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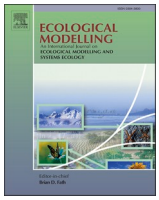

# A cellular necrosis process model for estimating conifer crown scorch

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ABSTRACT

Fire-caused tree mortality has major impacts on forest ecosystems. One primary cause of post-fire tree mortality in non-resprouting species is crown scorch, the percentage of foliage in a crown that is killed by heat. Despite its importance, the heat required to kill foliage is not well-understood. We used the "lag" model to describe time- and temperature-dependent leaf cell necrosis as a method of predicting leaf scorch. The lag model includes two rate parameters that describe 1) the process of cells accumulating non-lethal damage, and 2) damage becoming lethal to the cell. To parameterize models, we used a water bath to apply heat to newly expanded and one-year-old spring and autumn needles of *Pinus ponderosa* (ponderosa pine) and *Pseudotsuga menziesii* (Douglas-fir) at five temperatures (45, 50, 55, 60, and 65 °C), for 2 s to 2 h and 50 min. Electrolyte leakage measurements were used as indicators of percent cell survival. We fit the lag model to resulting survival curves and developed models of leaf scorch that can be applied across a range of temperatures and under fluctuating temperatures. Newly expanded foliage in spring was the most heat sensitive for both species examined. *P. menziesii* foliage sampled in spring was significantly more heat sensitive than foliage sampled in autumn, regardless of needle age. These findings indicate the importance of species, season, and age of foliage for crown scorch estimation. The models and methodologies developed in this study are directly applicable to fire effects models to improve precision of crown scorch estimates.

## 1. Introduction

Tree mortality is one of the primary processes by which fire influences plant communities and ecosystems (Bond et al., 2005). A main predictor of tree death in non-sprouting species is crown scorch, or foliage that is killed by heat but not consumed during a fire (Hood et al. 2018; JM Varner et al. 2021; Barker et al. 2022). Crown scorch is used as an input in most post-fire tree mortality models (Woolley et al., 2011; Fernandes et al. 2008). Despite its widely recognized importance, the heat required to scorch foliage is not well understood. A lethal temperature threshold of 60 °C is commonly used to estimate crown scorch, and the duration at which foliage must remain at this temperature (or exceed this temperature) to cause necrosis is assumed to range from instantaneous to 60 s (e.g. Van Wagner 1973; Michaletz and Johnson 2006, 2007; Bison et al. 2022). However, experimental studies have demonstrated that heat-induced leaf scorch depends on both the temperature of foliar tissue and the duration of time it remains at this temperature (Bigras 2000; Kelsey and Westlind 2017). A more comprehensive model of heat-induced leaf mortality is warranted to advance mechanistic understanding of fire-vegetation interactions and support improved predictions of fire effects to support fire management (Hiers et al. 2020).

The most commonly used crown scorch models for western US conifers were published by Van Wagner (1973). These models estimate a leaf mortality threshold ($T_L$) as 60 °C with support for this threshold attributed to Kayll (1968) and Methven (1971). However, even Kayll (1968) showed a time-temperature response of leaf mortality as opposed to a threshold response, with lower temperatures requiring longer durations of heating to kill foliage. In fact, no published study of crown scorch has found evidence of a threshold response, where foliage survives below a threshold and dies above the threshold. Despite numerous examples of time-temperature observations beginning in the earliest publications on the subject (e.g. Nelson 1952; Byram 1958; Hare 1961), no currently published process model combines temperature and duration at temperature to estimate crown scorch. Empirical models (e.g. Van Wagner 1973) circumvent the complex biological processes that drive crown scorch and generalize unsubstantiated thresholds across a range of species and conditions.

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Crown scorch occurs when foliage is heated to high enough temperatures for long enough to cause cell necrosis without the foliage igniting. Crown scorch is common following low- and moderate intensity surface fires (Michaletz and Johnson 2006). Traditional equations for crown scorch rely on fire behavior variables such as fire intensity (Kobziar et al. 2006) or flame height to estimate the height at which air temperature exceeds a $T_L$, indicating crown scorch height (Van Wagner 1973). These equations have been parameterized from experimental data and crown scorch height observations. While $T_L$ is qualitatively described as the temperature that kills foliage either instantaneously or after 60 s, neither the actual foliage temperatures nor the duration of heating is explicitly represented in these models. Instead, the temperature of canopy components as the fire passes, and the time required for foliage to scorch, are both implied by empirical relationships to fire behavior and weather variables (see Alexander and Cruz (2012) for thorough discussion of equations).

There are two physiological mechanisms which have been hypothesized to underly heat-induce foliar damage. First, foliage may undergo hydraulic failure. During a fire as air temperature increases and/or humidity decreases, the vapor pressure deficit (VPD; the difference between the amount of moisture in the air and how much moisture the air can hold when it is saturated) increases. Under high water stress and VPD, embolisms (air pockets in the water column of plant vascular tissue) may occur in foliage and branches. This can affect the hydraulic function of vascular tissues and cause instantaneous leaf scorch i.e. the "heat plume cavitation hypothesis"; (Kavanagh et al. 2010; Lodge et al. 2018). However, opinions differ as to the extent of leaf scorch this can cause during a fire and whether hydraulic failure can be easily repaired (Brodribb et al. 2016). More research is needed to determine how commonly hydraulic failure causes foliage necrosis and crown scorch in fires (Nolan et al. 2024). A second physiological mechanism of failure is that cells in a leaf can die from heat alone. Previous studies have observed leaf cell necrosis after applying heat using water baths, which maintains the hydraulic function of foliage while imposing damage from heat directly (Bilger et al. 1984; Balk et al. 1999; Bigras 2000). There is strong support in the literature that heat injury is a direct mechanism of leaf scorch and crown scorch in fire scenarios (Nolan et al. 2024). Consequently, while hydraulic failure may play a role in crown scorch, leaf tissue death by heat injury is the focus of this study.

When plant tissues are sufficiently injured by heat, cells undergo structural destabilization. The cell membranes or proteins embedded in the cell membrane become damaged, causing electrolytes to be irretrievably lost to intracellular space (Campos et al. 2003). This is termed electrolyte leakage and can be quantified by measuring the electrical conductivity (EC) of plant tissues (Hatsugai and Katagiri 2018). While damage to the cell membrane can be non-lethal initially, even minor damage often results in programmed cell death (Demidchik et al. 2014). Distinguishing between forms of cell death (e.g. programmed cell death versus cell necrosis) was outside the scope of the study (Shen et al. 2023). While cell membrane damage and resulting electrolyte leakage is not equivalent to cell necrosis, it is frequently used as an effective and efficiently-measured proxy for the likely extent of cell death in plant tissues (Igarashi et al. 2013; Bethke et al. 2016; Hatsugai et al. 2016, 2017; Hatsugai and Katagiri 2018). Electrolyte leakage was therefore used in this study to quantify heat injury to plant tissues.

Plant tissue injury from heat has best been characterised by Dickinson and Johnson (2004). They fit the "lag" model (Jung 1986), originally developed to describe mammalian cells, to experimental data from vascular phloem and cambium tissue collected from tree stems. In the lag model, cells within tissue that remain at high enough temperatures accumulate abnormalities or "lesions" resulting from the denaturation of proteins. These lesions are often non-lethal in the early stages of heat injury, as evidenced by a "lag" in cell necrosis that occurs at the onset of heating. As cell lesions accumulate through continued heating the likelihood of the lesions killing the cell increases (Jung 1986). Dickinson and Johnson (2004) demonstrated that the lag model fit experimental data of heat-caused cell necrosis in cambium tissue. Furthermore, the lag model can be used to estimate cell necrosis under fluctuating temperatures by calculating necrosis at short intervals, with cell necrosis accumulating at each time step. This "book-keeping" approach was evaluated by Dickinson and Johnson (2004), who demonstrated good agreement between predicted and observed cell necrosis ($R^2$ values between 0.53 and 0.95). The ability to calculate cell necrosis rates across fluctuating temperatures is critical to the determination of crown scorch in real-world wildland fire scenarios, since temperatures fluctuate widely depending on fire and atmospheric conditions (Atchley et al. 2024). Despite the apparent usefulness of the lag model, to our knowledge the lag model has never been applied to cell necrosis in leaf tissue.

Cell necrosis rates can vary by species and by season (Dickinson and Johnson 2004), contributing to differential crown scorch given the same fire intensity (Cansler et al. 2020). Minor differences in amino acid sequences can alter the thermal stability of proteins, which indicates a genetic mode of inheritance for determination of a species' thermal tolerance (Somero 1995). Seasonal differences in heat tolerance may result from changes in physiology as plants break or enter dormancy. These physiological changes differ between newly developed foliage and older foliage developed in previous years (Van Wagner 1967; Jolly et al. 2014). As such, there may be seasonal differences in heat tolerance between newly expanding foliage and older foliage within a single crown (Van Wagner 1967; Dickinson and Johnson 2004).

In this study, we used electrolyte leakage to estimate percent cell necrosis within leaf tissues resulting from different temperatures and durations of heating. We examined newly expanded and one-year-old needles of two western United States conifer species: *Pinus ponderosa* Lawson & C. Lawson var. *ponderosa* C. Lawson (ponderosa pine) and *Pseudotsuga menziesii* (Mirb.) Franco var. *glauca* (Beissn.) Franco (Douglas-fir) in two seasons, and modelled observations using the lag model. We chose *P. ponderosa* and *P. menziesii* because they have broad geographic distributions in western North America, regularly burn in wildfires and prescribed fires, and because their contrasting morphological foliage traits allowed investigation of potentially different thermal tolerances (e.g., *P. ponderosa* has thicker, longer needles than *P. menziesii*) (Greenberg and Collins 2021; Rodman et al. 2021). These species have also been the focus of extensive prior work on thermal tolerance which has revealed enduring questions about the mechanisms for fire-induced tree mortality (e.g. Wallin et al. 2003; Sparks et al. 2023). We used electrolyte leakage as an indicator of percent cell necrosis within leaf tissue, or leaf scorch. In doing so, we provide parameters for leaf scorch for these two widespread species in North America that could be incorporated into fire effects modelling systems for crown scorch estimation.

We had three specific objectives. 1) To determine if the lag model fits experimental data of leaf cell death by heat. If so, this lends support for the underlying assumption of the lag model- that heat-induced leaf tissue death (leaf scorch) is driven first by the formation of non-lethal lesions in cells, with lesions eventually killing the cells after sufficient time at lethal temperatures. 2) Develop models to describe the precise relationships between temperature and parameters of the lag under different conditions. These models can be used to calculate percent cell necrosis within foliage (i.e. leaf scorch) across different temperatures and under the fluctuating temperature regimes that occur in real-world situations. 3) Determine if species, season, and age of foliage significantly influence rates of cell necrosis. If so models of leaf scorch, and therefore crown scorch, should be parameterized for individual combinations of species, season, and age of foliage.

## 2. Materials and methods

### 2.1. Site description and sample collection

We sampled branches of *P. ponderosa* and *P. menziesii* saplings (DBH

2.5 to 12.7 cm) at Blue Mountain National Recreation Area on the Lolo National Forest in Missoula County, Montana, USA. We sampled saplings because there were enough saplings within a small area to minimize environmental variability, and because foliage from both species was easily accessible from the ground. Saplings are vulnerable to crown scorch even from low-intensity prescribed fires, as their crowns are often partially within the flaming zone and the degree of crown scorch can have significant bearing on their survival; saplings also contribute to fire spread via vertical and horizontal fuel continuity (Sayer et al. 2020). While foliar traits vary within trees by location within the crown and age class, these differences are small compared to interspecific variation (Weiskittel et al. 2008). Therefore, we feel that our results from saplings should extend to larger trees. The saplings were sampled within a 2 $km^2$ area. Elevation at the site ranged from 1107 m to 1197 m. Sites were north-facing with slopes ranging from 5 to 20 °. At the nearest weather station ("Missoula FTS Montana"; approx. 5.5 km away) from 2001 to 2022, the mean monthly air temperature was 7.5 °C, the mean monthly minimum air temperature was 1.3 °C, the mean monthly maximum air temperature was 14.7 °C, and the mean annual precipitation was 402.0 mm (Western Regional Climate Center 2024). Sampling took place between June and October of 2023. The Standardized Precipitation Index (SPI) for the region indicated no drought conditions during this period (Svoboda et al. 2002). However, mean monthly air temperature in Missoula County was slightly elevated, between 0.9 and 1.9 °C above historical normals (NOAA National Centers for Environmental information 2024).

The stands where *P. ponderosa* and *P. menziesii* trees were located differed somewhat in their structural characteristics and fire histories. The stand with *P. ponderosa* saplings was more open, with saplings established in large gaps free of overstory. This stand burned in the 2003 Black Mountain 2 wildfire (MTBS Project 2024). Char on the boles of larger trees was still evident. However, sampled trees did not show signs of past fire, and are believed to have established after the 2003 fire. The stand with *P. menziesii* saplings was more heavily shaded, and the *P. menziesii* saplings established beneath *P. ponderosa* in the overstory. No fires have occurred in this stand since at least 1984 (MTBS Project 2024).

At each sampling time, one branch from each sapling was cut, placed in tap water, and transported to the lab within 2 h. The late spring/early summer samples (hereafter referred to as "spring") were taken when the newly expanding needles were half the length of the previous year's needles (June 26, 2023 to July 9, 2023) and again from the same trees in autumn when the new needles were fully expanded (Sept 27, 2023 – Oct 11, 2023). Newly expanding/expanded needles ("new") and one-year-old needles ("1 yo") were removed from each branch – ten control and ten treatment needles for *P. menziesii* or three individual control and three individual treatment needles for *P. ponderosa*. Control and treatment samples were paired for calculations.

### 2.2. Experimental treatments

In the lab, samples were placed in an 8 L water bath (B2000 MyBath Digital Water Bath, Benchmark Scientific, Sayreville, NJ, USA), with temperature accuracy of ± 0.2 °C and temperature uniformity of ± 0.2 °C. The water bath was filled with de-ionized water (DI) heated to treatment temperatures of 45, 50, 55, 60, or 65 °C. Samples were immersed in the center of the heated water column for durations from 2 s up to 2 h and 50 min. Starting with 1 min treatments, shorter treatments were added until reduced treatment duration showed no increase in survival percentage and longer treatments were added until increased

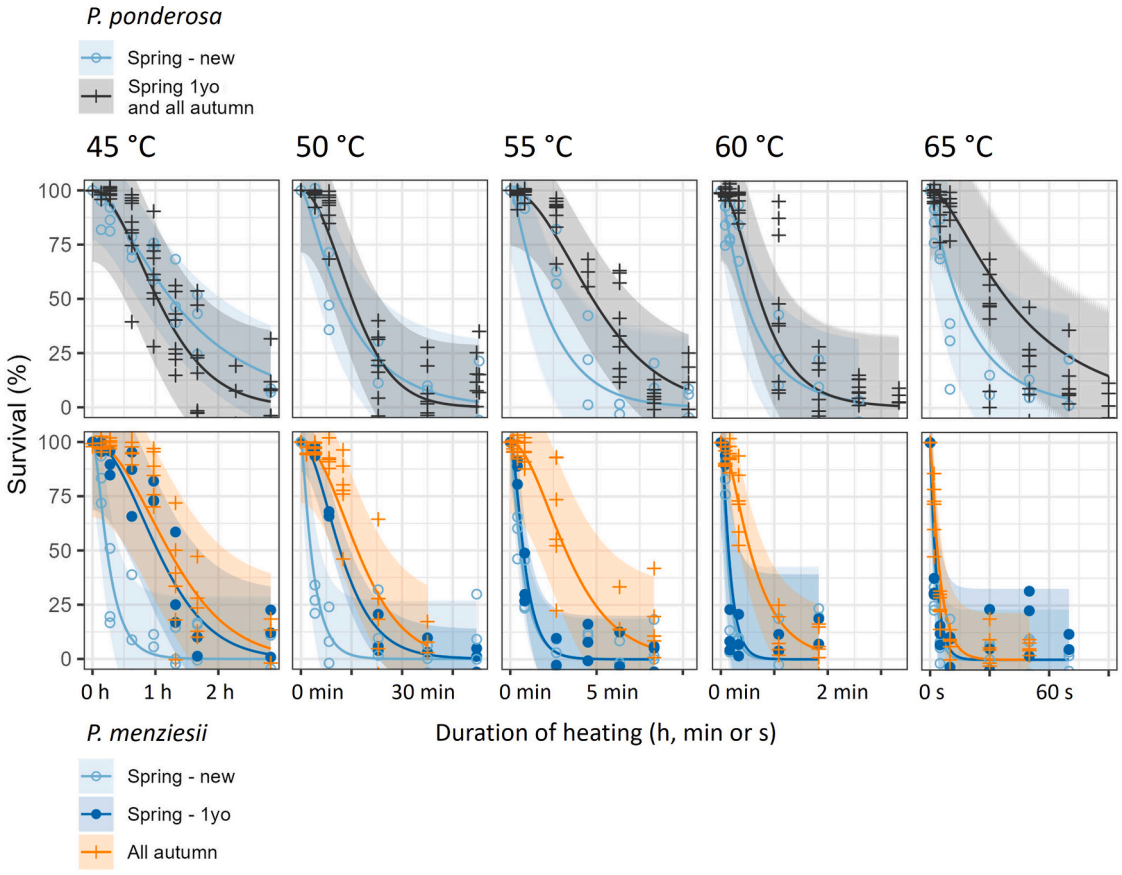


**Fig. 1.** Survival curves by species, season, and foliage age predicted using the lag model. P. ponderosa shown in upper row and P. menziesii in lower row. Shaded areas around lines show standard errors.

treatment duration showed waning reductions in survival percentage (i.e. survival curves approached apparent asymptotes; Fig. 1). Treatment temperatures required different durations of heating to complete survival curves, as the duration of heating required for cells to die varies considerably by temperature. When the treatment was completed, samples were submersed in room temperature DI (25 °C) to rapidly reduce the temperature, ensuring little additional heating beyond the desired treatment duration. While the treatments were being applied, the control samples were placed in DI at room temperature for the same duration of time as the duration of the treatment with which they were paired, to account for possible injury from samples being handled and submerged in DI water. According to equations for the free convective heating of horizontal cylinders using the dimensions of *P. menziesii* and *P. ponderosa* needles, samples should have equilibrated with the temperature of the water bath in < 0.1 s (Figure S1). Needle temperature was therefore assumed to be equal to the temperature of the water bath for the purposes of this study.

Cell survival percentage was estimated by calculating the change in electrolyte leakage of treatment needles relative to control needles from the same branch and age of needles. To estimate electrolyte leakage, we measured electrical conductivity (EC; mS $m^{-1}$). Temperature affects EC, so we compensated for changes in EC over temperature using industry standards for all EC measurements (Clesceri et al. 1998; Eq. (1)):

$$EC = EC_{observed} + EC_{observed} * [0.0191 * (T - 25)] \quad (1)$$

where $T$ is the temperature of the solution (°C).

After each treatment, heated and paired control samples were thoroughly rinsed with DI and left for 24 h in test tubes containing room temperature DI, following the methods of previous studies (Campos et al. 2003; Ahmad et al. 2016; Banerjee and Roychoudhury 2022). The EC of the resulting fluid ($EC_{initial}$) was then measured using a conductivity meter (AquaSearcher AB33EC Bench Meter, OHAUS Instruments Co. Ltd., Shanghai, China). The fluid was returned to the test tube and total EC was measured after boiling the needles for 20 min ($EC_{total}$). Boiling releases all ions from the tissue, allowing estimation of percent electrolyte leakage resulting from treatments (Campos et al. 2003; Ahmad et al. 2016; Banerjee and Roychoudhury 2022).

Percent difference in EC relative to control was calculated using Eq. (2):

$$heat\ injury\ (\%) = 100 * (R_t - R_0)/(1 - R_0) \quad (2)$$

where Rt $= EC_{initial}/EC_{total}$ of heat-treated samples and $R_0 = EC_{initial}/EC_{total}$ of control samples. Because all samples yield some electrolyte leakage, and leakage from control samples was subtracted from the treatment samples, it was not possible to calculate a heat injury index of 0. Values of $R_0$ ranged from 0.00 to 0.44, and values of $R_t$ ranged from 0.00 to 0.93. Resulting curves showed minimum heat injury values ranging from 7 to 58 %. Elevated minimum heat injury percentages likely resulted from damage to control samples from handling and being submerged in DI water. It was important to account for this damage when estimating percent cell necrosis resulting from heat alone. To do so, we scaled the curves from 0 to 100 using the R statistical program version 4.1.3 (R Core Team 2022) and a range transformation from the R package "caret" version 6.0–92 (Kuhn 2021). The scaled curves represented estimated percent cell necrosis resulting from heat injury. Percent cell survival was then estimated by subtracting percent cell necrosis from 100.

### 2.3. Data analysis

The lag model describes cells within "compartments" (Fig. 2). Each compartment contains a proportion of cells in the population. Initially all cells have no lesions (compartment $S_0$). When cells are heated to a damaging temperature, non-lethal lesions accumulate randomly in the population, and cells move into compartments for cells with progressively more lesions. The rate at which cells move out of compartment $S_0$ is described by Eq. (3):

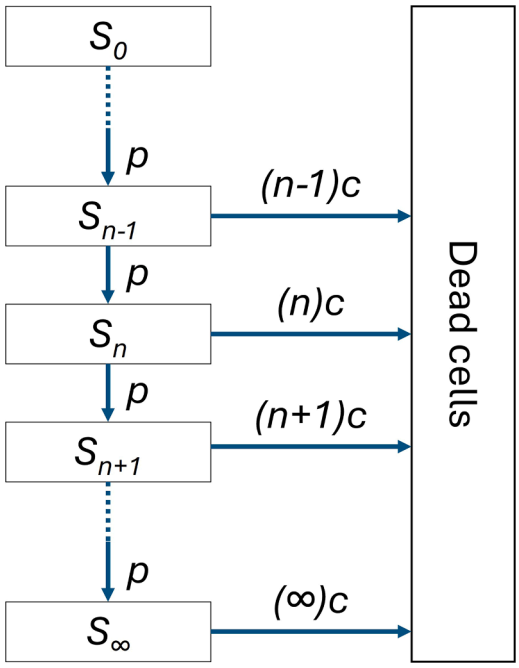


**Fig. 2.** Compartments described by the lag model. All cells have no lesions initially and belong to compartment $S_0$. A temperature dependent rate parameter $p$ ($s^{-1}$) defines the rate at which non-lethal lesions randomly accumulate in the cell population. Live cells that develop lesions move into successive compartments $S_{n-1}$ through $S_\infty$. A second temperature dependent rate parameter $c$ ($s^{-1}$) defines the rate at which non-lethal lesions are converted to lethal lesions. A cell with more lesions is therefore more likely to die. Figure adapted from Dickinson and Johnson (2004).

$$dS_0/dt = -pS_0 \quad (3)$$

where $p$ ($s^{-1}$) is the rate at which lesions accumulate in the cell population. The rate of change in the proportion of cells with lesions is:

$$dS_n/dt = pS_{n-1} - pS_n - ncS_n \quad (4)$$

where $S_n$ is the proportion of cells with $n$ lesions, and $c$ ($s^{-1}$) is the rate at which lesions change from non-lethal to lethal. The first term describes cells moving from compartment $S_{n-1}$ into compartment $S_n$, the second term describes the movement of cells out of compartment $S_n$ to $S_{n+1}$, and the last term describes the portion of cells moving out of compartment $S_n$ due to cell death. Note that the inclusion of $nc$ in the final term means that the more lesions a cell has the more likely it is to die at any given time.

Using Eqs. (3) and (4), the proportion of cells across all compartments $S_0$ to $S_\infty$ that survive ($S(t)$) after time $t$ (s) when heated at a constant temperature is calculated using Eq. (5):

$$S(t) = exp(p/c(1 - ct - exp(-ct))) \quad (5)$$

Together, the temperature-dependent rate parameters $p$ and $c$ define the shape of cell survival curves over time given a constant temperature. We estimated $p$ and $c$ by fitting the lag model to our experimental data. Following estimation of parameter values at each treatment temperature, the relationship between rate parameters and temperature were then described with a function that was fit to the data, allowing for interpolation of the shape of survival curves across a range of temperatures.

The parameters $p$ and $c$ describe the rate of lesion formation from heat damage and the rate of cell necrosis resulting from lesions, respectively. These parameters have been shown to exhibit

approximately log-linear relationships with temperature (Jung 1986; Dickinson and Johnson 2004). Developing models of functional relationships between temperature and these rate parameters under different conditions was one of the main objectives of this study. Therefore, linear regressions of log-transformed $p$ and $c$ against temperature were developed for use in species- and condition-specific models for connecting dynamic heat exposure to foliar damage (leaf scorch).

Previous studies that parameterized the lag model sometimes identified a bend or discontinuity in the log-linear relationship between either $p$ or $c$ and temperature (mammalian tissue, Jung 1986; plant bark tissue, Dickinson and Johnson 2004), and fit a second regression line to accommodate for these deviations. Our method of estimation favored a log-linear fit for these relationships and did not account for any discontinuities. However, we found that log-linear regression fit the modeled relationships well, and that introducing potential discontinuities was unnecessary. As such, we decided to forego more complex models in favor of parsimonious solutions.

The parameters $p$ and $c$ are collinear, which results in a flexible solution space. To identify a solution that provided high accuracy metrics for all models, we used a grid search approach followed by optimization. Data were jackknife resampled 100 times by leaving three randomly selected observations out of each round of grid search and optimization. This method was applied to each combination of species, season, and age of foliage separately. We merged datasets for combinations of species, season, and age of foliage that were not statistically significantly different from each other and repeated the procedure to generate final models for merged groups. All optimization in this study employed simulated annealing because it avoids local optima in favor of global optima, using the R package "optimization" version 1.0–9 (Husmann et al. 2017). All statistics were calculated use the base R package "stats" unless otherwise indicated.

The grid search was carried out on combinations of values of $p$ and $c$, evaluating the accuracy of the lag model for each combination of values. Within the ranges of values of $p$ and $c$ based on data from Dickinson and Johnson (2004), we generated 100 values of each variable. With each combination of these values, we predicted survival percentage using the lag model (Eq. (5)). Predictions were compared to observed survival percentages, with accuracy calculated as the normalized root mean squared error (NRMSE). This procedure resulted in 40,000,000 predictions total – 5,000,000 predictions per combination of species, season, and age of foliage – with 50,000 predictions per jackknifed data subset. Initial values of $p$ and $c$, as well as the minimum and maximum limits, to be used in optimization were selected for each jackknifed subset by examining the top 1 % of results from the grid search.

Using initial values and limits identified in the grid search, we optimized values of $p$ and $c$ for each jackknifed subset. Optimization was conducted on three models simultaneously: lag model fits to survival curves, linear regression of log-transformed $p$ against temperature, and linear regression of log-transformed $c$ against temperature. The algorithm minimized the maximum NRMSE of all models. In doing so, we identified a solution which minimized error for all applications of $p$ and $c$ to ensure good fits to raw data as well as generalizability across temperature for future applications. The results provided a distribution of each rate parameter, with 100 values of $p$ and 100 values of $c$ at each temperature. We used NRMSE to compare optimization results because it can be compared across response variables, and is independent of sample means (Faifer et al. 2020). However, we reported model predictions in root mean squared error (RMSE; Table S1) because it is in the same units as the response variable (percent survival), making interpretation more straightforward. We calculated RMSE by multiplying NRMSE by σ.

The mean predicted $p$ and $c$ were calculated and the lag model was used to predict survival percentage across temperatures (45, 50, 55, 60, and 65 °C). Significant differences between groups were identified by conducting analysis of variance (ANOVA) at each combination of temperature and duration of heating, followed by Tukey Honest Significance Difference tests. Mean standard errors ($\overline{se}_{lag}$) for lag model fits to observed survival were then calculated. We log-transformed the 100 predicted values of $p$ and $c$ from our jackknife procedure and used linear regression to model their relationships with temperature. Standard errors ($\overline{se}_{log-linear}$) from the linear models of log-transformed rate parameters against temperature were calculated. To determine which linear regression lines were statistically different from each other, we propagated error terms $\overline{se}_{lag}$ and $\overline{se}_{log-linear}$, and calculated final standard errors, following the methods of Ku (1966) and Efron (1981; Eq. (6)):

$$se_{final} = \sqrt{(1/T)^2 * log(\overline{se}_{lag})^2 + (\theta * T)^2 * \overline{se}^2_{log-linear}} \quad (6)$$

where $se_{final}$ is the final standard error, and $\theta$ is the model estimate for each linear regression of log-transformed rate parameters against temperature. Confidence intervals were calculated by multiplying the standard error by 1.96 ($\alpha = 0.05$), and adding to and subtracting from the estimate from each linear model of log-transformed rate parameters against temperature. Confidence intervals were compared between each model of species, season, and age of foliage. If confidence intervals overlapped across the entire range of temperatures for both $p$ and $c$, combinations were merged. The approach described above was repeated until no additional combinations could be merged, and final models for all groups were defined. Finally, we used optimization to estimate curves showing the temperature at which 50 % of cells are expected to die after 1 to 90 s of exposure ($LD_{50}$) using interpolations of log-linear models of rate parameters against temperature. We then compared $LD_{50}$ after 60 s of exposure because 60 °C for 60 s is assumed to cause crown scorch in many existing models (e.g. Van Wagner 1973). It is not currently known what percentage of cells within leaf tissue must necrotize before whole leaves are considered scorched. We estimated $LD_{50}$ because it is a well-established metric for describing lethal dose in plant physiology (Hammond et al. 2019), and a convenient threshold for discussing differences between treatment groups. Future research is needed to determine if $LD_{50}$ is an appropriate threshold for assuming whole leaf scorch.

## 3. Results

All observed survival data showed a decline in survival as the duration of heating increased (Fig. 2 and S2). Rates of decline in survival increased as temperature increased. A lag in necrosis was present for all combinations of species, season of sample, and age of foliage upon initiation of heating treatments. The lag model showed good agreement between predicted and observed survival for all groups (mean adjusted $R^2$ value of 0.79; Table S1). Linear regression of log-transformed lag model rate parameters $p$ and $c$ against temperature also showed good explanatory power for all models (Table 1; adjusted $R^2$ between 0.89 and 0.99; Fig. 3).

We found statistically significant differences between species, seasons of sampling, and ages of foliage. However, some categories were not significantly different from each other and were merged. Identifying the final groups required modelling rate parameters and testing for significance twice (Figs. S3 and S4), after which all groups were significantly different. Within categories of *P. ponderosa*, new foliage sampled in spring, new foliage sampled in autumn, and 1 yo foliage sampled in autumn were not significantly different from each other and were merged (Fig. 3). Within categories of *P. menziesii*, new foliage sampled in autumn, and 1 yo foliage sampled in autumn were merged.

*P. menziesii* exhibited higher necrosis rates than *P. ponderosa* under most circumstances, with all *P. menziesii* foliage categories becoming especially heat sensitive at higher temperatures (above 50 °C; Figs. 2 and S2). At the lowest temperatures examined in this study (45 and 50 °C), new *P. menziesii* foliage sampled in spring was the most heat sensitive (significance test p-value $< 0.001$). All other sampled foliage categories

**Table 1**
Predicted values of $log(p)$ and $log(c)$ from linear regressions against temperature and accuracy metrics by species, season, and foliage age. The natural logarithm was calculated using Napier's constant. The parameters $p$ and $c$ describe the rate of lesion formation from heat damage, and the rate of cell necrosis resulting from lesions, respectively. All groups had statistically significant differences (p-value $< 0.05$) between values of $p$ and $c$ compared to all other groups. Note: $p$ and $c$ estimates shown in units of $log(s^{-1})$.

| Species | Season and age of foliage | | 45 °C | 50 °C | 55 °C | 60 °C | 65 °C | Std. error | Adj $R^2$ |
|---|---|---|---|---|---|---|---|---|---|
| *P. ponderosa* | Spring - new | *p* | −8.09 | −6.70 | −5.30 | −3.90 | −2.50 | 0.003 | 0.95 |
| | | *c* | −6.94 | −4.71 | −2.48 | −0.24 | 1.99 | 0.007 | 0.89 |
| | Spring - 1 yo & autumn - all | *p* | −6.97 | −6.00 | −5.03 | −4.06 | −3.10 | 0.003 | 0.89 |
| | | *c* | −8.74 | −7.11 | −5.48 | −3.86 | −2.23 | 0.002 | 0.97 |
| *P. menziesii* | Spring - new | *p* | −6.75 | −5.27 | −3.78 | −2.29 | −0.81 | 0.001 | 0.99 |
| | | *c* | −5.27 | −3.48 | −1.70 | 0.08 | 1.86 | 0.003 | 0.96 |
| | Spring - 1 yo | *p* | −7.21 | −5.63 | −4.05 | −2.47 | −0.89 | 0.002 | 0.98 |
| | | *c* | −8.82 | −6.10 | −3.39 | −0.67 | 2.05 | 0.002 | 0.99 |
| | Autumn – all | *p* | −7.37 | −5.98 | −4.60 | −3.21 | −1.82 | 0.001 | 0.99 |
| | | *c* | −9.59 | −7.07 | −4.56 | −2.04 | 0.47 | 0.004 | 0.98 |

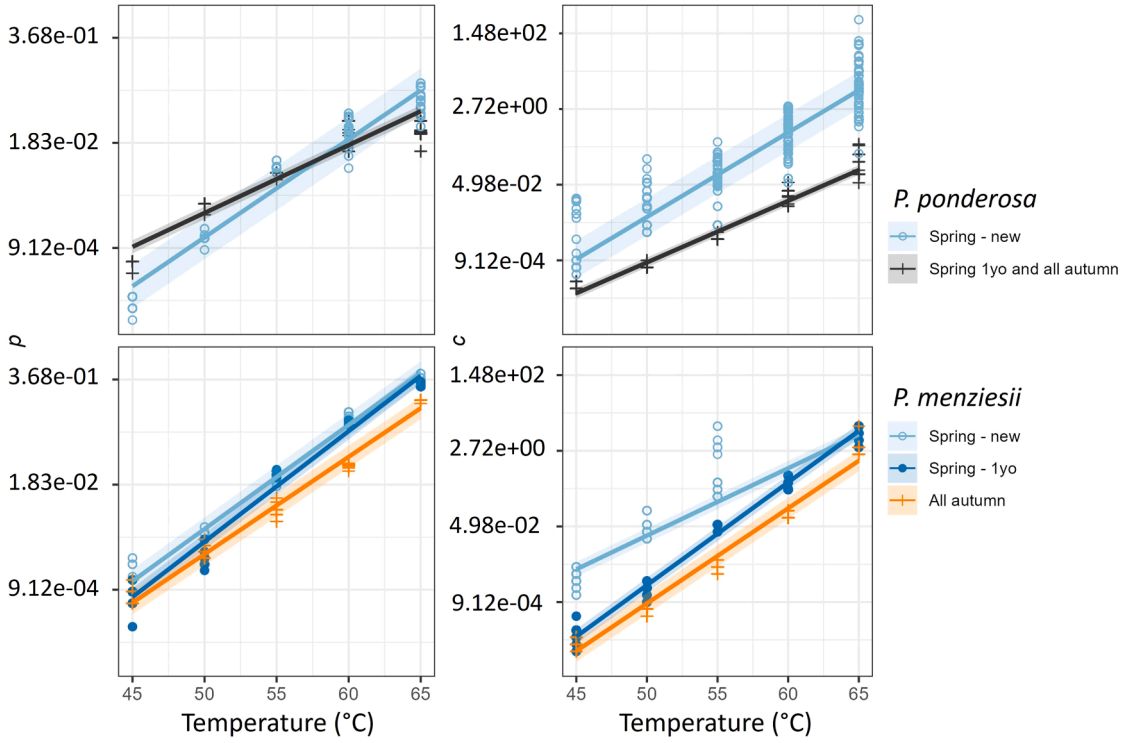


**Fig. 3.** Predicted values of the rate parameters $p$ and $c$ against temperature by species, season, and needle age. The parameters describe the rate of accumulation of non-lethal lesions ($p$) and the rate of conversion of these non-lethal lesions to lethal lesions ($c$) during heating at different temperatures. Shaded areas around lines show propagated standard errors. Note: y-axes are shown on a log scale.

were comparable in sensitivity at these temperatures. At 55 °C and higher, new and 1 yo *P. menziesii* foliage sampled in spring were similar in heat sensitivity. At the highest temperature examined in this study (65 °C), the merged *P. ponderosa* foliage category of 1 yo foliage sampled in spring and all autumn foliage was more tolerant of heat than any other category (p-value $< 0.001$). New *P. ponderosa* sampled in spring was second most heat tolerant, statistically significantly more heat tolerant than all *P. menziesii* foliage (p-value $< 0.001$).

As temperatures increased, 1 yo *P. menziesii* foliage sampled in spring showed the greatest increase in sensitivity as indicated by the greatest slope of regression lines in Fig. 3. The merged *P. ponderosa* foliage category of 1yo foliage sampled in spring and all autumn foliage exhibited the least increase in sensitivity (lowest slope) over the temperatures examined in this study. Examining $LD_{50}$ values at 60 s of exposure, categories of foliage ranked from most heat sensitive to least heat sensitive were as follows: new *P. menziesii* foliage sampled in spring, 1 yo *P. menziesii* foliage sampled in spring, *P. menziesii* foliage sampled in autumn, new *P. ponderosa* foliage sampled in spring, and all other *P. ponderosa* foliage (Fig. 4).

## 4. Discussion

Cell necrosis-caused crown scorch is driven by the denaturation of proteins in leaf cells at high temperatures, resulting in loss of function and loss of membrane stability. As proteins denature, cells survive or die depending on protein function (Demidchik et al. 2014). Heat injury in animal cells is cumulative (Jung 1994), with abnormalities or lesions accumulating as a result of damage to proteins until cells eventually die. The "lag" model describes this process based on two rate parameters, $p$ being the rate of lesion formation, and $c$ being the rate at which lesions become lethal to the cell. However, the lag model has never been tested using foliage. We found that the lag model fit our experimental data well. This supports the theory that leaf tissue death is driven first by non-lethal damage within cells, followed by necrosis. We developed a

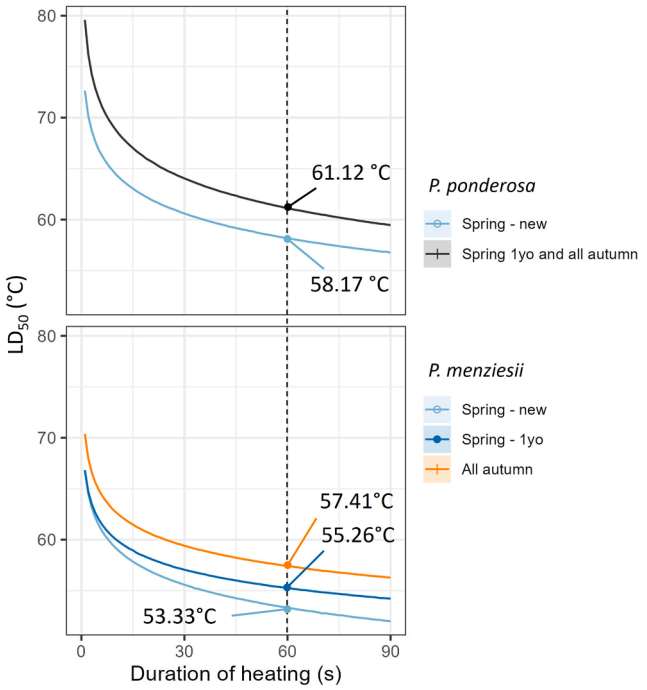


**Fig. 4.** $LD_{50}$ as the temperature at which 50 % of cells die after different durations of exposure to heat, presented in 1 s intervals, by species, season, and needle age. The dotted line highlights $LD_{50}$ at 60 s.

model describing time evolution of heat-induced leaf scorch that can be applied across a range of temperatures and under fluctuating temperatures. Since heat is considered the primary cause of leaf scorch, the models developed in this study can be used to improve crown scorch estimates from wildland fire given foliar temperatures over time. Lastly, we found significant differences between some combinations of species, season, and age of foliage, indicating that these variables can affect susceptibility to heat-induced crown scorch.

### 4.1. Crown scorch as a process

The temperature at which foliage dies ($T_L$), typically assumed to be 60 °C, is used in fire effects models to predict crown scorch height (Atchley et al. 2024). The basis for the value of $T_L$ lies in a number of studies that identified 60 °C as the temperature which kills pine needles after 60 s (*Pinus strobus* L.; Kayll 1968; *Pinus resinosa* Ait., and *P. strobus*; Methven 1971), although other studies suggest shorter times from instantaneous (Wade and Johansen 1986) to 30 s (Alexander et al. 2020). Crown scorch height equations implicitly incorporate assumptions about heating times, ambient wind penetration into the canopy (tied to convective cooling), fire line shape and plume intermittency, and real-world temperatures of canopy elements, rather than representing them explicitly (Atchley et al. 2024). This neglects to account for scorch that could result from foliage exposed to higher temperatures for shorter durations or lower temperatures for longer durations, thus oversimplifying how heat affects foliage survival. Existing crown scorch equations are also reliant on $T_L$ as a constant across species and situations that previous studies have shown are important factors in thermal sensitivity (Li et al. 1991; Somero 1995).

In the lag model, cells heated to lethal temperatures initially accumulate non-lethal damage, followed by cell necrosis. This delay or "lag" in cell necrosis was observed in our study, indicating that heat is initially non-lethal to plant cells as the lag model suggests (Jung 1986). This finding mirrors the results of Dickinson and Johnson (2004), who found that the lag model fit experimental data of cambium cells and refutes the notion of instantaneous leaf tissue death at a temperature and/or duration threshold. That the lag model was originally developed to model mammalian tissue (Jung 1986) yet fits experimental observations of plant cells suggests that protein denaturation and loss of membrane stability may underpin cell death by heat across taxa and tissue types. Future research should examine lag model fits to experimental data in other taxa to test this assertion.

### 4.2. Factors affecting thermal tolerance

Species differences in thermal tolerance have long been acknowledged (e.g. Nelson 1952). These differences are partially determined by differences in amino acid sequences of proteins, which confer varied levels of structural stability at high temperatures (Somero 1995). Other heritable differences in membrane structure and composition have also been proposed as possibly contributing to thermal tolerance (Somerville and Browse 1991).

With few exceptions, we found that *P. menziesii* was more heat sensitive than *P. ponderosa*, especially at temperatures above 55 °C. *P. menziesii* is therefore expected to exhibit greater crown scorch volume given the same initial ambient conditions, phenological stages, and fire intensities. This is supported by conventional wisdom, which suggests that *P. menziesii* should be more heat sensitive than *P. ponderosa* based on foliage traits (Fernandes et al. 2008; Varner et al. 2021). For example, *P. ponderosa* has much longer, thicker needles than *P. menziesii* (Keane et al. 1990; Weiskittel et al. 2008). Thicker needles correspond to higher leaf mass per area and lower specific leaf area and surface area to volume ratio, which positively correlate with heat tolerance (Rigolot 2004). However, while foliage trait values exist, potential differences in foliage heat tolerance to fire has received very little attention. This may be due to other traits related to branch morphology and crown architecture that also influence whole-tree fire tolerance (Fernandes et al. 2008; Hood et al. 2018). $LD_{50}$ after 60 s for *P. menziesii* deviated substantially from the commonly used $T_L$ of 60 °C (53.33 to 57.41 °C; Fig. 4), suggesting crown scorch height may be underpredicted for this species using current fire effects models (e.g. Reinhardt 2003; Heinsch and Andrews 2010; Hood and Lutes 2017). Conversely, $LD_{50}$ after 60 s for *P. ponderosa* was reasonably close (58.17 to 61.12 °C). This is perhaps unsurprising, since the standard 60 °C threshold is based on studies using material from pine species (Nelson 1952; Kayll 1968; Methven 1971). Here, we assumed 50 % cell necrosis would result in whole leaf scorch. To our knowledge this has never been tested. $LD_{50}$ is a well-established metric of lethal dose in plant physiology. However, further research is needed to determine if 50 % cell necrosis is a reliable threshold for estimating whole leaf scorch.

Season and age of foliage also affect thermal tolerance of these two species in different ways. *P. menziesii* foliage was overall more heat sensitive in spring than autumn. This suggests that spring fires may result in greater percentages of crown scorch in *P. menziesii* than autumn fires given the same fire intensity and initial physiological and environmental conditions. In the western US, conifer carbon stores are replenished in late summer, such that loss of foliage has a greater impact on tree mortality when trees are entering the growing season compared to entering dormancy (Kramer 2012). Presumably, with less carbon store replenishment in the early growing season and higher thermal sensitivity shown here, the potential for tree mortality of *P. menziesii* may be greater in the early growing season. Tree mortality may be even higher if burns occur after bud break during active new needle elongation, since more foliage would be exposed and unprotected during a fire (Harrington 1993). Season alone was less relevant for *P. ponderosa* and was only significant for new foliage. Additional research is needed to test how our needle-based results translate to tree-level mortality based on season of burn, as well as how season and age of foliage affect thermal tolerance in other species.

Age of foliage alone did not affect thermal tolerance of either species. However, season-by-age interactions were evident. In our study, new foliage sampled in spring was in the process of developing. The literature

is divided on whether developing foliage is more sensitive to heat injury than older foliage. For example, a study in *Capsicum annuum* L. (bell pepper; Anderson et al. 1990) found that immature foliage was less heat tolerant than fully expanded foliage, while a study in *Zea mays* L. (maize; Karim et al. 1999) showed that heat injury was less severe in developing foliage than in fully developed foliage. Studies reporting that developing foliage is more heat sensitive suggest that this may result from differences in the anatomy of cellular membranes (Wahid et al. 2007). We found that within categories of *P. ponderosa* foliage, developing spring foliage was significantly more sensitive to heat than all other season and age categories (Figs. 2 and S2). For *P. menziesii* foliage, developing spring foliage was the most heat sensitive followed by 1 yo foliage sampled in spring, and lastly any foliage sampled in autumn. Our findings indicate that by autumn, when new needles are fully developed, they are no less tolerant of heat than older foliage.

The greater thermal sensitivity of new conifer foliage in spring compared to autumn suggests that new foliage will be scorched more readily after a spring burn than after an autumn burn given the same fire behavior. One study showed that stands of *P. ponderosa* scorched after bud break in spring exhibited greater mortality than stands scorched in autumn given the same crown scorch volume (Harrington 1987). However, it is generally thought that if stands burned in spring before new needles emerge, mortality rates will be lower than if burned during the growing season (Crane and Fischer 1986). If true, these observations together with our findings suggest that increased mortality from spring burns (Harrington 1987) may result from disproportionate damage to developing foliage in our two study species.

The seasonal trends presented in this study may have important ecological repercussions. A higher proportion of damage to developing foliage in spring than in autumn may result in increased losses to carbon resources at a critical time. Newly developed foliage has greater photosynthetic capacity and greater nitrogen per unit mass than older foliage (Mediavilla and Escudero 2003). Consequently, loss of new foliage reduces nutrient pools more than loss of old foliage given the same mass lost. If stands burn after bud break in spring, scorch may therefore be expected to result in even greater impacts to a tree's carbon stores than might be expected given crown scorch volume estimates under existing models (e.g. Hood and Lutes 2017). Carbon reserves are especially important in the growing season (Hoch 2015). Needles can be retained on trees for as long as 5 y by *P. ponderosa* (Grulke and Retzlaff 2001) and 4 y by *P. menziesii* (Mulvey et al. 2013). This suggests a new-to-old foliage ratio of at least 1:4 and 1:3 within crowns, respectively. Scorch after bud break in spring is therefore expected to increase mortality of *P. menziesii* more than *P. ponderosa*, since developing foliage accounts for a greater proportion of the crown.

To our knowledge this is the first study to examine heat-induced leaf cell necrosis across phenological stages and species of conifer saplings, and to identify mechanisms of heat-driven leaf scorch that can be integrated into existing tree mortality prediction models. Our field-based study focused on two widespread species in the North American west. We were unable to control for all possible environmental factors, and these results should be considered specific to the species evaluated. We also limited our analysis to saplings, and these findings may differ for other size classes. Future studies should examine these species and others across a wider geographic range to capture possible relationships between inter-species variation, tree size class, plant traits, climate, and seasonal environmental changes.

## 5. Conclusion

We detailed heat-induced mechanisms of foliar cell necrosis (i.e. leaf scorch) in two ubiquitous western US conifers and characterize their dependence on both temperature and duration of heating. Our results dispute the characterization of crown scorch as a temperature threshold response. The lag model of heat injury fit experimental data well, supporting its broader application across taxa, across tissue types, and under more circumstances. The models detailed in this study can readily be applied to improve estimates of crown scorch in fire effects models for these species. While the models describe leaf scorch in only two species, our study demonstrates a novel approach for assessing foliar thermal tolerance that can be used to inform crown scorch estimates for other tree species. We found that species, season, and age of foliage can all influence thermal sensitivity. These findings refute the notion of the universal 60 °C threshold of crown scorch currently used in many fire effects models and highlights a need for crown scorch models to incorporate species and phenological data to improve accuracy.

## Data statement

The original data and code used to process, visualize, and finalize results can be accessed here: https://github.com/firelab/LeafScorch.

## CRediT authorship contribution statement

**Kate J. Fuller:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft, Writing – review & editing. **Leda N. Kobziar:** Writing – review & editing, Funding acquisition, Conceptualization. **Rodman R. Linn:** Writing – review & editing, Funding acquisition, Conceptualization. **Sharon M. Hood:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization.

## Declaration of competing interest

The authors of this manuscript have no competing interests to disclose.

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## Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ecolmodel.2025.111192.

## References

Ahmad, P., Abdel Latef, A.A., Hashem, A., Abd_Allah, E.F., Gucel, S., Tran, L-SP, 2016. Nitric oxide mitigates salt stress by regulating levels of osmolytes and antioxidant enzymes in chickpea. Front. Plant Sci. 7. https://www.frontiersin.org/articles/10.3389/fpls.2016.00347.

Alexander, M.E., Cruz, M.G., 2012. Interdependencies between flame length and fireline intensity in predicting crown fire initiation and crown scorch height. Int. J. Wildland. Fire 21, 95. https://doi.org/10.1071/WF11001.

Alexander M.E., Cruz M.G., Taylor S.W. (2020) Crown scorch height. 'Encyclopedia of Wildfires and Wildland-Urban Interface (WUI) Fires'. (Ed SL Manzello) pp. 1–7. (Springer International Publishing: Cham) doi:10.1007/978-3-319-51727-8_72-1.

Anderson, J., McCollum, G., Roberts, W., 1990. High temperature acclimation in pepper leaves. Hort. Sci. 25, 1272–1274.

Atchley, A.L., Hoffman, C.M., Bonner, S.R., Ritter, S.M., O'Brien, J., Linn, R.R., 2024. Evaluating crown scorch predictions from a computational fluid dynamics wildland fire simulator. Fire Ecol. 20, 71. https://doi.org/10.1186/s42408-024-00291-x.

Balk, J., Leaver, C.J., McCabe, P.F., 1999. Translocation of cytochrome c from the mitochondria to the cytosol occurs during heat-induced programmed cell death in cucumber plants. FEBS Lett. 463, 151–154. https://doi.org/10.1016/S0014-5793(99)01611-7.

Banerjee, A., Roychoudhury, A., 2022. Rhizofiltration of combined arsenic-fluoride or lead-fluoride polluted water using common aquatic plants and use of the 'clean' water for alleviating combined xenobiotic toxicity in a sensitive rice variety. Environ. Pollut. 304, 119128. https://doi.org/10.1016/j.envpol.2022.119128.

Barker, J.S., Gray, A.N., Fried, J.S., 2022. The effects of crown scorch on post-fire delayed mortality are modified by drought exposure in California (USA). Fire 5, 21.
Bethke, G., Thao, A., Xiong, G., Li, B., Soltis, N.E., Hatsugai, N., Hillmer, R.A., Katagiri, F., Kliebenstein, D.J., Pauly, M., Glazebrook, J., 2016. Pectin biosynthesis is critical for cell wall integrity and immunity in Arabidopsis thaliana. Plant Cell 28, 537–556. https://doi.org/10.1105/tpc.15.00404.
Bigras, F.J., 2000. Selection of white spruce families in the context of climate change: heat tolerance. Tree Physiol. 20, 1227–1234. https://doi.org/10.1093/treephys/20.18.1227.
Bilger, H.-W., Schreiber, U., Lange, O.L., 1984. Determination of leaf heat resistance: comparative investigation of chlorophyll fluorescence changes and tissue necrosis methods. Oecologia 63, 256–262. https://doi.org/10.1007/BF00379886.
Bison, N.N., Partelli-Feltrin, R., Michaletz, S.T., 2022. Trait phenology and fire seasonality co-drive seasonal variation in fire effects on tree crowns. New Phytol. 234, 1654–1663. https://doi.org/10.1111/nph.18047.
Bond, W.J., Woodward, F.I., Midgley, G.F., 2005. The global distribution of ecosystems in a world without fire. New Phytol. 165, 525–538. https://doi.org/10.1111/j.1469-8137.2004.01252.x.
Brodribb, T.J., Skelton, R.P., McAdam, S.A.M., Bienaimé, D., Lucani, C.J., Marmottant, P., 2016. Visual quantification of embolism reveals leaf vulnerability to hydraulic failure. New Phytol. 209, 1403–1409. https://doi.org/10.1111/nph.13846.
Byram, G.M., 1958. Some Basic Thermal Processes Controlling the Effects of Fire on Living Vegetation. US Forest Service, Southeastern Forest Experiment Station.
Campos, P.S., nia, Quartin V, chicho, Ramalho J, Nunes, M.A., 2003. Electrolyte leakage and lipid degradation account for cold sensitivity in leaves of coffea sp. plants. J. Plant Physiol. 160, 283–292. https://doi.org/10.1078/0176-1617-00833.
Cansler, C.A., Hood, S.M., van Mantgem, P.J., Varner, J.M., 2020. A large database supports the use of simple models of post-fire tree mortality for thick-barked conifers, with less support for other species. Fire Ecol. 16, 25. https://doi.org/10.1186/s42408-020-00082-0.
Clesceri, L., Greenberg, A., Eaton, A., 1998. Standard Methods for the Examination of Water and Wastewater. American Public Health Association, Washington, D.C., USA.
Crane M.F., Fischer W.C. (1986) Fire ecology of the forest habitat types of central Idaho. U.S. Department of Agriculture, Forest Service, Intermountain Research Station, INT-GTR-218. (Ogden, UT) doi:10.2737/INT-GTR-218.
Demidchik, V., Straltsova, D., Medvedev, S.S., Pozhvanov, G.A., Sokolik, A., Yurin, V., 2014. Stress-induced electrolyte leakage: the role of K+-permeable channels and involvement in programmed cell death and metabolic adjustment. J. Exp. Bot. 65, 1259–1270. https://doi.org/10.1093/jxb/eru004.
Dickinson, M.B., Johnson, E.A., 2004. Temperature-dependent rate models of vascular cambium cell mortality. Can. J. For. Res. 34, 546–559. https://doi.org/10.1139/x03-223.
Efron, B., 1981. Nonparametric estimates of standard error: the jackknife, the bootstrap and other methods. Biometrika 68, 589–599. https://doi.org/10.2307/2335441.
Faifer, M., Laurano, C., Ottoboni, R., Toscani, S., Zanoni, M., 2020. Frequency-domain nonlinear modeling approaches for power systems components—A comparison. Energies 13, 2609.
Fernandes, P.M., Vega, J.A., Jiménez, E., Rigolot, E., 2008. Fire resistance of European pines. For. Ecol. Manage. 256, 246–255.
Greenberg, C.H., Collins, B., 2021. Fire Ecology and management: Past, present, and Future of US Forested Ecosystems. Springer.
Grulke, N.E., Retzlaff, W.A., 2001. Changes in physiological attributes of ponderosa pine from seedling to mature tree. Tree Physiol. 21, 275–286. https://doi.org/10.1093/treephys/21.5.275.
Hammond, W.M., Yu, K., Wilson, L.A., Will, R.E., Anderegg, W.R.L., Adams, H.D., 2019. Dead or dying? Quantifying the point of no return from hydraulic failure in drought-induced tree mortality. New Phytol. 223, 1834–1843. https://doi.org/10.1111/nph.15922.
Hare R.C. (1961) Heat effects on living plants. USDA, Forest Service, Southern Forest Experiment Station, Occasional Paper 183.
Harrington, M.G., 1987. Ponderosa pine mortality from spring, summer, and fall crown scorching. Western J. Appl. Forestry 2, 14–16. https://doi.org/10.1093/wjaf/2.1.14.
Harrington, M., 1993. Predicting Pinus ponderosa mortality from dormant season and growing-season fire injury. Int. J. Wildland Fire 3, 65. https://doi.org/10.1071/WF9930065.
Hatsugai, N., Hillmer, R., Yamaoka, S., Hara-Nishimura, I., Katagiri, F., 2016. The μ subunit of Arabidopsis Adaptor Protein-2 is involved in effector-triggered immunity mediated by membrane-localized resistance proteins. Molecular Plant-Microbe Interact.: MPMI 29, 345–351. https://doi.org/10.1094/MPMI-10-15-0228-R.
Hatsugai, N., Igarashi, D., Mase, K., Lu, Y., Tsuda, Y., Chakravarthy, S., Wei, H.-L., Foley, J.W., Collmer, A., Glazebrook, J., Katagiri, F., 2017. A plant effector-triggered immunity signaling sector is inhibited by pattern-triggered immunity. EMBO J. 36, 2758–2769. https://doi.org/10.15252/embj.201796529.
Hatsugai, N., Katagiri, F., 2018. Quantification of plant cell death by electrolyte leakage assay. Bio Protoc 8, e2758. https://doi.org/10.21769/BioProtoc.2758.
Heinsch, F.A., Andrews, P.L., 2010. BehavePlus fire modeling system, version 5.0: design and features. Gen Tech Rep RMRS-GTR-249 Fort Collins, CO: US Department of Agriculture, Forest Service. Rocky Mount. Res. Station 111, 249.
Hiers, J.K., O'Brien, J.J., Varner, J.M., Butler, B.W., Dickinson, M., Furman, J., Gallagher, M., Godwin, D., Goodrick, S.L., Hood, S.M., 2020. Prescribed fire science: the case for a refined research agenda. Fire Ecol. 16, 1–15.
Hoch, G., 2015. Carbon reserves as indicators for Carbon limitation in trees. In: Lüttge, U., Beyschlag, W. (Eds.), 'Progress in Botany'. Springer International Publishing, Cham, pp. 321–346. https://doi.org/10.1007/978-3-319-08807-5_13. Progress in Botany.
Hood, S., Lutes, D., 2017. Predicting post-fire tree mortality for 12 western US conifers using the first order fire effects model (FOFEM). Fire Ecol. 13, 66–84. https://doi.org/10.4996/fireecology.130290243.
Hood, S., Varner, J., Mantgem, P., Cansler, C., 2018. Fire and tree death: understanding and improving modeling of fire-induced tree mortality. Environ. Res. Lett. 13, 113004. https://doi.org/10.1088/1748-9326/aae934.
Husmann, K., Lange, A., Spiegel, E., 2017. The R Package Optimization: Flexible Global Optimization with Simulated-Annealing. CRAN Citation.
Igarashi, D., Bethke, G., Xu, Y., Tsuda, K., Glazebrook, J., Katagiri, F., 2013. Pattern-triggered immunity suppresses programmed cell death triggered by fumonisin b1. PLoS One 8, e60769. https://doi.org/10.1371/journal.pone.0060769.
Jolly, W.M., Hadlow, A.M., Huguet, K., 2014. De-coupling seasonal changes in water content and dry matter to predict live conifer foliar moisture content. Int. J. Wildland Fire 23, 480. https://doi.org/10.1071/WF13127.
Jung, H., 1986. A generalized concept for cell killing by heat. Radiat. Res. 106, 56–72. https://doi.org/10.2307/3576561.
Jung, H., 1994. A generalized concept for cell killing by heat: effect of acutely induced thermotolerance and decay of thermosensitization. Radiat. Res. 139, 280–289.
Karim, M.D.A., Fracheboud, Y., Stamp, P., 1999. Photosynthetic activity of developing leaves of Zea mays is less affected by heat stress than that of developed leaves. Physiol. Plant. 105, 685–693. https://doi.org/10.1034/j.1399-3054.1999.105413.x.
Kavanagh, K.L., Dickinson, M.B., Bova, A.S., 2010. A way forward for fire-caused tree mortality prediction: modeling A physiological consequence of fire. Fire Ecol. 6, 80–94. https://doi.org/10.4996/fireecology.0601080.
Kayll, A., 1968. Heat tolerance of tree seedlings. Proceedings of the 8th Tall Timbers Fire Ecology Conference. Tallahassee, Florida, USA, Tallahassee, Florida, USA, pp. 89–106. https://talltimbers.org/wp-content/uploads/2018/09/89-Kayll1968_op.pdf.
Keane, R.E., Arno, S.F., Brown, J.K., 1990. Simulating cumulative fire effects in Ponderosa pine/Douglas-fir forests. Ecology 71, 189–203.
Kelsey, R.G., Westlind, D.J., 2017. Physiological stress and ethanol accumulation in tree stems and woody tissues at sublethal temperatures from fire. Bioscience 67, 443–451. https://doi.org/10.1093/biosci/bix037.
Kobziar, L., Moghaddas, J., Stephens, S.L., 2006. Tree mortality patterns following prescribed fires in a mixed conifer forest. Can. J. For. Res. 36, 3222–3238.
Kramer, P., 2012. Physiology of Woody Plants. Elsevier.
Ku, H.H., 1966. Notes on the use of propagation of error formulas. J. Res. Natl. Bur. Stand. (1934) 70C.
Kuhn M. (2021) caret: classification and regression training. https://CRAN.R-project.org/package=caret.
Li, P.H., Davis, D.W., Shen, Z.-Y., 1991. High-temperature-acclimation potential of the common bean: can it be used as a selection criterion for improving crop performance in high-temperature environments? Field. Crops. Res. 27, 241–256. https://doi.org/10.1016/0378-4290(91)90064-3.
Lodge, A.G., Dickinson, M.B., Kavanagh, K.L., 2018. Xylem heating increases vulnerability to cavitation in longleaf pine. Environ. Res. Lett. 13, 055007.
Mediavilla, S., Escudero, A., 2003. Photosynthetic capacity, integrated over the lifetime of a leaf, is predicted to be independent of leaf longevity in some tree species. New Phytol. 159, 203–211. https://doi.org/10.1046/j.1469-8137.2003.00798.x.
Methven, I.R., 1971. Prescribed fire, crown scorch and mortality: field and laboratory studies on red and white pine. Canadian Forestry Service Department of the Environment, PS-X-31. Chalk River, Ontario, Canada.
Michaletz, S.T., Johnson, E.A., 2006. A heat transfer model of crown scorch in forest fires. Can. J. For. Res. 36, 2839–2851.
Michaletz, S.T., Johnson, E.A., 2007. How forest fires kill trees: a review of the fundamental biophysical processes. Scand. J. For. Res. 22, 500–515. https://doi.org/10.1080/02827580701803544.
MTBS Project (2024) MTBS data access: fire level geospatial data. http://mtbs.gov/direct-download.
Mulvey, R.L., Shaw, D.C., Filip, G.M., Chastagner, G.A., 2013. Swiss needle cast of Douglas-fir. Forest Insect Disease Leaflet (FIDL) 181.
Nelson, R.M., 1952. Observations on heat tolerance of southern pines. USDA For Serv Res Paper SE-14, Southeast For Exp Stn.
NOAA National Centers for Environmental information (2024) NOAA National Centers for Environmental information, climate at a glance: county mapping. https://www.ncei.noaa.gov/access/monitoring/climate-at-a-glance/county/mapping.
Nolan, R.H., Reed, C.C., Hood, S.M., 2024. Mechanisms of fire-caused tree death are far from resolved. Tree Physiol. 44, tpae073. https://doi.org/10.1093/treephys/tpae073.
R Core Team (2022) R: a language and environment for statistical computing. http://www.R-project.org/.
Reinhardt, E.D., 2003. The Fire and Fuels Extension to the Forest Vegetation Simulator. United States Department of Agriculture, Forest Service, Rocky Mountain ….
Rigolot, E., 2004. Predicting postfire mortality of *Pinus hal*epensis Mill. And Pinus pinea L. Plant Ecol. 171, 139–151.
Rodman, K.C., Veblen, T.T., Andrus, R.A., Enright, N.J., Fontaine, J.B., Gonzalez, A.D., Redmond, M.D., Wion, A.P., 2021. A trait-based approach to assessing resistance and resilience to wildfire in two iconic North American conifers. J. Ecol. 109, 313–326. https://doi.org/10.1111/1365-2745.13480.
Sayer, M.A.S., Tyree, M.C., Kuehler, E.A., Jackson, J.K., Dillaway, D.N., 2020. Physiological mechanisms of foliage recovery after spring or fall crown scorch in young longleaf pine (*Pinus palustris* Mill.). Forests 11, 208. https://doi.org/10.3390/f11020208.
Shen, S., Shao, Y., Li, C., 2023. Different types of cell death and their shift in shaping disease. Cell. Death. Discov. 9, 1–12. https://doi.org/10.1038/s41420-023-01581-0.
Somero, G.N., 1995. Proteins and Temperature.

Somerville, C., Browse, J., 1991. Plant lipids: metabolism, mutants, and membranes. Science 252, 80–87.
Sparks, A.M., Blanco, A.S., Wilson, D.R., Schwilk, D.W., Johnson, D.M., Adams, H.D., Bowman, D.M.J.S., Hardman, D.D., Smith, A.M.S., 2023. Fire intensity impacts on physiological performance and mortality in Pinus monticola and Pseudotsuga menziesii saplings: a dose–response analysis. Tree Physiol. 43, 1365–1382. https://doi.org/10.1093/treephys/tpad051.
Svoboda, M., LeComte, D., Hayes, M., Heim, R., Gleason, K., Angel, J., Rippey, B., Tinker, R., Palecki, M., Stooksbury, D., Miskus, D., Stephens, S., 2002. The Drought Monitor. Bull. Am. Meteorol. Soc. 83, 1181–1190. https://doi.org/10.1175/1520-0477-83.8.1181.
Van Wagner, C.E., 1967. Seasonal Variation in Moisture Content of Eastern Canadian Tree Foliage and the Possible Effect on Crown Fires. Department of Forestry and Rural Development, Petawawa Forest Experiment Station.
Van Wagner, C., 1973. Height of crown scorch in forest fires. Can. J. For. Res. 3, 373–378.
Varner, J.M., Hood, S.M., Aubrey, D.P., Yedinak, K., Hiers, J.K., Jolly, W.M., Shearman, T.M., McDaniel, J.K., O'Brien, J.J., Rowell, E.M., 2021a. Tree crown injury from wildland fires: causes, measurement and ecological and physiological consequences. New Phytol. 231, 1676–1685.
Varner, J.M., Hood, S.M., Aubrey, D.P., Yedinak, K., Hiers, J.K., Jolly, W.M., Shearman, T.M., McDaniel, J.K., O'Brien, J.J., Rowell, E.M., 2021b. Tree crown injury from wildland fires: causes, measurement and ecological and physiological consequences. New Phytol. 231, 1676–1685. https://doi.org/10.1111/nph.17539.
Wade, D.D., Johansen, R.W., 1986. Effects of fire on Southern Pine: observations and recommendations. General Technical Report SE, US Department of Agriculture, Forest Service, Southeastern Forest Exp. Station 041, 1–14. https://doi.org/10.2737/SE-GTR-41.
Wahid, A., Gelani, S., Ashraf, M., Foolad, M., 2007. Heat tolerance in plants: an overview. Environ. Exp. Bot. 61, 199–223. https://doi.org/10.1016/j.envexpbot.2007.05.011.
Wallin, K.F., Kolb, T.E., Skov, K.R., Wagner, M.R., 2003. Effects of crown scorch on Ponderosa pine resistance to bark beetles in Northern Arizona. Environ. Entomol. 32, 652–661. https://doi.org/10.1603/0046-225X-32.3.652.
Weiskittel, A.R., Temesgen, H., Wilson, D.S., Maguire, D.A., 2008. Sources of within-and between-stand variability in specific leaf area of three ecologically distinct conifer species. Ann. For. Sci. 65, 1–10.
Western Regional Climate Center (2024) RAWS USA Climate Archive. https://raws.dri.edu.
Woolley, T., Shaw, D.C., Ganio, L.M., Fitzgerald, S., 2011. A review of logistic regression models used to predict post-fire tree mortality of western North American conifers. Int. J. Wildland Fire 21 (1), 1–35.