-acre study site is located in southwestern Vermont in the northern range of the Taconic Mountains and is characterized by slate bedrock from the Lake Saint Catherine formation. This publicly accessible site, The Lewis Deane Nature Preserve, is known for its ecologically diverse plant communities, and beech is one of many tree species that compose the second-growth forest. Beech sprouting is minimal, and the study site, in regards to BBD, is considered an aftermath forest (a forest in which significant mortality due to BBD has occurred previously).The site is relatively homogenous with respect to soil quality. Soil samples collected near infected and healthy trees (both the A and B horizons) showed no significant variation in pH, percent organic matter, or available phosphorus, potassium, calcium, sodium, or other common metals (Mg, Zn, Mn, Cu, Fe) (data not shown).

2.2. Monitoring of Beech Bark Disease and Radial Growth

The study site was completely traversed to locate all beech trees. Each study tree (those with DBH > 5 cm) was numbered and marked with an identifying tag. The 1–5 BBD ranking system developed by Griffin et al. [41] was used to evaluate the extent of BBD presented by each beech tree over the course of the 15-year study. Examples of some key characteristics of the onset and progression of the disease are shown in Figure 1. A rank of 1 refers to little-to-no sign of either pathogen (beech scale or fungus), 2 represents presence/sign of some scale and visible bark cracking, but a tree with vigor and a relatively intact canopy. At a rank of 3, the bark is heavily cracked, with visible fungal colonies and some crown or limb loss, with noticeable loss of canopy (25%–75% intact). Rank 4 is characterized by severe cracking of bark, large girdling cankers, significant crown loss, and

less than 25% of the canopy being intact; 5 means the tree is dead from BBD [41]. Rankings were then converted to uninfected (rank = 1), infected (rank = 2, 3 or 4), or dead (rank = 5). Trees were assessed seven times during the study, six times during the fall, and one year in the winter, delayed due to weather conditions. A total of 206 total trees were included in the study, yielding 1442 potential observations for which 22.2% have missing values.

Undergraduate researchers were individually trained each year by the same principal investigator to maintain consistency in both ranking and measuring DBH from year to year. Students would rank trees independently of the PI and values were compared to improve consistency prior to independent data collection by students. Tree diameter was measured with a steel tape. If the bark of trees was highly calloused or cracked (even almost girdled at times), the DBH was taken as close to the estimated breast height as possible where the bark was still smooth or less compromised.

Figure 1. Healthy beech trees (**1**) are becoming increasingly rare in the United States. During the initial infection stage of BBD (**2**), Beech have small patches of white (sign of the overwintering beech scale) that can eventually become quite large. Holes left by the boring insects are then colonized by nectria fungi, showing characteristic rusty welts (**3**,**4**). Eventually, the tree is killed by BBD (**5**). Note that both bark and canopy conditions influence the field rankings of BBD (despite these photos only showing bark). Photos taken by Natalie Coe and Robin Sleith.

2.3. Model Development

We developed a tree-specific hierarchical Bayesian model of growth and disease transmission. The base model assumes log-normally distributed annual relative growth rates that are correlated within year and proportional to a power function of tree DBH [49]. The base growth model is given by:

$$\text{annual diameter growth} = \mu * \text{DBH}^{\beta}, \quad (1)$$

where μ is the growth coefficient and β is the growth exponent. Tree growth is hypothesized to be affected by the presence of BBD, thus the annual growth rate for infected trees is multiplied by a BBD growth coefficient (ϕ):

$$\text{annual diameter growth} = \phi * \mu * \text{DBH}^{\beta}. \quad (2)$$

Trees that died from the disease were assumed to have zero growth. Uninfected trees were assumed to have an annual probability of becoming infected that varied by year due to variations in temperature and precipitation, fluctuations in scale migration, and other environmental variables. Infected trees were assumed to have a fixed annual probability of dying from the disease.

Tree DBH and infection status were modeled latently for every tree in the study based on the preceding relationships. Observed values for these variables were modeled assuming observer error. Observed DBH was modeled as a normal distribution with mean equal to the latent DBH value and a common variance parameter representative of observer error. Observed infection status (healthy, infected, or dead) was modeled as a multinomial distribution with probabilities for each category determined by the latent infection status of the

tree, P(Observed status | latent status). Model variables, latent and observed, and model parameters are given in Table 1. A diagram of the dynamic process model is given in Figure 2.

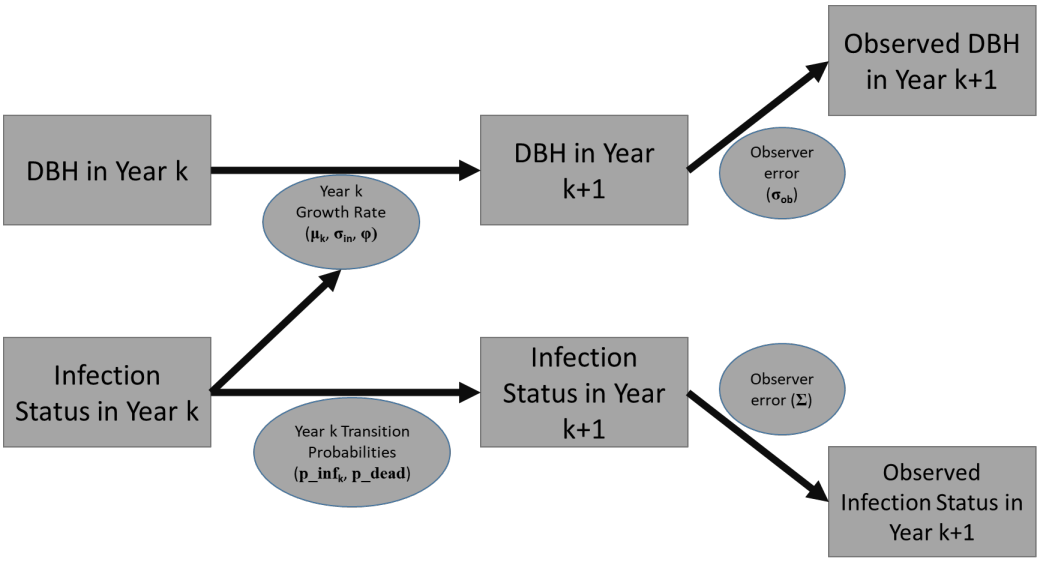

Figure 2. Process model for the spread of BBD and its impact upon radial tree growth. The diagram also shows the relationship between latent and observed values.

Table 1. Model variables and parameters.

Name	Symbol	Definition
Overall growth rate coefficient	μ	Mean growth rate coefficient (unitless) across all years.
Average growth rate coefficient by year	μ_k	Growth rate coefficient in year k.
Growth rate exponent	β	Exponent for growth model.
BBD coefficient	ϕ	Proportion of average growth achieved by infected trees relative to healthy trees.
Within year variability	σ^2_{win}	Variability in growth rates within a year.
Between year variability	σ^2_{btw}	Variability in growth coefficients (μ_k) between years.
Annual growth by tree by year	$gr_{i,k}$	Growth of tree i in year k.
Latent DBH	$\text{DBH}_{i,k}$	Diameter at breast height in cm for tree i in year k.
Observer variability for DBH	σ^2_{obs}	Variability in observer error when measuring DBH.
Observed DBH	$\text{DBH}_{\text{obs}i,j}$	Observed DBH for tree i in year j.
Probability of infection by year	$p_{\text{inf}k}$	Probability of a healthy tree becoming infected during year k.
Probability of death	p_{dead}	Probability of an infected tree dying within the coming year.
Latent infection status	$I_{i,k}$	Infection status (healthy, infected, or dead) of tree i in year k.
Infection status observation probability matrix	Σ	3×3 matrix. $\Sigma_{m,n}$ = probability of observing infection status n given latent status m where status ranges from 1 = healthy, 2 = infected, 3 = dead.
Observed Infection Status	$I_{\text{obs}i,j}$	Observed infection status of tree i in year j.

Tree number (i) $\in \{1:206\}$. Year of observation (j) $\in \{2005, 2007, 2008, 2009, 2011, 2016, 2019\}$. Year ($k$) $\in \{2005:2019\}$.

Following Hobbs and Hooten [43] a full model diagram is given in Figure 3.

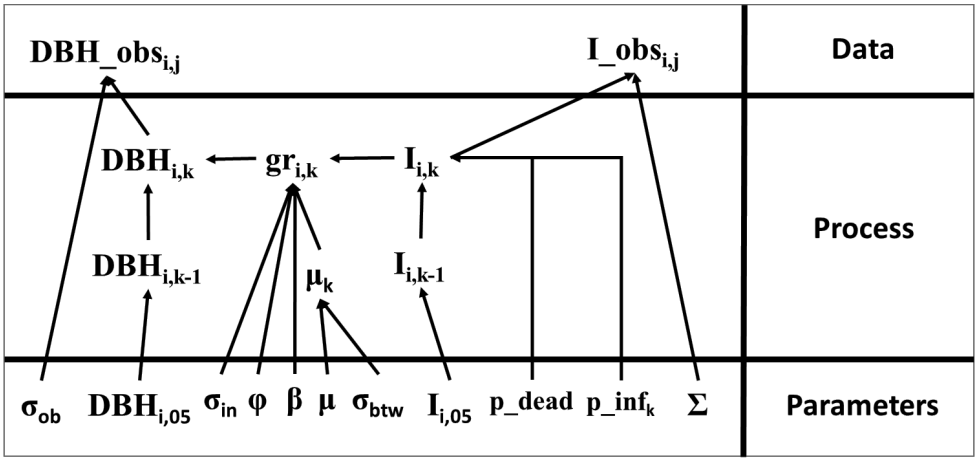

Figure 3. Full model diagram for the analysis following Hobbs and Hooten [43]. $1 \leq i \leq 206$ is the tree ID number and $05 \leq k \leq 19$ is the year after 2000.

By applying Bayes' Theorem to the observed data given the model likelihood, the full posterior distribution for the latent variables and model parameters is given by Equation (3). Beta distribution priors were set for the infection observation error matrix based on the judgment of the investigators to represent approximately a 10–20% probability of identifying a healthy tree as sick and vice versa. Errors for classifying a healthy tree as dead or a dead tree as healthy were estimated to be significantly lower with prior probabilities centered around 1%. Prior values for latent DBH and infection status for trees in 2005 were set based on the observed values for those variables and the prior observer error parameters.

$$\begin{aligned} &\mathrm{P}\left(\mu_k, \mu, gr_{i,k-1}, \sigma^2_{\mathrm{ob}}, \sigma^2_{\mathrm{win}}, \sigma^2_{\mathrm{btw}}, \phi, \beta, \mathrm{DBH}_{i,j}, I_{i,k-1}, p_{\mathrm{infk}}, p_{\mathrm{dead}} | \mathrm{DBH}_{\mathrm{obs}i,j}, I_{\mathrm{obs}i,j}\right) \propto \\ &\prod_i \prod_j \prod_k \mathrm{Normal}\left(\mathrm{DBH}_{\mathrm{obs}i,j} | \mathrm{DBH}_{i,j}, \sigma^2_{\mathrm{obs}}\right) * \mathrm{LogNormal}\left(gr_{i,k-1} | \mu_k, \sigma^2_{\mathrm{win}}\right) \\ &* \mathrm{TruncNormal}\left(\mu_k | \mu, \sigma^2_{\mathrm{btw}}\right) * \mathrm{Multinomial}\left(I_{i,k} | I_{i,k-1}, p_{\mathrm{infk}}, p_{\mathrm{dead}}\right) \\ &* \mathrm{Multinomial}\left(I_{\mathrm{obs}i,j} | I_{i,j}, \Sigma\right) * \mathrm{Uniform}(\sigma_{\mathrm{win}} | 0.0001, 3) * \mathrm{Uniform}(\sigma_{\mathrm{btw}} | 0.0001, 3) \\ &* \mathrm{Uniform}(\sigma_{\mathrm{obs}} | 0.0001, 3) * \mathrm{Uniform}(\phi | 0, 2) * \mathrm{Uniform}(\beta | 0, 1) \\ &* \mathrm{Uniform}(\mu | 0.0001, 10) * \mathrm{Beta}(p_{\mathrm{infk}} | 3, 11) * \mathrm{Beta}(p_{\mathrm{dead}} | 14, 100) * Beta(\Sigma) \end{aligned} \quad (3)$$

2.4. Model Estimation, Specification, Validation, and Convergence

Posterior distributions were estimated using a Markov Chain Monte Carlo algorithm (Gibbs sampling) [50,51]. Three chains were produced and the Gelman-Rubin R statistic [52] was used to determine a sufficient number of iterations to achieve convergence ($R < 1.01$). Chains of 600,000 iterations were run with the first 100,000 discarded for burn-in. The remaining 500,000 were thinned by a factor of 100, yielding three chains each with 5000 values drawn from the joint posterior distribution.

Model validity and specification were checked using multiple methods from the literature [43,53]. First, we produced a simulated dataset by taking random draws from the joint probability distribution for the model in Figure 3 with known parameters. We then ran our Bayesian analysis on the simulated data to compare posterior marginal distributions with the known parameters. We also ran posterior probability checks using the following statistics: mean, 10th percentile, 90th percentile, and variance for observed DBH in 2011, 2016, and 2019, and observed proportion of trees that were healthy, sick, and dead in 2011, 2016, and 2019. We used a pivotal discrepancy measure (PDM) by calculating standardized values (z-scores) for the difference in observed and latent growth rates by tree for 2011, 2016, and 2019 [53]. Finally, we compared the probability distribution of the predicted average annual relative growth rates from 2011 to 2019 to observed relative growth rates to check for discrepancies between the predicted distribution and the observed distribution.

3. Results

3.1. Overview of Data

Data were gathered for a population of 206 trees in the years 2005, 2007, 2008, 2009, 2011, 2016, and 2019. Of these, 24.7% of DBH measurements and 14.2% of infection status assessments were missing. Furthermore, 71.5% of missing values were recorded in 2016 and 2019, due in part to the inability to locate trees that had likely fallen due to either disease, high winds, or a combination of both, and to poor weather keeping researchers from accessing the site. In 2005, 117 trees out of 198 assessed (59.1%) showed little to no sign of infection, while the remaining 81 trees were in various stages of BBD. By 2011, only 12.6% of assessed trees were still determined to be uninfected.

The distribution of tree diameters for 2007 is shown in Figure 4.

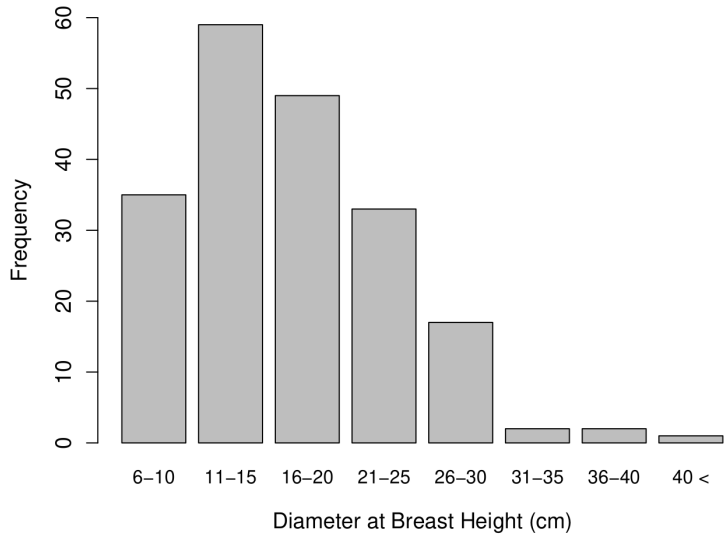

Figure 4. Frequency histogram of DBH in 2007, the year with the most complete data. The distribution of DBH did not change significantly over the period of the study.

Here, 190 growth rates were estimated from the data for observed DBH. Estimated absolute growth rates ranged from 0 to 0.87 cm/yr for healthy trees and 0 to 0.69 cm/yr for infected trees, while relative growth rates ranged from 0 to 0.048 cm/cm/yr for healthy trees and 0 to 0.047 cm/cm/yr for infected trees. (Due to observer error, some trees were estimated to have a negative growth rate. These are not reported here but are shown below.) The distribution of growth rates for uninfected and infected trees is shown in Figure 5.

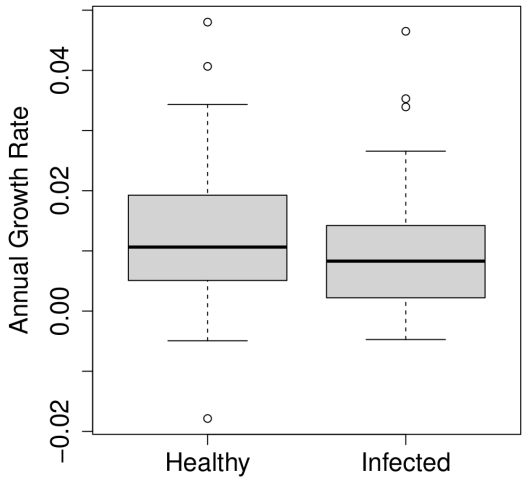

Figure 5. Distribution of relative growth rates for uninfected and infected trees.

3.2. Model Validation, Specification, and Convergence

There was no evidence of model misspecification. The simulated distributions of DBH in 2011, 2016, and 2019 did not differ significantly from the observed data. The observed mean DBH, 10th and 90th percentiles for DBH, and variance for DBH were all within the 95% credible intervals for the posterior distributions. This was also true for the observed proportions of healthy, infected, and dead trees in 2011, 2016, and 2019.

The joint posterior distributions estimated from the three sets of simulated data all contained the known coefficient values for all important parameters within a 90% credible interval. Annual average relative growth rates (μ_k) showed some variability, which is expected given their collinearity (a higher average growth rate estimate in one year will increase the probability of a lower growth estimate for the following year) and the high number of missing DBH values, and observed error for infection status had some additional variability in cases where the observer error rate was very low (<1%). Neither of these problems should impact estimates of overall growth, disease transmission, or the impact of BBD upon growth. Additionally, our PDMs (z-scores) were symmetric around zero and similar to the standard normal distribution except they were marginally leptokurtic (heavier tails). The Gelman–Rubin statistic to test for chain convergence was less than 1.01 for all parameters, indicating that the Markov chains had converged.

3.3. Parameter Estimates

Location statistics—mean, median, and 95% highest-density credible interval—for key model parameters are reported in Table 2 and marginal posterior distributions are shown in Figure 6. Figure 7 shows the posterior distributions for the observed infection status probabilities along with the prior distributions, and Figure 8 shows the multinomial probabilities for assessed infection status based on latent status.

Table 2. Parameter location statistics derived from the Monte Carlo Markov chain. The range from the 2.5% value to the 97.5% value represents a highest-density 95% credible interval.

	ϕ	μ	σ_{btw}	σ_{win}	σ_{obs}	β	p_{dead}
Mean	0.494	6.18×10^{-3}	1.85×10^{-2}	0.610	0.595	0.864	1.99×10^{-2}
Sd	6.83×10^{-2}	5.03×10^{-3}	7.82×10^{-3}	5.03×10^{-2}	1.77×10^{-2}	9.46×10^{-2}	4.28×10^{-3}
2.5%	0.371	3.2×10^{-4}	8.39×10^{-3}	0.504	0.561	0.648	1.23×10^{-2}
Median	0.490	5.03×10^{-3}	1.68×10^{-2}	0.614	0.594	0.880	1.96×10^{-2}
97.5%	0.640	1.86×10^{-2}	3.8×10^{-2}	0.700	0.630	0.993	2.9×10^{-2}

The overall mean proportional growth rate for uninfected trees was 0.0062 yr^{-1}, meaning that trees on average added 0.62% to their DBH. There was little evidence of a power law relationship with the 95% credible interval for β extending to 0.99. The impact of BBD infection upon growth was significant (median for $\phi = 0.490$, 95% credible interval $= [0.371, 0.640]$). Trees that were infected but not dead saw an estimated reduction in growth of 51.0%.

There was significant variability between years in average proportional growth rates with the standard deviation for the truncated normal distribution (σ_{btw}) estimated to be 0.0168 (approximately three times the size of the mean). The variability between trees in actual growth rates within a given year was also high, with the estimated standard deviation for the lognormal distribution (σ_{win}) equal to 0.614 cm.

The probability of a healthy tree becoming diseased in a given year varied between an estimated low of around 4% in 2009 to as high as 40% in 2010 and 2011 (results not displayed) with most years having a median estimate of around 15%. These estimates have large bounds on them because of collinearity in these variables, the high number of missing variables, and the observer error in determining infection status. Infected trees were estimated to be dying at a rate of 2% per year.

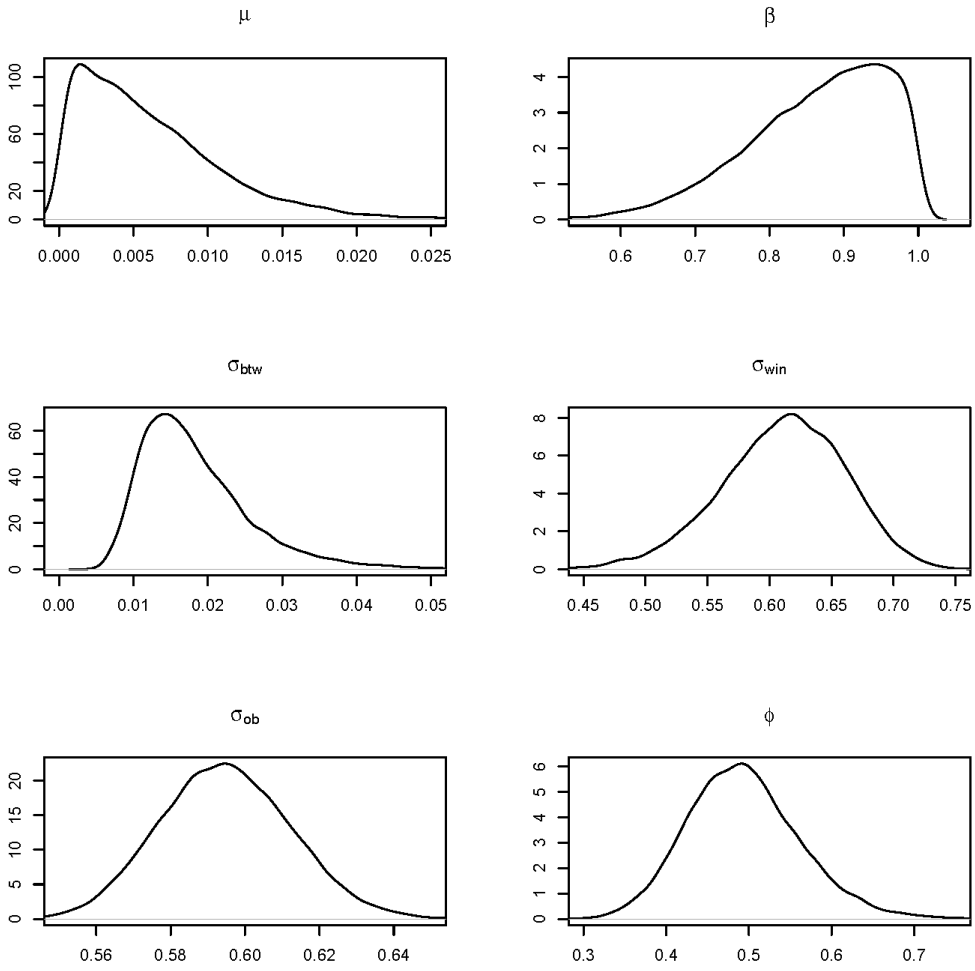

Figure 6. Marginal posterior distributions for model parameters. Distributions are estimated from the joint posterior distribution as represented by the Monte Carlo Markov Chain. The *x*-axis shows the likely range for each model coefficient.

3.4. Observed Error Estimates

Observed variability in estimating tree DBH was estimated to be 0.594 cm, which is reasonable given the precision of the measurements (nearest cm) and the variability in determining where to place the tape. The bounds on this parameter are relatively narrow (95% credible interval $= [0.561, 0.630]$) because of the high number of observations giving us significant confidence that observers are frequently off by more than 0.5 cm in measuring DBH.

Our work also yielded estimates of observer error when assessing tree infection status. Figure 7 shows that the data were informative in many cases, as the posterior distribution differs significantly from the prior distribution. Most notably, an observer had around a 10% chance of determining a healthy tree to be infected and a 6% probability of determining that an infected tree was healthy. Around 18% of the time, a tree that was still alive was determined to be dead. Our model verification using simulated data suggests that these probabilities are hard to estimate, but there is clear evidence that a modeling approach that incorporates growth rates and infection status gives us the ability to assess observer error in both contexts.

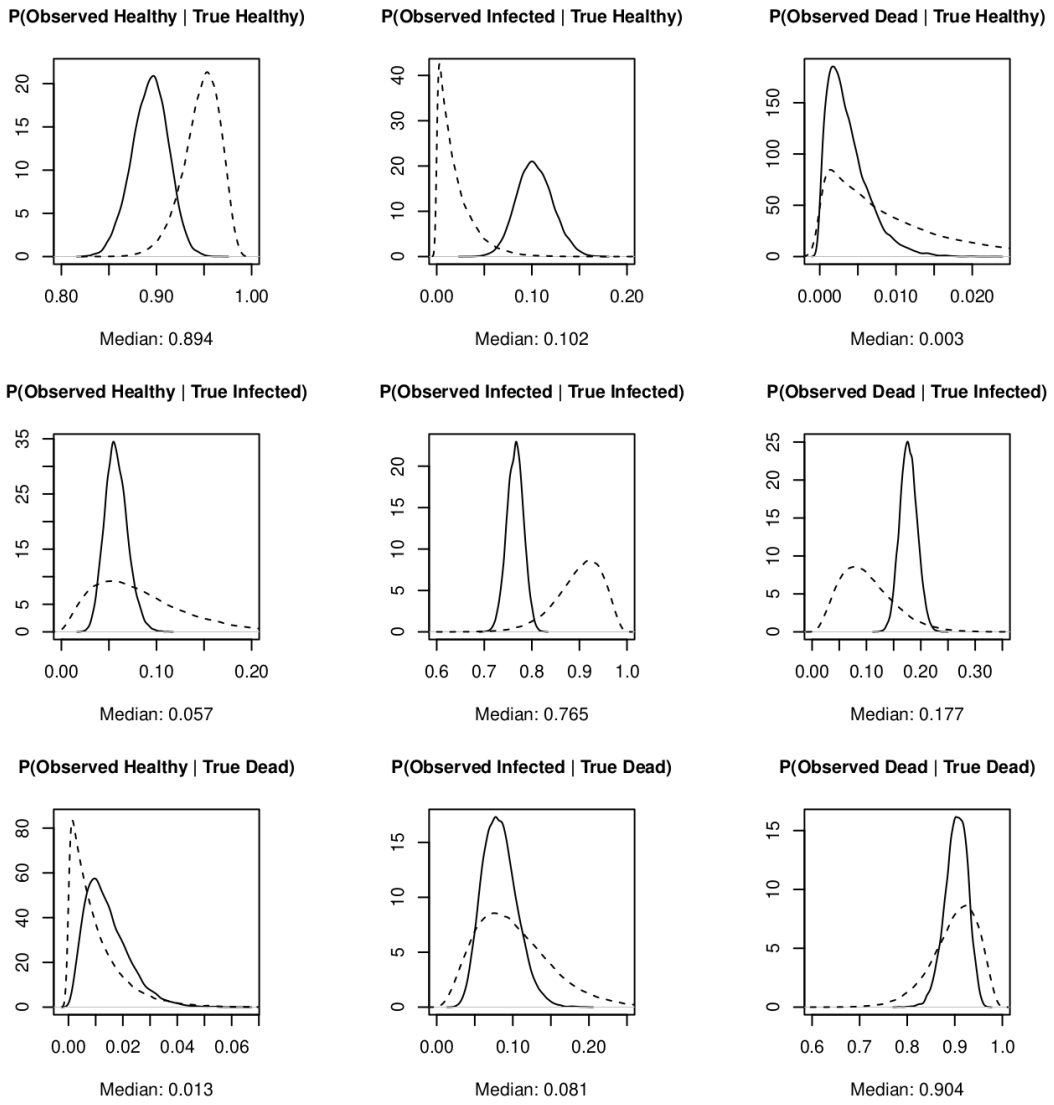

Figure 7. Posterior distributions for observed infection status probabilities conditional on the latent infection status. Dashed lines show the prior distributions.

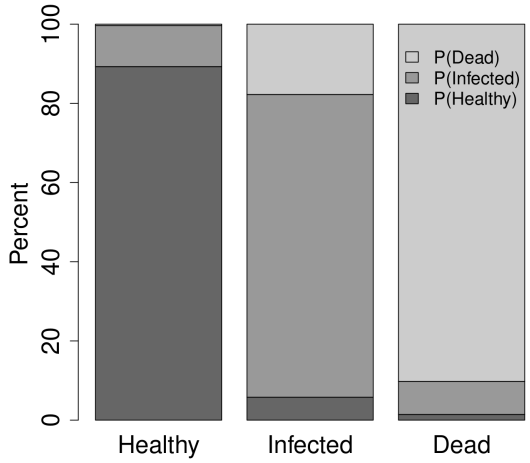

Figure 8. Mean observation probabilities for infection status based on latent infection status. Note that the *x*-axis is the latent (true) value, while the *y*-axis is the observed value. For example, 20% of trees that were infected were observed to be dead instead.

4. Discussion

Observer Error and Missing Data

Tree growth can be impacted by multiple environmental factors such as solar radiation, air temperature, rain/moisture, wind velocity, and competition [54,55]. Radial growth has been shown to be directly impacted by precipitation and temperature [56] as well as drought, the intensity of which can be site and species specific [57]. Studies on European beech have also illustrated that radial growth rates, while historically most influenced by soil conditions, are now more strongly influenced by climate change [58]. This drives the need to evaluate trees (both free of disease and infected with BBD) in a context in which rainfall is not a variable factor.

We chose a relatively homogenous study site to control for fluctuations in climate, soil quality, and management practices to be able to examine the potential impact of BBD on the radial growth of American beech. Additionally, American beech was the dominant tree species throughout the study site, with little to no competition with other tree species.

Canopy health is related to overall leaf number and leaf area, which is in turn, connected to radial growth [59]. Beech is noted for marcescence and can take many years to succumb to BBD, maintaining reasonably large and healthy canopies in the earlier stages of infestation. However, as BBD progresses, the canopy diminishes markedly. For this reason, one might expect a consequential radial growth decline in infected trees. However, in a recent study in European Beech, researchers did not find a relationship between leaf phenology and radial growth [60]. Indeed, defoliation can result in an increase in photosynthetic rates. This is a potentially significant consideration in the progression of beech bark disease and may explain in part the ability of many beech to remain alive for decades after infection.

Given the previous consideration, a priori, it was unclear whether and to what degree BBD would negatively impact radial growth. Our data show a 51% decrease in annual radial growth as a direct result of disease infestation. The change is not only statistically significant but substantial when one considers that nearly all of the American beech in North America are currently at some stage of this disease.

Although ours is the first study to document the reduction of radial growth as a direct consequence of pathogen infestation in the American beech, studies in other tree/pathogen scenarios have also shown this. A study of the impact of Swiss-Needle Cast on growth of Douglas-Fir in New Zealand places losses for mean top height, basal area, and stem volume at 25%, 27%, and 32%, respectively. Although mortality rates were not affected, the monetary loss due to these decreases is estimated to be approximately $NZ 1000/hectare on average [61]. These impacts are clearly substantial, both from a forestry standpoint and from the perspective of ecological services. Bert et al. [62] have made a similar argument in regard to the effect of powdery mildew on oak trees, showing that radial growth not only negatively correlated with infection severity but that the impacts of disease were seen even after the infection was no longer prevalent. Interestingly, a study examining the impact of woolly adelgid on the radial growth of eastern hemlocks showed some confounding results. A negative impact of disease on growth was only seen at half of the sites monitored, pointing again to the potential role of increased photosynthetic rates in survivors or perhaps a reduction in competition brought by the death of neighboring trees [62].

The role of forest pests is just beginning to be integrated into forest growth models. As explained by Dietze and Matthes, "the long-term growth reductions due to forests insects and pathogens (FIPs) are absent from models, [which] could lead to the systematic overestimation of carbon uptake and storage across wide regions" [63]. To this end, even non-lethal pathogens could have measurable effects on overall tree health and should also be considered in developing growth models [62].

To be able to discern the impact of BBD on radial growth, coring was not practical due to bark damage that could disrupt the quality of the growth rings. Errors in recording DBH can result from misreading of the tape, misrecording the data in the field, data entry error, placement of the tape itself (not being perpendicular to the vertical axis of the tree),

and even variation in the tension of the tape [64]. To avoid these errors, individuals were trained directly by the same principal investigator throughout the course of this study, but each academic year brings new students who, once trained, work independently. Butt and colleagues [65] have gone so far as to compare the accuracy of DBH measurements by trained volunteers versus experts to determine if these could lead to changes in biomass estimates. They found a DBH sampling error with volunteers to be 2.3 mm versus 1.4 mm for the experts, but only when excluding errors greater than one centimeter. If all data were included, volunteer data had a mean sampling error of 9.9 mm.

Our model acknowledges observer error in both disease assessment and measured DBH and minimizes its impact upon parameter estimates. Regardless of the monitoring data collected, there are sources of observer error that can be considered independent of the ecological system. These include the training and expertise of student researchers, time taken to ascertain a ranking or measurement, and the difficulty of finding or reaching a particular plot or tree based on physical, environmental, and weather conditions [66]. Burg and colleagues [67] point to observation bias in reference to high-alpine summits, noting the influence of a demanding climb on "observer-dependent factors". Within our study, although the change in elevation was quite small, relatively speaking, this phenomenon was also observed. Missing data is a common occurrence in forestry research, and looking to find ways to both minimize the instances but also deal with the inevitability of gaps has gained increasing interest over the past decade, especially as researchers consider combining multiple types of data, with various observations made over large areas by multiple means (e.g., data gathered through remote sensing as well as in the field) [68].

Longer-term studies similar to ours may also help to minimize the effect of observer errors that could result in potentially biased or erroneous short-term changes. While this does not override the existence of such errors, it does minimize their impact upon model predictions and parameter estimation [69].

5. Conclusions

It is critical to now factor in the effects of pathogens when modeling forest production, especially in light of climate change [70]. We agree with Pinkard and colleagues that "some models can encapsulate some of the processes, but no model can comprehensively account for the range of physiological responses to pest attack experienced by trees." It is essential to develop models that can accurately predict the impacts of forest pathogens on tree growth to be able to ascertain short and long term effects on woody biomass and ecosystem services.

We have been able to develop a model that predicts the average probability of a healthy tree becoming infected, and the reduction in annual radial growth rate. We can also predict the number of trees that will die from BBD. By incorporating observer error in assessing both the DBH and the status of the tree, we have increased both the rigor and applicability of our new modeling approach. "Methods research usually addresses either the improvement of data-gathering so as to reduce biases or increase precision, or the development of statistical methods that can account for biases or uncertainty in the collected data" [71]. We have attempted to do both in how our study has been designed and how our data have been evaluated. Ecological statistics are simultaneously empowered and limited by the care and accuracy of collected field data. Working in concert, the statistician and field researcher can develop models that utilize all available data without being hampered by missing data points. As invasive pathogens become more persistent and additional pathogens emerge [72], it is critical to be able to optimize currently available approaches to new threats in projecting the impact of disease on tree health, growth, and biomass.

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