Temperature-dependent rate models of vascular cambium cell mortality

Matthew B. Dickinson and Edward A. Johnson

Abstract: We use two rate-process models to describe cell mortality at elevated temperatures as a means of understanding vascular cambium cell death during surface fires. In the models, cell death is caused by irreversible damage to cellular molecules that occurs at rates that increase exponentially with temperature. The models differ in whether cells show cumulative effects of heating. The temperature dependencies of the models' rate parameters were estimated from cell-count data after exposing live-bark tissues from four Canadian Rocky Mountain tree species to a range of fixed temperatures in a water bath. Based on both models, lodgepole pine's (*Pinus contorta* Dougl. ex Loud.) growing season vascular cambium cells experienced lower mortality rates at elevated temperatures than those of aspen (*Populus tremuloides* Michx.), Engelmann spruce (*Picea engelmannii* Parry ex Engelm.), and Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco). Growing and dormant season differences were marginal. With reservations for lodgepole pine, both models predicted cell survival after exposures to rising and falling temperatures such as would be experienced by live tissues during fires. A simulation involving conduction heat transfer from flames and vascular cambium cell mortality suggests that differences among species in thermal tolerance are small compared with the effects of bark thickness. Although stem vascular cambium cell mortality was complete when tissues reached 60 °C during simulated surface fires, it may not be warranted to apply the 60 °C threshold to other tissues exposed to contrasting temperature regimes during fires.

Résumé : Nous avons utilisé deux modèles basés sur le taux du processus pour décrire la mortalité des cellules à des températures élevées afin de comprendre la mortalité des cellules cambiales durant un feu de surface. Dans les modèles, la mort des cellules est causée par les dommages irréversibles au niveau moléculaire qui surviennent à des taux qui augmentent exponentiellement en fonction de la température. Les modèles diffèrent selon que les cellules subissent ou non les effets cumulatifs de la chaleur. Les paramètres de taux des modèles qui sont dépendants de la température ont été estimés à partir des données provenant du décompte des cellules après avoir exposé des morceaux d'écorce vivante de quatre espèces d'arbres des Montagnes Rocheuses canadiennes à un éventail de températures fixes dans un bassin d'eau. Sur la base des deux modèles, les cellules du cambium vasculaire présentes au cours de la saison de croissance qui ont été soumises à des températures élevées ont connu des taux de mortalité plus faibles chez le pin lodgepole (*Pinus contorta* Dougl. ex Loud.) que chez le peuplier faux-tremble (*Populus tremuloides* Michx.), l'épinette d'Engelmann (*Picea engelmannii* Parry ex Engelm.) et le douglas de Menzies (*Pseudotsuga menziesii* (Mirb.) Franco). Les différences entre la saison de croissance et la période de dormance étaient marginales. Avec quelques réserves pour le pin lodgepole, les deux modèles prédisent la survie des cellules après une exposition à une augmentation et à une diminution de la température telles que subissent les tissus vivants lors d'un feu. Une simulation impliquant le transfert de chaleur des flammes par conduction et la mortalité des cellules du cambium vasculaire indique que les différences de tolérance thermale entre les espèces sont faibles comparativement aux effets dus à l'épaisseur de l'écorce. Même si toutes les cellules du cambium vasculaire de la tige étaient mortes lorsque les tissus atteignaient 60 °C lors de la simulation d'un feu de surface, il n'est peut-être pas justifié d'appliquer le seuil de 60 °C à d'autres tissus exposés à des variations de température lors d'un feu.

[Traduit par la Rédaction]

Introduction

Surface fires often kill the vascular cambium near the base of a tree. As the fire front passes a tree, flames heat the bark, and heat is transferred into the stem (Martin 1961; Fahnestock and Hare 1964; Hare 1965; Reifsnyder et al. 1967; Gill and Ashton 1968; Vines 1968; Costa et al. 1991; Gutsell and Johnson 1996; Dickinson and Johnson 2001). It is well established that bark thickness is the principal determinant of heat transfer to the vascular cambium in a fire (Mar-

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tin 1961; Hare 1965; Gill and Ashton 1968). Vascular cambium temperatures rise and fall as the fire passes a tree, and the tissue is killed at some combination of exposure time and temperature (Martin et al. 1969; Mercer et al. 1994; Mercer and Weber 2001).

It has often been assumed that vascular cambium tissues are killed at a threshold temperature (e.g., 60 °C; Brown and DeByle 1987; Steward et al. 1990; Gutsell and Johnson 1996). The problem with this temperature threshold concept is that longer exposures to temperatures below the threshold or shorter exposures to temperatures above the threshold may also result in cambium death (Hare 1961; Kayll 1963). Thus a more appropriate approach could be to incorporate into the heat transfer model a temperature-dependent rate of cell death. Unfortunately, to our knowledge, there are no data that describe vascular cambium tissue mortality in response to the rise and fall of temperatures caused by heat transfer into the stem as a fire passes a tree.

Here we use two temperature-dependent rate-process models to describe mortality within populations of cells exposed to elevated temperatures. One describes a negative exponential decline in cell survival at fixed temperatures (no-lag model), while the other includes a mechanism that causes a lag before negative exponential declines in cell mortality begin (lag model). The models are adapted from research on the heat treatment of mammalian cell populations (Dewey et al. 1977; Jung 1986) and assume that temperatures are high enough and last for a short enough time that cellular acclimation and repair processes play an insignificant role and are, thus, consistent with the rapid heating of the vascular cambium during forest fires. Acclimation and repair processes relevant to chronic exposures to sublethal temperatures (e.g., Kato et al. 1995; Uchida et al. 1993) are not included.

We use the two cell mortality models to address the following questions: (1) Which model gives the best fit to the empirical data on live-bark cell survival at different fixed temperatures for four subalpine tree species? (2) Can the models predict live-bark cell survival after a rise and fall of temperatures as would be experienced in the passage of a wildfire? (3) Finally, how important is variation in the temperature dependence of the rate process model parameters when considered in the context of a rise and fall in vascular cambium temperatures caused by conduction heat transfer during surface fires?

Cell mortality models

The two models we use assume that temperature-dependent rate processes damage the molecular constituents of cells and thereby cause cell death (Johnson et al. 1974; Levitt 1980). Protein denaturation is thought to play a central role in cell death because parameters estimated from cell mortality data fall in the range for protein denaturation (Rosenberg et al. 1971). The mechanisms by which cells and tissues are killed are complex and, often, poorly known (e.g., Levitt 1980), and accordingly, the models are valuable as hypotheses to the extent that they fit cell mortality data.

The no-lag model envisions that cells are unaffected by heating until being killed in a one-step process. The chance of a cell being killed is the same (constant), no matter how long a cell has been exposed. In other words, a proportion of cells are killed in a given time period with the proportion (rate) being determined by the (unvarying) temperature. This process is defined by

$$[1] \qquad \frac{dS}{dt} = -k\,S(t)$$

where $S(t)$ is the cell survival at time t (s), and k is the rate of cell death (s^{-1}). The solution to eq. 1 is

$$[2] \qquad S(t) = \exp(-kt)$$

One drawback of the no-lag model is that it fails to account for a time lag often seen at the onset of the negative exponential rate of cell death (Lorenz 1939; Dewey et al. 1977; Bauer and Henle 1979; Onwueme 1979; Gould 1989). Consequently, the lag model postulates that cell death at elevated temperature entails two processes. First, elevated temperatures produce nonlethal lesion (i.e., protein denaturation) at random in the cell population. These lesions are not reversible and accumulate in cells as a Poisson process. Second, a separate process transforms a single nonlethal lesion in a cell death event. This is also a Poisson process with possibly a different rate from the first process. Thus, cells with the most lesions are more likely to be killed.

The lag model can be given as a compartment model (Fig. 1). At time 0, all cells have no lesions and are in compartment S_0. When the temperature is elevated, cells accrue lesions randomly at a temperature-dependent rate p (s^{-1}). The rate of efflux of cells from the first compartment S_0 is

$$[3] \qquad \frac{dS_0}{dt} = -pS_0$$

The rate of change in the proportion of cells in a compartment with 1 to n lesions (i.e., S_1 to S_n) is

$$[4] \qquad \frac{dS_n}{dt} = pS_{n-1} - pS_n - ncS_n$$

where S_n is the fraction of cells with n lesions, and c is the rate (s^{-1}) describing the conversion of nonlethal lesions to lethal lesions (i.e., cell death). The first term in eq. 4 is the influx of live cells to S_n from S_{n-1}, the second term is efflux of live cells from S_n to S_{n+1}, and the third term gives the efflux caused by cell death. The solution to eqs. 3 and 4 over all compartments from S_0 to S_∞ (Fig. 1) gives the proportion of the starting population of cells which survive to time t after exposure to a constant elevated temperature and with initial conditions $S_0 = 1$ and $S_n = 0$:

$$[5] \qquad S(t) = \exp\left(\frac{p}{c}(1 - ct) - \exp(-ct)\right)$$

In effect, cells are killed before they accumulate large numbers of lesions. As such, S_∞ is a mathematical convenience with no biological meaning.

Methods

Study area

Live bark was sampled from canopy trees of trembling aspen (*Populus tremuloides* Michx.), Engelmann spruce (*Picea engelmannii* Parry ex. Engelm.), Douglas-fir (*Pseudotsuga*

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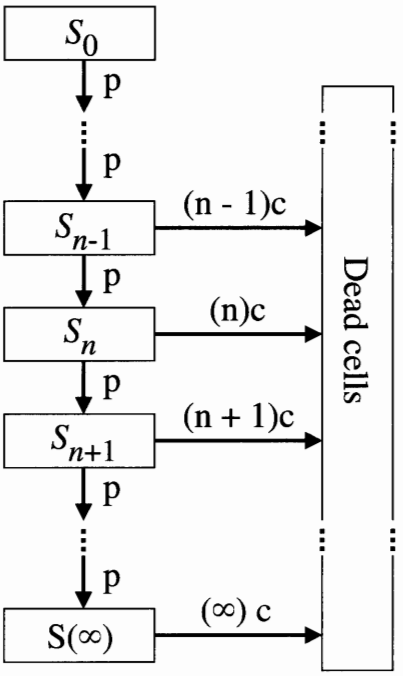
Fig. 1. Compartment model for the lag model of cell survival (see Jung 1986). All cells initially have no lesions, i.e., $S_0 = 1$. Nonlethal lesions ($n = 1 \rightarrow \infty$) accumulate at random in the population of cells at a rate p that is proportional to temperature. Nonlethal lesions are converted to lethal events for cells at a rate c that also is proportional to temperature, i.e., cells with more nonlethal lesions are more likely to be killed than cells with fewer such lesions. A subset of compartments are shown in the figure; ellipses indicate missing compartments.



menziesii (Mirb.) Franco), and lodgepole pine (*Pinus contorta* Dougl. ex Loud.). All trees were within 5 km of the Barrier Lake Station of the Kananaskis Field Stations (51°01′N, 115°02′W, elevation 1335 m). Aspen were in almost pure closed-canopied stands with an understory of *Elymus innovatus* Beal and a diversity of other herbaceous species. Engelmann spruce were in spruce-dominated closed-canopied stands with minor component of lodgepole pine, with an understory of *Shepherdia canadensis* (L.) Nutt., *Cornus canadensis* (L.), and *Ledum groenlandicus* Oeder, and a ground cover of feathermosses (*Pleurozium* spp.). Lodgepole pine were in pine-dominated closed-canopied stands with minor components of spruce and an understory of *Arctostaphylos uva-ursi* (L.) Speeng., *Vaccinium* spp., and *Linneus borealis* L. The Douglas-fir were in an open stand with Engelmann spruce and an understory of *Elymus*.

In fall–winter (September–March), 65 aspen and 55 spruce trees were sampled, and in spring–summer (April–August), 58 aspen, 82 spruce, 111 pine, and 55 Douglas-fir were sampled to estimate parameters of the cell-death models. During the spring–summer period, 92 aspen, 111 spruce, 234 pine, and 108 Douglas-fir were sampled to compare cell survival after a rise and fall of temperatures against predictions of the cell-death models.

Sample preparation

A sample of live bark was removed by chisel from each tree, wrapped in paraffin film, and transported immediately (within 0.5 h) to the laboratory. Live bark contains vascular cambium and phloem tissues. The samples were kept at ambient temperature during transport. Four cross sections (5 mm long, 2 mm wide, and 0.6 mm thick) were cut with a razor from each sample and placed in 0.05 mol/L potassium phosphate buffer (7.2 pH) at 20 °C (see Caldwell 1993). The cross sections were divided into control and treatment groups. The control groups were kept in buffer at room temperatures until being infused with stain at the same time as their treatment group.

Constant temperature treatment

Cross sections from each tree were put in open vials containing 20 mL of buffer solution that had been allowed to equilibrate in a water bath at the treatment temperature. Treatment temperatures were those of the buffer solution in the vials, not the water bath's temperature. Treatment temperatures ranged from 43 to 65 °C. Cross sections were thermally thin with a time constant of about 2 s (i.e., the time required for sample temperature to reach 63% of a temperature rise defined by the difference between an initial and treatment temperature). As such, the samples reached the treatment temperature rapidly. Samples were removed from the vials at evenly spaced intervals and cooled rapidly. The longest heating time was chosen with the goal that a small proportion cells would remain alive.

A heat treatment consisted of two cross sections from each of three to five trees heated at the same temperature for the same amount of time. A replicate consisted of all the heat treatments (i.e., different times of exposure to the same temperature) conducted on the same day. A single estimate of the rate constants k (no-lag model) and c (two-hit model) was made for each replicate (see below).

Cell counts

Control (unheated) and treatment (heated) cross sections were placed in neutral red stain (20 ppm) (Lorenz 1939) after each heat treatment. The stain was made fresh in buffer each day. Neutral red is a vital stain that is sequestered in live cells with intact membranes. The stain was vacuum infiltrated to ensure consistent staining throughout the tissue sections. The sections remained in the stain for 1 h. After staining, the cross sections were placed under cover slips and viewed with a compound light microscope at a magnification of × 40. Cells that stained bright pink were counted as alive. Three counts were made on different portions of each cross section over the entire field of view; counts were composed primarily of phloem parenchyma, since vascular cambium cells stained poorly. Survival was estimated by dividing the mean count for a heated cross section (treatment) by the mean count from an unheated cross section (control).

Model parameters and goodness of fit

The k parameter of the no-lag model (eq. 2) was estimated from the cell survival data for each replicate temperature treatment by a nonlinear least-squares routine (PROC NLIN, SAS Institute Inc., Cary, N.C). The rate parameters p and c of the lag model (eq. 5) were estimated as follows. The p parameter (s^{-1}) was calculated from

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$$[6] \qquad p = \frac{k_B T}{h} \exp\left(\frac{\Delta S}{R}\right) \exp\left(-\frac{\Delta H}{RT}\right)$$

where T is temperature (K), k_B is the Boltzman constant (1.38×10^{-23} J·K^{-1}), h is Planck's constant (6.63×10^{-34} J·s), ΔS is the activation entropy (J·mol^{-1}·K^{-1}), R is the universal gas constant (8.31 J·mol^{-1}·K^{-1}), and ΔH is the activation enthalpy (J·mol^{-1}). Activation entropy and enthalpy were set to 482 J·mol^{-1}·K^{-1} and 248 410 J·mol^{-1}, respectively, values that correspond to the denaturation kinetics of an average protein in Rosenberg et al. (1971). With p determined, a nonlinear least-squares routine (PROC NLIN) was then used to estimate the c parameter in eq. 5 from cell survival data. Note that we do not correct the rate parameter estimates for the delay experienced by samples in reaching the prescribed treatment temperature (Perkin et al. 1977). The delay was short in our experiments because the samples were thermally thin (see above), and the correction was trivial when the appropriate analyses were conducted. The delay begins to result in appreciable bias at treatment temperatures greater than those we report here.

Equation 6 is derived from the thermodynamics and statistical mechanics of simple biochemical reactions (Johnson et al. 1974) and does not have a precise interpretation in the context of cell populations (Jung 1986; Caldwell 1993). Our strategy for calculating parameters is due to the fact that unique estimates of both parameters could not be obtained simultaneously because they were highly correlated (see Reich 1981).

How well the models described the cell survival data was examined by comparing the mean difference between observed and fitted survival values by species and season. The heating times for each replicate fixed temperature experiment were rescaled so that data from low temperatures could be compared with data from high temperatures. The rescaling was accomplished by dividing the heating time by the maximum heating time for that replicate. We expected from the literature that observed survival would lag behind fitted survival for the no-lag model early in the heating treatments.

Rate parameters estimated from the no-lag (k, eq. 2) and lag models (c, eq. 5) were examined for their dependencies on temperature, species, and season. Differences between seasons in the temperature dependence of the rate parameters were determined by regression analyses (PROC GLM, SAS Institute Inc., Cary, N.C.) in which temperature was the dependent variable, and the natural log of the appropriate rate parameter was the independent variable. A difference between seasons indicates that the slope or intercept of the relationships or both differ. Differences between species in the natural-log transformed rate parameters k and c were determined by planned contrasts after regression (PROC GLM) in which species and temperature were dependent variables. Again, a difference between species indicates that the slope or intercept of the relationships or both differ. Thermodynamic parameters (ΔS and ΔH) describing the temperature dependence of the rate parameters k and c were estimated for each species, season, and temperature range from eq. 6 by a nonlinear least-squares routine (PROC NLIN).

Validation of the cell-survival models for rising and falling temperatures

Cross sections of live bark were heated over a rise and fall in temperatures, as would be experienced in forest fires (Martin 1961; Fahnestock and Hare 1964). Cross sections from each tree were placed in a beaker with 100 mL of buffer and heated on a hot plate to a peak temperature ranging from 43 to 65 °C. The beaker was then removed from the heat and allowed to cool. The buffer was stirred continuously during heating and cooling. Temperatures of the buffer solution were recorded every second by means of a datalogger and a resistance-type thermometer encased in a high-conductivity sheath. Control cross sections from the same bark sample were stirred in 100 mL of buffer at 20 °C for the duration of the corresponding heating treatment. Control and heated cross sections were stained, and cell survival was determined as previously described.

Cell survival after the rise and fall of temperatures was compared with survival predicted by the no-lag and lag models. The temperature regime used to predict survival was that measured during the heating process (see above). The no-lag and lag model differential equations (eqs. 1 and 3, and 4, respectively) were rewritten as difference equations and used in a numerical bookkeeping routine. Analytical solutions to the no-lag (Zsakó 1970) and lag (Jung 1986) models for nonconstant temperature regimes exist but remain a challenge for the rise and fall of temperatures experienced by the stem vascular cambium in surface fires. Survival was initially 1.0 in both models, and the time stepped at 1-s intervals. From eq. 1, survival in the next time step for the no-lag model is

$$[7] \qquad S(t+1) = S(t) - k(T)\,S(t)\Delta t$$

where $S(t)$ is current survival, $k(T)$ is the rate parameter (s^{-1}) at the temperature of the current time step (see below), and Δt is the time step (s). The proportion of cells in each compartment and overall survival in the lag model were similarly updated each time step. For the lag model, the proportion of cells with no lesions in the next time step (from eq. 3) is

$$[8] \qquad S_0(t+1) = S_0(t) - p(T)\,S_0(t)\Delta t$$

where $S_0(t)$ is the proportion of cells with no lesions at the current time step, $p(T)$ is the rate parameter governing the occurrence of lesions (s^{-1}) at the temperature of the current time step (see below), and Δt is the time step (1 s). The proportion of cells with $n \geq 1$ lesions becomes

$$[9] \qquad S_n(t+1) = S_n(t) - [p(T)S_{n-1}(t) - p(T)S_n(t) - nc(T)S_n(t)]\Delta t$$

where $S_n(t)$ is the proportion of cells with n lesions at the current time step, and $c(T)$ is the rate parameter that governs cell death at the current temperature. The total number of compartments in the lag model was set to $n_{max} = 100$ because cells accumulated many fewer than 100 lesions before being killed. Survival in the lag model is the sum of the proportion of cells in compartments S_0 and S_1 to S_{100} at the end of each time step (see Fig. 1). The rate parameters c and k were calculated from eq. 6 using the activation entropy and enthalpy values estimated from fixed temperature exposures for the species and season of interest (Table 1) and the tem-

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Table 1. The thermodynamic parameters that describe the temperature dependence of the rate parameters in Figs. 4–6.

		No-lag model, k		Lag model, c	
Species	Range (°C)	ΔS	ΔH	ΔS	ΔH
Fall–Winter					
Aspen (N = 17)	43 to 65	372 (34)	217 524 (11 303)	230 (62)	174 019 (20 245)
Spruce (N = 11)	43 to 65	455 (99)	246 032 (32 297)	473 (102)	255 392 (33 255)
Spring–summer					
Aspen (N = 10)	43 to 65	665 (42)	312 522 (13 879)	822 (85)	365 925 (27 753)
Spruce (N = 12)	53 to 65	575 (60)	283 968 (19 732)	823 (112)	367 364 (36 714)
Douglas-fir (N = 15)	43 to 65	528 (65)	270 036 (21 118)	564 (128.6)	285 164 (42 029)
Pine (N = 17)	43 to 65	518 (52)	267 984 (17 039)	—	—
	43 to 53	—	—	–272 (185)	20 195 (59 406)
	53 to 65	—	—	1139 (230)	478 828 (76 127)

Note: Activation entropy (ΔS, J·mol^{-1}·K^{-1}) and enthalpy (ΔH, J·mol^{-1}) and their standard errors (in parentheses) for the no-lag (k) and lag (c) cell-survival models were estimated from rate parameter estimates and eq. 6. The number of replications is given in parentheses beside each species' name. Separate estimates of the lag model parameters for temperatures above and below 53 °C are given for lodgepole pine.

perature at that time step. The rate parameter p at each time step was calculated from eq. 6 using literature values of the thermodynamic parameters (above).

Linear regressions were used to compare predicted with observed cell survival after the rise and fall of temperatures. Observed survival values of zero were removed from the data because survival converged on zero when peak temperatures were high. Survival could not fall below zero because survival is a ratio of cell counts from control and heated tissue sections. In contrast, observed survival values >1 were commonly observed because of sampling error after samples were heated to low peak temperatures. If survival values of zero had been retained in the analyses, intercepts would have been biased towards the expected value of zero, and the proportion of variance explained would have been inflated.

Heat transfer into tree stems

The importance of variation in the temperature-dependent rate parameters and the relevance of the threshold concept of tissue necrosis can only be examined in the context of realistic vascular cambium temperature regimes. This means that conduction of heat into the stem from the flames must be modeled as well as the cooling process once the flames pass. We use the infinite-slab model to simulate unsteady-state conduction into a tree stem (Martin 1961; Hare 1965; Reifsnyder et al. 1967; Gill and Ashton 1968). Conduction is unsteady because the temperature gradient through the stem is not constant with time, reflecting passage of the fire. The tree trunk is assumed to be an evenly heated flat surface (slab) that is infinitely thick, and thus, heat transfer is one-dimensional. The one-dimensional assumption is reasonable for all but small saplings during surface fires in which heat transfer through the middle of the stem is important (Jones 2003). A flat surface means that fissuring is assumed to be unimportant, an assumption that is not reasonable for trees, often of large diameters, with highly fissured bark (Smith and Sutherland 2001). Model assumptions and their consequences are reviewed in Dickinson and Johnson (2001) and Jones (2003).

Fig. 2. Examples of the fit for aspen between the no-lag (dotted line) and lag (solid line) models and empirical cell survival (closed symbols ± 1 standard error) during the spring–summer period. Fixed water bath temperatures (°C) are given next to each pair of lines. Note that the timescale is shorter for the higher temperature treatments.

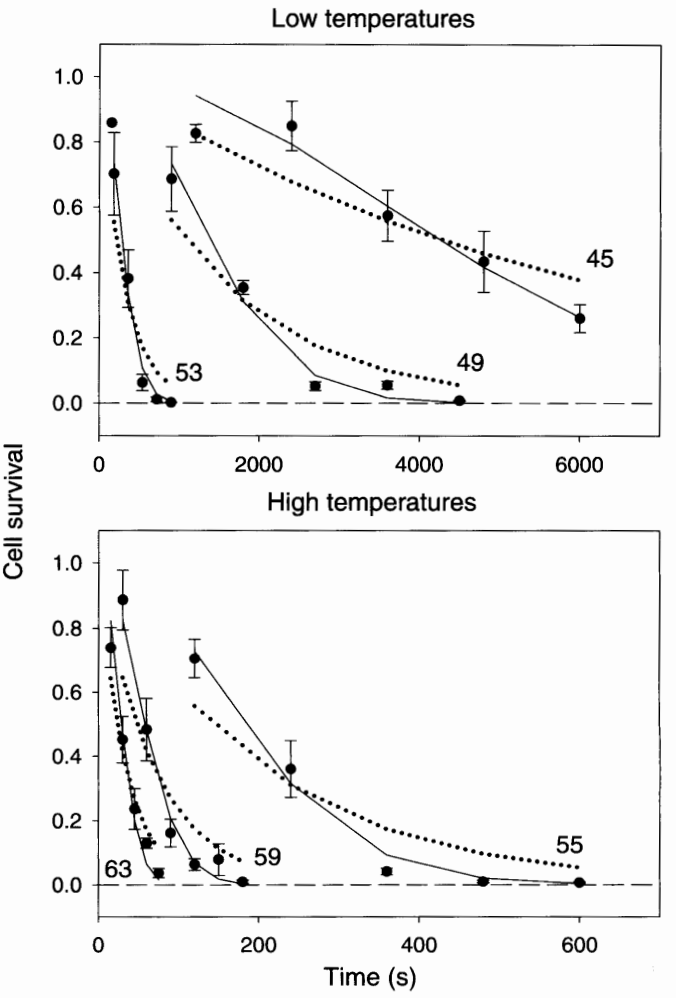

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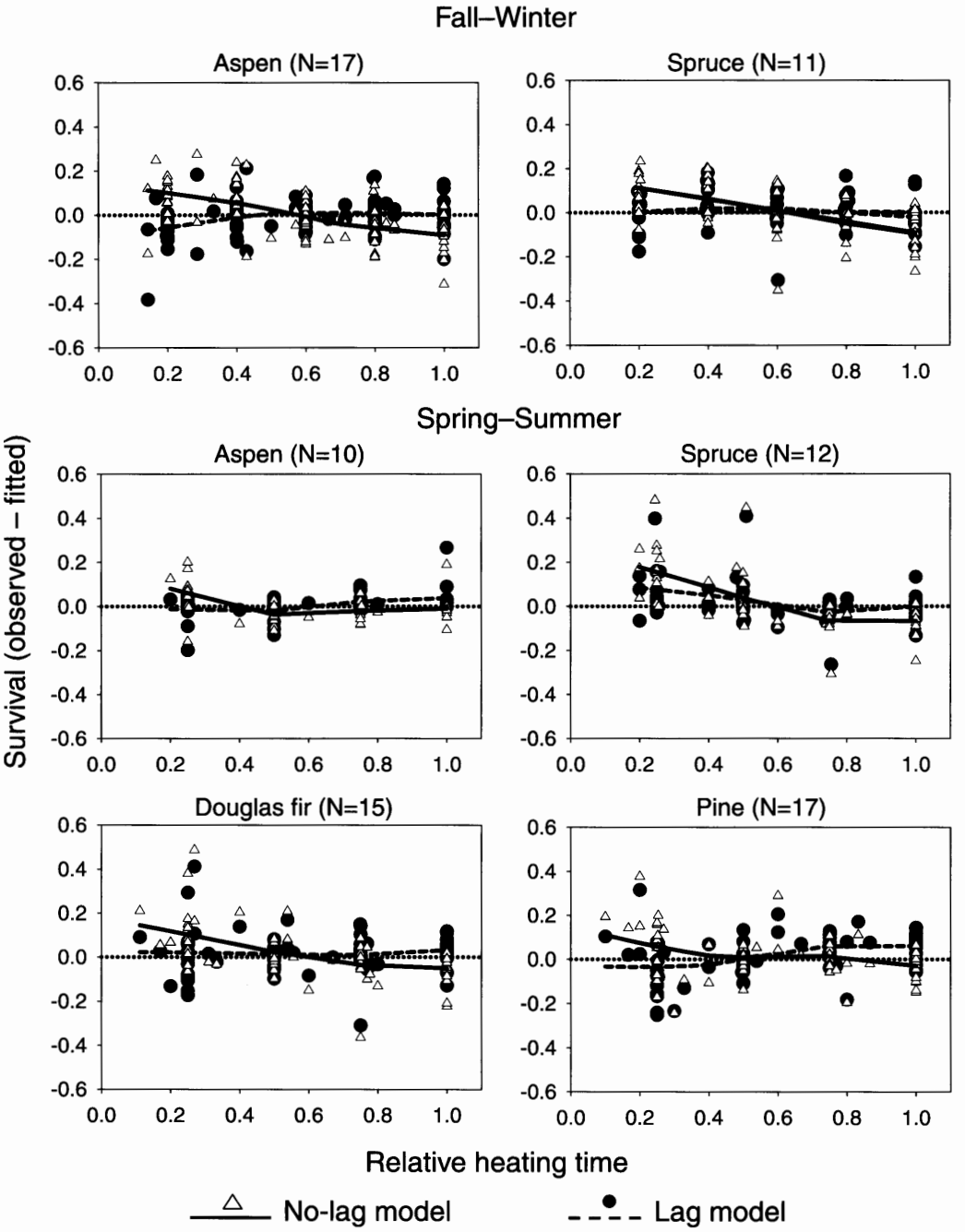
Fig. 3. The difference between observed and fitted cell survival for the lag and no-lag rate-process models. Relative heating time is the actual heating time divided by the longest time of exposure for a given replicate (i.e., a series of exposures to a given fixed temperature). Locally weighted regression curves (f = 0.7) are given to describe trends. Perfect agreement between observed and fitted cell survival corresponds to the broken line where the difference between observed and expected survival equals zero. We expect that any lag in cell mortality rates would be revealed during relatively short exposures.



The infinite-slab conduction model is

$$[10] \quad \frac{\partial T}{\partial t} = \alpha \frac{\partial^2 T}{\partial x^2}$$

where T is temperature (°C) at a given depth x (m) measured in from the surface of the bark, t is time (s), and α is thermal diffusivity ($m^2 \cdot s^{-1}$). Equation 10 states that the change in temperature through time at some depth within the tree trunk, $\partial T/\partial t$, is proportional to the gradient in temperature through the trunk, $\partial^2 T/\partial x^2$, at some depth. The thermal diffusivity tells how easily the temperature gradient raises the temperature of the material. Equation 10 has generally been solved analytically to approximate the rise in vascular cambium temperatures during a fire. However, cell survival is determined during the entire stem heating and cooling

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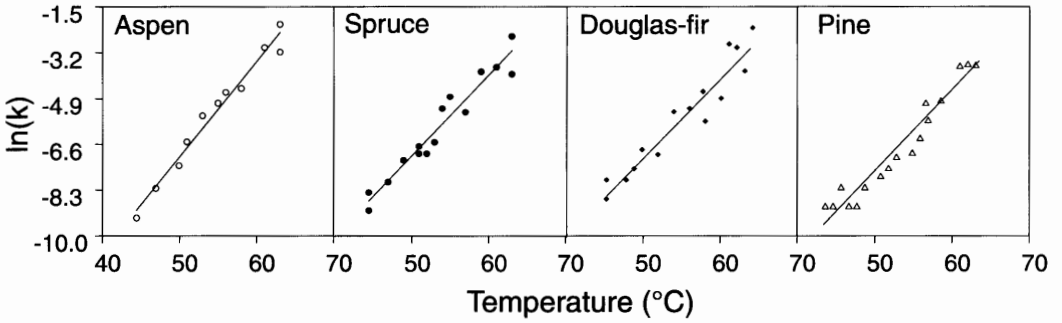
Fig. 4. Effect of species and temperature on the rate parameter k of the no-lag model estimated from tissue collected during the spring and summer. The lines are the linear least-squares regression of the log-transformed rate parameter estimates on temperature. Planned contrasts between species are shown.



Contrast (df = 1)	MS	F	P
Aspen vs. Spruce	1.0	0.6	0.44
Aspen vs. Fir	0.3	0.2	0.69
Aspen vs. Pine	12.8	7.4	0.01
Spruce vs. Fir	0.3	0.2	0.67
Spruce vs. Pine	7.6	4.5	0.04
Fir vs. Pine	11.5	6.7	0.01

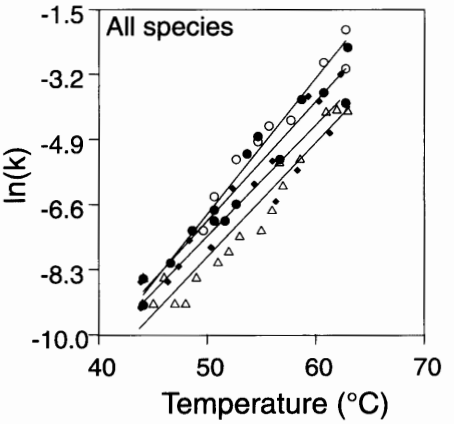
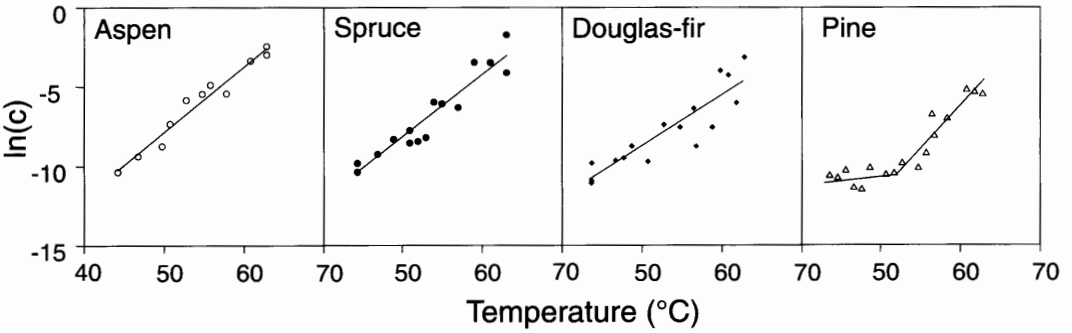


Fig. 5. Effect of species and temperature on the rate parameter c of the lag model estimated from tissue collected during the spring and summer. The lines are the linear least-squares regression of the log-transformed rate parameter estimates on temperature. Planned contrasts between species are shown. The contrasts for lodgepole pine only involve temperatures above 53 °C.



Contrast (df = 1)	MS	F	P
Aspen vs. Spruce	1.0	1.2	0.28
Aspen vs. Fir	10.8	12.5	<0.01
Aspen v.s Pine	43.3	50.3	<0.01
Spruce vs. Fir	6.3	7.3	<0.01
Spruce vs. Pine	38.0	44.0	<0.01
Fir vs. Pine	3.4	15.5	<0.01

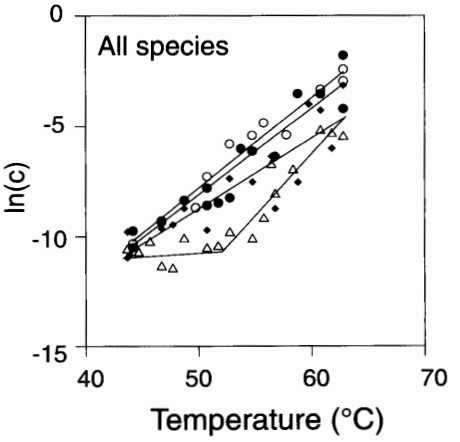


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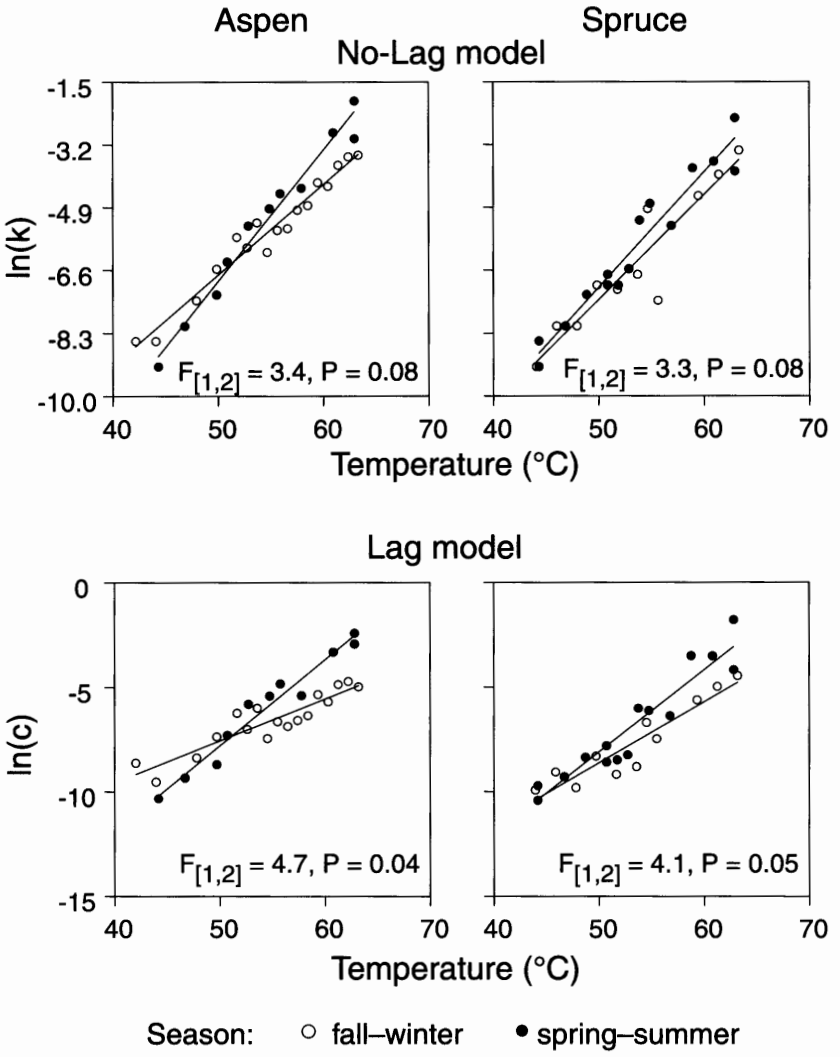
Fig. 6. Effect of season and temperature on rates of cell death. The lines are the linear least-squares regressions of the log-transformed estimates of the rate parameters k and c from the no-lag and lag models, respectively, on temperature. The F tests for differences between seasons are given.



process, a situation for which useful analytical solutions remain a challenge. Accordingly, we use the forward difference form of eq. 10 (cf. Rego and Rigolot 1990) to model both the relatively rapid heating and the much slower cooling of the vascular cambium:

$$[11] \quad T(i,t+1) = T(i,t) + \frac{1}{\alpha}\frac{\Delta t}{(\Delta x)^2}[T(i-1,t) - 2T(i,t) + T(i+1,t)]$$

where i is the depth increment of Δx (mm), and t is the time increment of Δt (s). A time-varying temperature was prescribed (see below) at the surface, i.e., $T(0, t) = T(t)$. Thermal diffusivity varies minimally among dead outer bark, live bark, and wood (Dickinson and Johnson 2001), and thus, layers of material were not distinguished. For eq. 11 to be computationally stable, the following condition must be met:

$$[12] \quad \Delta t \leq \frac{(\Delta x)^2}{2\alpha}$$

A suitable value was Δt = 2.5 s.

For simplicity, we assume that the bark surface temperature rises immediately to a constant temperature determined by the balance among radiant and convective heating by the flame, heat losses from reradiation into the surroundings, and conduction into the tree. We set bark surface temperatures during the residence time of the flames at 320 °C, an average bark surface temperature measured during experimental fires conducted by Pinard and Huffman (1997). If heat transfer into the stem were governed by this bark-surface temperature regime, however, the model would substantially over predict vascular cambium temperatures because the heat sink associated with the vaporization of water constrains material temperatures near 100 °C until the material dries. Thus, an approximate solution is to constrain bark temperatures below the bark surface at 100 °C (Rego and Rigolot 1990).

Although the most of the bark may not experience temperatures above 100 °C, a certain proportion of the outer bark is desiccated during fires. To capture some of the effect

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Fig. 7. Observed and predicted cell survival after tissues were exposed to rising and falling temperatures in a water bath.

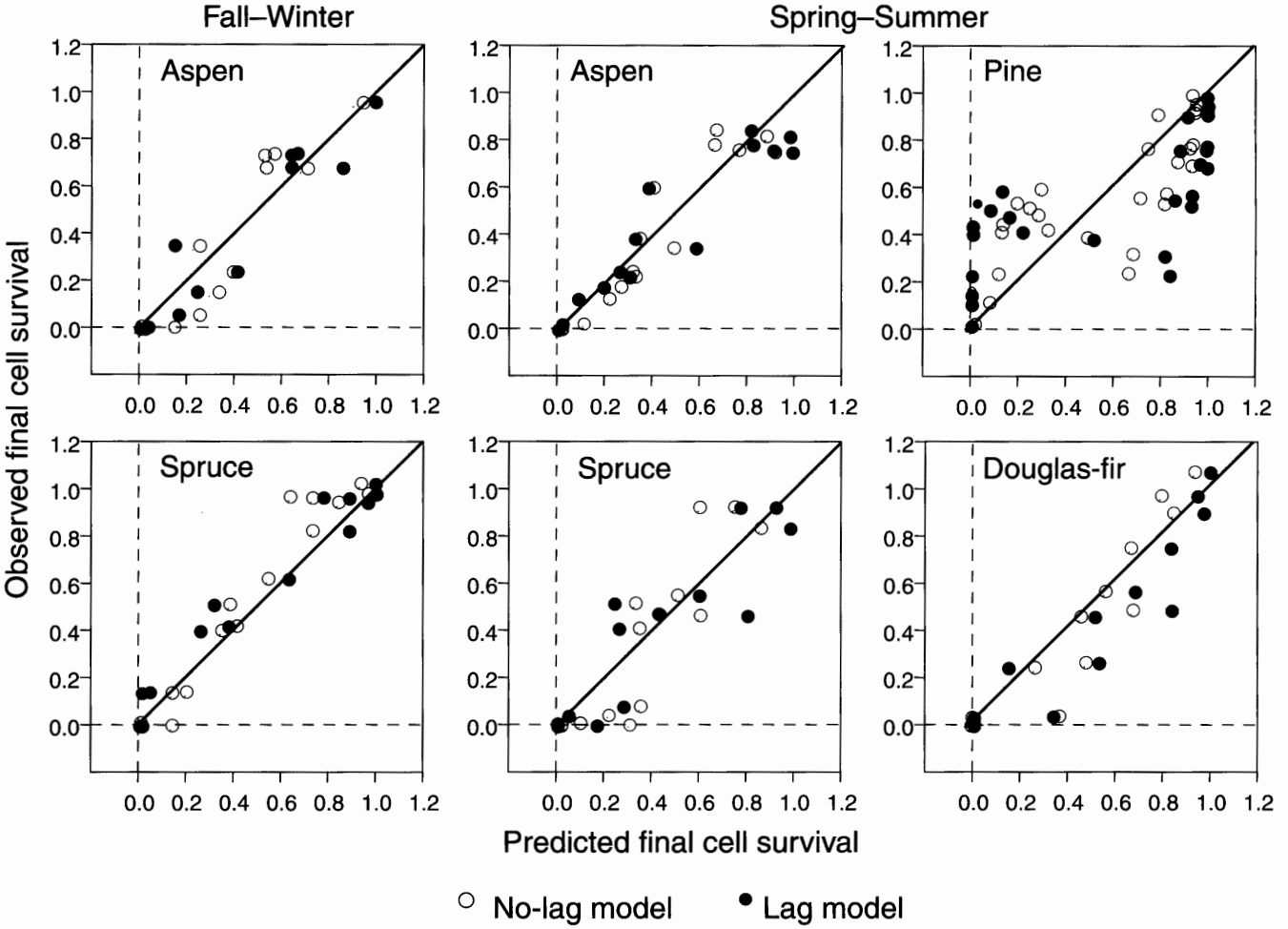

Table 2. Fit between observed cell survival and survival predicted by the no-lag and lag models after exposure to rising and falling temperatures.

		95% confidence interval				
	Regression model	Intercept	Slope	N	R^2	P
Fall–winter						
Aspen, no lag	observed = 0.05 + 0.84(modeled)	–0.28 to 0.37	0.23 to 1.44	12	0.48	0.012
Aspen, lag	observed = 0.14 + 0.64(modeled)	–0.11 to 0.39	0.22 to 1.06	12	0.53	0.007
Spruce, no lag	observed = 0.01 + 1.14(modeled)	–0.15 to 0.17	0.89 to 1.39	12	0.91	<0.0001
Spruce, lag	observed = 0.15 + 0.87(modeled)	0.05 to 0.24	0.74 to 1.00	12	0.95	<0.0001
Spring–summer						
Aspen, no lag	observed = 0.02 + 1.01(modeled)	–0.24 to 0.27	0.55 to 1.47	16	0.61	0.0003
Aspen, lag	observed = 0.06 + 0.83(modeled)	–0.14 to 0.27	0.51 to 1.16	16	0.68	<0.0001
Spruce, no lag	observed = –0.08 + 1.22(modeled)	–0.26 to 0.10	0.81 to 1.63	14	0.78	<0.0001
Spruce, lag	observed = 0.02 + 0.90(modeled)	–0.12 to 0.16	0.63 to 1.17	14	0.81	<0.0001
Douglas-fir, no lag	observed = –0.10 + 1.10(modeled)	–0.31 to 0.10	0.74 to 1.47	14	0.78	<0.0001
Douglas-fir, lag	observed = –0.06 + 0.92(modeled)	–0.26 to 0.13	0.62 to 1.23	14	0.78	<0.0001

Note: Results for lodgepole pine are not included because observed survival is not a linear function of predicted survival (Fig. 7).

of desiccation, the fall in temperatures at the bark surface after flames extinguished was determined by a Newtonian cooling equation (e.g., Weber et al. 1995):

$$[13] \quad T_s(t) = T_a + \exp(-\gamma t)(T_i - T_a)$$

where $T_s(t)$ is the bark surface temperature at time t, T_a is ambient temperature (set at 20 °C), T_i is the temperature of the bark's surface at the beginning of the cooling period (320 °C), and γ is called the Newtonian cooling parameter (s^{-1}). The Newtonian cooling parameter (0.005 s^{-1}) was esti-

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mated from bark surface cooling after experimental flames extinguished (M. Pinard and J. Huffman, unpublished data; see Pinard and Huffman 1997). Thus, after the flames go out, bark surface temperatures fall at a declining rate as they approach ambient temperature.

Results

All species tested showed a decline in cell survival with time at fixed temperatures. Here we show example curves for aspen (Fig. 2). In no case can the decline in cell survival be called instantaneous (very steep slope) except at the highest temperature.

The lag model tended to fit the cell survival data better than the no-lag model (Fig. 3). The no-lag model overpredicted cell mortality rates shortly after heating began as would be expected if a lag in the onset of rapid cell mortality rates were present. In contrast, the lag model predictions were close to observed values except for spruce during the summer period and lodgepole pine.

Figures 4 and 5 show that the k and c parameters of the two rate-process models increase exponentially with temperature. Temperature was a highly significant factor in all analyses ($P < 0.0001$). For the k parameter (Fig. 4), planned contrasts between lodgepole pine and all other species were significant. For the c parameter (Fig. 5), lodgepole pine showed a change in the temperature dependence of the rate parameter at about 53 °C. Planned contrasts (Fig. 5) between species (including fixed temperatures above 53 °C for lodgepole pine) showed a significant difference between lodgepole pine and the other three species and between Douglas-fir and aspen and Engelmann spruce. Aspen and spruce (Fig. 6) showed no significant seasonal change in the temperature dependence of the k parameter of the no-lag model but showed small but significant differences in the c parameter of the lag model. The thermodynamic parameters estimated from the data in Figs. 4–6 are given in Table 1; these parameter values are used from here on to describe rate parameter temperature dependencies (eq. 6) for predicting and simulating cell mortality.

The performance of both cell survival models for rising and falling water bath temperatures (Fig. 7, Table 2) was reasonable for all species except lodgepole pine. For aspen, spruce, and Douglas-fir, the 95% confidence intervals for the slopes of the linear regressions between observed and modeled final cell survival included unity. As well, the 95% confidence intervals for the y intercept included zero with the exception of the lag model for spruce during the fall–winter period.

Vascular cambium temperature regimes used for simulating the effects of fires were generated with the conduction heat transfer model (eqs. 8–10) for trees with a range of bark thicknesses. These temperature regimes were then used to run the cell survival models (eqs. 7–9). Figure 8 gives examples of vascular cambium temperature regimes and resulting cell mortality.

Figure 9 shows that there were only small differences in final cell survival among aspen, spruce, and Douglas-fir during the spring–summer period. This is not unexpected given the small differences among species in the temperature dependence of the no-lag and lag model rate parameters (Figs. 4 and 5). Lodgepole pine, however, showed somewhat greater final cell survival than the other species. Recall (Fig. 7) that we had limited confidence in our predictions of lodgepole pine cell survival after a rise and fall of temperatures. In line with small seasonal differences in the temperature dependence of cell mortality rates for aspen and spruce (Fig. 6), there were minor differences between seasons in final cell survival over a range of bark thicknesses (not shown).

There were also only small effects of increasing total heat transfer (increasing flame residence time (t_r) and thermal diffusivity (α)) on the relationship between final cell survival and peak vascular cambium temperature. However, increases in total heat transfer cause a given level of final cell survival to occur in stems with increasing bark thickness. Figure 10

Fig. 8. Effect of a simulated surface fire on the vascular cambium. (A) Example of vascular cambium temperature regimes simulated with the conduction heat-transfer model (eq. 11) for three bark thicknesses. Flame residence time is 45 s, and bark thermal diffusivity is 0.1 $mm^2 \cdot s^{-1}$. (B) Decline in cell survival during exposures to temperature regimes in Fig. 8A. Cell survival was predicted with the no-lag model (eq. 7). Thermodynamic parameters for aspen during the growing season (Table 1) were used to describe the temperature dependence of the rate parameters.

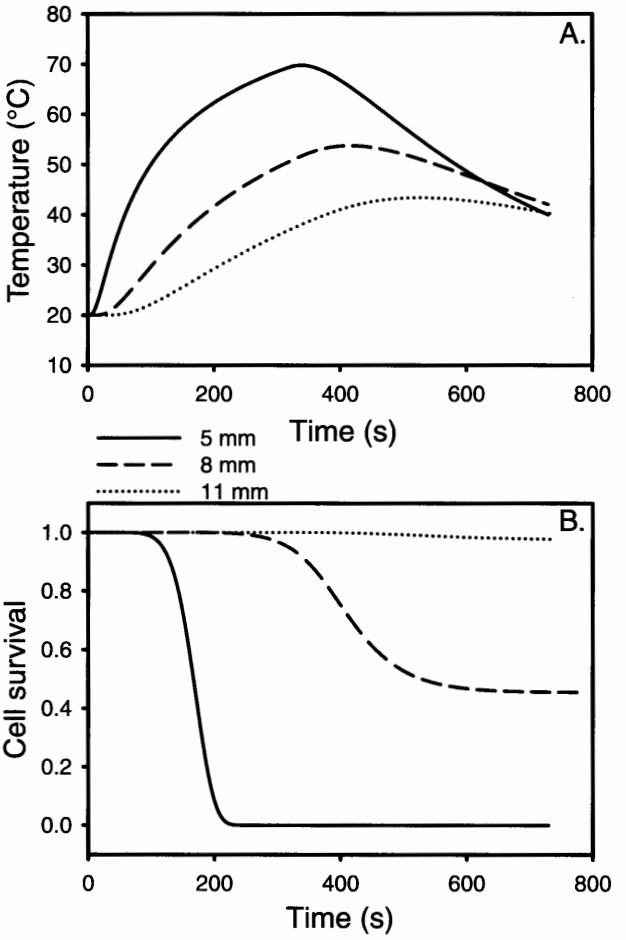

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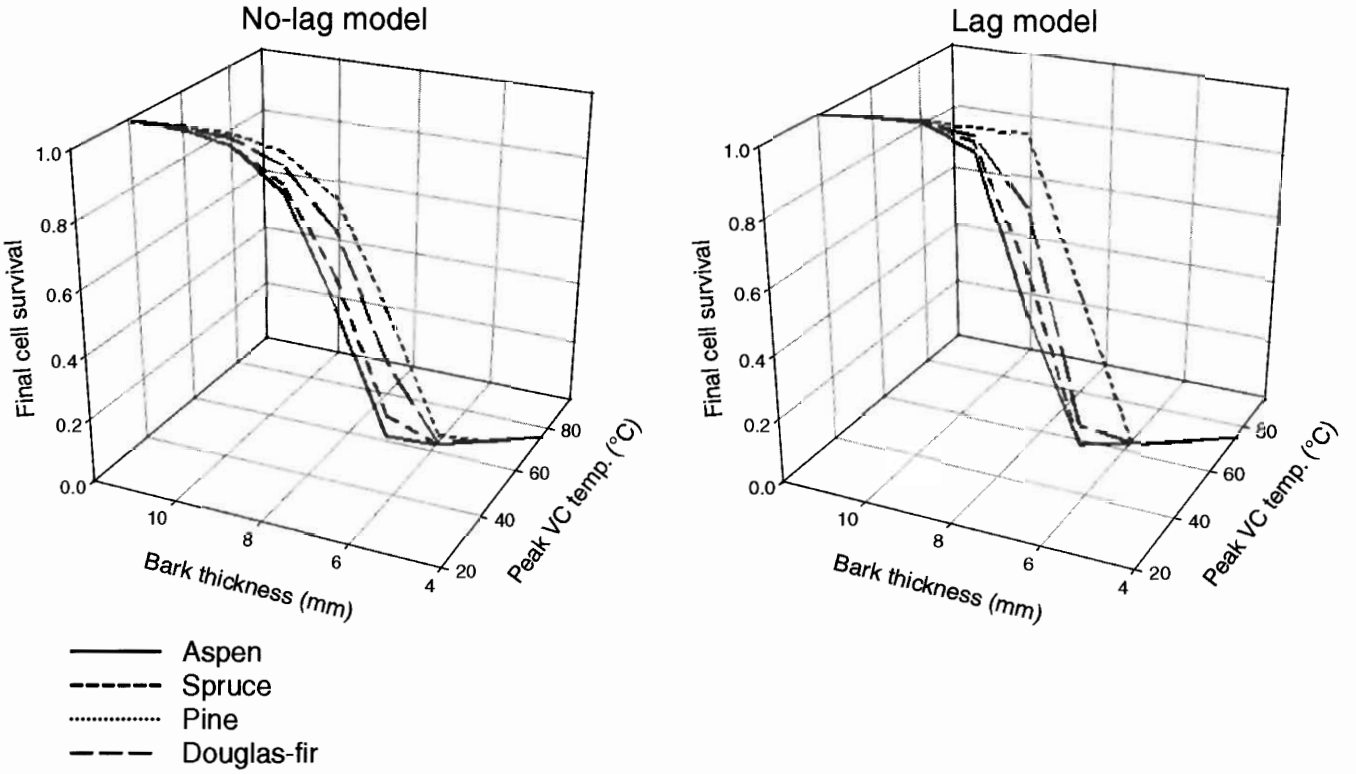
Fig. 9. Differences among species during the spring–summer period in modeled vascular cambium cell survival after a fire. Vascular cambium (VC) temperature regimes for the range of bark thicknesses were generated with the one-dimensional conduction heat-transfer model for use as the boundary condition in the cell-survival models (see Methods, Fig. 7). The flame residence time was 45 s, and bark thermal diffusivity was 0.1 $mm^2 \cdot s^{-1}$ in the simulation. Thermodynamic parameters from Table 1 were used to describe the temperature dependence of the rate parameters in the cell survival models.



shows an example using aspen during the spring–summer period; the pattern is consistent among species.

Discussion

All four species tested showed a strong dependence between temperature and the rates governing cell mortality (Figs. 4–6). Thus, the argument that cells die at some threshold is, at best, a simplification. Live-bark cells appear to behave as other animal and plant cells (Johnson et al. 1974; Levitt 1980) in that there is an exponential dependence between mortality rates and temperature. Also, aspen, spruce, Douglas-fir, and lodgepole pine exhibited a lag before the onset of exponential declines in cell survival at fixed elevated temperatures (Fig. 3). This lag has been found previously in both plant (e.g., Lorenz 1939; Caldwell 1993) and animal cells (Dewey et al. 1977; Jung 1986) and led to the development of the lag model.

There was little difference among aspen, spruce, and Douglas-fir in the temperature dependence of the k and c rate parameters of the no-lag and lag models (Figs. 4 and 5). In contrast, lodgepole pine tissues had better survival rates at elevated temperatures than the other species. For lodgepole pine, the temperature dependence of the rate parameter c from the lag model showed an increase in slope at about 53 °C. A break in rate parameter temperature dependence has been found in other plants (e.g., Caldwell 1993) and in animal cells (e.g., Dewey et al. 1977; Jung 1986). Perhaps different mechanisms of cell damage operate above and below the break. The break was not apparent for the k rate parameter of the no-lag model, but this is not entirely surprising because the fit of the no-lag model to the cell survival data is more approximate.

Aspen and spruce showed small increases in thermal tolerance at high temperatures during the fall–winter period (dormant season, Fig. 6). The seasonal differences were marginally significant for the rate parameter c of the lag model but not significant for the rate parameter k of the no-lag model. The seasonal differences provide mixed support for the possibility that winter dormancy may, in some situations, confer tolerance for both freezing and high temperatures (Levitt 1980). In contrast to our results, if fires selected for tissue thermal tolerance, one would expect greater thermal tolerance during the growing season because most of area burned has occurred during the late summer (July and August) in recent decades (Johnson and Wowchuk 1993) and, presumably, during some portion of the growing season over recent millennia. Alternatively, to the extent that these species' population dynamics are dominated by stand replacing fires (e.g., lodgepole pine, Engelmann spruce) (Johnson and Fryer 1989; Johnson et al. 1994), no differences among seasons in thermal tolerance because of adaptation to fire would be expected.

A simulation model involving conduction heat transfer and tissue response suggests that the differences among species in temperature-dependent rates may be roughly equivalent to a 1–4 mm change in bark thickness (Fig. 9). This result is robust to changing heat transfer conditions

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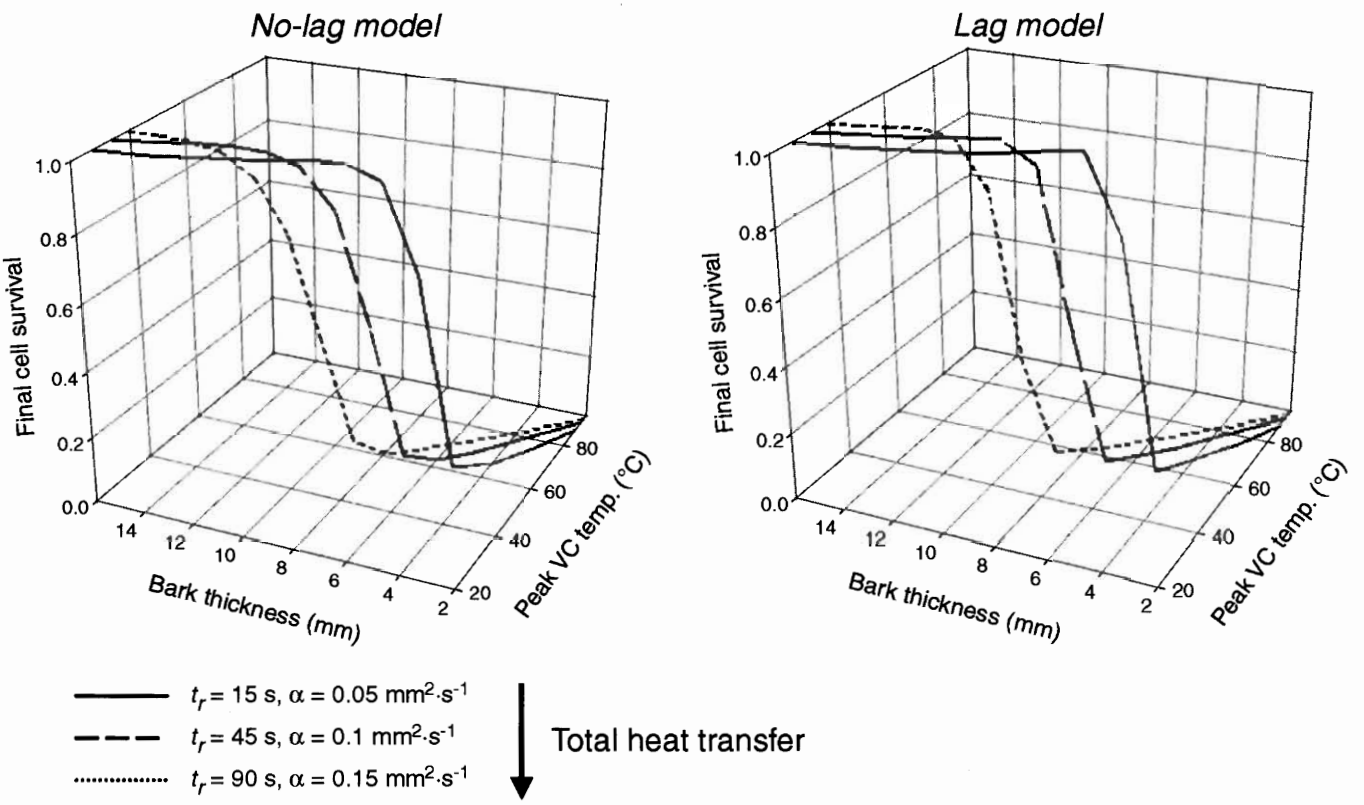
Fig. 10. Modeled effect of changing flame residence time (t_r) and thermal diffusivity (α) on aspen cell survival during the spring–summer period. Vascular cambium (VC) temperature regimes for the range of bark thicknesses and combinations of flame residence times and thermal diffusivities were generated with the one-dimensional conduction heat-transfer model for use as the boundary condition in the cell-survival models (see Methods, Fig. 7). Thermodynamic parameters from Table 1 were used to describe the temperature dependence of the rate parameters in the cell survival models.



(Fig. 10). Increasing total heat transfer (by using residence time and thermal diffusivity) results in a given level of final cell survival occurring in trees with thicker bark but does not change the shape of the relationship between final cell survival and temperature. Lodgepole pine has the thinnest bark of the four species (Ryan and Reinhardt 1988) and has the lowest temperature-dependent rate (higher survival at each temperature); could this be of some ecological importance?

Our results would seem to support the use of a 60 °C threshold for vascular cambium necrosis within stems exposed to surface fires. Cell mortality was complete, or nearly complete, for all species when peak vascular cambium temperatures reached 60 °C during simulated fires (Fig. 9). This result is robust to substantial variation in heating regime (see Fig. 10 for an example). Despite these results, it is not warranted to apply the 60 °C threshold to other tissues such as roots, foliage, branches, buds, and seeds. Not only may there be differences among tissues in thermal tolerance (e.g., Martin and Cushwa 1966; Levitt 1980), but tissues are heated in contrasting ways. For instance, roots may be heated over long periods by smoldering combustion, while foliage and thin branches may be heated quickly in the plume because they are often not well insulated (Dickinson and Johnson 2001).

There are several caveats associated with our results. First, the vascular cambium cells stained poorly, and we could not quantify rates of cell death in that tissue. Consequently, we cannot say for certain that our results from phloem parenchyma reflect patterns of cell death in the cambium. As well, it is unclear to what extent heating tissue samples in buffer solution mimics the heating of intact tissues in fires. And of course, more species need to be studied. Finally, it should be borne in mind that our heat-transfer model includes important components of the overall stem heating process, but the model is simplified in a number of ways. For example, the model does not include bark fissuring and the small differences in thermal properties among species and bark layers and wood (Dickinson and Johnson 2001; Jones 2003). As such, the value of the heat-transfer model is primarily that it provides vascular cambium temperature regimes that approximate those that occur in fires, enabling us to make realistic comparisons of tissue thermal tolerance among species.

Finally, we simulated the effect of a surface fire on stem mortality in two Kananaskis Valley stands of lodgepole pine and Engelmann spruce initiated by fire 55 and 222 years prior to sampling (Fig. 11). Individual trees recruited into these stands in the years after the fires. Tree diameters were converted to the bark thicknesses shown in Fig. 11 by equations in Ryan and Reinhardt (1988). Shown are the species-specific bark thickness thresholds for stem survival in the simulated fire. No stems in the younger stand survived the fire. However, two Engelmann spruce stems in the older stand escaped stem death from the flames because of their thicker bark. Note that the difference in thermal tolerance

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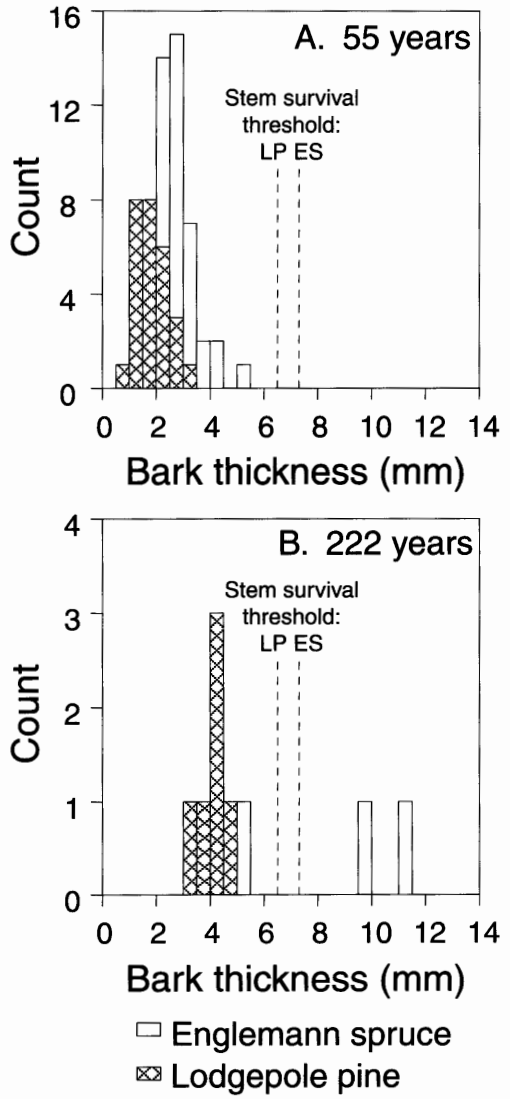
Fig. 11. Simulation of stem mortality from a surface fire in two Kananaskis Valley stands. The simulated surface fire had a residence time of 45 s, and bark thermal diffusivity was set to 0.1 $mm^2 \cdot s^{-1}$ (the intermediate fire in Fig. 10). We used the no-lag cell death model with spring–summer parameter values (see Table 1) and vascular cambium temperature regimes generated by the conduction heat transfer model to determine stem survival thresholds. The stem survival threshold was that bark thickness in which 63.2% of cells were killed (in analogy to the time constant of the no-lag rate process at a fixed temperature, i.e., where proportional cell survival declines to 1/e). The threshold is arbitrary and its precise value is not likely to be important given the steep slope in the relationship between final cell survival and bark thickness (Figs. 9 and 10). (A) Bark thickness distributions for Engelmann spruce and lodgepole pine in a 55-year-old stand before the simulated surface fire. All stems were predicted to be killed (i.e., all stems are below the stem survival threshold). (B) Bark thickness distributions in a 222-year-old stand indicating survival of two Engelmann spruce stems.



between lodgepole pine and Engelmann spruce is small when considered in the context of the large effects of variation in bark thickness caused by differences among species, time since stand initiation, the timing of stem recruitment, and stem growth rates. Shorter flame residence times would move the stem survival thresholds left along the bark thickness axis in Fig. 11, while longer residence times would result in the death of trees with thicker bark. Although two Engelmann spruce stems survived the effects of the simulated flames, they might still be killed by the plume of hot gases that rises above the flames (e.g., Mercer and Weber 2001).

Conclusion

One of the advantages of mechanistic models is that they clearly show one's postulates about the governing processes and, thus, which variables are important. Recently there has been a renewed interest in heat transfer into tree stems (e.g., Costa et al. 1991; Gutsell and Johnson 1996; Dickinson and Johnson 2001; Potter and Andresen 2002). However, all of these studies have been concerned with predicting the temperature at the cambium. They all leave mute the mechanisms of cambium death.

Here, for the first time to our knowledge, we have used a temperature-dependent rate process to explain vascular cambium cell death. Although we have examined only four species, they show that there are often significant differences in the rates of cell death. The rate-process models indicate that caution is warranted when using threshold temperatures to predict tissue death, particularly for tissue temperature regimes that differ from those in the stem (e.g., roots, branches, buds, and foliage). This topic has not yet been explored. Further, with a cambium-death model, we are getting closer to being able to construct a more complete stem-death model (Jones 2003). However, several interesting topics still need to be investigated, e.g., effect of bark moisture, combustion, swelling, and ridges on heat transfer and the role that embolism in the transport system may play in stem death.

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