

# Heat transfer and vascular cambium necrosis in the boles of trees during surface fires

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**ABSTRACT:** Heat-transfer and cell-survival models are used to link surface fire behavior with vascular cambium necrosis from heating by flames. Vascular cambium cell survival was predicted with a numerical model based on the kinetics of protein denaturation and parameterized with data from the literature. Cell survival was predicted for vascular cambium temperature regimes measured during experimental heating of Bolivian tree species by wick fires. Predicted cell survival was a threshold function of peak temperatures because (1) vascular cambium temperatures rise to a maximum and then decline relatively slowly and (2) cell mortality rates rise exponentially with temperature. Peak vascular cambium temperatures, in turn, were described by an equation derived from a dimensional analysis of the variables governing conduction heat transfer (i.e., the temperature gradient through the stem, flame residence time, bark and wood thermal diffusivity, and bark thickness). The heat-transfer and cell-survival models are combined to provide an index of vulnerability to vascular cambium necrosis in surface fires.

## 1 INTRODUCTION

As flames from a surface fire pass a tree, heat-transfer processes cause a rise and fall of vascular cambium temperatures in the bole, i.e., the trunk (Martin 1961, Fahnestock & Hare 1964, Hare 1965, Reifsnyder *et al.* 1967, Gill & Ashton 1968, Vines 1968). Elevated vascular cambium temperatures may lead to stem death if they cause necrosis around the circumference of the bole (see review in Dickinson and Johnson 2001). In this paper, vascular cambium temperature regimes measured during experimental fires for a group of Bolivian tree species are used to predict cell survival by way of a numerical model parameterized with data from the literature. Cell survival after the vascular cambium has cooled is related to peak vascular cambium temperatures by way of an equation (the "vascular cambium heating variable") derived from a dimensional analysis of the variables governing conduction heat transfer. Dimensional analysis was used because the one-dimensional conduction heat-transfer model often used in the ecological literature fit the data only moderately well. Finally, the relative vulnerability of the Bolivian species to vascular cambium necrosis during surface fires is described by way of the vascular cambium heating variable and the tissue necrosis threshold.

## 2 DATA

Wick fires were used to generate vascular cambium temperature regimes for 30 trees from 12 Bolivian, seasonal-forest tree species. Tree and flame characteristics are described in Pinard and Huffman (1996). The wick fires involved igniting a kerosene-soaked rope nailed around each tree's bole. Temperature regimes were measured with a thin thermocouple inserted along the vascular cambium. A bark surface and vascular cambium temperature regime is given in Figure 1. The thinner the bark, the higher the maximum temperature and more rapid the temperature rise and fall.

Vascular cambium temperature regimes were used in a cell-survival model to generate predictions of final survival, that is, cell survival after the vascular cambium had cooled below a temperature at which tissues are damaged ( $\sim 43^{\circ}\text{C}$ ). Parameters of the cell-survival model were estimated from data on seedling stem parenchyma cell survival after varying exposure times to constant, elevated temperatures in a water bath (Lorenz 1939). Typical cell survival data from Lorenz (1939) are given in Figure 2.

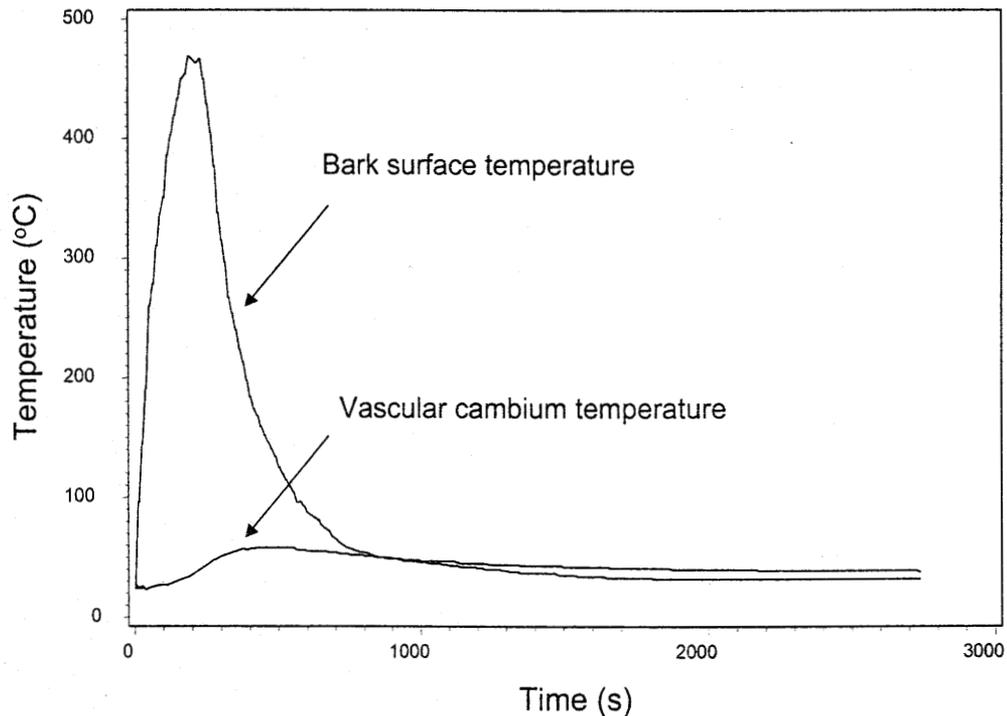


Figure 1. Bark surface and vascular temperature regimes from a wick fire.

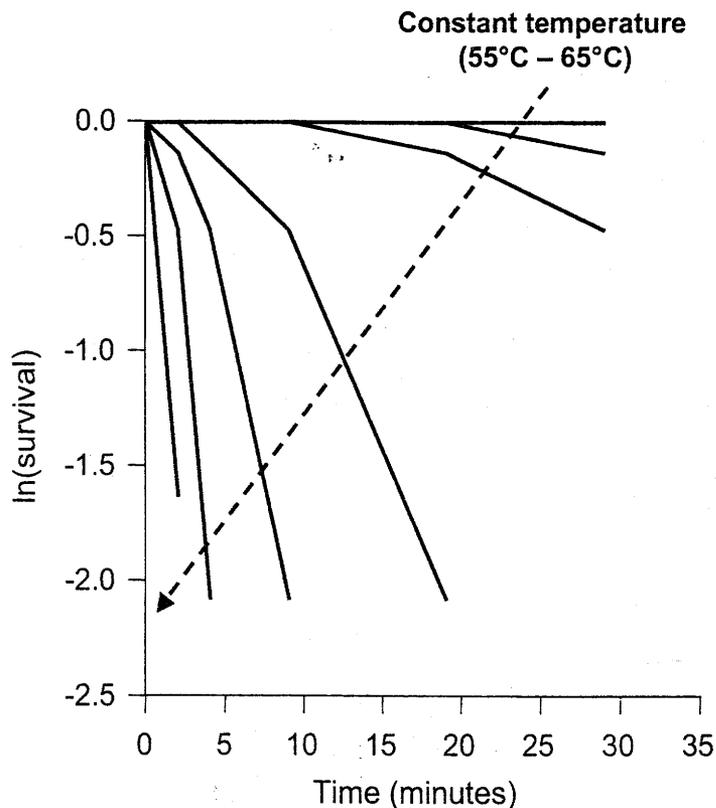


Figure 2. *Pinus resinosa* cell survival at constant, elevated temperatures.

### 3 MODELS

#### 3.1 Cell survival

Two approaches to predicting tissue response to elevated temperatures appear in the fire ecology literature. In the first, tissue necrosis is predicted to occur if a threshold temperature for necrosis is reached (Brown & DeByle 1987, Steward *et al.* 1990, Gutsell & Johnson 1996). The basis of this threshold is that physiologically active plant tissues will not generally survive if exposed to 60°C for short periods of time (Van Wagner 1973). The problem with the threshold approach is that longer exposures to temperatures below the threshold also result in necrosis (Hare 1961, Kay 1963). In the second approach, an exponential relationship between temperature and the time required for tissue necrosis at constant elevated temperatures was used to model necrosis after time-varying temperature regimes (Martin *et al.* 1969, Mercer *et al.* 1994, Mercer & Weber 2001). There is no mechanistic link in these studies between the effects of constant-temperature exposures and the effects of exposures to time-varying temperature regimes. A more intuitive approach to predicting tissue necrosis in fires is offered in this paper.

The cell-survival model, originally developed in the context of cancer research, describes tissue necrosis as the end point of the process of mortality within populations of cells exposed to elevated temperatures. Cell death is envisioned as being determined largely by temperature-dependent rate processes affecting the cell's molecular constituents (Johnson *et al.* 1974, Levitt 1980). Protein denaturation is thought to play a central role because the thermodynamic parameters estimated for the process of cell death often are within the range expected for protein denaturation (Rosenberg *et al.*

1971). Temperatures are assumed to be sufficiently high and last for a short enough time that cellular acclimation and repair processes play an insignificant role. Thus, the models should be well suited for describing fire effects.

The conceptual base of the cell-survival model is a random distribution of energy states within a population of cells. At a given time, a proportion of the cells are at or above an energy threshold at which an undefined cell-killing molecular transformation can occur. The proportion of cells above the threshold increases with temperature. The model has been termed the "one-hit" model in its application to cell survival because cells in the model are either unaffected or are killed by heating. The random distribution of energy states among cells results in a first-order rate process:

$$\frac{dS}{dt} = -kS(t) \quad (1)$$

where  $S(t)$  is proportional cell survival at time  $t$  (s) and  $k$  is a rate constant ( $s^{-1}$ ).

The temperature-dependence of the rate constant  $k$  must be described for modeling cell survival during any realistic vascular cambium temperature regime:

$$k = \frac{k_B T}{h} \exp\left(\frac{\Delta S}{R}\right) \exp\left(-\frac{\Delta H}{RT}\right) \quad (2)$$

where  $T$  is temperature (K),  $k_B$  is the Boltzman constant ( $1.38 \times 10^{-23} \text{ J K}^{-1}$ ),  $h$  is Planck's constant ( $6.63 \times 10^{-34} \text{ J s}$ ),  $\Delta S$  is the activation entropy ( $\text{J mol}^{-1} \text{ K}^{-1}$ ),  $R$  is the universal gas constant ( $8.31 \text{ J mol}^{-1} \text{ K}^{-1}$ ), and  $\Delta H$  is the activation enthalpy ( $\text{J mol}^{-1}$ ). Equation 6 is derived from the thermodynamics and statistical mechanics of simple biochemical reactions (Johnson *et al.* 1974) and does not have a precise meaning in the context of cell populations and tissues (Jung 1986, Caldwell 1993). For simple biochemical reactions, the activation entropy can be thought of as a measure of the favorableness of a reaction and the enthalpy as an energy barrier.

The rate parameter can be estimated from data on cell survival at a range of exposure times to a constant, elevated temperature. For constant temperatures, the integral of Equation 1 is:

$$S(t) = \exp(-kt) \quad (3)$$

where  $S(t)$  is survival after tissues are exposed for time  $t$  (s). Once  $k$  is available for a range of constant, elevated temperatures, the thermodynamic parameters of Equation 2 can be estimated.

The cell-survival model is used to predict cell survival after the vascular cambium has cooled following a fire. Equation 1 was written as a difference equation and the decline in cell survival was determined numerically at a 1 s time step:

$$S(t) = S(t-1) - kS(t-1)\Delta t \quad (4)$$

where  $S(t-1)$  is survival (initially 1) at the last time step and  $\Delta t$  is the time step (s). The temperature at the present time step determines the rate parameter according to Equation 2. The decline in cell survival predicted by the model for characteristic vascular cambium temperature regimes is given in Figure 3.

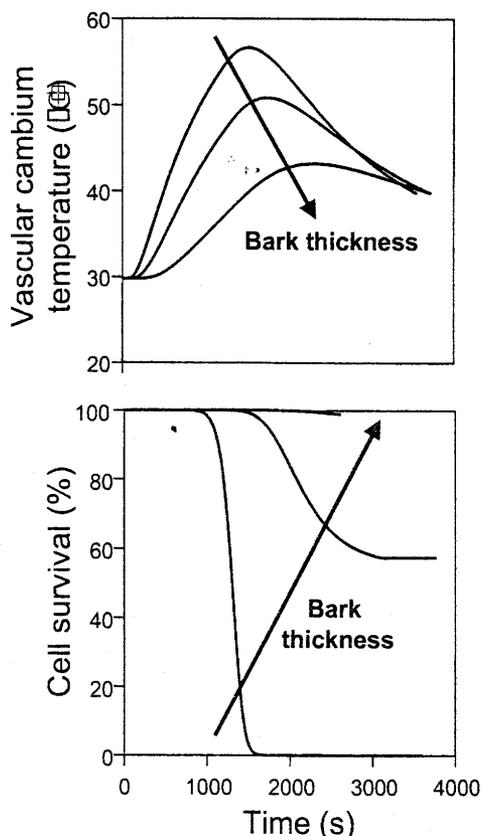


Figure 3. Characteristic vascular cambium temperature regimes and corresponding decline in modeled cell survival.

### 3.2 Dimensional analysis of conduction heat transfer

It was found that rates of cell mortality increased exponentially with temperature (see below). Accordingly, I speculated that peak vascular cambium temperature would serve as a suitable correlate of final cell survival. A dimensional analysis (e.g., Legendre and Legendre 1983) of the variables governing conduction heat transfer was used to derive an equation for describing peak vascular cambium temperatures from the wick-fire dataset. Knowing the most important variables in a given process, dimensional analysis can suggest the proper relationships among variables along with their relative weights. Intuitively, I expected that bark surface temperature, residence time of the flames, and thermal diffusivity (Equation 3) would scale directly with peak vascular cambium temperature. In contrast, bark thickness would be expected to scale inversely with peak vascular cambium temperature. Dimensional analysis of the above variables yields the following dimensionless groups:

$$\Pi_1 = \frac{T_{vc}}{T_s} \quad (5)$$

where  $T_v$  is the temperature rise from the initial bole temperature to the peak vascular cambium temperature during heating ( $^{\circ}\text{C}$ ) and  $T_s$  is the average temperature rise above ambient at the surface of the bark during the residence time of the flames ( $^{\circ}\text{C}$ ), and

$$\Pi_2 = \frac{x}{t_r^{\frac{1}{2}} \alpha^{\frac{1}{2}}} \quad (6)$$

where  $x$  is bark thickness (m),  $t_r$  is flame residence time (s), and  $\alpha$  is thermal diffusivity ( $\text{m}^2 \text{s}^{-1}$ ). The gradient in temperature through the bark is described by  $\Pi_1$  while  $\Pi_2$  describes heat transfer resulting from the temperature gradient.

If the variables in the two dimensionless groups adequately describe temperature rise at the vascular cambium, the product of the two groups should yield a constant  $j$  (dimensionless):

$$j = \left( \frac{x}{t_r^{\frac{1}{2}} \alpha^{\frac{1}{2}}} \right) \left( \frac{T_{vc} - T_i}{T_s} \right) \quad (7)$$

Substituting  $T_p - T_i$  for temperature rise at the vascular cambium  $T_{vc}$ , where  $T_p$  is peak vascular cambium temperature and  $T_i$  is initial vascular cambium temperature, and solving for peak vascular cambium temperature, Equation 8 becomes:

$$T_p = j \frac{t_r^{\frac{1}{2}} \alpha^{\frac{1}{2}} T_s}{x} + T_i \quad (8)$$

where both sides of the equation have the dimension of temperature. The following group of variables, from the right hand side of Equation 9, will be called the vascular cambium heating variable  $X_{vc}$ :

$$X_{vc} = \frac{t_r^a \alpha^b T_s^c}{x^d} \quad (9)$$

where the exponents  $a$ ,  $b$ ,  $c$ , and  $d$  either are those suggested by the dimensional analysis ( $a = 0.5$ ,  $b = 0.5$ ,  $c = 1$ , and  $d = 1$ ) or are estimated empirically from data.

#### 4 RESULTS AND DISCUSSION

Rates of cell mortality increased exponentially with temperature in Lorenz's (1939) data (Figure 4). Variation among Lorenz's species in cell death kinetics was trivial in the context of the effects of variation in bark thickness among the Bolivian trees. Accordingly, the means of the thermodynamic parameters of the temperature-dependence equation (Equation 2) estimated for the 5 species ( $\Delta S = 1035 \text{ J mol}^{-1} \text{ K}^{-1}$ ,  $\Delta H = 444303 \text{ J mol}^{-1}$ ) were used in predicting cell survival from Pinard and Huffman's (1996) vascular cambium temperature regimes.

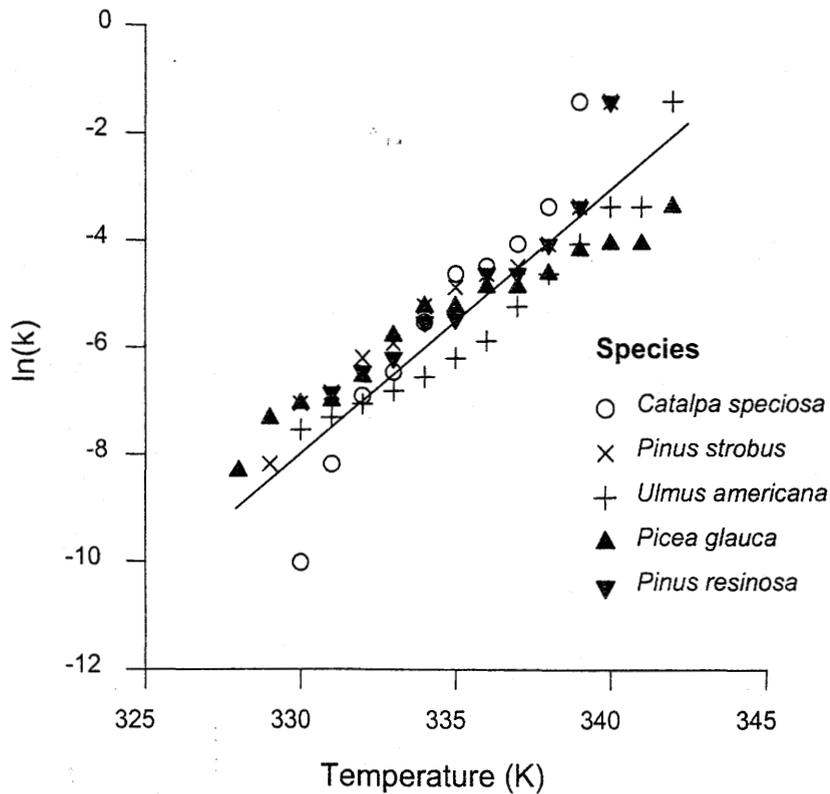


Figure 4. Temperature dependence of the rate parameter  $k$  of the cell survival model estimated from data in Lorenz (1939).

The vascular cambium heating variable, with exponents estimated from data, provided a satisfactory correlation with peak vascular cambium temperatures ( $R^2 = 0.93$ ; Figure 5). Long residence times (4-6 minutes) and elevated bark-surface temperatures may have resulted in a substantial vaporization heat sink, a process not included in the simple conduction heat-transfer model. Accordingly, it is not surprising that the exponents differ from theory. In surface fires, residence times may often be short enough (1-2 minutes) that the theoretical exponents adequately describe the process.

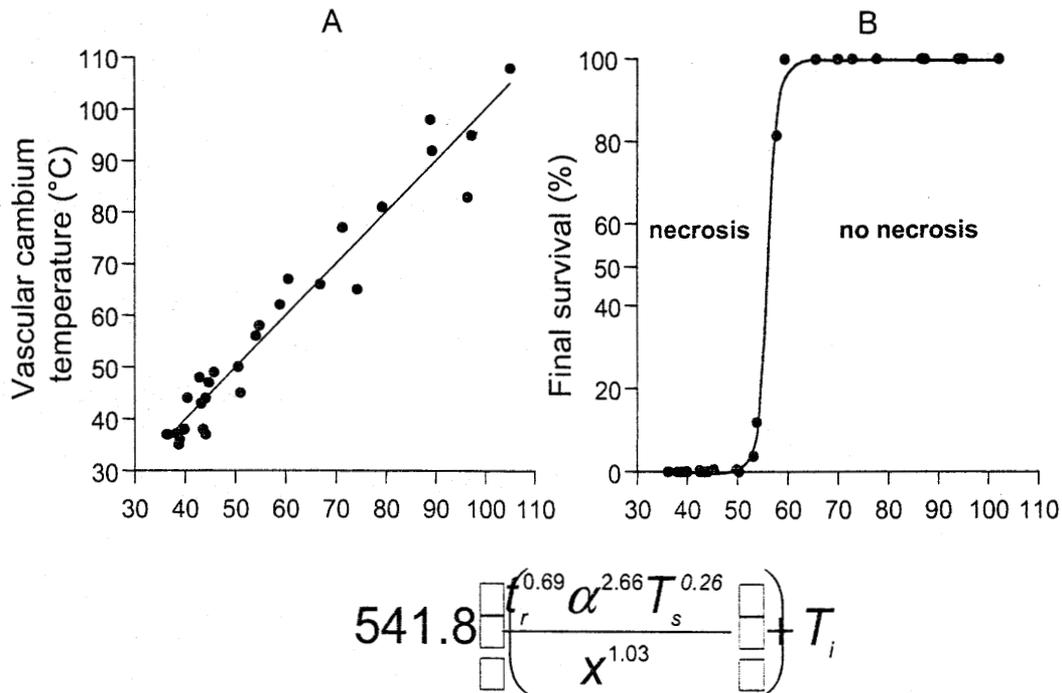


Figure 5. Relationship between peak vascular cambium temperature (A) and final cell survival (B) and the vascular cambium heating variable.

Final cell survival is predicted to follow a threshold relationship with peak vascular cambium temperatures (Figure 5). The threshold resulted from (1) the characteristic rise and fall of vascular cambium temperatures determined by heat transfer during and after the wick fires and (2) the exponential relationship between the rates of cell mortality and temperature.

Cell survival reaches 50% when the peak vascular cambium temperature is 56 °C according to Pinard and Huffman's (1996) vascular cambium temperature data and the cell survival model parameterized with Lorenz's (1939) cell-survival data. Accordingly, 50% mortality would appear to be a convenient threshold for modeling vascular cambium necrosis from heating by surface fires. The threshold relationship between cell survival and peak vascular cambium temperatures (Figure 5) lends credence to the use of a threshold in modeling vascular cambium necrosis. However, the threshold has meaning only in the statistical sense and its numerical value will change depending on the temperature regimes to which the tissue is exposed. For example, root heating occurs on a very different time scale than vascular cambium heating by flames. Appropriate thresholds cannot be determined until (1) appropriate estimates of the parameters of the cell survival model are available for a range of tissues and (2) characteristic tissue temperature regimes are adequately described.

The vascular cambium heating variable and the threshold temperature for vascular cambium necrosis should provide a means of describing the relative vulnerability of trees to surface fires (Figure 6). Relative vulnerability is dependent on a particular combination of variables describing fire behavior (residence time and bark surface temperatures) and tree characteristics (thermal diffusivity and, particularly, bark thickness). In Figure 6, thermal diffusivity is held at an average value because calculations suggested that it varied little among species. In addition, bark surface tempera-

ture is held constant at the average for the wick fires. The models suggest that there should exist a threshold bark thickness for vascular cambium necrosis for a fire of a given residence time. Variation in the behavior of a spreading fire should result in a blurring of that threshold.

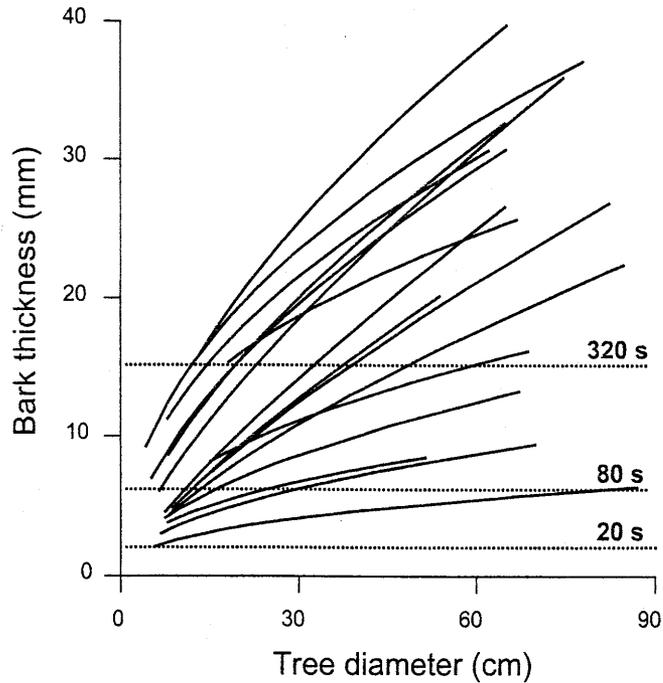


Figure 6. Bark thickness equations for 16 Bolivian species from Pinard and Huffman (1986). Broken lines give threshold bark thicknesses for vascular cambium necrosis for fires of different residence times. Residence times of 20-140 s were reported for surface fires in the Amazon (Cochrane *et al.* 1999).

## 5 SUMMARY

Vascular cambium necrosis is predicted to occur at a threshold value of a grouping of variables (the vascular cambium heating variable) that govern conduction heat-transfer into the boles of trees during surface fires. The vascular cambium heating variable includes characteristics of fires (e.g., residence time) and trees (e.g., bark thickness and thermal diffusivity) and was calibrated with data on peak vascular cambium temperatures. A peak temperature of about 56 °C defined the necrosis threshold. The vascular cambium heating variable and the necrosis threshold can be used to quantify the relative vulnerability of different species and sizes of trees to vascular cambium necrosis in surface fires.

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## REFERENCES

- Brown, J. K., and N. V. DeByle. 1987. Fire damage, mortality, and suckering in aspen. *Canadian Journal of Forest Research* 17:1100-1109.
- Caldwell, C. R. (1993) Estimation and analysis of cucumber (*Cucumis sativus* L.) leaf cellular heat sensitivity. *Plant Physiology* 101:939-945.
- Cochrane, M. A., A. Alencar, M. D. Schulze, C. M. Souza Jr., D. C. Nepstad, P. Lefebvre, E. A. Davidson. 1999. Positive feedbacks in the fire dynamic of closed canopy tropical forests. *Science* 284:1832-1935.
- Dickinson, M. B., and Johnson, E. A. (2001) Fire effects on trees. *Forest fires: behavior and ecological effects* (eds E. A. Johnson and K. Miyanishi), pp. 477-525. Academic Press, New York.
- Fahnestock, G. R., and R. C. Hare. 1964. Heating of tree trunks in surface fires. *Journal of Forestry* 62:799-805.
- Gill, A. M., and D. H. Ashton. 1968. The role of bark type in relative tolerance to fire of three central Victoria eucalypts. *Australian Journal of Botany* 16:491-498.
- Gutsell, S. L., and E. A. Johnson. 1996. How fire scars are formed: coupling a disturbance process to its ecological effect. *Canadian Journal of Forest Research* 26:166-174.
- Hare, R. C. (1961) *Heat effects on living plants*. Occasional Paper 183. USDA Forest Service, Southern Forest Experimental Station, New Orleans, LA.
- Hare, R. C. 1965. Contribution of bark to fire resistance of southern trees. *Journal of Forestry* 63:248-251.
- Johnson, F. H., H. Eyring, and B. J. Stover. 1974. *The theory of rate processes in biology and medicine*. Wiley, New York.
- Jung, H. 1986. A generalized concept for cell killing by heat. *Radiation Research* 106:56-72.
- Kayll, A. J. 1968. Heat tolerance of tree seedlings. Proceedings of the Eighth Tall Timbers Fire Ecology Conference. Tall Timbers Research Station, Tallahassee, Florida.
- Legendre, L., and P. Legendre. *Numerical Ecology*. Elsevier, New York.
- Levitt, J. 1980. Responses of plants to environmental stresses, Volume 1, chilling freezing, and high temperature stresses. Academic Press, New York.
- Lorenz, R. W. 1939. High temperature tolerance of forest trees. Technical Bulletin 141, Minnesota Agricultural Experiment Station.
- Martin, R. E. 1961. Thermal and other properties of bark and their relation to fire injury of tree stems. Ph. D. dissertation, University of Michigan.
- Martin, R. E., C. T. Cushwa, and R. L. Miller. 1969. Fire as a physical factor in wildland management. In Proceedings of the Ninth Annual Tall Timbers Fires Ecology Conference Tall Timbers Research Station, Tallahassee, Florida, pp. 271-288.
- Mercer, G. N., Gill, A. M. & Weber, R. O. (1994) A time-dependent model of fire impact on seed survival in woody fruits. *Australian Journal of Botany* 42:71-81.
- Mercer, G. N., and R. O. Weber. 2001. Fire plumes. In E. A. Johnson, and K. Miyanishi (eds) *Forest fires: behavior and ecological effects*. Academic Press, in press.
- Pinard, M. A., and J. Huffman. 1997. Fire resistance and bark properties of trees in a seasonally dry forest in eastern Bolivia. *Journal of Tropical Ecology* 13:727-740.
- Reifsnyder, W. E., L. P. Herrington, and K. W. Spalt. 1967. Thermophysical properties of bark of shortleaf, longleaf, and red pine. Yale University School of Forestry, Bull. No. 70.
- Rosenberg, B., G. Kemeny, R. C. Switzer, and T. C. Hamilton. 1971. Quantitative evidence for protein denaturation as the cause of thermal death. *Nature* 232:471-473.
- Steward, F. R., Peter, S. & Richon, J. B. (1990) A method for predicting the depth of lethal heat penetration into mineral soils exposed to fires of various intensities. *Canadian Journal of Forest Research* 20:919-926.
- Van Wagner, C. E. 1973. Height of crown scorch in forest fires. *Canadian Journal of Forest Research* 3:373-378.
- Vines, R. G. 1968. Heat transfer through bark, and the resistance of trees to fires. *Australian Journal of Botany* 16:499-514.