A Generalized Methodology for Developing Whole-Stand Survival Models

Charles E. Rose Jr., Michael L. Clutter, Barry D. Shiver, Daniel B. Hall, and Bruce Borders

ABSTRACT. A large source of variability in yield predictions is due to estimation of future surviving trees per unit area. Previous whole-stand survival modeling efforts have concentrated on modeling the empirical survival curve. Modeling hazard functions, an approach to survival analysis commonly used in fields such as medicine and sociology, can be applicable to plantation survival estimation. We offer a generalized method for deriving whole-stand survival models that are capable of modeling complex underlying hazard functions. We use our knowledge of the empirical hazard function to limit our selection to appropriate functions. Integrating selected functions results in initial condition difference equations that, when fitted to our data, provide biologically reasonable whole-stand survival predictions and adequately represent the underlying hazard function. Our method is relatively easy to implement and can model a whole-stand survival curve with a complex underlying hazard function. FOR. SCI. 50(5):686–695.

Key Words: Survival analysis, hazard function, differential equation.

WHOLE-STAND SURVIVAL MODELS are important in accurately describing growth and yield for plantations because the basal area growth model is sensitive to underlying mortality. However, development of whole-stand survival models has received little attention relative to individual tree survival models. Forest mortality can result from, but is not limited to, competitive factors (e.g., light, nutrients, water), insects, pathogens, or catastrophic events (e.g., flooding, tornado, hurricane). Here we focus on modeling noncatastrophic whole-stand forest plantation survival.

Let the distribution of T, time to mortality, be $F(t) = Pr[T \leq t]$, then $f(t)$ is the corresponding density function, and the survival function is defined as $S(t) = Pr(T > t) = 1 - F(t)$. The hazard function, which is the instantaneous rate of mortality assuming the individual has survived to time t (Collett 1994), is defined as $h(t) = f(t)/[1 - F(t)]$. Analogous to the continuous time hazard function, the discrete time hazard function is defined as $q(t) = [F(t + 1) - F(t)]/[1 - F(t)]$. Survival curves are nonincreasing over time, whereas hazard functions can increase, decrease, remain constant, or assume a combination of these shapes.

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A common cumulative distribution function (cdf) used in survival analysis is the Weibull because its hazard function can monotonically increase, monotonically decrease, or remain constant, depending on the value of the shape parameter. However, the Weibull distribution imposes strong restrictions on the data and is unable to model complex hazard functions such as the "bathtub" (Hjorth 1980) or "ladle" shapes. In survival literature, a bathtub shape hazard function is commonly referred to as U shaped. An observed bathtub shape hazard function usually contains an interval of the event's survival function (i.e., the event is not observed for all subjects) that typically includes the initial survival curve for the subjects. It is often reasonable, given the observed event for all these subjects, for the hazard function to appear ladle shaped. Here, we use bathtub- and ladle-shaped hazard functions interchangeably.

Of the three general shapes of hazard functions, the monotonically increasing hazard function is the most common. Increasing monotonic hazard functions are probably more common because many studies focus on an interval of the subject's lifespan in which gradual aging takes place beyond the juvenile stage. There is usually no empirical motivation because of the interval focus to find distributions that are capable of producing more complex shaped hazard functions. However, a bathtub-shaped hazard function is a reasonable assumption when viewing the entire lifespan of many, if not most, biological organisms. Consider a tree's lifespan; assume four distinct phases: seedling to sapling (hazard generally decreases), sapling to juvenile (hazard generally stable), juvenile to adult (hazard might be stable or start to increase), and adult to maturity (hazard increases). Taking an interval from any of these phases will likely result in a specific type of hazard curve, but when viewed as an entity it is reasonable to assume a bathtub, or alternatively, a ladle-shaped hazard curve. This doesn't mean that a high vigor mature tree will have a high hazard. However, it does mean the hazard is generally increasing for this high vigor mature tree as it ages.

Whole-stand survival models have commonly been developed using a derivative of the generalized Gamma distribution (e.g., Weibull and exponential) or the difference equation approach. These approaches, either implied or explicitly stated, make assumptions about the relative rate of instantaneous mortality. The Weibull is a flexible distribution that has been widely used for whole-stand survival models (Pinder et al. 1978, Glover and Hool 1979, Somers et al. 1980, Pienaar and Shiver 1981, Belli and Ek 1988, Amateis et al. 1997). The hazard function for the two-parameter Weibull distribution is $h(t) = c/t(t/b)^c$, where t is time and b, c are scale and shape parameters, respectively. As previously mentioned, the Weibull hazard function can produce the three most common hazard shapes: monotonically increasing, monotonically decreasing, or remaining constant. Often these three common hazard shapes are applicable because the study data only contain an interval of a stand's lifespan (e.g., data may pertain to plots that are established after the initial seedling mortality). Parametric whole-stand survival models (e.g., based on the Weibull

distribution) use suppositions with respect to the distribution's ability to model the empirical survival trends. Conversely, difference equation models use suppositions about the relative rate of instantaneous mortality change. Although these two approaches appear distinct, often the difference equation approach leads, after integration, to a parametric whole-stand survival model. We illustrate this using a simple difference equation for the relative rate of mortality that assumes the instantaneous hazard rate is constant, i.e.,

$$-\frac{1}{N}\frac{dN}{dA} = \beta$$

where N is the number of trees per unit area, A is age, and β a parameter. After integration using the initial conditions of $N_2 = N_1$ when $A_2 = A_1$, the result is

$$\frac{N_2}{N_1} = S(A_2) = e^{-\beta(A_2 - A_1)}$$

Thus, this difference equation results in the exponential distribution and implies that the instantaneous hazard rate is constant for all ages, densities, and site indices (Clutter et al. 1983). Exponential distributions have been used in several whole-stand survival studies (Devine and Clutter 1985, Martin et al. 1999); however, the exponential distribution imposes the strong assumption of a constant hazard rate. Nonetheless, a constant hazard rate can be reasonable because of the study data age range. For example, Devine and Clutter (1985) used survival data from 161 plots, of which only two plots had measurement data less than 5 years of age. Hence, we wouldn't expect to detect early stand survival trends. Clutter and Jones (1980) presented a more flexible difference equation in which they assumed that the relative rate of instantaneous mortality is proportional to age and initial trees per acre, which are raised to a power, i.e.,

$$\frac{1}{N}\frac{dN}{dA} = \alpha A^\delta N^\phi$$

Integrating over the initial conditions specifying that $N_2 = N_1$ at $A_2 = A_1$ yields $N_2 = [N_1^\beta + \eta(A_2^\varphi - A_1^\varphi)]^{1/\beta}$, where β, φ, η are parameters, and the other terms are previously defined. This flexible whole-stand survival model has subsequently been used with slight modifications in several studies (e.g., Pienaar and Rheney 1993, Martin and Brister 1999).

It is common to model whole-stand mortality for a stand's lifespan using a system of equations (e.g., Matney and Farrar 1992, Amateis et al. 1997). These systems disaggregate the lifespan of a stand's survival into distinct phases, typically some combination of the seedling, sapling, juvenile, adult, and mature phases. Disaggregating whole-stand survival into phases is often motivated by the difficulty in developing a biologically reasonable function that can model survival throughout stand development and by data limitations.

We demonstrate a method for deriving biologically reasonable whole-stand survival models, which are capable of

modeling complex underlying hazard functions. Our method is capable of modeling whole-stand survival for a stand's lifespan using a single equation. In addition, we hypothesize the continuum of whole-stand forestry survival generally has an underlying bathtub-shaped hazard function.

Data

Data were obtained from the Consortium for Accelerated Pine Production Studies (CAPPS), which is overseen by the Warnell School of Forestry at the University of Georgia. CAPPS' purpose is to investigate the effects of intensive forest management on the productivity of loblolly pine (*Pinus taeda* L.) plantations in the southeastern United States. The CAPPS plots were established throughout Georgia from 1986–1994. Study protocol directed establishment of two complete blocks at each location, each block containing four 0.15-ha treatment plots. A 0.15-ha treatment plot was established at each location using bareroot seedlings on a 2.44-m × 2.44-m spacing. A 0.05-ha measurement plot was centered within each of the treatment plots. The following four cultural treatments were randomly assigned to the blocks at each location.

1. Herbicide (H): plot sprayed with nonsoil-active herbicide as needed to maintain complete control of woody and herbaceous vegetation.

2. Fertilization (F): apply recommended rates of fertilizer annually, if necessary, to ensure that nutrients are not a limiting factor.

3. Herbicide and Fertilization (HF): apply both herbicide and fertilization treatments.

4. Control treatment (C): no cultural treatment other than mechanical site preparation.

The study protocol called for replication of all treatment plots every 2 years for the first 10 years of the study. The actual study deviates from the protocol because of funding limitations so that replications have been repeated at different intervals for different locations. Plots have been measured annually with survival data beginning at age 2, and data are summarized by plot age and treatment (Table 1). Plot site indices are unknown because estimates were not obtained prior to site preparation. It is possible to estimate site indices using height equations developed for this re-

Table 1. The CAPPS study plot distribution by year planted for the spectrum of plots and by treatment.

Year planted	Plot age	Plots	Control	Fertilized	Herbicide	HF[a]
				Plots by treatment		
1986	14	26	8	4	8	6
1987	13	28	8	6	8	6
1988	12	36	10	8	10	8
1989	11	24	8	4	8	4
1992	8	20	6	4	6	4
1994	6	12	4	2	4	2
Total		146	44	28	44	30

[a]HF is the herbicide and fertilizer treatment.

gion; however, we currently feel it is unjustified to estimate site indices at base age 25 because the plots range from 6 to 14 years.

Model Development

We used the Kaplan-Meier product limit estimator (KM) (Kaplan and Meier 1958) and corresponding discrete hazard estimates as guides for developing whole-stand survival models. The KM and hazard estimates provide insight into the complexity of survival and hazard shapes necessary for modeling our whole-stand survival data. The KM product limit estimator is defined as

$$\hat{S}(t) = \prod_{i=1}^{j} \left[1 - \frac{d_i}{n_i} \right]$$

for $t_i \leq t \leq t_{j+1}$. Here, n_i and d_i are the subjects at risk (n_i) and that die (d_i) at time t_i and $\hat{S}(t) = 1$ for $t < t_1$.

Computed KM survival estimates and corresponding discrete hazards are presented in Table 2. The KM survival estimates for the spectrum of plots illustrate that the underlying discrete hazard decreases from age 2 to 5 and then increases from ages 5 to 14. In addition, KM survival and discrete hazard estimates were computed by treatment (Figure 1). Survival curves by treatment illustrate that the H and F treatments result in the most favorable and unfavorable survival, respectively. In addition, the HF treatment has favorable early survival, but mortality increases rapidly after about age 8. The estimated hazard function trend for the spectrum of plots appears to be bathtub shaped, but the oldest plots are 14 years, and it is difficult to infer the future trend of the hazard function. Nevertheless, it is reasonable to assume that the hazard function will continue to increase with time, which is consistent with most biological organisms (Pinder et al. 1978). Multiple censoring occurs for our data and is primarily due to plots being less than 14 years of age (Table 2). However, some censoring occurs for individual trees because their status could not be determined from

Table 2. The Kaplan-Meier product-limit estimator for the cumulative survival and discrete time estimated hazards using the CAPPS study data.

Age[a]	N[b]	Alive[c]	Dead[d]	Censored	Survival	Hazard
2	11,956	11,425	531	1	0.9556	0.0444
3	11,424	11,269	155	5	0.9426	0.0136
4	11,264	11,193	71	2	0.9367	0.0063
5	11,191	11,134	57	8	0.9319	0.0051
6	11,126	11,061	65	955	0.9265	0.0058
7	10,106	10,034	72	19	0.9199	0.0071
8	10,015	9,940	75	1,598	0.9130	0.0075
9	8,342	8,274	68	77	0.9055	0.0082
10	8,197	8,115	82	872	0.8965	0.0100
11	7,243	7,171	72	1,345	0.8876	0.0099
12	5,826	5,745	81	1,899	0.8752	0.0139
13	3,846	3,772	74	1,943	0.8584	0.0192
14	1,829	1,788	41		0.8391	0.0224

[a]Age = ending age; beginning age = age–1.
[b]N = total number of trees at the beginning age.
[c]Alive = the total number of trees surviving to the ending age.
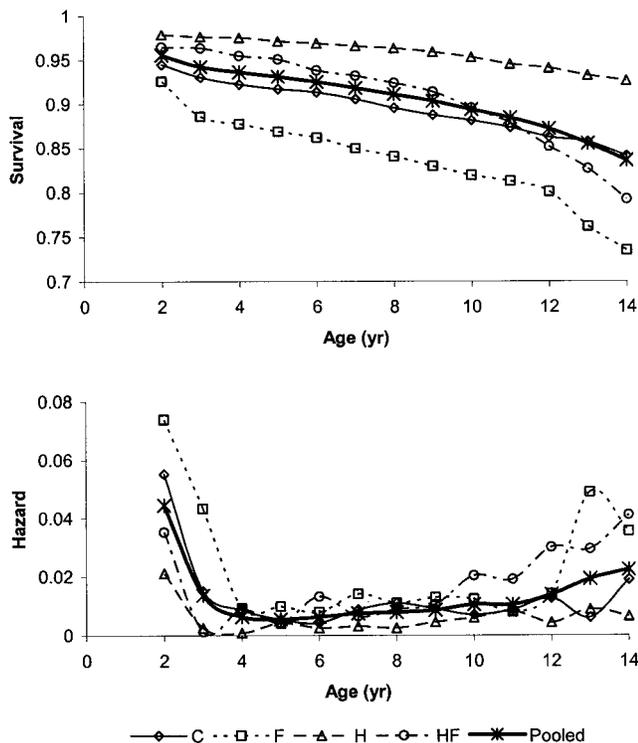[d]Dead = the number of trees dying during the interval.

Figure 1. The CAPPS study empirical survival, mortality, and hazard functions by treatment (C, control; F, fertilized; H, herbicide; and HF, herbicide and fertilized) and spectrum of plots (pooled).

one measurement occasion to the next (e.g., tree tag number is no longer in data set but no indication given that it had died). In addition, an entire fertilized treatment plot was censored at age 9 because of a catastrophic flood. It is assumed trees are randomly censored and the distribution of censoring times is independent of exact survival times. This means that censored values come from the same survival distribution as those that are not censored, and the amount and pattern of censoring should be comparable across groups. As Table 2 reveals, approximately 35 trees are censored unrelated to plot age or catastrophic flooding. Censoring related to these trees was investigated, and there is no evidence that these "missing" trees are more likely to be dead than "nonmissing" trees. If these approximately 35 trees are in fact dead, then we would be overestimating our survival curve. Hence, to determine the consequence of if these trees are dead, we recomputed the empirical survival and hazard functions treating these trees as dead. There is no substantial difference among the survival and hazard functions, and the hazard function still exhibits a bathtub shape. We also looked at cohort mortality, where our cohorts were defined as plots established in the same year by treatment. There are no substantial differences in the survival curves of our cohorts. We originally defined our cohorts as plots established in the same year by location, but this led to approximately 4.5 plots per cohort. Because our plots are 0.05 ha, we feel this is too small a sample to obtain an accurate picture of the mortality because it is a rare event.

All evidence from the discrete hazard estimates indicates a bathtub-shaped hazard function for our data.

We began whole-stand survival modeling by considering parametric survival functions that are capable of modeling a bathtub-shaped hazard function. Two of the considered functions are the four-parameter generalized Weibull distribution and exponential power distribution. The generalized Weibull distribution has the following survival function:

$$S(t) = e^{-\alpha(t^\beta + \kappa)^\lambda}$$

where α, β, κ, and λ are parameters, and t is time. If $\kappa = 0$ and $\lambda = 1$, then the generalized Weibull distribution reduces to the two parameter Weibull distribution. The two-parameter exponential power distribution survival function is given by:

$$S(t) = \exp[1 - \exp((\lambda t)^\alpha)]$$

where λ and α are parameters, and t is time. As a preliminary investigation, we fit these parametric survival functions to the complete CAPPS data set. The four-parameter generalized Weibull distribution failed to converge when using numerous nonlinear methods and starting parameters. The power exponential converged, but results were not wholly satisfactory because it did not adequately reflect the empirical hazard curve at older ages. In light of our preliminary parametric survival functions investigation, and because we wanted to develop a highly adaptable method for generating numerous hazard shapes, we focused on the differential equation whole-stand survival modeling method.

We assumed that whole-stand plantation survival could be modeled using a generalized differential equation to describe the relative rate of mortality, i.e.,

$$\frac{1}{N}\frac{dN}{dt} = f(t) X$$

where N is the number of trees per unit area, $f(t)$ is a function of time, and X can be a function of any whole-stand attribute(s). This model assumes that the instantaneous hazard rate is a function of time, but note that if $f(t)$ and X equal β and 1, respectively, then we are assuming that the instantaneous hazard rate is constant. Our focus is on methodology and not investigation of potential covariates (e.g., site index or stand density); therefore, we narrowed the scope for viable survival models by assuming X equals 1 or N^d. However, note that N^d can distinguish among density classes. Our search for a viable $f(t)$ function began by assuming from empirical evidence (Figure 1) that the function should be flexible enough to model a bathtub-shaped hazard function. During preliminary investigation, we developed numerous $f(t)$ functions with constraints that the function had a closed form solution on integration and $f(t)$ could model bathtub-shaped curves. Our preliminary investigation led us to the chosen functional form of:

$$f(t) = \frac{1}{1 + at} + \frac{b}{1 + t} + ct$$

Where t is time, and a, b, and c are parameters. This function has the flexibility to model, but is not limited to, monotonically increasing or decreasing hazards as well as a bathtub-shaped hazard function (Figure 2). Our motive for choosing $f(t)$ is its ability to model bathtub-shaped curves. We can establish if the resulting survival function is capable of modeling an underlying bathtub-shaped hazard function by noting the concavity and number of inflection points in the survival curve. To obtain a bathtub-shaped hazard curve, it is obvious that the hazard curve must first decrease over time, level off, and then increase. A bathtub-shaped hazard curve corresponds to a survival curve with at least two inflection points. Furthermore, the concavity of the survival curve between the two inflection points must be convex to the first inflection point (starting at $t = 0$), then concave from the first inflection to the second inflection point, and then convex again after the second inflection point. Substituting $f(t)$ into the differential equation and integrating for both values of X using the initial condition that when $N_2 = N_1$ then $t_2 = t_1$ results in:

$$N_2 = N_1 \left(\frac{1 + at_2}{1 + at_1} \right)^{1/a} \left(\frac{1 + t_2}{1 + t_1} \right)^b e^{c/2(t_2^2 - t_1^2)}, \; X = 1 \quad (1a)$$

and

$$N_2 = \left[N_1^d + \frac{d}{a} \ln\left(\frac{1 + at_2}{1 + at_1} \right) \right.$$
$$\left. + b' \ln\left(\frac{1 + t_2}{1 + t_1} \right) + c'(t_2^2 - t_1^2) \right]^{1/d}, \; X = N^d \quad (2a)$$

Here, $b' = -db$ and $c' = -dc/2$. Both models possess desirable properties of path invariance, and when $t_2 \rightarrow t_1$ then $N_2 \rightarrow N_1$. Clearly, Models 1a and 2a have the flexibility to model multiple inflection points. Note if $d \neq 1$, then Model 2a will distinguish among density classes for survival because the survival rate will depend on N_1. In addition, the Model 1a lower asymptote is 0 if c is negative. The lower asymptote for Model 2a is not as clear; however, it is capable of having a lower asymptote at 0. We modified
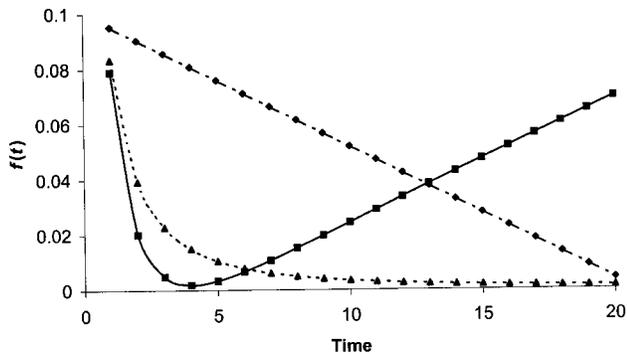

Figure 2. **Examples of viable shapes using the chosen function,**

$$f(t) = \frac{1}{1 + at} + \frac{b}{1 + t} + ct$$

used in the whole-stand survival model differential equation.

Models 1a and 2a asymptotes to allow for a biologically reasonable lower asymptote, which has been demonstrated to be a reasonable assumption for plantation loblolly pine of the southeastern United States (Harrison and Borders 1996, Martin and Brister 1999). Lower asymptotes for both models were redefined as $N_i - N_{min}$, where $i = 1, 2$ and N_{min} is the specified lower asymptote. We fit Models 1a and 2a by using the complete data set and by cultural treatment to determine the effects of cultural treatments on estimated parameters.

All models were evaluated by examining the residuals, error sum of squares (SSE), mean square error (MSE), root mean square error (RMSE), and fit index, which is defined as:

$$FI = 1 - \frac{\sum_{i=1}^{n}(Y_i - \hat{Y}_i)^2}{\sum_{i=1}^{n}(Y_i - \bar{Y})^2} = 1 - \frac{SSE}{SSTO}$$

Here, Y_i, $\hat{Y}_i$, and $\bar{Y}$ are the actual, predicted, and mean *TPH*; *SSE* is sums of squares of error; and *SSTO* is total sums of squares. In addition, fitted functional behavior was examined, both within the range of the data and extrapolating to a reasonable age.

Results

We fit all models using the Gauss-Newton method with N_2 as the dependent variable. Starting values for Model 1a parameters were determined using reasonable model properties. These properties are: (1) parameter $a > 0$, which insures the divisor will never be 0; (2) b competes with a in a sense, so we started with a negative number; and (3) $c < 0$, which will produce a lower asymptote of 0. Based on these criteria, initial parameter estimates for Model 1a were specified as $a = 1$, $b = -1$, and $c = -0.1$. Using these starting values, Model 1a converged in four iterations with a fit index of 0.9467. Initial values for Model 2a were not as easily determined, but it is reasonable to expect a, $d > 0$. Parameters b' and c' appear to have a tradeoff, i.e., values these parameters are multiplied by will always be positive, so it is reasonable to start with opposite signs for these two parameters. Using $a = 1$, $d = 1$, $b' = -1$, and $c' = 1$ as starting values, Model 2a converged in 11 iterations with a fit index of 0.9472. Several choices of the starting values were used for Models 1a and 2a, and convergence was achieved to the same estimated parameters each time.

Next, we fit Models 1a and 2a separately for each cultural treatment. The treatments' specific estimated parameters for Models 1a and 2a were noticeably different from the parameters estimated using the complete data set. Therefore, we refitted Models 1a and 2a, now referred to as Models 1b and 2b, and allowed the F, H, and HF cultural treatments to vary from the baseline parameters. Cultural treatment effects were coded as F = 1 if treatment equals F and 0 otherwise, similarly for the H and HF treatments.

These results were applicable for Models 1b and 2b. Here for Model 2b, we redefine $b = b'$ and $c = c'$, in:

$$a = a_0 + a_F \,(F) + a_H(H) + a_{HF} \,(HF)$$

$$b = b_0 + b_F \,(F) + b_H \,(H) + b_{HF} \,(HF)$$

$$c = c_0 + c_F \,(F) + c_H \,(H) + c_{HF} \,(HF)$$

$$d = d_0 + d_F \,(F) + d_H \,(H) + d_{HF} \,(HF)$$

We used the parameter estimates for Models 1a and 2a as the starting values for the baseline parameters of Models 1b and 2b, respectively. In addition, we used 0 as the cultural treatment initial values for Models 1b and 2b. Model 1b was fitted and insignificant cultural treatment parameters were removed using a stepwise procedure ($\alpha = 0.05$). We were unsuccessful in achieving convergence for the full Model 2b using the estimated parameters from Model 2a and setting the initial cultural treatment parameters to 0. Therefore, we fitted Model 2b by entering cultural treatment parameters into the model using a forward selection procedure.

Results for all models revealed some evidence of heteroscedasticity and nonnormality in the residuals; however, there were no large influential observed values. We fit all models with weighted least squares (WLS) using several weights. There was no substantial improvement in the residual plots when using WLS, and predictions were virtually unchanged. We investigated using robust regression; there was a minor improvement in the normality of the residuals, but predictions remained virtually identical. Because our focus here is on model development and prediction, we fitted Models 1b and 2b without adjusting for minor heteroscedasticity and nonnormality of the residuals. Our final fitted models, which allow for systematic cultural treatment effects and a modified lower asymptote, are

$$N_2 = N_{min} + (N_1 - N_{min})\left(\frac{1 + at_2}{1 + at_1}\right)^{1/a}\left(\frac{1 + t_2}{1 + t_1}\right)^{b} e^{c/2(t_2^2 - t_1^2)}$$

$$(1b)$$

where the cultural treatment parameters are defined as

$$a = a_0 + a_F \,(F) + a_H \,(H) + a_{HF} \,(HF)$$

$$b = b_0 + b_F \,(F) + b_H \,(H)$$

$$c = c_0 + c_F \,(F) + c_{HF} \,(HF)$$

and

$$N_2 = N_{min} + \left[(N_1 - N_{min})^d + \frac{d}{a}\ln\left(\frac{1 + at_2}{1 + at_1}\right)\right.$$
$$\left. + b\ln\left(\frac{1 + t_2}{1 + t_1}\right) + c(t_2^2 - t_1^2)\right]^{1/d}$$

$$(2b)$$

where

$$a = a_0 + a_H \,(H) + a_{HF} \,(HF)$$

$$b = b_0 + b_F \,(F)$$

$$c = c_0 + c_H \,(H) + c_{HF} \,(HF)$$

$$d = d_0 + d_H \,(H) + d_{HF} \,(HF)$$

Here, $N_j = TPH/100$ (TPH = trees per hectare, and $j = 1, 2$); $N_{min} = 2.5$, which is the lower asymptote for $TPH/100$; t_i is plot age at time i; and a, b, c, and d are parameters. We originally fit all models by defining $N_j = TPH/100$, because convergence may be more easily achieved for nonlinear models when predictor variables are roughly on the same scale. However, convergence was easily achieved using $TPH/100$ or TPH, and we arbitrarily chose $TPH/100$ in our final models. The N_{min} of 2.5 corresponds to approximately 100 trees per acre, which has been deemed a reasonable lower limit for loblolly pine of this region (Harrison and Borders 1996). However, we recognized that the lower asymptote is subjective, and we fit Models 1b and 2b with lower asymptotes of 1.0, 2.5, and unconstrained. We chose to leave $N_{min} = 2.5$ because Models 1b and 2b converged easily, and predictions were almost identical when using these lower asymptotes.

Models 1b and 2b parameter estimates, standard errors, and p-values are presented in Table 3. Summary fit statistics reveal that Model 2b explains more of the variation in survival. RMSE for Models 1b and 2b are 0.4590 and 0.4570, respectively. This means that for the average TPH (approximately 1,600), there is less than a 3% error. The fit indices for Models 1b and 2b are 0.9507 and 0.9511, respectively. There is no substantial difference between Models 1b and 2b according to these criteria. Hence, to further assess model performance, the mean survival and corresponding hazard functions were computed for the spectrum of plots and stratified by treatment.

Models 1b and 2b predicted mean survival and hazard functions for the spectrum of plots illustrate that both models adequately mirror the empirical survival and hazard functions trends (Figure 3). Model 1b more closely mirrors the empirical hazard function for the early ages, but Model 2b exhibits more overall flexibility. Mean survival and the corresponding hazard function were computed by treatment for both models. Models 1b and 2b adequately reflect the underlying hazard function associated with each survival curve. However, there is increased flexibility in solutions when using Model 2b versus Model 1b. This increased flexibility is a result of Model 2b being able to differentiate among density classes.

Models 1b and 2b extrapolation properties were examined by predicting survival by treatment for ages 1–30. Both models behave similarly within the data range (ages 2–14); however, model extrapolation properties are substantially different. Model 1b provides reasonable survival curves by treatment and has a lower asymptote of 250 TPH. In contrast, Model 2b has reasonable extrapolation properties only for the H treatment. The HF treatment predictions decline rapidly beyond the data range. Moreover, the C and F treatments are only able to predict to age 17 using the

Table 3. Models 1b and 2b estimated parameters, standard errors, and *p*-values when fitted to the CAPPS study data.

	Model 1b				Model 2b		
Parameter[a]	Estimate	SE	$Pr > \mid t \mid$		Estimate	SE	$Pr > \mid t \mid$
a_0	0.6487	0.0274	<0.0001		–0.03343	0.0052	<0.0001
a_F	–0.1305	0.0451	0.0039				
a_H	0.0991	0.0419	0.0181		0.1996	0.0758	0.0085
a_{HF}	–0.0562	0.0131	<0.0001		0.1121	0.0565	0.0473
b_0	–1.4088	0.0319	<0.0001		–2.9202	0.4302	<0.0001
b_F	–0.2041	0.0600	0.0007		–0.4098	0.1412	0.0038
b_H	0.1796	0.0474	0.0002				
c_0	–0.00140	0.000329	<0.0001		–0.05387	0.00359	<0.0001
c_F	–0.00225	0.000827	0.0066				
c_H					0.04180	0.00703	<0.0001
c_{HF}	–0.00270	0.000552	<0.0001		0.02839	0.01070	0.0081
d_0					0.8372	0.0683	<0.0001
d_H					0.2240	0.0543	<0.0001
d_{HF}					0.1135	0.0365	0.0019

[a] Parameter subscripts for cultural treatments are F = fertilizer, H = herbicide, and HF = herbicide and fertilizer.
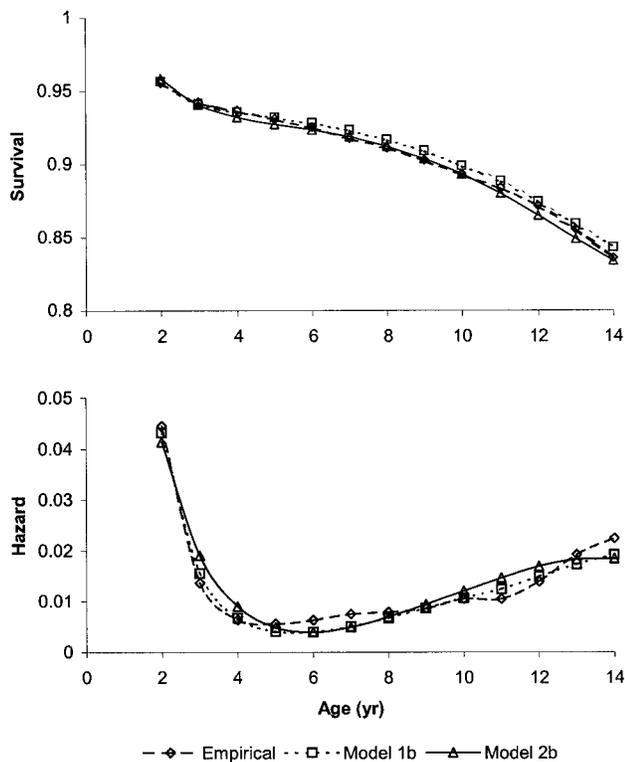

Figure 3. Models 1b and 2b fitted, and the empirical hazard and survival functions for all plots using the CAPPS study data.

estimated parameters because the bracketed term for Model 2b when predicting survival for these treatments is negative after age 16, which results in a negative fractional power. Model 1b provides more reasonable extrapolation predictions when fitted to our data. However, both models behave adequately within the data range.

To further evaluate Models 1b and 2b, we conducted a simulation to demonstrate model performance when using an independent data set. The simulation was conducted by dividing the CAPPS data set into fit and validation data sets.

We removed approximately one-third of the plots by cultural treatment for the validation data set and fit Models 1b and 2b to the remaining plots. After fitting Models 1b and 2b using the fit data set, estimated parameters were used to predict *TPH* for the validation data set. Then the mean error, absolute mean error, and fit index were computed using the validation data set. We performed 1,000 simulations using this scenario and then computed the mean error and mean absolute error for the 1,000 simulations. Results demonstrate that Models 1b and 2b perform excellent when applied to the validation data (Table 4). The small range between the minimum and maximum values for all criteria demonstrates the robustness of Models' 1b and 2b.

Our final model evaluation criteria compared our Model 1a with two common whole-stand survival models. The Weibull distribution and the Clutter and Jones (1980), hereafter referred to as C&J, models are probably the most

Table 4. Simulation results for the fit and validation data sets.

	Mean[a]	Minimum[b]	Maximum[b]
Model 1b			
Fit data			
Fit index	0.9506	0.9369	0.9609
Validation data			
Fit index	0.9474	0.9057	0.9653
Mean error[c]	–0.01081	–0.1152	0.1032
Mean absolute error[c]	0.2247	0.1939	0.2575
Model 2b			
Fit data			
Fit index	0.9510	0.9362	0.9630
Validation data			
Fit index	0.9481	0.9038	0.9668
Mean error[c]	–0.00118	–0.09013	0.108066
Mean absolute error[c]	0.2289	0.1999	0.2622

[a] The mean is the mean for the 1,000 fit and validation data sets.
[b] The minimum and maximum values correspond to these 1,000 data sets.

[c] Mean error $= \dfrac{\sum\limits_{i=1}^{n}(Y_i - \hat{Y}_i)}{n}$, mean absolute error $= \dfrac{\sum\limits_{i=1}^{n}|(Y_i - \hat{Y}_i)|}{n}$.

widely used whole-stand survival models; therefore, these models were fitted to our data for comparison purposes. We chose Model 1a for demonstration purposes. Models 1b, 2a, or 2b could have been chosen, and we could have allowed the C&J and Weibull parameters to vary by cultural treatment if appropriate. However, here our focus is demonstrating extrapolation properties. We failed to achieve convergence using our data for the Weibull or C&J survival models, probably because the empirical survival curve has two inflection points (Figure 3). Since we could not achieve convergence and because it is common to have whole-stand plantation survival data that begins at age 4 or 5, we fitted Weibull and C&J survival models and our Model 1a after eliminating all data prior to age 4. All three models easily achieved convergence using the age 4–14 survival data. Results revealed that all three models fit well, but the C&J model has a more favorable fit index (0.9842), albeit negligible, than the Weibull (0.9834) and Model 1a (0.9834). Our motive for fitting these models to the age 4–14 data was not to determine which model fit the data better but to establish which model would more accurately predict stand survival and corresponding hazards for ages 1–2, 2–3, and 3–4. The predicted survival curves and corresponding hazard curves for these models, using $N_1 = TPH = 1,600$ (approximately the average density for our data), for the ages 2–14 reveal that Model 1a extrapolated extremely well for these data (Figure 4). Model 1a extrapolation prediction performance is demonstrated by comparing its predicted age 1–2, 2–3, and 3–4 hazards with the empirical hazards. In contrast, the Weibull and C&J models extrapolated poorly when predicting the age 1–2 and 2–3 hazards using our data.
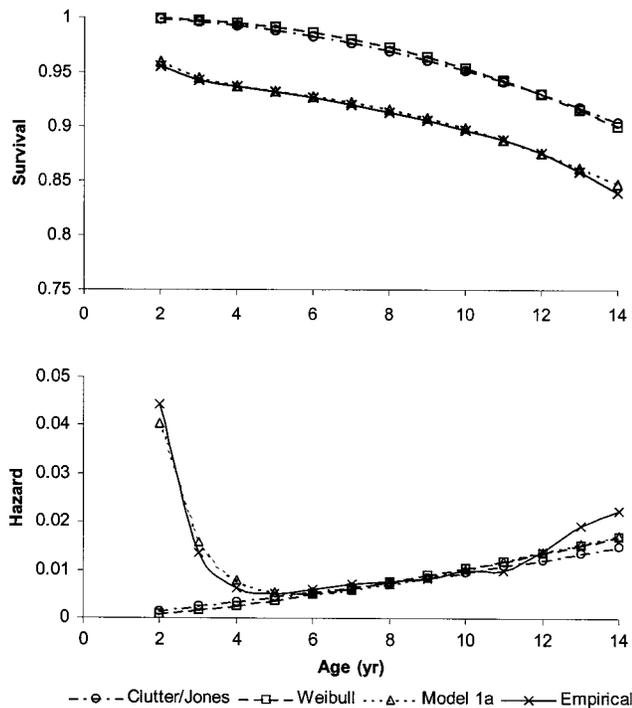

Figure 4. The empirical, Model 1a, C&J, and Weibull predicted survival and hazard functions. The models were fitted to all plots using age 4–14 data.

Discussion

Whole-stand survival models capable of describing complex underlying hazard functions were developed and demonstrated to provide biologically reasonable predictions. In addition, our empirical hazard curves illustrated that nonproportionality exists among the treatments, which was modeled by including, where appropriate, treatment-specific parameters. Our modeling method is based on the premise that $f(t)$ should be flexible enough to model the hazard function trend exhibited by the data.

It is easily demonstrated that functional forms such as $f(t) = at^b$ (i.e., simple linear or nonlinear functions of time) are incapable of modeling complex hazard functions. These types of functions can include additional covariates such as site index or stand density (e.g., for site index we could define the function as $f(t) = (SI) at^b$). However, additional covariates in simple linear or nonlinear models are not necessarily enough to model complex-shaped hazard functions in whole-stand survival models. To model complex hazard functions, the additional covariates would have to enter the function in such a way that the survival function is capable of modeling more than one inflection point, which is not easily accomplished. In contrast, $f(t)$ can be modeled as the addition of two (or more) linear/nonlinear functions that will produce bathtub or more complex hazard curves. Furthermore, the functional form does not need numerous parameters to model complex hazard functions (e.g., Model 1a has only three parameters). Models 2a and 2b demonstrate that placing a different assumption on the generalized differential equation using the same $f(t)$ function as Model 1a can produce more flexible hazard curves. Nevertheless, Model 2b provided only slightly more flexible solutions and has an extrapolation cost when fitted to our data. Model 2b extrapolation cost is likely to be reduced or eliminated as more data from older plots become available. Presently, within the data range, Models 1b and 2b are acceptable, but outside the data range, Model 1b is preferred. Our method for developing whole-stand survival models based on the empirical hazard function is relatively straightforward and can model complex hazard curves. In contrast, traditional whole-stand survival models generally lack the flexibility of our methodology and consequently only model more restrictive hazard curves.

We demonstrated the extrapolative power that can result when using the empirical hazard function to model whole-stand survival. Hazard function shape can be determined using empirical evidence if the data covers a wide enough spectrum or from *a priori* knowledge. Extrapolative power was demonstrated by comparing the fit and early stand predicted survival using Model 1a with the Weibull and C&J survival models when fitted to the age 4–14 data. Although the fit was improved marginally using the C&J, the extrapolation predictions for the ages 1–2, 2–3, and 3–4 survival and hazards are substantially improved using Model 1a. Model 1a was developed to model bathtub-shaped hazard functions and may be predisposed to this shape for our data even though it is capable of modeling

other hazard shapes. However, note that the empirical survival curve appears to have an inflection point at approximately age 5 (Figure 3). We estimated the slope between each successive age group using the empirical survival curve and noted a slope close to 0 at age 5; hence, there is an inflection point located at about age 5. Before this inflection point the curve is convex, and after the inflection point it is concave. If we do not have empirical data before this time period, we can use our *a priori* knowledge to determine the basic shape of the hazard during the early stand life. Note that modeling complex hazard functions implies the survival curve will have multiple inflection points. Our Model 1a is capable of modeling multiple inflection points, whereas the Weibull is incapable of modeling multiple inflection points and C&J is not flexible enough.

We demonstrated that a whole-stand survival model may provide adequate future extrapolation predictions but may not behave reasonably biologically for early survival extrapolation predictions because its hazard function is more restrictive. Hence, extrapolating backwards and forward (if the data set warrants) is equally important in establishing if a whole-stand survival model has biologically reasonable behavior. Therefore, computing and plotting the empirical hazard function can aid in establishing the complexity of the function that may be necessary to adequately model whole-stand survival.

Conclusion

Modeling whole-stand forest survival is enhanced when using the empirical hazard function behavior to aid in selecting an appropriate survival function. Use of the hazard function to aid in forestry survival model selection is not novel. Preisler and Slaughter (1997) demonstrated they could limit their individual-tree survival model selection to a model that was capable of reflecting the empirical hazard function behavior. Our study demonstrates that improved whole-stand survival models may result by considering the underlying empirical hazard function. We demonstrated this by using the empirical hazard function to limit our selection to an appropriate function that could model a bathtub-shaped hazard function. Survival analysis literature commonly refers to a U-shaped hazard function as bathtub shaped, and we have used the convention. However, if we imagine the hazard function curve for our data from age 1–75 years, the first 20–30 years or so may appear bathtub shaped, but the hazard function for the entire 75 years is likely to be ladle shaped. Therefore, it is important to understand biologically reasonable hazard function behavior outside the data range if the model is to be used for extrapolation. Although Models 1b and 2b consist of 10 and 11 parameters, respectively, it is not the number of parameters that allows the models to reflect an underlying bathtub-shaped hazard function. Our models, when fitted to the data without cultural treatment parameters, still exhibit the underlying bathtub hazard function behavior. Cultural treatment parameters allow additional model flexibility, but it is the $f(t)$ function that allows our model to exhibit a bathtub-

shaped hazard trend if the data warrants. Moreover, additional covariates in the X or $f(t)$ functions of the difference equation may provide more flexibility in the solutions. However, flexibility gained from additional covariates will not necessarily approach the flexibility gained from the $f(t)$ function.

The lifespan for plantation whole-stand survival is commonly modeled using a system of equations and/or tables, which often creates a cumbersome and difficult system to implement. Here we demonstrated that one equation could adequately model the lifespan for whole-stand survival when derived from knowledge gained through the empirical hazard function. Our method is relatively easy to implement and can model a whole-stand survival curve that exhibits complex hazard function behavior.

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