Title: Evaluation of mixed-effects models for predicting Douglas-fir mortality Jeremiah D. Groom¹ Department of Forest Engineering Resources & Management **Oregon State University** Corvallis, Oregon 97331 jgroom@odf.state.or.us David W. Hann Department of Forest Engineering, Resources, and Management Oregon State University Corvallis, Oregon 97331 david.hann@oregonstate.edu Hailemariam Temesgen Department of Forest Engineering, Resources, and Management Oregon State University Corvallis, Oregon 97331 hailemariam.temesgen@oregonstate.edu ¹Present address: Oregon Department of Forestry 2600 State Street Salem, Oregon 97310 Phone: 503-945-7394 Fax: 503-945-7490

36 ABSTRACT

37 We examined the performance of several generalized linear fixed- and mixed-effects individualtree mortality models for Douglas-fir stands in the Pacific Northwest. The mixed-effects models 38 39 accounted for sampling and study design overdispersion. Inclusion of a random intercept term 40 reduced model bias by 88% relative to the fixed-effects model; however, model discrimination 41 did not substantially differ. An uninformed version of the mixed model that used only its fixed 42 effects parameters produced predicted mortality values that exceeded the fixed-effects model 43 bias by 31%. Overall, we did not find compelling evidence to suggest that the mixed models fit our data better than the fixed-effects model. In particular, the mixed models produced fixed-44 effects parameter estimates that predicted unreasonably high mortality rates for trees 45 approaching 1 m in diameter at breast height. 46 47 48 49 50

52 INTRODUCTION

Tree mortality is a critical component of stand growth and yield models. It is also highly
variable and difficult to predict (Lee, 1971; Dobbertin and Biging, 1998). The nature of data
collected to model and quantify mortality, however, may challenge the assumptions inherent in
statistical tools used to estimate mortality. In this study we examine a generalized linear mixedeffects method to account for data structure and lack of independence.

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Lee (1971) and Staebler (1953) described tree mortality as either regular or irregular. Irregular 59 mortality includes death occurring from insects, disease, fire, snow damage, and wind. This type 60 61 of mortality typically is episodic, brief, and difficult to predict. Regular mortality is more predictable, and includes influences such as competition for light, moisture, and nutrients. As 62 stands become more crowded, a degree of mortality usually occurs. Trees may die for several 63 64 possibly co-occurring reasons: suppression where stands are differentiating, weakening due to insects and disease, and buckling where stems become tall and thin (Oliver and Larson, 1996). 65 Trees in stands characterized by regular mortality exhibit a preponderance of mortality amongst 66 smaller-diameter individuals that are over-topped by neighbors (Peet and Christensen, 1987). 67 Mortality rates become low for established trees until larger diameters are reached and the 68 69 mortality rate increases again (Buchman et al., 1983; Harcombe, 1987; Monserud and Sterba, 1999). Although both classes of mortality may affect stands, only single-tree regular mortality 70 71 models are routinely incorporated in most growth and yield simulators such as FVS (Dixon, 72 2011) and ORGANON (Hann, 2011).

74 Single-tree mortality models have been developed using a variety of data and approaches. Logistic models are common for data sets where revisit frequency consists of equal-length time 75 periods (Hamilton, 1986; Bigler and Bugmann, 2003; Jutras et al., 2003; Moore et al., 2004; 76 Adame et al., 2010). However, if the time periods differ, a common solution is to use the logistic 77 78 model but insert time as a power upon survival probabilities or use a complimentary log-log link 79 function (e.g., Monserud, 1976; Eid and Tuhus, 2001; Moore et al., 2004; Temesgen and Mitchell, 2005; Fortin et al., 2008). For stands where remeasurement occurred multiple times, 80 researchers either avoid pseudoreplication at the level of the tree by omitting all but the last 81 82 remeasurement for each tree (Hamilton, 1986) or include the remeasurement information (Temesgen and Mitchell, 2005; Fortin et al., 2008). 83

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Data used in these analyses are from nested samples, with the highest level referred to as 85 installations. Each installation contains one or more plots; each plot contains many trees with 86 87 repeated measurements. Analyses performed on individual tree mortality data has recently begun to account for the structured nature and non-independence by using generalized linear 88 mixed-effects models. Logistic models by Adame et al. (2010) and Jutras et al. (2003) include 89 90 random intercepts for study plots or stands. A complimentary log-log model by Fortin et al. (2008) included an adjusted intercept with random effects for study plot and specific time 91 92 interval nested within plot.

93

94 Prediction performance for nonlinear mixed-effects models may be improved (less bias and 95 greater precision) when compared to corresponding fixed-effects models conditional on the 96 availability of previous information on the subject; however, in absence of random-effects

97 information, predictions using just the fixed portions of the parameterization from the nonlinear

98 mixed-effects model exhibit greater bias and less precision than even the original fixed-effects

99 model (Monleon, 2003; Temesgen et al., 2008; Garber et al., 2009). Setting the random effect to

200 zero follows from prediction theory only for linear mixed models, but it has a different meaning

101 for nonlinear models. Consider a linear mixed model where X is a $(n \times p)$ design matrix where n

102 is the number of observations and p is the number of fixed-effects parameters, β is a vector of

103 linear slope values, Z is a $(n \times r)$ design matrix where r is the number of random effects

parameters, γ represents G-sided random effects parameterization, and ε is the random error:

105
$$y = X\beta + Z\gamma + \varepsilon$$
, where $E(\gamma) = E(\varepsilon) = 0$

106 Then, conditional on the random effect, and because the expectation is a linear operator,

107 $E(\mathbf{y} \mid \boldsymbol{\gamma}) = X\boldsymbol{\beta} + Z\boldsymbol{\gamma}$

108 Unconditionally,

109 $E(y) = E(X\beta + Z\gamma + \varepsilon) = X\beta + ZE(\gamma) = X\beta$

110 Thus, in a linear model, the unconditional expectation can be calculated from the conditional

- 111 expectation by setting the random effect to zero:
- **112** E(y) = E(y | y = 0)
- 113
- 114 For a nonlinear model, this is not the case. The nonlinear mixed model can be written as:
- 115 $y = f(X, \beta, Z, \gamma) + \varepsilon$, where $E(\gamma) = E(\varepsilon) = 0$.
- 116 Conditional on installation:

117 $E(\mathbf{y} \mid \boldsymbol{\gamma}) = \mathbf{f}(X, \boldsymbol{\beta}, \boldsymbol{Z}, \boldsymbol{\gamma})$

- 118 Unconditionally:
- 119 $E(\mathbf{y}) = E[E(\mathbf{y} \mid \boldsymbol{\gamma})] = E[f(X, \boldsymbol{\beta}, \boldsymbol{Z}, \boldsymbol{\gamma})]$

Unlike linear models, for nonlinear models, the unconditional model is not the same as theconditional model with the random effects set to zero:

122 $E(y) \neq E(y \mid \gamma = 0)$ because $E[f(X, \beta, Z, \gamma)] = \int f(X, \beta, Z, \gamma) d\mu(\gamma) \neq f(X, \beta, Z, \gamma = 0)$, where $\mu(\gamma)$ is

- 123 the distribution function of γ .
- 124

125 The model for E(y) is known as the population-average model and the model for E(y | y) is known as the subject-specific model. For nonlinear mixed models, those versions are different. 126 Choosing which type of model and inference is appropriate for each objective is fundamental 127 128 when dealing with nonlinear mixed models. For a tree from a completely new stand that does not have information to estimate the random effects and, therefore, condition on the stand effect, 129 the proper model is a population average model. When using the subject-specific model with $\gamma =$ 130 0 (i.e., the subject-specific model for the average stand), prediction performance is expected to 131 decline. Again, in linear mixed models this is not an issue, because setting $\gamma = 0$ yields the 132 population-average model. 133

134

Forest management requires models that are useful beyond their study areas. Generalized or 135 136 nonlinear mixed-effects models can increase bias when applied to novel data (e.g., Robinson and Wykoff, 2004). Mixed models require estimated information about a hierarchical level that may 137 138 be unknown for novel data sets. One technique to extend generalized linear or nonlinear mixed-139 effect model applicability is to utilize minimal data from new stands for estimating the random effects parameters. This allows the application of nonlinear mixed effects models beyond their 140 141 original data frames (Monleon, 2003; Temesgen et al., 2008; Garber et al., 2009). However, this 142 technique may be limited by the response variable type. In those studies it worked for tree

height, a continuous static variable. Our study's response variable, individual tree mortality, is
rare, binomial, dynamic, and requires several years of data collection to observe. Thus,
incorporating subsample information from new plots to inform mixed-effects model predictions
is generally unfeasible.

147

148 The objectives of this study are to 1) determine whether a generalized linear mixed model fit to repeatedly remeasured Douglas-fir (Pseudotsuga menziesii [Mirb.]) trees can improve mortality 149 estimation over a previous nonlinear estimation approach (Hann et al., 2003; Hann et al., 2006), 150 151 and 2) compare the predictive abilities of mixed-effects models to nonlinear least squares estimation in the presence and absence of random effects information. We expect biased 152 predictions from the mixed model that lacks random effects information, but examine the degree 153 by which those results are useful relative to the nonlinear least squares predictions. Taken 154 together, our goal is to examine how well models met our objectives and whether we produce a 155 156 model that is useful for current Douglas-fir growth and yield simulators.

157

158 METHODS

159 Study Area and Data Acquisition

Data used in this analysis were obtained from randomly located installations on nine land ownerships and represent a subset of data described in Hann et al. (2003; 2006). One of the uses of the overall data collection effort was to calibrate the ORGANON stand development model (Hann, 2011) for intensively managed Douglas-fir in the Pacific Northwest region of the USA and Canada. What follows is a description of the subsetted data. The data were from 304 permanent sample installations from Southwest British Columbia, Western Washington, and

166	Northwestern Oregon. The 820 plots within those installations contained 195,795 revisit data
167	collected from 70,720 Douglas-fir trees. Trees were revisited one to 18 times over the course of
168	data collection. Time between revisits was not equal among trees or plots, and varied from 3 to 7
169	years (median = 5 years). The fixed-area plots varied in size from 0.041 to 0.486 ha
170	(mean=0.069). The average breast height age was 27.8 years and ranged from 3 to 108 years.
171	Plots included in this study were not subject to thinning or fertilization experimental treatments.
172	
173	We further reduced the data set according to two criteria. The first criterion only permitted data
174	from installations that had two or more plots. This criterion was necessary for creating mixed-
175	effects mortality predictions (described below), and it removed 12,616 trees, 38,314
176	observations, and 67 single-plot installations from the data set. The second criterion was that we
177	retained only trees with $DBH < 101.6$ cm. We removed larger-DBH trees to allay model
178	convergence issues likely arising from a paucity of mortality information leading to a lack of fit
179	at that extreme. This removed eight observations and five trees ($<0.01\%$ of data) and permitted
180	model convergence. The resulting data set included 157,473 revisits of 58,099 trees in 753 plots
181	located within 201 installations.

183 Mortality estimation

184 We based this analysis on a general equation of mortality given differing plot revisit schedules as185 described by Hann et al. (2006):

187 [1]
$$PM = 1.0 - [1.0 + e^{-(X\beta)}]^{-PLEN} + \varepsilon_{PM}$$

189 Where PLEN is the length of the growth period in 5-year increments (i.e., length of a growth 190 period in years divided by 5), PM is the 5-year mortality rate, and ε_{PM} is the random error on 191 PM. The response variable distribution is $y \sim Bernoulli(\pi)$ where the observed response was y192 and π is the corresponding response probability. Several different parameterizations have been 193 examined for $X\beta$. Hann et al. (2006) modeled $X\beta$ as:

194

195 [2]
$$X\beta = \beta_0 + \beta_1 DBH + \beta_2 CR + \beta_3 BAL + \beta_4 DFSI$$

196

The variable DBH is diameter at breast height (cm) at 1.3 m, CR is tree crown ratio, $BAL(m^2/ha)$ 197 is basal area per ha in trees with diameters larger than that of the subject tree on the plot, and 198 DFSI is the Douglas-fir site index (Hann and Scrivani, 1987) in meters. We examined the 199 predictive ability of this model in three ways. We wished to investigate whether the mixed-200 effects approach would provide a reasonable mortality prediction for older trees, so we included 201 the square of DBH (DBH²) as a predictor variable (e.g., Monserud and Sterba, 1999; Hann and 202 203 Hanus, 2001). CR was subsampled on many of the plots in the modeling data set and would require the imputation of the missing values if used in a mortality equation. This would introduce 204 prediction error issues which we decided to avoid by removing CR from the analysis. We 205 206 retained BAL to represent competition experienced by an individual tree (Wykoff et al., 1982; 207 Wykoff, 1986; Temesgen and Mitchell, 2005). The parameterization we used in this analysis 208 was:

209

210 [3]
$$X\beta = \beta_0 + \beta_1 DBH + \beta_2 DBH^2 + \beta_3 BAL + \beta_4 DFSI$$

212 We present a generalized linear fit of this model, fit via a maximum likelihood estimator (PROC GLIMMIX, SAS Inc. 2008). This model produced results identical to those from the nonlinear 213 approach employed by Hann et al. (2006) to estimate tree mortality. We refer to this model as 214 NLS given its equality to the original procedure. We also examined two generalized linear 215 models with the same parameterization as [3]. One corrected for model overdispersion by 216 217 altering the model variance. The other corrected for overdispersion and included a random effect term for the model intercept grouped by installation. We selected installation as a grouping level 218 instead of plot due to our desire to validate models using a leave-one-out approach (described 219 220 below). We refer to these models as GXR and GXME respectively.

221

We constructed GXR and GXME using the generalized linear mixed-model procedure Proc 222 GLIMMIX (SAS Institute Inc. 2008). The procedure made use of a pseudo-likelihood estimator 223 instead of a maximum likelihood estimator due to the presence of R-sided mixed effects 224 (Schabenberger, 2007). The advantages of GLIMMIX over other SAS procedures (e.g., Proc 225 NLMIXED) included the ability to incorporate more than one random effect into the model (G-226 sided random effect) and to include a multiplicative overdispersion parameter (R-sided random 227 228 effects). A disadvantage of GLIMMIX is that its pseudo-likelihood estimator may produce biased estimates in certain contexts (Breslow and Lin, 1995). The main structural difference 229 230 between the marginal (fixed-effects or population-averaged; i.e., NLS, GXR) and the mixed-231 effects model GXME is the incorporation of the G-sided random effects terms $Z\gamma$ into the mixed-effects model structure: 232

233

234 [4]
$$PM = 1.0 - [1.0 + e^{-(X\beta + Z\gamma)}]^{-PLEN} + \varepsilon_{PM}$$

The $Z\gamma$ term alters the model linear predictors. We created a model with an installation grouped random intercept by structuring the linear predictors of our model as:

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239 [5]
$$\eta = X\beta + Z\gamma = \beta_0 + b_i + \beta_1 DBH_{ij} + \beta_2 DBH^2_{ij} + \beta_3 BAL_{ij} + \beta_4 DFSI_{ij}$$

240

The linear predictors included a population-level intercept β_0 , a deviation from that intercept of amount *b* for installation *i*, and the remaining parameter estimates for observations *j* in installations *i*. The modified logit function is:

244
$$\pi_{ij} = f(\eta) = 1 - \{1 + exp(-[\eta])\}^{-PLEN}$$

 $b_i \sim N(0, \theta_b^2)$

246 [6]
$$Var(y_{ij}|\pi_{ij}) = \pi_{ij}(1-\pi_{ij})$$

247 In GLIMMIX, the variance of observations, conditional on the random effects, is: 248 $var[Y|\gamma] = A^{1/2}RA^{1/2}$

249 The diagonal matrix A contains the variance functions of the model (i.e., equation [6]) and

expresses the variance function for the i^{th} observation (Littell et al., 2006, p. 535). G-sided

251 random effects will therefore affect the values for A. The random effects matrix $R = \phi I$ where I

is an identity matrix and ϕ is a dispersion scale parameter. In binomial models where there is no

- overdispersion, $\phi = 1$. However, if data are overdispersed, the variances can be accordingly
- increased by changing this parameter. We tested for model overdispersion using the Pearson's
- statistic (Littell et al., 2006). We additionally weighted our tree remeasurement data by their

respective plot sizes (Flewelling and Monserud, 2002). Model weighting is accomplished by calculating ϕ/w_i , where w is the weight associated with observation *i*. To summarize, GXME was constructed in PROC GLIMMIX with linear mixed- and fixed-effects predictors from [5] used in the nonlinear equation [4]. A random intercept was estimated by installation and we included an R-sided random effect to account for overdispersion. Observations for the model were weighted by plot size.

262

A difficulty with using the estimates for GXME to predict mortality for trees that are not part of a current installation is that no hierarchical parameter values for that installation would be available. The random effects parameters remain uninformed. We explored the utility of applying the uninformed mixed model by examining the predictive ability of an additional model, GXFE. This model incorporates the fixed-effects parameter estimates from GXME but discards its random effects parameterization.

269

We validated models NLS, GXME, and GXFE using a leave-one-out approach. GXR was 270 excluded as model validation relies on parameter point estimates and its parameter point 271 estimates (not error) should be identical to those for NLS. In this instance we repeatedly fit 272 273 models to subsets of the data. Each subset included all but one of the plots (model set). The resulting model was used to predict the response of each of the excluded sites' observations 274 (prediction set). In order to facilitate inclusion of models that relied on random effects at the 275 level of installations, we reduced the data set to include only installations with two or more plots. 276 277 With one plot excluded, the model was still able to estimate a random effect for that installation. 278

We used model estimates from the model data set to produce residual values for the validation
set. We used the Hosmer-Lemeshow test to determine model goodness-of-fit (Hosmer and
Lemeshow, 2000) and compared model discrimination by using receiver operating characteristic
(ROC) curve analysis and examining the area under the ROC curves. We examined model and
bias for the overall validation data set and for different values of BAL, DFSI, and DBH. We
calculated mean bias using the following equation:

285 [8]
$$Bias = \frac{\Sigma(y_j - \hat{\pi}_{ij})}{n}$$

The symbol y_j is a single mortality observation (1 or 0), $\hat{\pi}_{ij}$ is the fitted value, and *n* is the number of observations.

288

289 RESULTS

The data set included the mortality of 9982 trees (6.3% of total). Deaths appeared to be skewed towards smaller DBH categories while mortality appeared to increase at higher BAL volumes, indicating that trees may have been more likely to perish if the stand typically had more trees with basal area greater than the tree in question (Figure 1).

294

295 Model coefficients for the three models were estimated from the full sample data set (Table 1).

296 The inclusion of R-sided random effects variables reduced overdispersion. The Pearson's

statistic for the condition distribution for the NLS model was 10.88, substantially different from

- a value of 1. The Pearson's statistics for GXR and GXME were 1.00 indicating that the
- 299 inclusion of the R-sided or R- and G-sided random effects corrected for the overdispersion. As a
- 300 consequence, GXR fixed-effects parameter standard errors were greater than NLS standard

errors. A difference among models was the parameter values for DBH², which increased by 60%
when comparing NLS to GXME.

303

Predicted values generated from the mixed-effects model with random variables improved bias compared to the nonlinear model. However, the mixed model's bias suffered when only its fixed effects were considered (Table 2). On average, GXME, with random effects and overdispersion terms, exhibited a bias that was 22% the values of model NLS. Model GXFE's bias was 4 times greater than the value of NLS.

309

The area under the ROC curve was 2.3% higher for GXME than for NLS or GXFE, indicating 310 that the mixed model exhibited a slightly greater degree of model discrimination. The values for 311 312 NLS and GXFE were nearly identical. The Hosmer-Lemeshow goodness-of-fit test statistics were significant (df = 8, p<0.001) for all models considered, indicating that no models fit data at 313 an acceptable level (e.g., $\chi^2 \le 15.5$). Pearson's residuals increased with DBH > 20 cm and BAL 314 $< 40 \text{ m}^2/\text{ha}$; a pattern did not appear evident between residuals and DFSI. Among the models, 315 GXFE's score was substantially higher than either NLS or GXME, and NLS had the lowest score 316 of the three. Pearson's correlations among variables was highest between DBH and DBH² 317 (0.935), the next highest was between DBH² and DFSI (0.191). 318

319

Bias was generally lowest for model GXME across all values of all predictor variables with a
few close exceptions (Figure 2). Values and patterns of bias were similar for NLS and GXFE
across variables, although the bias values for GXFE were generally but not always more
extreme. In particular, bias for GXFE was more than twice as great as other models at DBH <

20 cm. Comparisons of observed and predicted values of mortality (Figure 3) demonstrate the
generally closer fit of the mixed model predicted values to observed mortality. Relative to
GXFE, NLS better predicts tree mortality at DBH values < 20 cm and is fairly equivalent at other
DBH values. NLS mortality predictions were closer to observed values at all BAL categories
except 50-59 m. NLS also outperformed GXFE at four of the six DFSI categories (not including
30-34m and > 45m).

330

We compared predicted model performance to observed values to determine where model 331 shortcomings were (Figure 4). Of note, GXME appeared to best match observed mortality at 332 DBH values < 20 cm while the other models generally underpredicted tree mortality. However, 333 all models except for NLS predicted a dramatic increase in mortality beyond 90 cm DBH. The 334 20% observed mortality at 97 cm DBH represented one of five trees of that size class perishing. 335 We examined fixed-effects parameter values for GXME for trees with DBH < 90 cm to 336 determine if this mortality was exhibiting a strong influence on DBH² and found that results 337 were virtually unchanged. 338

339

340 DISCUSSION

We report partial success at meeting our study objectives. The mixed-effect models accounted for overdispersion in the data and accordingly increased parameter standard errors. The mixedeffects model GXME additionally reduced prediction bias relative to NLS. However, the predicted fits at observed parameter values were of concern; the DBH² parameter of the mixedeffects model GXME and its related models predicted an unreasonably high mortality rate for trees with DBH > 90 cm. The larger-DBH predictions for NLS were more reasonable. The

GXME model appeared to best fit the data at DBH < 40 cm, a range that included the bulk of ourdata.

349

The inclusion of R-sided random effects assisted in reducing model overdispersion. Although unreported, the estimated standard errors of parameter estimates resulting from earlier analyses such as Temesgen and Mitchell (2005) and Hann et al. (2003; 2006) would have been too small. For those authors the models were used in validation trials so the means, not standard errors, affected validation outcomes. The increase in error terms could indicate that previouslysupported parameters were not contributing to the model, although all of our parameters remained supported in all models.

357

Once we included a random intercept in the model along with an R-sided random effect, the term 358 for DBH² increased markedly. Bias for the mixed-effects model was improved relative to the 359 marginal model. However, when we examined predicted fits for the mixed model's fixed-effects 360 parameters without taking into account the individual installation information (random intercept) 361 the bias increased to an amount four times greater than the marginal model. Clearly, it would be 362 363 difficult to justify this model's use. This finding is similar to results reported by several other authors (Monleon 2003; Temesgen et al. 2008; Garber et al. 2009), and confirms our expectation 364 that this would be the case. 365

366

Other authors provide examples of studies in which mixed models produce an improvement in predictive ability, and minimal data collection allowed for an application of the mixed models to novel stands (Monleon, 2003; Temesgen et al., 2008; Garber et al., 2009). Obtaining ancillary

mortality data to estimate random effects is prohibitively difficult. Given the modest
improvements in prediction from the G-sided mixed model, the anticipated poor performance of
the uninformed mixed model, and our lack of ability to apply the mixed model to novel stands,
we find no advantage here with utilizing the generalized linear mixed-effects models for
predicting Douglas-fir mortality.

375

Our issues with model bias when fixed-effects parameter estimates were extracted from the generalized mixed model indicate a problem with our application, not a problem with the model. We wished to obtain a finding we could generalize between subjects when the mixed models were best able to generalize results within subjects. We imagine that if we desired inference to additional plots within installations, our mixed model would have proven more useful than the marginal model.

382

All of our models examined failed the goodness-of-fit test; it appears this may be in part due to results for larger-diameter trees that were among the largest trees in a stand. We interpret this to indicate that our model did not fit mortality data well at these larger ranges where we had a relative paucity of data. Other possible contributing issues include overfitting the model or providing insufficient fixed-effects parameters. Among models, the goodness-of-fit scores were lowest for GXME with GXFE a distant third.

389

Across models, bias was highest at low DBH and high BAL values (both well-represented in the data set). With DFSI, bias was high for the smallest category which corresponded with few data relative to other categories. Bias patterns differed across models as well. GXME tended to

exhibit a different and reduced pattern of bias across all three predictor variable categories. The
models that were not incorporating installation-specific effects into their estimates tended to
behave similarly with model GXFE frequently providing the most extreme bias per variable
category.

397

The intensity of the effect DBH² had on mortality prediction at greater DBH values surprised us. 398 Although our predicted U-shaped mortality curve is in spirit similar to that discussed by 399 Harcombe (1987) and found by Monserud and Sterba (1999) for Norway Spruce and Hann and 400 401 Hanus (2001) for Douglas-fir, grand fir, white fir, incense-cedar, ponderosa pine, and California black oak, only the predicted mortality for large DBH values from the model NLS appeared 402 reasonable. The mixed-effects based models predicted mortality rates at 95 cm DBH that are 403 simply too extreme; if those estimates were real, old-growth (> 180 year) Douglas fir stands 404 would not exist. However, the models, particularly GXME, did appear to predict observed 405 mortality for trees <80 cm DBH. GXFE appeared most severely underpredict the 5-year 406 mortality rate. 407

408

409 CONCLUSION

Our generalized linear mixed model of Douglas-fir mortality did not outperform a similar model lacking mixed effects. In particular, the incorporation of mixed effects resulted in alterations to fixed effects that produced unreasonably high mortality rates for trees approaching 1 m in diameter. The practical application of predicting mortality rates for novel stands did not improve with the utilization of a mixed model. We believe this will generally be the case for tree mortality estimation when random effects information is unavailable, a condition that should be

416	common. The correction for model overdispersion was appropriate and represented an
417	improvement in parameter variance estimation, but overall we cannot recommend the mixed
418	model as a suitable replacement for the original model form.
419	
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425	
426	LITERATURE CITED
427	Adame, P., del Río, M., Cañellas, I. 2010. Modeling individual-tree mortality in Pyrenean oak
428	(Quercus pyrenaica Willd.) stands. Ann. For. Sci. 67: 810, 10 pp.
429	
430	Bigler, C., Bugmann, H. 2003. Growth-dependent tree mortality models based on tree rings.
431	Can. J. For. Res. 33: 210-221. doi: 10.1139/X02-180.
432	
433	Breslow, N.E., Lin, X. 1995. Bias correction in generalised linear mixed models with a single
434	component of dispersion. Biometrika 82: 81-91.
435	
436	Buchman, R.G., Pederson, S.P., Walters, N.R. 1983. A tree survival model with application to
437	species of the Great Lakes region. Can. J. For. Res. 13:601-608.
438	

439	Dixon, G.E. 2011. Essential FVS: A user's guide to the Forest Vegetation Simulator. United
440	States Department of Agriculture Forest Service, Forest Management Service Center,
441	Fort Collins, CO. Available from:
442	http://www.fs.fed.us/fmsc/ftp/fvs/docs/gtr/EssentialFVS.pdf [Accessed 10 March 2012].
443	
444	Dobbertin, M., Biging, G.S. 1998. Using the non-parametric classifier CART to model forest
445	tree mortality. For. Sci. 44(4): 507-516.
446	
447	Eid, T., Tuhus, E. 2001. Models for individual tree mortality in Norway. For. Ecol. Manage.
448	154 :69-84.
449	
450	Flewelling, J.A., Monserud, R.A. 2002. Comparing methods for modeling tree mortality.
451	USDA For. Serv. Proc. RMRS-P-25: 168-177.
452	
453	Fortin, M., Bédard, S., DeBlois, J., Meunier, S. 2008. Predicting individual tree mortality in
454	northern hardwood stands under uneven-aged management in southern Québec, Canada.
455	Ann. For. Sci. 65: 205-217.
456	
457	Garber, S. M., Temesgen, H., Monleon, V.J., Hann, D.W. 2009. Effects of height imputation
458	strategies on stand volume estimation. Can. J. For. Res. 39: 681-690. doi: 10.1139/X08-
459	188.

461	Harcombe, P.A. 1987. Tree life tables: simple birth, growth, and death data encapsulate life
462	histories and ecological roles. Bioscience 37(8): 557-568.
463	
464	Hamilton, D.A., Jr. 1986. A logistic model of mortality in thinned and unthinned mixed conifer
465	stands of Northern Idaho. For. Sci. 32(4): 989-1000.
466	
467	Hann, D.W. 2011. ORGANON User's Manual, Edition 9.1. Department of Forest Resources,
468	Oregon State University, Oregon. Available from
469	http://www.cof.orst.edu/cof/fr/research/organon/downld.htm#man [accessed 10 March
470	2012].
471	
472	Hann, D.W., Hanus, M.L. 2001. Enhanced mortality equations for trees in the mixed conifer one
473	of southwest Oregon. Research Contribution 34, Forest Research Laboratory, Oregon
474	State University, Corvallis. P. 34.
475	Hann, D.W., Scrivani, J.A. 1987. Dominant height-growth and site-index equations for Douglas-
476	fir and ponderosa pine in southwest Oregon. Research Bulletin 59, Forest Research
477	Laboratory, Oregon State University, Corvallis.
478	
479	Hann, D.W., Marshall, D.D., Hanus, M.L. 2003. Equations for predicting height-to-crown-base,
480	5-year diameter-growth rate, 5-year height-growth rate, 5-year mortality rate, and
481	maximum size-density trajectory for Douglas-fir and western hemlock in the coastal
482	region of the Pacific Northwest. Research Contribution 40, Forest Research Laboratory,
483	Oregon State University, Corvallis. P. 53.

485	Hann, D.W., D.D. Marshall, M.L. Hanus. 2006. Reanalysis of the SMC-ORGANON equations						
486	for diameter-growth rate, height-growth rate, and mortality rate of Douglas-fir. Research						
487	Contribution 49, Forest Research Laboratory, Oregon State University, Corvallis.						
488							
489	Hosmer, D.W., Lemeshow, S. 2000. Applied Logistic Regression, 2 nd edition. John Wiley &						
490	Sons, Inc. 392 pp.						
491							
492	Jutras, S., Hökkä, H., Alenius, V., Salminen, H. 2003. Modeling mortality of individual trees in						
493	drained peatland sites in Finland. Silva Fennica 37(2): 235–251.						
494							
495	Lee, Y. 1971. Predicting mortality for even-aged stands of lodgepole pine. The Forestry						
496	Chronicle 47:29-32.						
497							
498	Littell, R.C., Milliken, G.A., Stroup, W.W., Wolfinger, R.D., Schabenberger, O. 2006. SAS®						
499	for Mixed Models, 2 nd Ed. Cary, NC. 840 pp.						
500							
501	Monleon, V.J. 2003. A hierarchical linear model for tree height prediction. Joint Statistical						
502	Meetings - Section on Statistics & the Environment: 2865-2869.						
503							
504	Monserud, R.A. 1976. Simulation of forest tree mortality. For. Sci. 22(4): 438-444.						
505							

506	Monserud, R.A., Sterba, H. 1999. Modeling individual tree mortality for Austrian forest species.
507	For. Ecol. Manage. 113: 109-123.
508	
509	Moore, J.A., Hamilton, D.A., Jr., Xiao, Y., Byrne, J. 2004. Bedrock type significantly affects
510	individual tree mortality for various conifers in the inland Northwest, U.S.A. Can. J. For.
511	Res. 34 : 31-42. doi: 10.1139/X03-196.
512	
513	Oliver, C.D., Larson, B.C. 1996. Forest Stand Dynamics, Updated Ed. John Wiley and Sons,
514	Inc. Page 227. 544 pp.
515	
516	Peet, R.K., Chriestensen, N.L. 1987. Competition and tree death: most trees die young in the
517	struggle for the forest's scarce resources. Bioscience 37(8): 586-594.
518	
519	Robinson, A.P., Wykoff, W.R. 2004. Imputing missing height measures using a mixed-effects
520	modeling strategy. Can. J. For. Res. 34: 2492-2500. doi: 10.1139/X04-137
521	
522	SAS Institute Inc. 2008. SAS/STAT® 9.2 User's Guide. Cary, NC: SAS Institute Inc.
523	
524	Schabenberger, O., 2007. Growing up fast: SAS® 9.2 enhancements to the GLIMMIX
525	procedure. SAS Global Forum 2007: Statistics and Data Analysis. Paper 177-2007.
526	

527	Staebler, G.R. 1953. Mortality estimation in fully stocked stands of young-growth Douglas-fir.
528	U.S. Forest Service, Pacific Northwest Forest and Range Experiment Station. Research
529	Paper 4. 8 pp.
530	
531	Temesgen, H., S.J. Mitchell. 2005. An individual-tree mortality model for complex stands of
532	southeastern British Columbia. West. J. Appl. For. 20(2): 101-109.
533	
534	Temesgen, H., Monleon, V.J., Hann, D.W. 2008. Analysis and comparison of nonlinear tree
535	height prediction strategies for Douglas-fir forests. Can. J. For. Res. 38: 553-565.
536	doi:10.1139/X07-104.
537	
538	Wykoff,W.R. 1986. Supplement to the User's Guide for the Stand Prognosis Model—Version
539	5.0. Gen. Tech. Rprt. INT-208. USDA For. Serv., Ogden, UT 36 p.
540	
541	Wykoff, W.R., N.L. Crookston, and Stage, A.R. 1982. User's guide to the Stand Prognosis
542	Model. Gen. Tech. Rprt. INT-133. USDA For. Serv., Ogden, UT. 112 p.
543	

Table 1. Fixed and random effects estimates and standard errors (SE) for the generalized linear
least squares models NLS, GXR, and GXME. The overdispersion parameter (Residual) indicates
the size of the underlying residual effect's variance and the standard error of that effect.

	NLS		GXR		GXME	
	Estimate	StdError	Estimate	StdError	Estimate	StdError
Fixed Effects						
Intercept	-4.5118	0.02807	-4.5118	0.09267	-5.0958	0.2891
DBH	-0.2105	0.00251	-0.2105	0.00829	-0.2719	0.00677
DBHSQ	0.00168	7.8E-05	0.00168	0.00026	0.00279	0.00017
BAL	0.00421	1.8E-05	0.00421	6.1E-05	0.00495	8.3E-05
DFSI	0.04897	0.00068	0.04897	0.00224	0.05996	0.00804
Random						
Effects						
Residual (Subje	ct = Tree)		10.884	0.03879	10.275	0.03665
Intercept (Subject = Installation)					0.6353	0.07953

557 Table 2: Comparisons of model performance at predicting the probability of tree mortality over a

558	five-year period	(PM ₅).	Comparisons	include model bia	as, area under the	ROC curve (AUC), a
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and the Hosmer-Lemeshow goodness-of-fit test statistic (H-L Test). Number of observations =

560 157,473.

Models	Bias (P _{5-year mort})	AUC	H-L Test
NLS	0.002643908	0.845	366.8
GXME	-0.000604775	0.864	388.8
GXFE	0.0110345	0.844	1505.6



Figure 1. Histograms of observations (live + dead) by variable name. The clear bars represent all data of a particular category; black
bars represent the number of dead observations.



573 Figure 2. Prediction bias associated with models NLS, GXME, and GXFE across the range of data values for DBH, BAL, and DFSI.



579 Figure 3. Five-year predicted and observed probability of mortality. Mortality probabilities are presented by diameter, BAL, and

⁵⁸⁰ DFSI classes.



Figure 4. Predicted mortality rates by DBH and average parameter values at specific DBHvalues.