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Modeling slash pine mortality rates incorporating responses to silvicultural treatments and fusiform rust infection rates

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A R T I C L E I N F O

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Bedding
Cronartium quercuum
Differential equations
Dominant height
Maximum likelihood
Vegetation control

A B S T R A C T

The evaluation of stand survival at any age is an essential task that allows foresters to estimate stand dynamics and ultimately, the value of a forest stand. Therefore, any growth and yield system developed with the aim of predicting or projecting standing value requires precise estimates of the number of trees surviving at any point in time. When describing survival through modeling the mortality rate, differential equations using height increments rather than time increments have been shown to improve the overall fit of survival models. Using a long-term data set of slash pine plantations (*Pinus elliottii* Engelm.) in which silvicultural treatments (i.e., bedding and complete vegetation control) were applied during the establishment phase, and fusiform rust (*Cronartium quercuum* Berk.) infection rates were recorded, a survival/mortality model of this nature was constructed. Height increments were taken from a proposed dominant height model that included explicit treatment effects. In addition, the proportion of trees infected with fusiform rust at an age of 5 years was added as a predictor describing the mortality rate. Our results show that stand survival is better described by a model in which time increments are used rather than height increments, and although silvicultural treatments were essential for modeling dominant height, mortality was not greatly affected by these treatments and therefore, no additional parameter modifiers associated to these treatments were needed. On the other hand, the inclusion of average fusiform rust infections was essential to describe mortality rates in these stands, with higher infection rates associated to higher mortality rates.

1. Introduction

Forest stand dynamics and the associated financial value of a forest are largely determined by the stand survival at any point in time. It is therefore crucial that growth and yield systems developed to predict or project stand-level metrics (e.g., cubic volume, tons, or value), include a precise estimate of the number of trees surviving at any age. In forest plantations, density-dependent mortality is the most common process modeled. There are numerous challenges associated with modeling other stochastic factors such as diseases, pest infestations, and extreme weather events (Lee, 1971). In the southeastern United States, the stand-level relative rate of density dependent mortality has been modeled using differential equations since the 1980s (Bailey et al., 1985; Clutter and Jones, 1980; Pienaar and Shiver, 1981). This rate has been described as either a constant over time or as a function of current stand density, age and/or site quality. An extensive review of mortality functions was presented by Zhao et al., (2007a) concluding that no single equation was the best for all areas and management scenarios.

A different approach to model survival in which mortality rates are modeled with respect to dominant height instead of time, also using differential equations, was proposed by García (2009). García argued that using dominant height instead of age was more appropriate to describe mortality rates since dominant height is directly describing size, while age, although related to size, is not a direct measurement of size. One advantage of following this modeling approach is the ability to obtain a model that is independent of site quality (a quantity expected to reflect the site conditions over time), but that at the same time is able to accommodate changes in dominant height trajectories, providing a good

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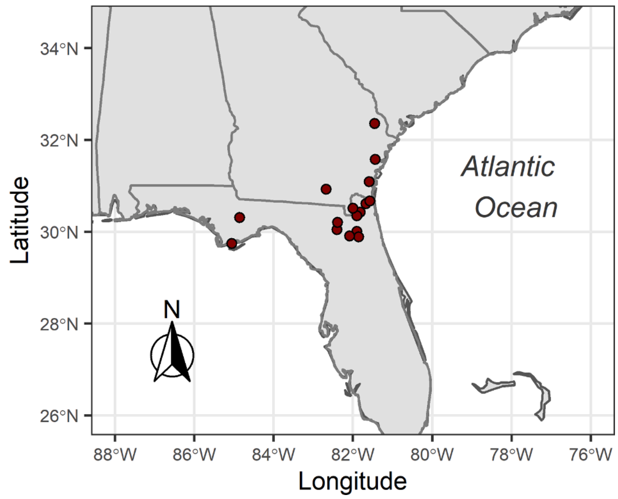

Fig. 1. Location of the 16 installations of the study.

fit even when the data is scarce (García, 2010; García et al., 2011; Tewari and Singh, 2018).

In the southeastern United States, pine plantations are intensively managed through silvicultural prescriptions aiming to reach the maximum production potential on a given site (Fox et al., 2007). Along with this intensive silviculture and management, growth and yield models have been constructed to accurately capture the effects of silvicultural treatment applications (Bailey and Burgan, 1989; Clutter and Jones, 1980; Gyawali and Burkhart, 2015; Martin et al., 1999; Pienaar and Rheney, 1995). Silvicultural treatment effects are usually incorporated directly into growth and yield models by adding a response value to a control (or base) model (Pienaar and Rheney, 1995) or modifying the parameters of the model (Mason and Milne, 1999).

Particularly for slash pine (*Pinus elliottii* Engelm.), the second most important planted species in the southern United States (Barnett and Sheffield, 2004), several authors have proposed dominant height and survival models that include the effect of silvicultural treatments mainly by adding additional treatment factors or modifying the parameters of the model according to the treatment applied (Bailey et al., 1985; Bailey and Burgan, 1989; Pienaar and Rheney, 1995; Ramirez et al., 2022). In light of the methodology proposed by Garcia (2009), in which mortality is modeled with respect to dominant height instead of time, it is of interest to determine whether a mortality model for a forest stand in which silvicultural treatments were applied, requires additional modifications if the dominant height model already includes the treatment effects.

Survival models for slash pine plantations usually include fusiform rust (*Cronartium quercuum* Berk.) presence and/or infection rates as an important component determining the mortality rate (Bailey and Burgan, 1989; Devine and Clutter, 1985; Nance et al., 1981). Fusiform rust is one of the most relevant pathogens affecting slash pine plantations in the southeastern United States, and despite loss estimates of around 84 million dollars (2020 US dollars) per year (Susaeta, 2020), infection rates have not declined significantly for this important species (Randolph, 2016). Although mortality is not always directly caused by the cankers produced in an infected tree, several studies have shown evidence of higher mortality rates in infected trees compared to uninfected trees (Jones, 1972; Sluder, 1977), and infection rate at young ages (less than5 years) has been identified as a good predictor of future mortality and volume loss in slash pine plantations (Wells and Dinus, 1978).

The main objective of this paper was to develop a survival/mortality model for slash pine plantations including the effect of silvicultural treatments and the effects of fusiform rust infection on the mortality rate. The methodology proposed by Garcia (2009) involving dominant height increments was tested, and model performance was compared with other mortality models that included the treatment effect explicitly and described the mortality rate using time increments. Our hypothesis regarding the different modeling techniques tested was that similar performance could be achieved when modeling mortality rates with respect to height without including additional explicit treatments effects and when modeling mortality rates with respect to time but including explicit treatment effects.

Table 1
Dominant height (H_D) and trees per hectare (TPH), average and standard deviation values per plot.

Age (years)	Treatment	No. plots	Mean $H_D(m)$	Standard deviation H_D(m)	Mean TPH	Standard deviation TPH
5	Control	18	3.3	0.7	1181.9	157.2
8		18	6.2	1.1	1137.2	194.4
11		18	9.1	1.6	1108.9	194.4
14		18	12.2	2.0	1062.9	221.3
17		18	14.5	2.4	1046.3	218.5
20		18	16.4	2.5	1048.0	231.2
23		17	18.3	2.9	1017.2	241.2
26		16	19.8	3.4	990.8	250.5
31		12	20.9	4.1	972.8	244.4
5	Bedding	19	3.9	0.7	1252.2	127.8
8		19	7.1	1.0	1228.6	142.4
11		19	10.1	1.3	1191.7	132.5
14		19	13.2	1.5	1150.6	149.6
17		19	15.5	2.0	1141.0	151.2
20		19	17.1	2.4	1124.1	167.6
23		18	18.9	2.9	1095.0	190.0
26		17	20.2	3.3	1032.3	225.6
31		11	20.9	4.2	1042.3	272.2
5	Vegetation control (Chem)	17	4.8	0.7	1181.2	130.7
8		17	8.3	0.9	1144.2	171.1
11		17	11.3	1.1	1130.5	186.0
14		17	14.3	1.5	1083.0	242.0
17		17	16.5	1.8	1076.3	249.8
20		16	18.3	2.3	1032.3	251.0
23		15	20.1	2.4	992.1	259.8
26		14	21.5	2.8	964.4	256.1
31		10	22.8	3.2	968.7	276.5
5	Bedding + Vegetation control (Chem)	18	5.3	0.5	1200.9	139.4
8		18	8.9	0.7	1163.8	147.7
11		18	11.9	0.8	1146.5	163.7
14		18	14.9	1.1	1110.0	172.0
17		18	17.1	1.4	1083.0	191.0
20		17	18.6	1.8	1063.3	195.5
23		15	20.4	2.2	1005.3	246.5
26		15	21.9	2.4	963.3	243.2
31		11	22.8	3.0	1038.5	226.9

2. Methods

2.1. Data

A long-term slash pine study established in 1979 by the Plantation Management Research Cooperative (PMRC) at the University of Georgia in the southeastern United States was used to test the proposed hypothesis. The study was established on 16 different installations throughout the Lower Coastal Plain in northern Florida and southern Georgia (Fig. 1). The main silvicultural treatments considered were bedding, consisting of a double pass with a bedding harrow during site preparation, complete competing vegetation control (using herbicides), and the combination of these two treatments. The vegetation control treatment included an herbicide application before site preparation (3 % solution of Roundup) and repeated localized applications of Roundup or Garlon to remove most of the competing vegetation until crown closure (Zhao et al., 2009). These silvicultural treatments were replicated at least once on each one of the installations and dominant height, stand density measurements, and average fusiform rust infection rates per plot were available every 3 years starting from age 5 and up to age 31 for the longest series. In Table 1 a summary of the dominant height and number

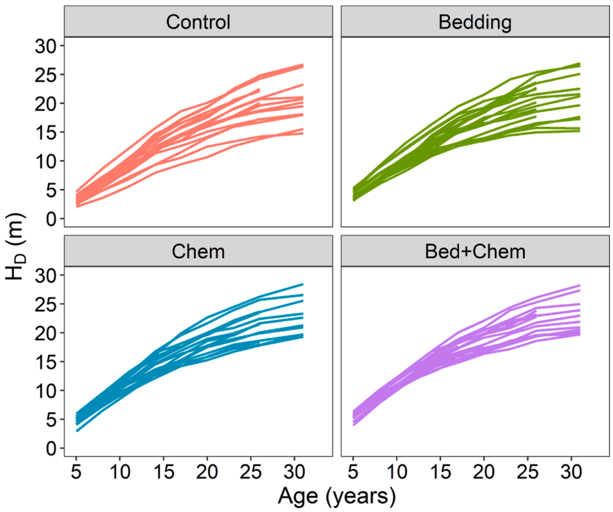

Fig. 2. Dominant height trajectories for slash pine by treatment.

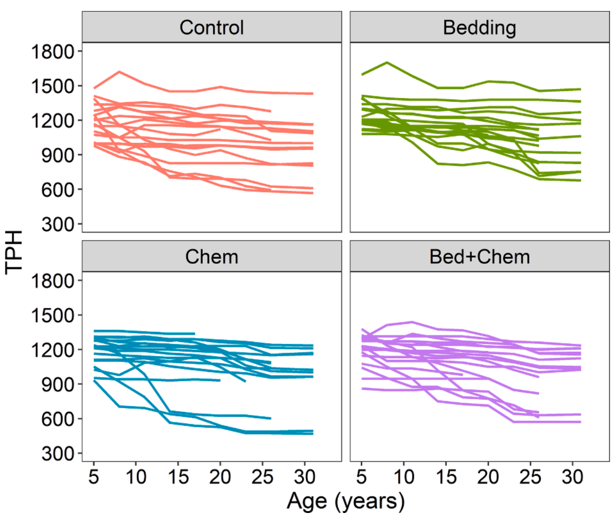

Fig. 3. Trees per hectare (TPH) over time by treatment.

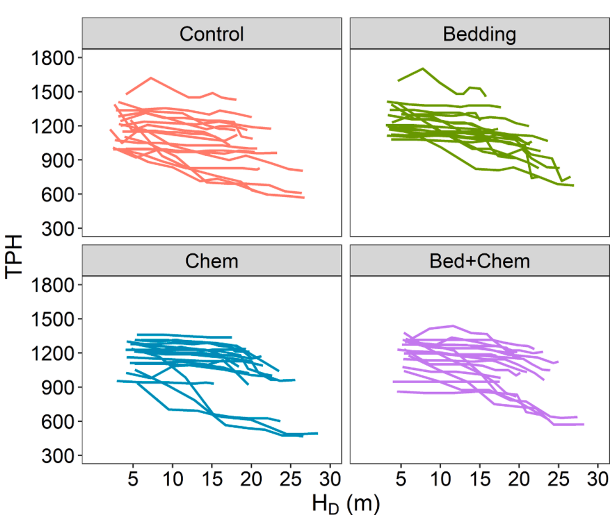

Fig. 4. Trees per hectare (TPH) versus dominant height (H_D) by treatment.

of trees per hectare for each of the treatments is presented. More details about this study are provided by Zhao et al., (2009), Zhao et al., (2007b) and Ramirez et al., (2022). Dominant height (H_D) trajectories are plotted in Fig. 2 and trees per hectare (TPH) with respect to time and dominant height are plotted in Fig. 3 and Fig. 4, respectively.

2.2. Models

2.2.1. Dominant height model

A dominant height model was proposed to subsequently estimate dominant height increments and test the hypothesis related to using height increments versus time increments in the mortality model. The Gompertz model which has been used previously to model forest stand height trajectories (Medeiros et al., 2017; Zang et al., 2016), was chosen to describe the dominant height trajectories. The basic assumption of this model is that growth is proportional to size with a constant of proportionality μ, and that the effectiveness of the growth mechanism decays over time, generating an exponential decay (France and Thornley, 1984). In mathematical terms this can be described with the following system of equations:

$$\frac{dH_D}{dt} = \mu H_D \tag{1}$$

$$\frac{d\mu}{dt} = -K\mu \tag{2}$$

Where dH_D/dt is the change in dominant height (H_D) over a period of time (dt), t is time in years, and μ and K are parameters determining the dominant height trajectory. When Eq. (2) is solved as a separable differential equation and the value of μ is replaced back in Eq. (1), the following differential equation is obtained.

$$\frac{dH}{dt} = \mu_0 H e^{-Kt} \tag{3}$$

Dominant height has been found to be positively affected by bedding and vegetation control (Ramirez et al., 2022). To incorporate silvicultural treatment responses into the Gompertz model (Eq. (3)), parameter modifiers were proposed to be added to this model. Bedding is a silvicultural treatment that improves growth in the early stages of a stand by enhancing rooting along with improved soil moisture conditions and nitrogen availability (Morris and Lowery, 1988). To incorporate these effects into the model, a modifier was added to the parameter μ_0 which is the parameter directly associated with growth. For the vegetation control treatment, a modifier was added into the parameter K since this treatment does not directly improve growth, but reduces the limiting factors on site for the crop trees. Therefore, it was expected that this treatment would affect the decay rate (K) at which the growth rate μ_0 decreases. The dominant height model with the modifiers can be expressed as:

$$\frac{dH_D}{dt} = \mu_0 b_1^{Z_1} H_D e^{-Kb_2^{Z_2} t} \tag{4}$$

Where b_1 and b_2 are the parameter modifiers to be estimated, and Z_1 and Z_2 are dummy variables equal to 1 if bedding or vegetation control was applied, respectively, and zero otherwise.

Model from Eq. (4) was compared to the (null) model without treatment effects (Eq. (3)) to evaluate the effectiveness of the proposed model to incorporate treatments effect into dominant height.

2.2.2. Survival model

Marked differences in survival trajectories were observed in the studied plots, with some of them experiencing high mortality while others experienced little to no mortality (Fig. 3 and Fig. 4). These differences were found to be strongly associated with the average percentage of fusiform rust infected trees at year 5. Overall, when this percentage was less than 15 %, less mortality was recorded, while higher

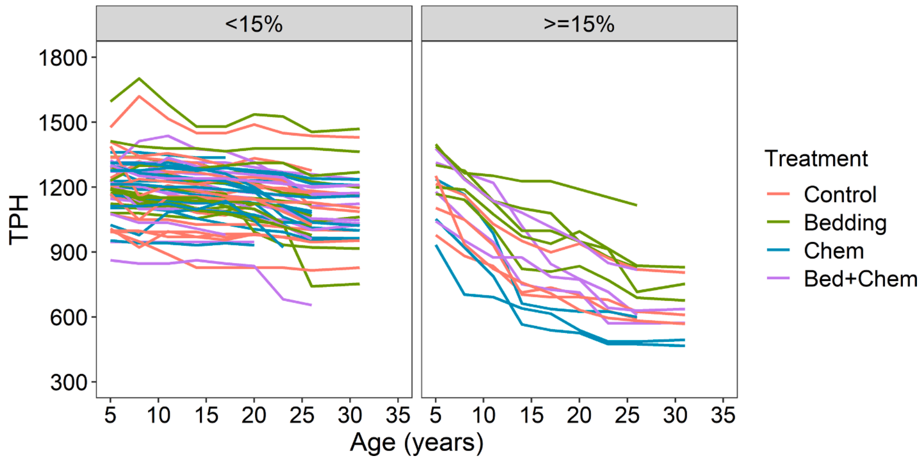

Fig. 5. Trees per hectare (TPH) over time for different groups of fusiform rust infection rate (less than 15% or greater than or equal to 15%) at age 5.

Table 2
Average fusiform rust infection rates at age 5 differentiated by treatment.

Treatment	No. plots	No. plots FR < 15 %	No. plots FR ≥ 15 %	Avg FR infection % (FR < 15 %)	Avg FR infection % (FR ≥ 15 %)	Avg FR infection % (all plots)
Control	18	14	4	2.01	28.45	7.89
Bedding	19	14	5	2.01	22.34	7.36
Veg. control	17	14	3	3.56	38.30	9.69
Bed + Veg. control	18	14	4	2.75	36.68	10.29

Table 3
Base mortality model forms tested.

Equation	Model form	Model with Fusiform rust	Reference
Eq. 5	$\frac{dN}{dt} = \alpha N$	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {}^*FR)N$	(Devine and Clutter, 1985)
Eq. 6	$\frac{dN}{dt} = \alpha Nt$	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {}^*FR)Nt$	(Zhao et al., 2007a)
Eq. 7	$\frac{dN}{dt} = \alpha Nt^{\delta}$	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {}^*FR)Nt^{\delta}$	(Pienaar and Shiver, 1981)
Eq. 8	$\frac{dN}{dt} = \alpha N^{\gamma} t$	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {}^*FR)N^{\gamma} t$	(Zhao et al., 2007a)
Eq. 9	$\frac{dN}{dt} = \alpha N^{\gamma} t^{\delta}$	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {}^*FR)N^{\gamma} t^{\delta}$	(Clutter and Jones, 1980)

N: trees per hectare at time t, t: time in years, FR: Fusiform rust infection rate per plot at year 5 (varying from 0 to 1), α, α_0, α_1, γ, δ: parameters to be estimated.

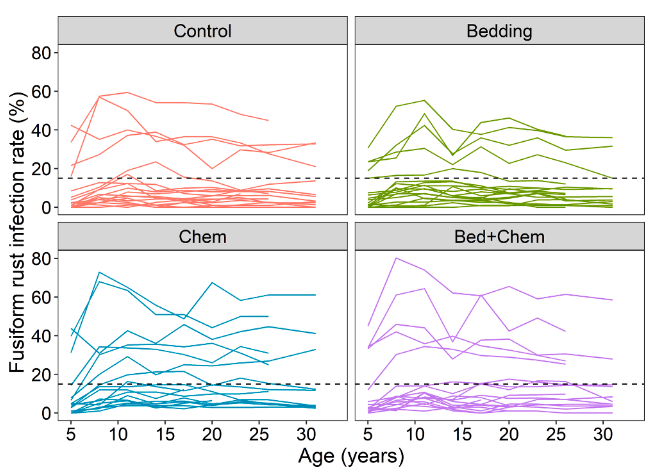

Fig. 6. Fusiform rust average infection rate per plot, trajectory for the duration of the study. The dashed line represents the 15% infection rate threshold.

Table 4
Mortality model modifications to include treatment effects.

Equation	Model form	Description
Eq. 10	$\frac{dN}{dt} = f(N, t, \theta)$	Base model from Table 3
Eq. 11	$\frac{dN}{dH_D} = f(N, t, \theta)$	Modified model following García (2009) using dH_D
Eq. 12	$\frac{dN}{dH_D} = f(N, H_D, \theta)$	Modified model following García (2009) using dH_D and H_D (no t involved)
Eq. 13	$\frac{dN}{dt} = f(N, t, \theta) \bullet c_1^{Z_1} c_2^{Z_2}$	Base model with explicit treatments effect
Eq. 14	$\frac{dN}{dH_D} = f(N, t, \theta) \bullet c_1^{Z_1} c_2^{Z_2}$	Modified model following García (2009) with explicit treatments effect
Eq. 15	$\frac{dN}{dH_D} = f(N, H_D, \theta) \bullet c_1^{Z_1} c_2^{Z_2}$	Modified model following García (2009) with explicit treatments effect (no t involved)

N: trees per hectare at time t, t: time in years, H_D: dominant height (meters), Z_1: dummy variable equal to 1 if bedding was applied and 0 otherwise, Z_2: dummy variable equal to 1 if vegetation control was applied and 0 otherwise, c_1, c_2, θ: parameters to be estimated.

mortality rates were observed for plots in which the fusiform rust infection rate was higher than 15 % (Fig. 5). This was in line with the differential mortality rates reported by other authors assessing mortality in slash pine plantations in the southeastern United States (Jones, 1972; Sluder, 1977; Wells and Dinus, 1978). Therefore, all mortality models proposed in this research included average fusiform rust infection rate (varying from 0 to 1) as a predictor variable. In Table 2 the average fusiform rust infection proportion differentiated by treatment and by the cutoff value of 15 % is presented. It is important to note that this classification of mortality trajectories for plots with less or more than 15 % fusiform rust infection was done to visually inspect more clearly the different survival trajectories. Nevertheless, since the actual proportion of infected trees was used in the models tested, taking this infection rate as a continuous value, this cutoff value is not relevant for modeling purposes. The recorded fusiform infection rates for each plot during the whole period of study are presented in Fig. 6 differentiated by silvicultural treatments.

Five different models found in the literature for slash pine were fitted first for the control plots, and the model with the best fit was then selected to be modified to include treatment effects and test the mentioned hypothesis related to the modeling approach. The models considered in this research are referenced in Table 3, where Eq. 5, Eq. 7, and Eq. 9 have been used previously in slash pine (Clutter and Jones, 1980; Devine and Clutter, 1985; Pienaar and Shiver, 1981) and Eq. 6 and Eq. 8 have been used for modeling mortality in other pine species (Zhao et al., 2007a). All these models were modified to include the effect of fusiform rust infestation by modeling the mortality rate (α) as a linear function of the average fusiform rust infection rate at year 5.

The model that had the best fit from Table 3 was selected to be modified following Garcia's (2009) approach. The different model variations are presented in a general form in Table 4, with all the variables as described before, θ representing the parameters from the base model in Table 3, and c_1, c_2 corresponding to the explicit treatment effects of bedding and vegetation control on the mortality rate, respectively. The inclusion of fusiform rust infection rates was maintained for these models.

Models where mortality was modeled with respect to dominant height instead of time (dN/dH_D instead of dN/dt) were obtained by combining the model in Eq. 10 with the best dominant height model from section 2.2.1 as follows:

$$\frac{dN}{dt} = f(N, t, \theta)\frac{dH_D}{dt} \tag{16}$$

$$\frac{dN}{dH_D} = f(N, t, \theta) \tag{17}$$

Where dH_D/dt in Eq. (16) is taken from the proposed dominant height model (Eq. (3) or Eq. (4)). The models in Eq. 12 and Eq. 15 are similarly derived, although the time variable is completely replaced by the dominant height variable (H_D) for these models.

2.3. Parameters estimation

Parameters were estimated using the maximum likelihood framework in the Julia programming language (Bezanson et al., 2017). The procedure consisted of solving the differential equation numerically as an initial value problem and then finding the combination of parameters that maximized the likelihood of observing the data collected (given the assumed model), that is, as an inverse problem with unknown starting values. Therefore, in addition to the parameters for each one of the models in Table 3 and Table 4, the starting values for each equation (either dominant height at age 5 or stand density at age 5, for each plot), were defined as additional parameters to be estimated as part of the optimization procedure. Given the 72 plots used in this research, there were 72 dominant height values and 72 starting densities estimated using a dummy variable approach, similar to what was proposed by Cieszewski and Bailey (2000) for dominant height. An approximation of the standard error for all the parameters was obtained by calculating the Hessian matrix during the optimization process. For all models, the variable N (TPH) was scaled by dividing the values by a factor of 1,000 to facilitate the optimization process.

The following statistics were calculated to evaluate model fit:

- Root Mean Square Error (RMSE):

$$RMSE = \sqrt{\frac{\sum_{i=1}^{n}(Y_i - \widehat{Y}_i)^2}{n-p}}$$

- Mean Difference (MD):

$$MD = \frac{\sum_{i=1}^{n}(Y_i - \widehat{Y}_i)}{n}$$

- Mean Absolute Difference (MAD):

Table 5
Fit statistics for dominant height model without treatments (Eq. (3)) and with treatments (Eq. (4)).

Model	RMSE (m)	MD (m)	MAD (m)	AIC
Eq. (3)-Without treatments	0.7057	−0.0046	0.5469	1436.52
Eq. (4)-With treatments	0.5724	0.0012	0.4463	1189.01

Table 6
Parameter estimates for the dominant height model (Eq. (4)).

Parameter	Estimated value	Standard error
μ_0	0.3786	0.0093
K	0.1140	0.0015
b_1	0.9174	0.0102
b_2	1.1039	0.0068
$\ln(\sigma)$	−0.5579	0.0288

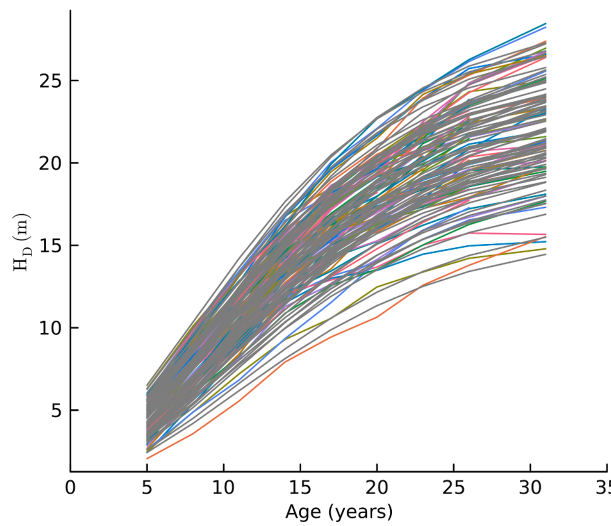

Fig. 7. Estimated dominant height trajectories.

$$MAD = \frac{\sum_{i=1}^{n}|Y_i - \widehat{Y}_i|}{n}$$

And for model comparison:

- Akaike Information Criteria (AIC):

$$AIC = -2loglik + 2p$$

Where n is the total number of observations, p is the number of parameters in each model, Y_i is the observed value, $\widehat{Y}_i$ is the predicted value.

3. Results

3.1. Dominant height model

Fit statistics for the two dominant height models evaluated are presented in Table 5. The proposed model with the addition of parameter modifiers representing treatments effects was effective at reducing the average error and bias. Therefore, model from Eq. (4) was chosen to test the hypothesis of using height increments for the mortality model in section 3.2. Global parameter estimates for this model are presented in Table 6. The local parameters corresponding to the initial state of the variable, which is the dominant height (H_D) at age five ($t = 5$), are not presented.

Eq. (4) expressed as a difference equation (projection form), is

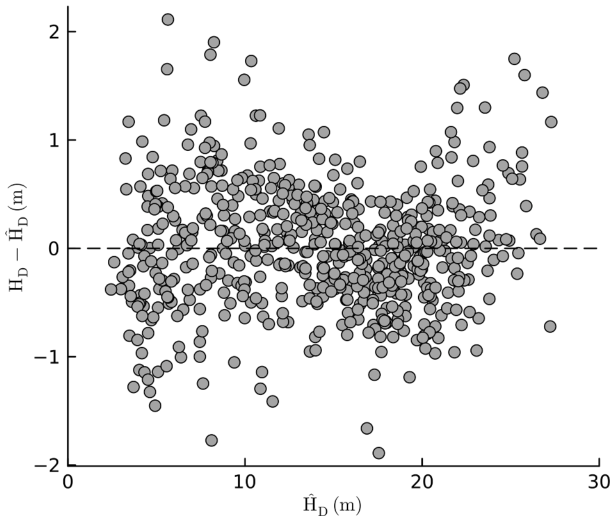

Fig. 8. Dominant height residuals vs predicted dominant height values.

Table 7
Fit statistics[1] for base survival models (control only, no-treatment effects).

Eq.	Model form	RMSE (TPH)	MD (TPH)	MAD (TPH)	AIC	Log-like
Eq. 5	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {*} FR)N$	41.55	0.22	30.08	−533.18	−269.59
Eq. 6	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {*} FR)Nt$	52.79	0.31	35.60	−459.88	−232.94
Eq. 7	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {*} FR)Nt^{\delta}$	35.54	0.02	26.37	−579.00	−293.50
Eq. 8	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {*} FR)N^{\gamma}t$	57.58	−0.001	38.37	−431.31	−219.66
Eq. 9	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {*} FR)N^{\gamma}t^{\delta}$	35.46	0.003	26.23	−577.68	−293.84

[1] Fit statistics were scaled to the original value by multiplying by 1,000. N: trees per hectare at time t, t: time in years, FR: Fusiform rust infection rate per plot at year 5 (varying from 0 to 1), α_0, α_1, γ, δ: estimated parameters.

Table 9
Parameter estimates for the final survival model including fusiform rust and treatment effects.

Parameter	Estimated value	Standard error estimate
α_0	−0.0091	0.0020
α_1	−0.2272	0.0472
δ	−0.4306	0.0798
$\ln(\sigma)$	−3.0871	0.0288

presented in Eq. (18). The predicted dominant height trajectories and residuals are presented in Fig. 7 and Fig. 8, respectively.

$$H_{D_2} = H_{D_1} exp\left[\frac{\mu_0 b_1^{Z_1}}{K b_2^{Z_2}}\left(e^{-K b_2^{Z_2} t_1} - e^{-K b_2^{Z_2} t_2}\right)\right] \tag{18}$$

Given the importance of fusiform rust infection on the survival of the slash pine stands evaluated, it is natural to raise the question of the effect fusiform rust infection on dominant height. Nevertheless, this effect was not evaluated in this research due to previous research showing little or a non-significant reduction in height growth due to fusiform rust infection in slash pine (Jones, 1972; Nance et al., 1981; Sluder, 1977). The main documented effect of fusiform rust in slash pine has been the rust-associated mortality (RAM), which generates volume losses due to low stocking at the end of the rotation (Wells and Dinus, 1978). Economic losses are also usually associated with the low quality timber affected by stem cankers (Sluder, 1977) rather than with a reduction in growth. Burton et al., (1985) argued that although they did find significant differences in height growth at an early age (less than 5 years), over the long-term, losses associated with this lower growth rate are shadowed by RAM since young infected trees are the most likely to die before rotation age.

3.2. Survival models

Fit statistics for the evaluated base survival models from Table 3 are presented in Table 7. The model with better performance was the model form from Eq. 7, which is the model proposed by Pienaar and Shiver (1981), and represents a reduced version of the model proposed by Clutter and Jones (1980) (Eq. 9 in Table 7) if parameter γ is equal to one ($\gamma = 1$). The full version of this model with $\gamma \neq 1$ generated a slightly better fit, with lower values of RMSE, MD and MAD. Nevertheless, this model had an additional parameter, and when using AIC as the model selection criteria, the model with the lowest (better) value was the model from Eq. 7.

Following what presented in Table 4, modifications were made to the model form from Eq. 7 to include treatments effects (i.e., bedding and vegetation control). The fit statistics for these models are presented in

Table 8
Fit statistics[1] for survival models including treatment effects.

Eq.	Model form	RMSE (TPH)	MD (TPH)	MAD (TPH)	AIC	Log-like
Eq. 19	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {*} FR)Nt^{\delta}$	45.63	−0.01	32.59	−1997.18	−1002.59
Eq. 20	$\frac{dN}{dH_D} = (\alpha_0 + \alpha_1 {*} FR)Nt^{\delta}$	48.89	0.00	34.73	−1914.32	−961.16
Eq. 21	$\frac{dN}{dH_D} = (\alpha_0 + \alpha_1 {*} FR)NH_D^{\delta}$	48.56	−0.10	34.58	−1922.35	−965.18
Eq. 22	$\frac{dN}{dt} = (\alpha_0 + \alpha_1 {*} FR)c_1^{Z_1}c_2^{Z_2}Nt^{\delta}$	45.53	0.01	32.57	−1995.77	−1003.88
Eq. 23	$\frac{dN}{dH_D} = (\alpha_0 + \alpha_1 {*} FR)c_1^{Z_1}c_2^{Z_2}Nt^{\delta}$	48.75	0.02	34.76	−1913.65	−962.82
Eq. 24	$\frac{dN}{dH_D} = (\alpha_0 + \alpha_1 {*} FR)c_1^{Z_1}c_2^{Z_2}NH_D^{\delta}$	48.49	−0.09	34.54	−1920.10	−966.05

[1] Fit statistics were scaled to the original value by multiplying by 1,000. N: trees per hectare at time t, t: time in years, FR: Fusiform rust infection rate per plot at year 5 (varying from 0 to 1), H_D: dominant height (meters), Z_1: dummy variable equal to 1 if bedding was applied and 0 otherwise, Z_2: dummy variable equal to 1 if vegetation control was applied and 0 otherwise, α_0, α_1, c_1, c_2, δ: estimated parameters.

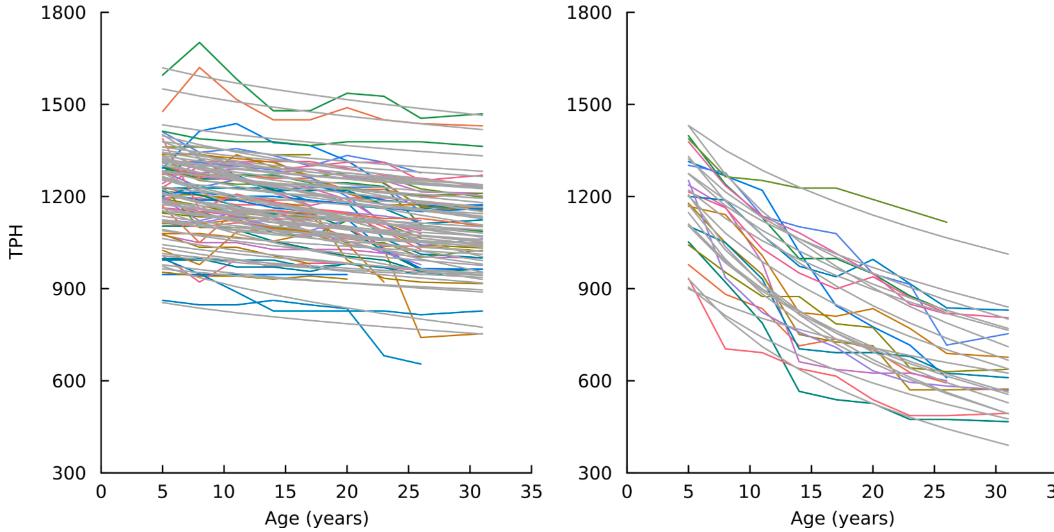

Fig. 9. Trajectories of TPH estimated with Eq. 19 for plots with less than 15% (left), and higher than 15% (right) fusiform rust infection rates.

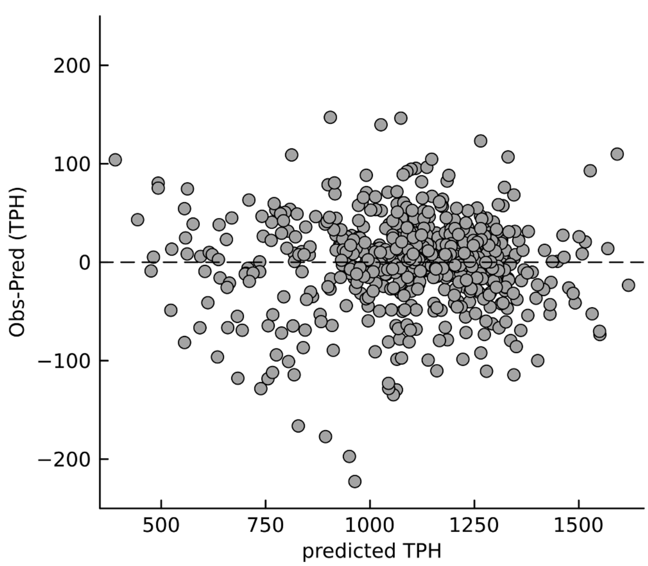

Fig. 10. Residuals for the recommended mortality model (Eq. 19).

Table 8. The model with improved performance was model in Eq. 19, where the mortality rate was modeled with respect to time, and no additional treatment factors were used. When explicit treatment effects were added (Eq. 22), the RMSE and MAD were reduced slightly compared to model in Eq. 19, nevertheless, this was at the expense of two additional parameters, which it is not justified from a statistical point of view.

Parameter estimates for the final model (Eq. 19) are presented in Table 9. Only global parameters are presented. The trees per hectare (*N*) trajectories for this model are presented in Fig. 9 along with the residuals in Fig. 10.

The residuals presented in Fig. 10 are well distributed around zero across the predicted TPH values. In Fig. 11 the histogram of these residuals and a Q-Q plot are presented and show no major deviations from a normal distribution, nevertheless some heavy tails are observed (Fig. 11).

The final recommended model in its difference (projection) form is as follows:

$$N_2 = N_1 \exp\left[\frac{\alpha_0 + \alpha_1 FR}{\delta + 1}\left(t_2^{\delta+1} - t_1^{\delta+1}\right)\right] \tag{25}$$

In the particular case of $t_1 = 0$ and $N_1 = N_0 =$ initial planting density, the prediction equation is as follows:

$$N_t = N_0 \exp\left[\frac{\alpha_0 + \alpha_1 FR}{\delta + 1} t^{\delta+1}\right] \tag{26}$$

4. Discussion

Stand survival in slash pine plantations including silvicultural treatment applications was best described when the mortality rate was modeled with respect to time increments rather than with respect to dominant height increments as proposed by Garcia (2009). Stankova and Diéguez-Aranda (2014) had attributed improvements in model fit when using Garcia's approach to the implicit inclusion of site quality factors when including dominant height into the model. Nevertheless, in all the models tested, the estimated local values per plot likely accounted for some of the site-specific variation. Garcia (2009) also mentioned his approach was particularly useful when dealing with scarce or low quality data that does not cover a wide enough range of growing conditions, which was not the case for this study where the installation locations were located throughout the slash pine range in southern Georgia and northern Florida.

The explicit treatment modifiers added to include the effect of bedding and or vegetation control as part of the survival function, were not successful in improving model fit significantly. Although some authors have found bedding to have a positive effect on pine plantations growth and survival when stands are located in poorly drained soils (Gent et al., 1986; Pritchett, 1979), we found that bedding did not have a significant effect on the mortality rate in this study, or more precisely, no effect was observed for the measurement period considered in this research, which started at year five. It is likely that the effect of bedding on mortality rates was more pronounced during the first years after planting and that no significant effect was observed in later years, when the overstory measurements commenced.

Regarding the vegetation control treatment, although growth gains have been reported for slash pine due to this treatment (Creighton et al., 1987; Lauer and Glover, 1998; Ramirez et al., 2022; Zhao et al., 2009), a less marked effect has been found for survival. Jokela et al., (2000) did not find a significant effect on slash pine survival rates at early ages (5 and 8 years) due to herbaceous weed control applications during site preparation, and Creighton et al. (1987) did report an improvement in survival rates due to vegetation control applications, although this was only when plantations were established on sites with a water deficit and high levels of competition. In contrast, the sites where these research

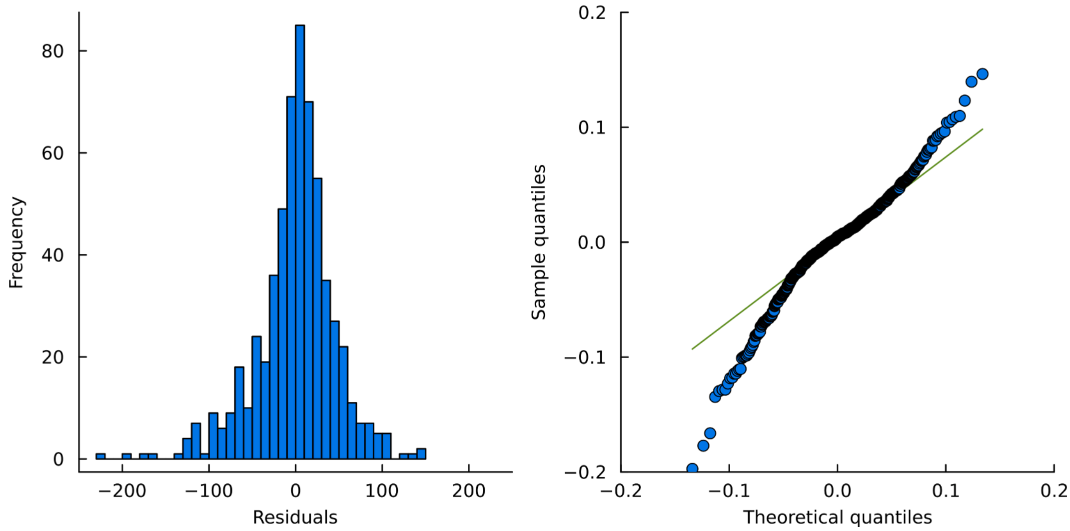

Fig. 11. Residuals histrogram and Q-Q plot for the recommended mortality model including treatments effect (Eq. 19).

plots were established were more likely to have excess water, explaining the non-significant effect of the vegetation control treatment on mortality observed in this research.

Fusiform infection rates have been found to be differential when combined with early silvicultural treatments. Burton et al., (1985) found for young stands (less than 5 years) that fusiform infection rates were higher when complete vegetation control and bedding were applied compared to a control. Although this was not an objective of our study, the fact that explicit treatment effects did not significantly improve model fit, suggest that in the long-term, the interaction between the silvicultural treatments and the fusiform rust infection rates might be less relevant.

Results from this study indicate that the most important variable to describe mortality in slash pine plantations was the fusiform rust infection rate. Wells and Dinus (1978) also found that the number of trees infected with fusiform rust at year five was a reliable predictor of rust-associated mortality at year 10. In our proposed model, the proportion of infected trees at year five was useful to model survival trajectories over time. The rate of infection at age five was maintained through the observed measurement period (Fig. 6), with low infection rates continuing for sites in which low infection rates were observed at year five and vice versa. Therefore, it is believed that fusiform rust infection has an impact on survival for a sustained period of time beyond year five, but the first measurement taken at year five was a good proxy of the fusiform rust infection impact through the whole period evaluated. Performing an assessment of fusiform rust at year 5 is then recommended to use the proposed mortality model. Nevertheless, in the absence of this assessment, localized historical infection rates could be used to approximate the infection rate at year 5, which is preferable to ignore fusiform rust infection completely.

The use of differential equations in the construction of the proposed dominant height and survival models facilitated the inclusion of silvicultural treatments and fusiform rust infection effects. Although both models can be integrated analytically and can be fitted in this form using non-linear least squares or maximum likelihood, modifying the model in its differential form allowed us to better evaluate which terms should be modified according to what each parameter represented in the model.

5. Conclusions

We tested for the effect of silvicultural treatments (bedding and vegetation control) as they affect the rate of stand mortality in slash pine plantations. Treatment effects were incorporated either implicitly, through the use of dominant height increments which already captured treatment effects, or explicitly through treatment terms in the mortality model form. The use of dominant height increments and explicit parameter modifiers in the mortality model were not effective in improving model fit, implying bedding and vegetation control did not affect mortality rates at or beyond age 5 in the slash pine plantations evaluated in this study. On the other hand, knowledge of fusiform rust infection rates was essential to accurately describe mortality trajectories, with higher fusiform rust infection rates implying higher mortality. Mortality was modeled using differential equations in which the change in the number of trees per hectare at a given time was described by the current number of trees, a power function of age, and the observed average fusiform rust infection rate at age 5. Although fusiform rust has been previously identified by several authors as one of the main drivers of mortality in slash pine plantations, risk of infection remains high in the southeastern United States (Randolph, 2016; Weng et al., 2018). Genetic improvement has proven to be efficient at reducing infestation rates in other pines in the region (Randolph, 2016), suggesting the need to prioritize genetic improvement for rust resistance for the species to reduce the impact in overall value (Susaeta, 2020).

CRediT authorship contribution statement

Laura Ramirez: Conceptualization, Methodology, Software, Formal analysis, Data curation, Writing – original draft, Visualization, Resources. **Cristian R. Montes:** Conceptualization, Methodology, Software, Writing – review & editing, Supervision. **Bronson P. Bullock:** Writing – review & editing, Supervision, Resources.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The authors do not have permission to share data.

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