

## VULNERABILITY OF FREMONT COTTONWOOD (*POPULUS FREMONTII* WATS.) INDIVIDUALS TO XYLEM CAVITATION

A. Joshua Leffler<sup>1</sup>, Laura E. England<sup>2</sup>, and Jon Naito<sup>3</sup>

**ABSTRACT.**—Embolism, the blockage of water transport in the xylem by air, is an important consequence of low water availability for all plant species. Riparian plants, since they typically experience mesic conditions, are not water stress tolerant and hence are vulnerable to xylem cavitation, the formation of emboli. We have constructed a composite vulnerability curve for *Populus fremontii* (Fremont cottonwood); assessed native state embolism, critical xylem pressure potential ( $\Psi_{\text{cav}}$ ), and safety margin; and determined predawn and midday leaf water potential ( $\Psi_{\text{L}}$ ) within a central New Mexico cottonwood population. Our results indicate (1) that this population of *P. fremontii* is extremely vulnerable to cavitation, with complete xylem blockage occurring at  $-2.25$  MPa, and (2) that native state embolism is between 19% and 42%.  $\Psi_{\text{cav}}$  was determined to be  $-1.36$  MPa. Measurements of predawn  $\Psi_{\text{L}}$  were typically near  $-0.5$  MPa while midday  $\Psi_{\text{L}}$  values averaged  $-1.7$  MPa. Estimates of midday xylem pressure potential ( $\Psi_{\text{px}}$ ) were  $-1.1$  MPa. These values suggest that these individuals maintain small safety margins (0.26 MPa) between  $\Psi_{\text{px}}$  and  $\Psi_{\text{cav}}$ . This small safety margin may be detrimental under increased variation in water availability caused by anthropogenic alteration of river systems.

*Key words:* cavitation, drought stress, embolism, plant water potential, *Populus fremontii*.

Water availability plays an important role in the physiology of virtually all plant species. Low water availability is known to influence patterns of physiological activity, distribution of species, and competitive interactions. Recently, the importance of xylem cavitation, the loss of water transport capability in a plant, has received increasing attention as an important consequence of drought.

Xylem cavitation is a process by which xylem elements become blocked by air, typically under conditions of low water availability. Negative xylem pressure ( $\Psi_{\text{px}}$ ) causes the upward movement of water from soil into the vascular tissue of plants (Nobel 1983, Tyree and Sperry 1988, Pockman et al. 1995). However, if  $\Psi_{\text{px}}$  becomes very negative, as when water availability is low, catastrophic loss of hydraulic conductance (flow of water through the xylem) may occur through the process of cavitation. If the critical xylem pressure ( $\Psi_{\text{cav}}$ , the  $\Psi_{\text{px}}$  that can be maintained without causing catastrophic cavitation [Tyree and Sperry 1988]) is reached, an explosive phase change (liquid water to water vapor), or cavitation, occurs (Tyree and Dixon 1986). Embolism ensues as dissolved gases from surrounding tissue come out of solution, filling the cavitated

vessels and blocking water flow (Tyree and Sperry 1988). Embolized vessels cannot necessarily be refilled and cause a reduction in the capacity to conduct water. Vulnerability curves can be constructed to describe the loss of hydraulic conductance as a function of  $\Psi_{\text{px}}$ .

Although cavitation is an important consequence of drought in all plants, riparian plants are thought to be less frequently harmed by cavitation because they have access to a stable water supply. Plants typically operate at  $\Psi_{\text{px}}$  near  $\Psi_{\text{cav}}$  (Tyree and Sperry 1988), but given a stable water supply, cavitation should be infrequent. Riparian plants are known to have small safety margins, the difference between  $\Psi_{\text{cav}}$  and  $\Psi_{\text{px}}$ , because of their presumed stable water supply (Blake et al. 1996). However, *Populus* spp., important riparian trees in the southwestern U.S., have suffered significant declines in the past century, and it is widely thought that drought stress caused by anthropogenic alteration of stream flow regimes has been the primary cause of this decline (Rood and Mahoney 1990, Howe and Knopf 1991, Tyree et al. 1994, Busch and Smith 1995). Increased frequency of cavitation as a result of reduced water availability may be an important factor limiting the establishment, growth,

<sup>1</sup>Department of Biology, University of New Mexico, Albuquerque, NM 87131. Present address: Ecology Center, Utah State University, Logan, UT 84322-5230.

<sup>2</sup>Department of Biological Sciences, North Carolina State University, Raleigh, NC 27695. Present address: Institute of Ecology, University of Georgia, Athens, GA 30602-2202.

<sup>3</sup>Biology Department, Reed College, Portland, OR 97202. Present address: Zaiji 392, Kora-cho, Inukamigun Shiga 522-0244 Japan.

and reproduction of *Populus* spp. Studies examining poplar vulnerability to cavitation are important for understanding water relations of the species and for management of riparian systems.

Previous studies have shown that *Populus* species are extremely vulnerable to drought-induced cavitation compared to other non-riparian, woody species (Tyree and Ewers 1991, Tyree et al. 1994, Pockman et al. 1995). In this study we investigated vulnerability to cavitation in *Populus fremontii* Wats. (Fremont cottonwood), the dominant native canopy tree in lowland southwestern riparian ecosystems. The 1st goal of this study was to construct a vulnerability curve for *P. fremontii* from a central New Mexico population and determine if our curve was similar to the Pockman et al. (1995) study, the only previous study on cavitation in *P. fremontii*, and to other *Populus* studies. Although comparisons between studies on the same species of *Populus* have been made previously (Blake et al. 1996), there are no published comparisons of *P. fremontii* among populations. The 2nd goal of this study was to calculate native embolism, the percent loss of hydraulic conductance, that has occurred in our study population under natural conditions. Native embolism has not been reported for *P. fremontii* and may provide clues to the actual impact of cavitation on water relations in situ; a vulnerability curve describes only potential consequences. Additionally, we wished to make repeated measurements of native embolism to determine if it increases during the growing season. Our final goal was to calculate and report  $\Psi_{\text{cav}}$  and the safety margin, values which further clarify the vulnerability of this species.

## MATERIALS AND METHODS

### Study Site and Plant Material

*Populus fremontii*, a native riparian tree of the southwestern U.S., is the predominant tree of western prairies (Rood and Mahoney 1990). Distributed from the western Texas/Mexico border (Vines 1960) to the northernmost population at Moffet Creek in northern California (Griffin and Critchfield 1972), Fremont cottonwood typically thrives in sand-gravel soils within 4 m of the water table (Tyree et al. 1994). Thus, it is a phreatophytic species, relying on groundwater as a primary water source (Rood and Mahoney 1990).

Our study population is located approximately 4 km east of Bernardo, New Mexico (34°25'12"N, 106°48'7"W, 1440 m) at the southern end of a 210-km corridor of riparian forest along the Rio Grande, an effluent river. Cottonwoods at our study site were 23–30 yr of age ( $27 \pm 1.6$  yr, mean  $\pm$  s). At this site *P. fremontii* is the dominant native canopy tree, while *Elaeagnus angustifolia* (Russian olive), *Tamarix ramosissima* (salt cedar), and *Salix exigua* (coyote willow) occur in the subcanopy.

Five trees were randomly chosen and sampled 3 times during June and July 1997. We began by excising a 1-yr-old branch from the lower canopy of each tree and transporting it to the laboratory (<10 min transit time) in a plastic bag to minimize cavitation in transit. In the lab branches were soaked in water for a minimum of 15 min (to reduce the likelihood of inducing embolism during sample preparation), and side branches and ends were trimmed under water to obtain a stem segment of 30 cm (Sperry et al. 1988). This procedure does not dissolve emboli because the water is not under pressure; pressurized water is required to refill xylem. We chose 30-cm segments because they are longer than the longest xylem vessel, ensuring that there are intact, or uncut, vessels present (Tyree and Dixon 1986, Sperry et al. 1988). Although vessel length was not measured in this study, other measurements on *Populus* spp. suggest that the majority of vessels are generally  $\leq 15$  cm, although vessels to 30 cm have been observed (*P. deltoides*, Lo Gullo and Salleo 1992; *P. balsamifera*, Hacke and Sauter 1996).

### Measurement of Cavitation

We utilized the air-injection method of inducing embolism (Sperry and Saliendra 1994) to determine vulnerability to cavitation of our study trees. The air-injection method utilizes positive pressure to force air across pit membranes and yields results similar to the more traditional dehydration method which allows air to be pulled across pit membranes as a stem dries (Sperry and Tyree 1990). These methods yield similar results because the pressure required to push and the tension required to pull air across pit membranes are similar in magnitude. The air-injection apparatus consists of a double-ended pressure chamber, which allows induction of emboli and measurement of conductance in the same

system (Cochard et al. 1992, Sperry and Salendra 1994), an improvement over the dehydration method that requires 2 different systems. The chamber had a test gauge port for measurement of pressure and a port connected to a regulated compressed nitrogen tank. The proximal end of the stem inside the chamber was connected to a source solution used for measuring hydraulic conductance (see below). A T-junction and additional tubing allowed air bubbles to escape from the stem during the measurement process (see Sperry and Salendra 1994 for a diagram of the apparatus).

Construction of vulnerability curves consisted of 3 stages: (1) refilling of field-cavitated vessels via perfusion, (2) induction of emboli, and (3) measurement of hydraulic conductance ( $K$ ). Prior to inducing embolism, we determined maximum hydraulic conductance ( $K_{\max}$ ) by refilling all cavitated vessels. This was accomplished by perfusion, the forcing of fluid through the stem to dissolve emboli (Tyree et al. 1994). We perfused the stem using a filtered (0.2  $\mu\text{m}$  Whatman) distilled water solution adjusted to pH 2 with HCl. This solution prevents microbial growth that could block water flow in xylem tissue (Sperry et al. 1988). The fluid was forced through the stem at  $\approx 150$  kPa for 20 min by pressurizing the vessel containing the solution. After 20 min  $K$  was measured ( $K$  = flow rate through stem/applied pressure). We repeated perfusions and measurements until flow had stabilized. This flow rate was taken as  $K_{\max}$ .

To construct a vulnerability curve, we induced emboli at air-injection pressures of 0.1–2.5 MPa and measured  $K$ , the rate of water movement through the stem. The 1st air injection was at 0.1 MPa, the 2nd at 0.25 MPa, and each subsequent injection was increased by 0.25 MPa. Air injection was performed for 10 min at each pressure. Between air injections we measured  $K$ . We connected the proximal end of the branch to the solution described above and induced flow by raising and maintaining the solution source approximately 50 cm above the stem (inducing a pressure of  $\approx 5$  kPa). Flow was allowed to equilibrate for 3 min, and then  $K$  was measured by collecting effluent from the stem in test tubes containing cotton. Effluent was collected for 1 min at 2-min intervals and was then weighed. During the measurement of  $K$ , we maintained a pressure of  $\approx 0.1$  MPa in the chamber to prevent

dissolution of induced emboli. Alternations of air injection at increasing pressure and measurements of  $K$  were conducted until  $K = 0$ .

Vulnerability curves were constructed for each stem by plotting injection pressure against percent loss of conductance (PLC):

$$\text{PLC} = 100 * (K_{\max} - K) / (K_{\max}) \quad (\text{Eq. 1})$$

where  $K$  is the hydraulic conductance at a given injection pressure and  $K_{\max}$  is the maximum hydraulic conductance from the perfusion step (Tyree and Dixon 1986, Sperry et al. 1994).

#### Native Embolism and Water Status

Native embolism is the blockage of the xylem present in situ. We measured native embolism for each branch prior to induction of cavitation. After branches were soaked and trimmed, we connected them to the solution source and measured  $K$ . After allowing the flow to equilibrate for 3 min, we measured the flow as previously described. Native embolism was calculated as a PLC by comparison with the perfusion flow rate ( $K_{\max}$ ).

We measured the water status of 5 study trees throughout June and July to determine if *P. fremontii* individuals experience  $\Psi_{\text{px}}$  that can cause significant cavitation. Using a Scholander pressure chamber (Scholander et al. 1965), we measured predawn and midday water potentials of 3 lower canopy terminal branch segments per tree on each of 7 d. Since pressure chamber measurements determine leaf water potential ( $\Psi_L$ ), an adjustment was made to calculate  $\Psi_{\text{px}}$ .  $\Psi_{\text{px}}$  was quantified by calculating the mean between predawn and midday  $\Psi_L$  (Alder et al. 1996). This calculation of  $\Psi_{\text{px}}$  is appropriate because predawn  $\Psi_{\text{px}}$  is  $\approx$  predawn  $\Psi_L$  and midday  $\Psi_{\text{px}}$  must be above midday  $\Psi_L$  but below predawn  $\Psi_{\text{px}}$  because the plant is transpiring at midday.

#### Calculation of $\Psi_{\text{cav}}$ and Safety Margin

The lowest water potential that does not cause catastrophic cavitation ( $\Psi_{\text{cav}}$ ) was calculated using the iterative procedure described in Alder et al. (1996). Briefly, a computer program was written to calculate  $\Psi_{\text{px}}$  from transpiration ( $E$ ), soil water potential ( $\Psi_s$ ), and the vulnerability curve function ( $K(\Psi_{\text{px}})$ ):

$$E = (\Psi_s - \Psi_{px})K(\Psi_{px}) \quad (\text{Eq. 2}).$$

The program then computed the decline in  $K$  caused by  $\Psi_{px}$  (from a function fit to the vulnerability curve) and continued this loop until a stable  $\Psi_{px}$  was found. This program was run at increasing values of transpiration until stable  $\Psi_{px}$  could no longer be achieved (after 1000 iterations). The last stable  $\Psi_{px}$  at the highest transpiration rate was assumed to be  $\Psi_{cav}$ . For this analysis,  $\Psi_s$  was assumed to be equal to predawn  $\Psi_{Lr}$  and arbitrary values of  $K$  and transpiration were used. These arbitrary values have no impact on final  $\Psi_{cav}$  (Alder et al. 1996). Our vulnerability curve was fit with a 3rd-order polynomial which had a coefficient of determination = 0.98.

The safety margin, which is a measure of proximity to water potentials that could cause catastrophic cavitation, was calculated as the difference between  $\Psi_{px}$  and  $\Psi_{cav}$  (Alder et al. 1996).

#### Statistical Analysis

We wished to determine if the 5 trees we measured differed in their vulnerability to cavitation and native embolism, and if vulnerability curves and native embolism changed over time. To do so we utilized a 2-way ANCOVA (SAS 1990). In this analysis individual was the main effect used to test for differences among individuals, repetition was the main effect testing for differences through time, and injection pressure was the covariate. We also tested for differently shaped vulnerability curves among individuals by testing for a PLC\*pressure interaction effect. Prior to this analysis, PLC was log-transformed because of the exponential relationship between PLC and injection pressure. We used a 2-way ANOVA to test for differences among individuals and through time for native embolism using the main effects above. For each analysis type III sums-of-squares were used in all calculations.

#### RESULTS AND DISCUSSION

All individuals measured in our population had statistically identical vulnerability curves; the individual effect in our ANCOVA model was not significant (Table 1). Furthermore, values did not vary over time (among repetitions), and individuals had the same slopes and intercepts for their vulnerability curve

TABLE 1. ANCOVA testing for differences among individuals and repetitions in vulnerability curves.

Source	df	F	P
Individual	4	2.16	0.080
Repetition	2	2.08	0.130
Injection pressure	1	381.10	0.001

(individual\*injection pressure was not significant; Table 1). Therefore, we describe the vulnerability of this population with 1 composite vulnerability curve (Fig. 1). Here, we discuss vulnerability curves using pressure values >0 because we constructed our curves using the air-injection technique (positive pressure). Other studies are also discussed in this manner for consistency of presentation. Cavitation generally remained below 10% at injection pressures up to 1.0 MPa. Between 1.0 and 1.75 MPa, the catastrophic nature of this process became evident as cavitation increased from 10% to 75%. Complete loss of hydraulic conductance occurred by 2.25 MPa. At 1.5 MPa, 50% loss of conductance occurred.

Our vulnerability curve is nearly identical to the only other published study on cavitation in *P. fremontii* (see Pockman et al. 1995). Using a variety of methods to induce cavitation,

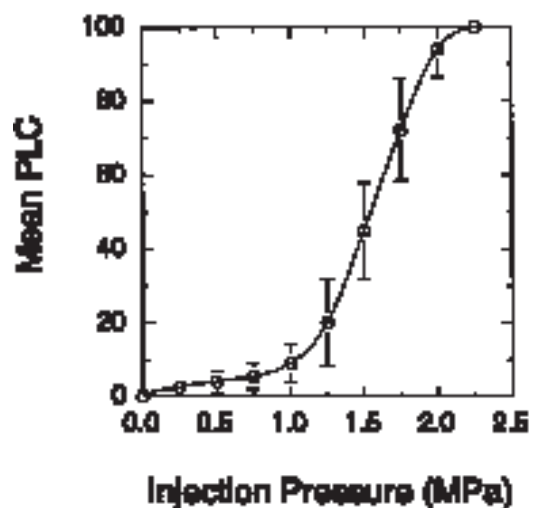


Fig. 1. Composite vulnerability curve using 3 measurements on each of 5 individuals constructed from percent loss of hydraulic conductance (PLC) as a function of injection pressure. Each datum is the mean  $\pm$  s among individuals. All values are positive because the air-injection technique, which used positive pressure, was utilized in vulnerability curve construction.



Pockman et al. (1995) constructed a curve that showed 10–20% loss of hydraulic conductance below 1.0 MPa, followed by a steep decline in conductance between 1.25 and 1.75 MPa. Fifty percent loss of conductance occurred at  $\approx 1.5$  MPa, and complete blockage was achieved by 2.0 MPa. Together, these 2 studies suggest that little variability in vulnerability exists among populations of this species, a result found for other *Populus* spp. (Blake et al. 1996). Variation in vulnerability among populations of *Quercus rubra* has, however, been observed (Tyree et al. 1992).

Other cavitation studies on the genus *Populus* have found similar vulnerability curves. Tyree et al. (1994) constructed vulnerability curves on *Populus* spp. from southern Alberta, Canada. Vulnerability curves of *P. balsamifera* and *P. angustifolia* were most similar to our samples, with little embolism below 1.0 MPa, followed by a rapid decline in hydraulic conductance and complete blockage by air above 2.25 MPa. Fifty percent loss of conductance was achieved at 1.7 MPa. However, *P. deltoides* was significantly more vulnerable, with 50% loss of hydraulic conductance occurring at 0.7 MPa.

Other *Populus* spp. have more resistant xylem. Sperry et al. (1994) constructed vulnerability curves for *P. tremuloides*, showing 50% loss of conductance from 1.5 to 3.5 MPa. *Populus tremuloides* occurs at higher elevations than *P. fremontii* (Elmore and Janish 1976). Its greater resistance to cavitation compared to our individuals likely reflects its exposure to freezing winter conditions. These results suggest that there can be variation in vulnerability to cavitation among species within a genus. This variation is likely related to environmental conditions experienced by individual species.

Native state embolism was not significantly different among individuals or repetitions within this population ( $P = 0.590$  and  $P = 0.780$ , respectively). Native state embolism measurements on individuals ranged from 19% to 42%

with a mean of 30%. We did not observe statistically significant changes in native state embolism over the course of this study, although embolism was highest in early July, the period with lowest water potential (Table 2). Additionally, our observation that native state embolism decreased in late July from the early July maximum suggests that limited refilling of embolized vessels may be possible, a phenomenon that has been recently described in several species (Canny 1997, McCully et al. 1998).

The native state embolism we report is low and less variable compared with other studies: 30–90% in *P. deltoides*, 20–75% in *P. balsamifera*, 20–50% in *P. angustifolia* (Tyree et al. 1994) and  $\geq 50\%$  in *P. tremuloides* and *P. balsamifera* (Sperry et al. 1994). These differences could arise from the timing of our study (June through July), conditions during 1997, or different adaptations to local conditions; additionally, they may suggest that our site experiences less water stress than those used in other studies.

Our leaf water potential ( $\Psi_L$ ) measurements suggested that conditions stressful enough to result in significant in situ cavitation occurred on a regular basis. Predawn  $\Psi_L$  was essentially constant at  $-0.5 \pm 0.02$  MPa during the period of measurement. There was, however, greater variation in midday  $\Psi_L$  values, ranging from  $-1.27$  MPa to  $-2.23$  MPa (mean =  $-1.7 \pm 0.06$  MPa).  $\Psi_{px}$ , as estimated from the average of predawn and midday  $\Psi_L$ , was  $-1.1$  MPa. Similar predawn and midday  $\Psi_L$  values have been observed in numerous studies on riparian tree physiology (Pallardy and Kozlowski 1981, Pezeshki and Hinckley 1982, Schulte et al. 1987, Tyree et al. 1994, Alder et al. 1996). It is likely that this pattern of plant water status is a general characteristic of riparian trees and may be caused by either little physiological control over water loss or a common midday  $\Psi_L$  set-point that induces stomatal closure.

$\Psi_{cav}$  calculations suggest that there is a very small margin of safety between  $\Psi_{px}$  and

TABLE 2. Water potential and native embolism data during summer 1997. Values are means of individuals used in this study with standard error in parentheses.

	Predawn $\Psi_L$ (MPa)	Midday $\Psi_L$ (MPa)	Native embolism (%)
Early June	-0.46 (0.02)	-1.69 (0.05)	27.4 (8.53)
Early July	-0.56 (0.06)	-2.01 (0.09)	34.8 (4.63)
Late July	-0.45 (0.02)	-1.61 (0.12)	28.4 (8.58)

$\Psi_{\text{cav}}$  in this species. Our iterative procedure produced a  $\Psi_{\text{cav}}$  of  $-1.36$  MPa. The difference between  $\Psi_{\text{px}}$  and  $\Psi_{\text{cav}}$  was only  $0.26$  MPa, suggesting that *P. fremontii* individuals operate very close to the threshold  $\Psi_{\text{px}}$  that could cause catastrophic cavitation. Although on average, individuals maintain  $\Psi_{\text{px}}$  above  $\Psi_{\text{cav}}$ , the range in midday  $\Psi_{\text{L}}$  we observed suggests that  $\Psi_{\text{cav}}$  is often exceeded, potentially resulting in catastrophic cavitation. Leaf xylem is likely more resistant to cavitation than stem xylem, since leaves are typically subjected to lower  $\Psi$  values than stems.  $\Psi_{\text{cav}}$  values for other riparian poplars include  $-1.05$  MPa for *P. deltoides* (Tyree et al. 1994) and  $-1.20$  MPa for *P. fremontii* (Blake et al. 1996 citing unpublished data from Pockman and Sperry). Blake et al. (1996) suggested that species which are highly vulnerable to cavitation maintain small safety margins. Data from our study are consistent with this finding. Additionally, species with small safety margins are thought to have stomata that are highly sensitive to water status (Schulte et al. 1987), a result we have also observed.

Several authors have suggested that various impacts of water stress are responsible for the decline of cottonwood populations throughout western North America (Rood and Mahoney 1990, Howe and Knopf 1991, Busch and Smith 1995). The extreme vulnerability of individuals to cavitation and the small safety margins reported in this and other studies are strong evidence to support this theory. High vulnerability and small safety margins likely evolved under conditions of higher water availability, conditions similar to those in riparian areas prior to damming and channelization of rivers. The greater variation in water availability that these populations now experience may cause  $\Psi_{\text{px}}$  to exceed  $\Psi_{\text{cav}}$  on a regular basis, causing significant damage to trees.

#### ACKNOWLEDGMENTS

We thank the Sevilleta Long Term Ecological Research Station, Middle Rio Grande Conservancy District, and U.S. Fish and Wildlife Service for permitting our research. Much appreciation goes to Will Pockman for technical assistance and Ann Evans and 2 anonymous reviewers for assistance with this manuscript. LEE and JN were researchers with the

Sevilleta LTER REU program (NSF BIR-9424121 awarded to Ann Evans); special thanks go to all those who contributed to this program, especially Ursula Shepherd. Additional support was provided to AJL by the University of New Mexico Graduate and Professional Student Association and the Department of Biology Graduate Student Association.

#### LITERATURE CITED

- ALDER, N.N., J.S. SPERRY, AND W.T. POCKMAN. 1996. Root and stem xylem embolism, stomatal conductance, and leaf turgor in *Acer grandidentatum* populations along a soil moisture gradient. *Oecologia* 105:293–301.
- BLAKE, T.J., J.S. SPERRY, T.J. TSCHAPLINSKI, AND S.S. WANG. 1996. Water relations. Pages 401–422 in R.F. Stettler, H.D. Bradshaw, Jr., P.E. Heilman, and T.M. Hinckley, editors, *Biology of Populus and its implications for management and conservation*. NRC Research Press, Ottawa, Canada.
- BUSCH, D.E., AND S.D. SMITH. 1995. Mechanisms associated with decline of woody species in riparian ecosystems of the southwestern U.S. *Ecological Monographs* 65:347–370.
- CANNY, M.J. 1997. Vessel contents during transpiration—embolisms and refilling. *American Journal of Botany* 84:1223–1230.
- COCHARD, H.P., P. CRUIZLAT, AND M.T. TYREE. 1992. Use of positive pressures to establish vulnerability curves. *Plant Physiology* 100:205–209.
- ELMORE, F.H., AND J.R. JANISH. 1976. Shrubs and trees of the southwestern uplands. Southwest Parks and Monuments Association, Tucson, AZ. 214 pp.
- GRIFFIN, J.R., AND W.B. CRITCHFIELD. 1972. The distribution of forest trees in California. USDA Forest Service Research Paper PSW-82.
- HACKE, U., AND J.J. SAUTER. 1996. Drought-induced xylem dysfunction in petioles, branches, and roots of *Populus balsamifera* L. and *Alnus glutinosa* (L.) Gaertn. *Plant Physiology* 111:413–417.
- HOWE, W.H., AND E.L. KNOPE. 1991. On the imminent decline of Rio Grande cottonwoods in central New Mexico. *Southwestern Naturalist* 36:218–224.
- LO GULLO, M.A., AND S. SALLEO. 1992. Water storage in the wood and xylem cavitation in 1-year-old twigs of *Populus deltoides* Bart. *Plant, Cell and Environment* 15:431–438.
- MCCULLY, M.E., C.X. HUANG, AND L.E.C. LING. 1998. Daily embolism and refilling of xylem vessels in the roots of field-grown maize. *New Phytologist* 138:327–342.
- NOBEL, P.S. 1983. *Biophysical plant physiology and ecology*. W.H. Freeman and Company, San Francisco, CA. 608 pp.
- PALLARDY, S.G., AND T.T. KOZLOWSKI. 1981. Water relations of *Populus* clones. *Ecology* 62:159–169.
- PEZESHKI, S.R., AND T.M. HINCKLEY. 1982. The stomatal response of red alder and black cottonwood to changing water status. *Canadian Journal of Forest Research* 12:761–771.
- POCKMAN, W.T., J.S. SPERRY, AND J.W. O'LEARY. 1995. Sustained and significant negative water pressure in xylem. *Nature* 378:715–716.

- ROOD, S.B., AND J.M. MAHONEY. 1990. Collapse of riparian poplar forests downstream from dams in western prairies: probable causes and prospects for mitigation. *Environmental Management* 14:451–464.
- SAS. 1990. SAS/STAT user's guide. SAS Institute, Cary, NC. 1686 pp.
- SCHOLANDER, P.F., H.T. HAMMEL, E.D. BRADSTREET, AND E.A. HEMMINGSEN. 1965. Sap pressure in vascular plants. *Science* 148:339–346.
- SCHULTE, P.J., T.M. HINCKLEY, AND R.F. STETTLER. 1987. Stomatal responses of *Populus* to leaf water potential. *Canadian Journal of Botany* 65:255–260.
- SPERRY, J.S., AND N.Z. SALIENDRA. 1994. Intra- and inter-plant variation in xylem cavitation in *Betula occidentalis*. *Plant, Cell and Environment* 17:1233–1241.
- SPERRY, J.S., AND M.T. TYREE. 1990. Water stress induced xylem embolism in three species of conifers. *Plant, Cell and Environment* 13:427–437.
- SPERRY, J.S., J.R. DONNELLY, AND M.T. TYREE. 1988. A method for measuring hydraulic conductivity and embolism in xylem. *Plant, Cell and Environment* 11:35–40.
- SPERRY, J.S., K.L. NICHOLS, J.E.M. SULLIVAN, AND S.E. EASTLACK. 1994. Xylem embolism in ring-porous, diffuse-porous, and coniferous trees of northern Utah and interior Alaska. *Ecology* 75:1736–1752.
- TYREE, M.T., AND M.A. DIXON. 1986. Water stress induced cavitation and embolism in some woody plants. *Physiologia Plantarum* 66:397–405.
- TYREE, M.T., AND F.W. EWERS. 1991. The hydraulic architecture of trees and other woody plants. *New Phytologist* 119:345–360.
- TYREE, M.T., AND J.S. SPERRY. 1988. Do woody plants operate near the point of catastrophic xylem dysfunction caused by dynamic water stress? *Plant Physiology* 88:574–580.
- TYREE, M.T., J. ALEXANDER, AND J. MACHADO. 1992. Loss of hydraulic conductivity due to water stress in intact juveniles of *Quercus rubra* and *Populus deltoides*. *Tree Physiology* 10:411–415.
- TYREE, M.T., K.J. KOLB, S.B. ROOD, AND S. PATIÑO. 1994. Vulnerability to drought-induced cavitation of riparian cottonwoods in Alberta: a possible factor in the decline of the ecosystem. *Tree Physiology* 14:455–466.
- VINES, R.A. 1960. Trees, shrubs and woody vines of the Southwest. University of Texas Press, Austin. 1104 pp.

Received 4 May 1999

Accepted 1 September 1999