Review

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Linking definitions, mechanisms, and modeling of drought-induced tree death

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Tree death from drought and heat stress is a critical and uncertain component in forest ecosystem responses to a changing climate. Recent research has illuminated how tree mortality is a complex cascade of changes involving interconnected plant systems over multiple timescales. Explicit consideration of the definitions, dynamics, and temporal and biological scales of tree mortality research can guide experimental and modeling approaches. In this review, we draw on the medical literature concerning human death to propose a water resource-based approach to tree mortality that considers the tree as a complex organism with a distinct growth strategy. This approach provides insight into mortality mechanisms at the tree and landscape scales and presents promising avenues into modeling tree death from drought and temperature stress.

Tree death in a changing climate

Tree death is a fundamental process in ecosystems and has substantial leverage in forest ecosystem responses to global environmental change [1,2]. Increases in tree mortality rates have been reported in temperate forests [3], boreal forests [4], and tropical forests [5] in concert with rising temperatures and drought. Tree death from drought and heat stress is expected to accelerate in coming decades with climate change, and widespread forest die-off events have been observed around the globe (Figure 1) [2]. Rapid ecosystem changes and vegetation loss constitute a large uncertainty in ecosystem responses to climate change [6–8]. Widespread tree death can influence ecological communities and succession, and ecosystem function and services, and can affect regional and global climate through biophysical and biogeochemical changes [9].

How trees die in response to drought and heat stress associated with climate change is an active area of research. Physiological pathways of tree death from drought and temperature stress have been intensely debated [10–20], often with the motivating goal of modeling tree mortality with climate change [12,17,18]. Larger questions about how we consider and approach tree death have not yet been answered. What does death mean in a complex organism? How much physiological detail is needed to model the behavior of a complex organism during stress? How do we integrate stress and repair across multiple tissues and timescales from days to decades? How do we quantify the manifold feedbacks among organ subsystems, biotic agents, and microenvironment during stress? How would we integrate even a hypothetically perfect understanding of an individual tree's death to whole-forest die-off at the scale of a climate–vegetation model's grid cell? The answers to these questions greatly influence approaches to experiments and models of tree mortality.

The challenges raised above justify careful consideration of the lens through which we approach definitions, mechanisms, and modeling drought and heat-induced tree mortality. Tree 'death' from drought and temperature stress is poorly defined. In this context it may be useful to consider what has been learned from studies of death of another complex organism – humans. Drawing on the human mortality literature, we present here a definition of tree death as a complex system failure due to lack of water resources. We explore the implications of this resource-based approach for ultimate and proximate mechanisms of how trees die during drought. We conclude with how this consideration of tree death could guide experimental studies and modeling approaches of tree mortality

Glossary

Cavitation: entry of air bubbles into conducting xylem elements, typically triggered in drought by high xylem water tensions, that blocks water conductance and transport.
Cavitation fatigue: decreased resilience of xylem elements to cavitation due to damage from previous cavitation events.
Embolism repair: refilling of cavitated xylem elements, perhaps under tension, to restore water conductance and xylem hydraulic capability.
Hydraulic capacitance: the tissue-specific ratio of change in a tissue's water content to change in its water potential
Hydraulic redistribution: nighttime passive movement of water in the soil and roots along water potential gradients from wetter soil to drier soil. This can result in upward, downward, or lateral movement of water in the soil at faster rates than in typical percolation.
Modularity: the degree to which a system's components can be separated into modules. Plants typically exhibit high modularity (e.g., branches) relative to animals, which can allow loss of some modules while maintaining overall function of the organism.
Stress contingency: the effect of a certain stress is contingent or dependent on the history and/or timing of previous stresses and repair of damage from those stresses.
Water storage capacity: the integrated volume of all water storage within a plant, including xylem and phloem transport, as well as cell water.

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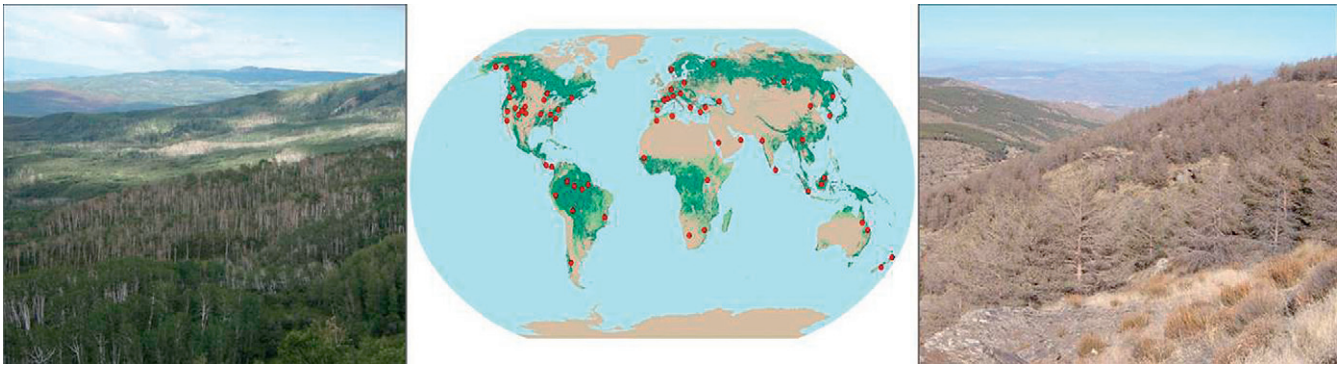

Figure 1. Widespread drought and heat-induced forest die-off. Global forest cover (dark green) and other wooded regions (light green) (Food and Agriculture Organization of the United Nations, 2006). Red dots mark locations of studies that documented drought- and heat-induced forest die-off (redrawn from [2]); photographs provide examples of die-off triggered by both drought and heat in concert– (R. Navarro-Cerrillo, left; W.R.L. Anderegg, right).

in response to global environmental change. Although we primarily consider the death of large, adult trees in this review, the physiology and approach examined here should largely apply to most vascular plants.

Learning from humans: death in a complex organism

Only one study to date has explicitly defined tree death: "Death is defined as thermodynamic equilibrium between the organism and the environment, in which plants no longer have energy gradients to drive metabolism or regenerate" [17]. Although completely true, lessons from the medical literature (Box 1) highlight that this definition is limited in utility for investigating tree death because it does not provide testable criteria for when an organism crosses the transition from life to death. Without such tests tied to the appropriate biological scale, it is not especially helpful for physiological experiments aimed at informing modeling of forest mortality.

A tree, like a human, is a complex organism with an array of regulatory mechanisms to keep critical systems operating within appropriate bounds and mechanisms to repair damage that may occur when these bounds are exceeded [21,22]. Ultimately, a tree can be viewed as a complex organism that transports water from the soil the atmosphere; its survival hinges on maintenance of this transport system, which is essential for maintaining hydration, and efficient exchange of water for the CO_2 required for photosynthesis in a dynamic and often water-limited environment.

We outline here a definition of tree death from drought and temperature stress, drawing on the standard definition of death in the medical literature, refined to apply to trees. We define tree death as the permanent cessation of functioning of the organism as a whole. We do not mean the whole organism, for example, the sum of its cells, tissues and organs, but rather the highly complex interaction of its organ subsystems (*sensu* [23]). In humans, such complex interactions of organ subsystems are mediated by a central nervous system, which does not apply to trees. Thus, we clarify our definition such that breakdown of highly complex interactions in organ subsystems occurs through irreparable damage to the interaction and coordination of tree organ subsystems in xylem and/or phloem transport within a tree. We emphasize that this is not simply 'hydraulic failure' as proposed elsewhere [11] and elaborate on this below. For resprouting species, the lack of meristem tissue vitality after death of aboveground tissues may be an additional useful component of the mortality definition, depending on research aims (e.g., useful for studies examining shrubs and less critical for simulation of ecosystem carbon fluxes with mature tree mortality).

For our purposes of tree death from drought and temperature stress, we highlight that our definition of death focuses more on whether a tree is past the point of recovery – when a tree is functionally dead. A formal definition of tree death allows for explicit comparison across studies (e.g., [24]) and can influence experimental results (e.g., [14]). For example, which physiological mechanisms mediated *Pinus edulis* mortality in a greenhouse experiment were influenced by defining death as needle browning, which may or may not provide an accurate determination of the timing of death [12,14]. Most importantly, a definition can guide an approach (i.e., whole-system failure rather than tissue-level or cell-level failure) to assess mortality mechanisms for modeling tree mortality.

Yet trees as a life-form also differ from humans in some important ways. Trees generally experience indeterminate growth and high degrees of modularity in their growth plan, despite great plasticity in that growth form [25]. Functionally, trees do not 'age' as animals do [26], leading to the possibility that some trees could be 'immortal' in the absence of stress [27]. Instead, mortality probabilities with age appear to be an accumulation of the effects of exogenous stresses from any number of sources such as fire, climate, insects, pathogens, and competition [19]. For example, dead cells comprise much of tree biomass at most points during a tree's life cycle. Taken together with the consideration of the tree as an interconnected organism, this suggests that tree mortality should be considered as a failure of system function rather than cellular function. The degree of modularity within trees further highlights that tree mortality should be considered with regard to long-distance transport and coordination among multiple organ subsystems within an individual. Long-distance transport (xylem and phloem) interconnects roots, leaves, and reproductive organs and allows coordinated response

Box 1. What is death?

Death is a defining feature of living organisms. In its most basic sense, death is the permanent termination of biological function within a living organism [82]. To most of us in daily life, determining and identifying death in an organism seems an easy and commonsense task. However, what defines death in humans, the most well-researched organism, is controversial and important, because this is the point at which medical treatment stops and organs may be removed for transplantation. Much confusion stems from a conflation of two questions [23,83]. First, what is death? Second, how can we determine that it has occurred? These questions are interrelated, but must be considered separately. The first question is a value judgment (normative) and must be answered by a clear and explicit definition of death. The second question is data driven (positive) and must furnish criteria for the transition of death to have occurred [83]. Thus, both questions must be answered separately and explicitly; the second question does not answer the first.

A long and rich literature on what constitutes human death has wrestled with these questions. Before the 1950s, human death was generally defined as failure of the cardiopulmonary system. When a person's heart was no longer beating and lungs no longer breathing, he or she was considered dead [84]. In 1968, the Ad Hoc Committee of the Harvard Medical School to Examine the Definition of Brain Death put forth that a person's whole brain must be dead for death to have occurred, predicated on the definition of death being a permanent shutdown of the central nervous system [84]. Since then, this 'whole-brain' criterion has been generally accepted in the medical and legal communities, with careful consideration of the aforementioned distinction between death's definition and criteria [23], though many criticisms and reevaluations still occur (e.g., [84]).

Several critical lessons for tree death can be learned from the literature on human death. First, 'death' must be explicitly defined beforehand. The implicit assumption 'I'll know death when I see it' hinders research advancement, intercomparison, and prediction of death. Second, the scale of a definition of mortality must be considered in complex organisms. Individual cells and even tissues can live for days after the organism has been determined to be 'dead', even in humans [85]. This means that the timing of cell-level, tissue-level, and organism-level death can all differ. Clear definitions of death must be tied to the appropriate biological scale. Finally, definitions of mortality must provide both a definition and criteria or tests of mortality to be useful.

of the tree to the environment through transport of water, carbon, nutrients, and chemical signals.

Mechanisms of drought-induced tree mortality: a resource-based, systems approach

Gradients in water potential within a continuum of water in conducting tissues drive transport in trees [28,29]. Water movement from roots to leaves via transpiration provides the water for all other transport within a tree, including stomatal movements and the pressure flow of phloem and distribution of hormones such as auxins, abscisic acid (ABA), and cytokinins [21,30]. Furthermore, water loss to the atmosphere is unavoidable; trees face the challenge of maintaining hydration while poised between the dry soil and the drier atmosphere. Plant water storage capacity (W_p) across all tissues can be thought of as an analogy to soil water reserves such that reserves at time i are equal to uptake (U_i) from the soil to the distal meristem minus evapotranspiration (ET_i) plus storage (S_i), which includes water in cells and all water in xylems conduits and phloem [31]:

$$W_{p,i} = U_i - ET_i + S_i \quad [1]$$

In unstressed conditions with ample soil water reserves. water loss from evapotranspiration can be easily met by water uptake from the soil. Thus, $dW_{p,i}/dt \geq 0$, depending on whether the tree is accumulating water, for instance in expanding tissues, undergoing growth, or increasing storage reserves such as heartwood [32] (Figure 2a).

We define drought as a dynamic state that occurs when the rate at which water can be acquired from accessible soil water reserves is insufficient to meet tree water requirements. During moderate or tolerable water stress, water becomes more difficult to extract from the soil and U_i declines. Trees typically reduce evapotranspiration during drought via many mechanisms, including stomatal closure [33–35], leaf shedding [36], seasonal canopy loss [37], and cavitation of leaf, stem, and root xylem [38]. Thus, although there may be short-term disconnects between water uptake and evapotranspiration, such as in the daily cycle of transpiration leading to more negative water potentials, the tree typically maintains ample hydration and water reserves, such that $dW_{p,i}/dt \approx 0$ over this period of drought stress (Figure 2b).

Due to constraints imposed by soil physics, a tree cannot access all water in the soil [39]. However, a tree cannot stop losing water altogether. Stomatal closure is never able to completely halt water loss. Water can still escape through the cuticle, whose permeability increases with higher leaf temperatures and leaf age [40–42]. Other organs, stems, buds, and roots have some permeability to water vapor [43,44]. Thus, as drought becomes more severe an individual tree must approach a limit where it cannot extract enough water from the soil to meet evapotranspiration losses (Figure 2c). At this point, $dW_{p,i}/dt < 0$ and the tree enters a period of 'water debt'. During this phase, a tree's water status (e.g., xylem tension or water potential) can change dramatically with a small change in soil and plant water content. The trajectories and severity of this water debt are likely to be influenced by the characteristics of the drought, such as drought severity, duration, and timing relative to an area's drought regime, as well as atmospheric temperature and moisture deficit, the size of the tree, root distribution, xylem properties, and hydraulic capacitance (Figure 3a). This water debt at time i (WD_i), integrated from the start of a drought period or across several droughts, can be expressed as:

$$WD_i = \int_0^i U\,dt - \int_0^i ET\,dt \quad [2]$$

Modeling water uptake and evapotranspiration will require some simulation of plant hydraulics, stomatal functioning, water use strategy and risk avoidance mechanism and strategy, as well as soil properties such as the soil water characteristic curve, many of which are already present in current hydraulics and ecosystem models.

As the tree falls below this sustainable level of water reserves, numerous physiological processes result in breakdown of transport and coordination in the organism. These include drawdown of hydraulic capacitance and water storage capacity [31,32], tension-induced cavitation in xylem vessels [29], possible xylem conduit collapse [45], phloem transport impairment [46], breakdown in xylem–phloem coupling [47], failure to take up nutrients from soil [48], and failure to defend against pathogen or insect attack [49]. More research is needed to identify

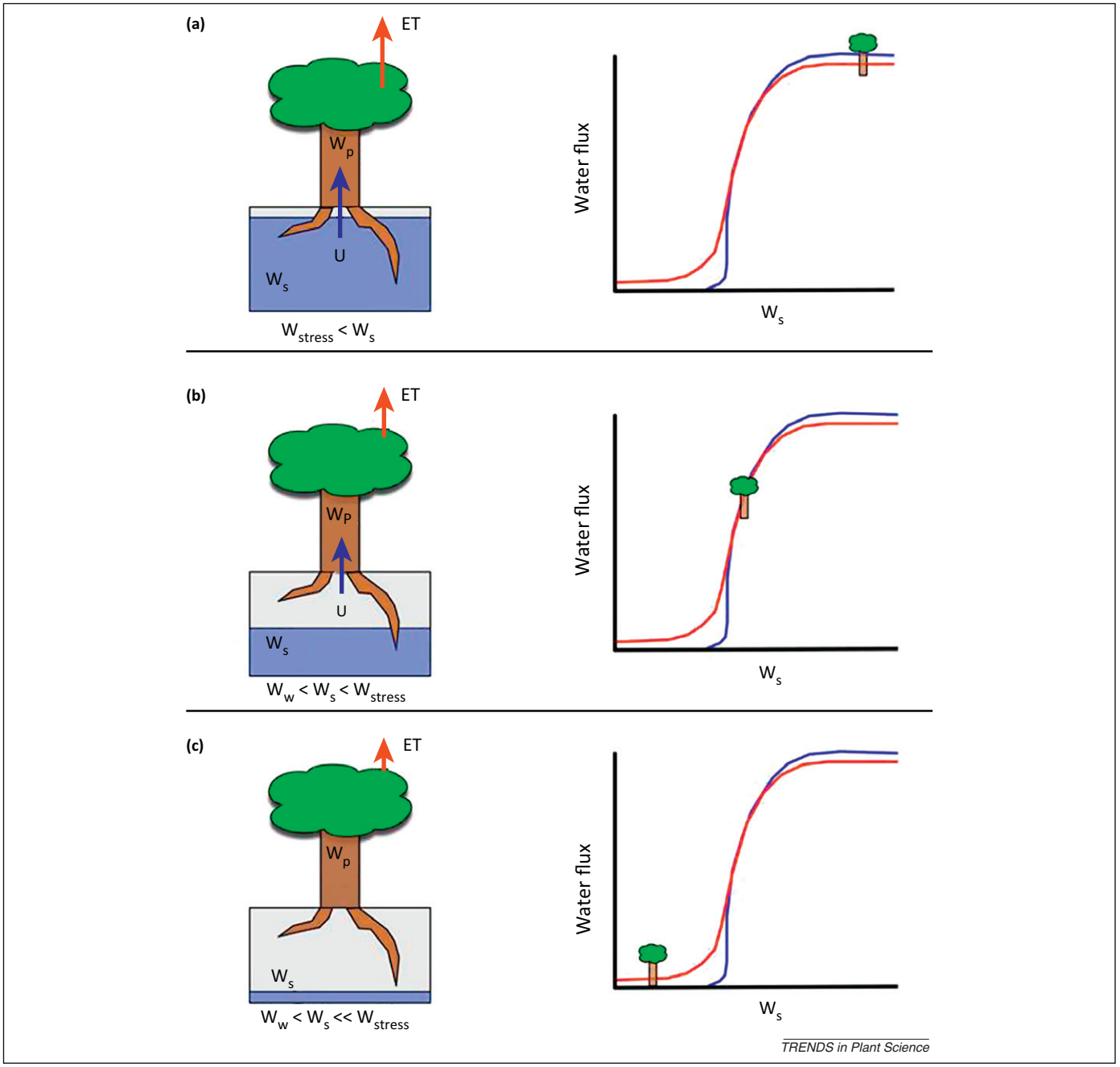

Figure 2. Tree water reserves during stages of drought. **(a)** A tree can access a certain amount of water reserves in the soil (W_s) and take up this water at a certain rate (U, blue), while losing water to the atmosphere at another rate (ET, red). The plant also holds an internal water reserve (W_p) to maintain hydration. At this stage, W_s exceeds the levels of soil water that induce damaging plant stress (W_{stress}) (e.g., extensive cavitation) and no drought occurs. **(b)** Soil water falls below W_{stress}, but is above the wilting or leaf turgor loss point (W_w), and the tree curtails U and ET while maintaining adequate plant water reserves. **(c)** Soil water falls to the point where U can no longer meet ET water loss for a sustained period of time. Water debt increases as drought proceeds, leading to higher levels of system damage.

and quantify these processes, especially in tissues that link the two essential interfaces of the tree with the environment: leaves and fine roots.

We suggest that it may be productive to consider the above physiological processes as proximate mechanisms of tree death. These proximate mechanisms have been the focus of much previous research as an attempt to locate the 'Achilles heel' of a tree species during drought – the single point at which an individual subsystem has failed. By contrast, the accumulation of water debt as leading to a whole-system transport failure (a water resource-based approach) can be viewed as an ultimate mechanism of tree death. We emphasize that the process of transport failure is not 'hydraulic failure', because transport failure may affect multiple transport systems within the plant and their interactions, including xylem transport, phloem transport, xylem–phloem interactions, and short-distance signaling. To illustrate a whole-system failure leading to mortality, we consider the complex web of physiological processes leading to a widespread drought-induced die-off of trembling aspen (*Populus tremuloides*) in the western USA (Figure 1, left) [50]. A severe and hot drought from 2000 to 2003 triggered a die-off that commenced in 2004 and continued through 2011 [20,51,52]. Initial research

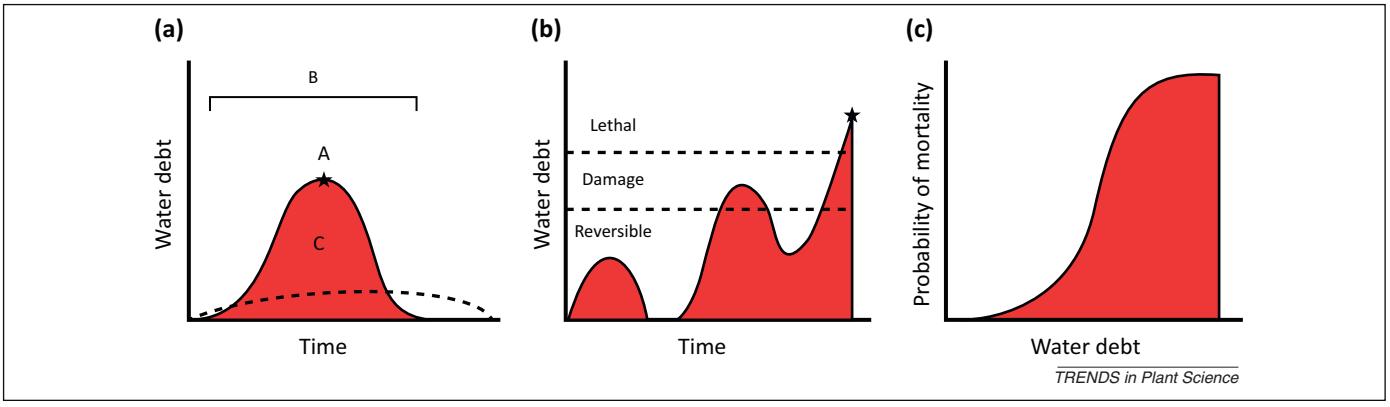

Figure 3. Visualizing tree water debt over multiple timescales. **(a)** Water debt within a tree over time with two hypothetical pathways of sustained drought (dashed line) and severe drought (solid line). Point A signifies the maximal severity of the drought's water debt, line B is the duration of the drought, and red area C is the integral of drought severity and duration. **(b)** Water debt over a period longer than a single drought. Dashed lines indicate where irreversible and lethal system damage could occur. **(c)** Cumulative density of mortality probability of a given tree as a function of water debt.

found that the die-off was associated with high degrees of branch and root xylem cavitation, which occur in drought experiments as well as in the field [20]. However, drought also led to decreases in carbon uptake and branch and fine root growth, despite little observed change in non-structural carbohydrates, which probably increased subsequent years' hydraulic vulnerability and frequencies of insect and fungal attack [53,54]. Tree branches in stressed and dying areas exhibited much lower degrees of cavitation refilling and substantial 'cavitation fatigue', whereby previously cavitated xylem embolizes under less tension in subsequent droughts [55] (W.R.L. Anderegg *et al.*, unpublished). Patterns of mortality associated with canopy openness and insect dynamics suggest that biotic agents and changes in microclimate such as temperature and radiation also feed back to accelerate die-off [51,56].

For modeling aspen die-off, the Achilles heel of a single proximate mechanism is by no means clear. Instead of a web of proximate mechanisms, we posit that it is more productive to view this breakdown in coordination and transport as a whole-system failure ultimately triggered by the accumulation of water deficit. We focus below on transport failure of the xylem as being the primary modulator of plant water resources [21]. Examining tree death from this water resource-based approach yields several insights that are largely absent from previous mortality mechanisms, which we discuss below (e.g., [11,18]).

Tree death as a systems failure accumulated over multiple timescales

Multiple lines of recent evidence suggest that tree transport failure is dynamic, reversible within constraints, and contingent on previous stresses. Only in the past few years has the accumulation of stress and damage received much attention and this understanding remains limited enough to preclude thorough treatment in vegetation models. Xylem repair by regrowth and repair of embolism in xylem elements provide two examples of contingent function in plant transport systems. Xylem regrowth was identified as the primary means of repair of hydraulic conductivity and gas exchange in drought-sensitive *Callitris* conifer species, limiting recovery time following experimental drought [57]. Xylem cavitation refilling is thought to be possible, though the physiological mechanisms are poorly understood [58,59]. In general, however, cavitation repair requires water transport, phloem function, and available carbohydrates [60–62]. Cavitation as an element of transport failure may also be reversible only to a point. Even when repaired, cavitation fatigue can lead to increased vulnerability to future cavitation in some species [55]. Cavitation fatigue and xylem repair through regrowth are two physiological process that demonstrate that both accumulation and order of events determine xylem system performance [63] (Figure 3b).

Daily cycles in water stress highlight the dynamic changes in transport system function and repair. In most environments, a strong diel pattern of water use by the tree leads to large day/night differences in water potential. When plant-available soil water is limited, transpiration can exceed water uptake from the soil, leading to cavitation or drawdown of water storage capacity, but water storage may be replenished or damage may be repaired during the night if sufficient water is available [64]. As soil and plant water depletion progresses, the possibility for replenishment and repair during the night is diminished. Nighttime transpiration may provide a mechanism for system repair and transport, but this too diminishes rapidly during drought [65,66]. Hydraulic redistribution is another possible mechanism to keep tissues such as fine roots alive during daytime water stress, but at a certain point in water deficit, it cannot buffer against daytime stress or aid in nighttime repair enough to maintain transport system health [48]. Thus, daily cycles in transport system stress and function emphasize the roles of temporal dynamics in accumulating and repairing system stress, leading to 'ratcheting' or build-up of stress and repair on timescales from days to months.

Seasonal and interannual variation further underscore the influence of multiple timescales of drought stress leading to mortality. Growing evidence suggests that trees that experienced severe drought stress were subsequently more vulnerable and had higher mortality rates in a second drought [5,67,68]. For example, a rainfall-exclusion experiment in South East Asia led to changes in hydraulic performance that lasted for multiple years [69]. These studies suggest that drought can trigger long-term changes

in tree physiology that affect future survival to drought, either in increased sensitivity or in acclimation to drought stress. This accumulated and contingent stress perspective fits well with our understanding of aging and mortality probabilities in plants, as discussed above.

Transport failure as a whole-system process in a tree suggests that a single-system lethal 'threshold' for mortality is unlikely. Considering the tree as an integrated complex system, the complex state and interactions between multiple subsystems will ultimately determine the probability of tree death as a cumulative density function of the amount of stress (Figure 3c). Whether the amount of stress, conceptualized here as water debt, is a function of the absolute maximal debt (Figure 3a, point a), the time spent in debt (Figure 3a, distance b), or the integral of the two (Figure 3a, area c) is a key unknown.

Finally, assessing system recovery potential is a critical step in determining mortality. Our approach enumerated here provides two necessary criteria for assessing mortality (Box 1): (i) transport subsystems no longer effectively link parts of the tree; and (ii) transport subsystems cannot be reestablished following alleviation of stress. We suggest that measurement of the dynamics of plant water potential may be a key observation for identifying death according to the first criterion. Analogous to electrical activity in the human brain, continuous water potential measurements should reveal dynamic changes over the day and with environmental conditions when the tree is alive and cease when the tree is dead. This mortality criterion could be assessed via technologies such as stem psychrometry. In addition, the potential for system recovery from stress is key to determining the transition to death and predicting tree death. Re-watering following mortality from experimental drought stress and assessing the recovery of physiological function could help illuminate both proximate mechanisms and transition points in accumulated water debt. Drought/re-watering experiments have been performed in both herbaceous [70] and woody plants [71], and can shed light on the conditions and limits of recovery [63,72]. However, these experiments are rare to date [73] and relatively few have considered lethal or near-lethal drought stress in trees (but see [57,63]). In natural systems, restoration of average climate conditions or soil moisture for an adequate amount of time (perhaps $\geq$1 year) could accomplish a similar function to experimental re-watering.

Concluding remarks: future steps in experiments and modeling

A clear definition of mortality and application of criteria for death would help improve the comparability and utility of greenhouse drought–mortality experiments. We suggest that such studies should provide an explicit definition of mortality, clear criteria and tests, and quantitative estimates of drought intensity/duration to best facilitate comparison. Although desirable, perfect criteria and measurements of mortality are not currently achievable. However, examining coordination of water potential in response to environmental changes such as the diurnal cycle, as well as re-watering to assess plant recovery, provide promising first steps towards experimental determination of death.

Considering tree death as a complex system failure in the tree's transport capability due to accumulated water debt has several advantages over focusing on specific proximate physiological mechanisms of death. Lack of knowledge of the appropriate proximate mechanisms, their interactions, how they connect to survival probabilities, and how species-level mechanisms scale to ecosystems or modeled plant functional types may limit our ability to predict and model drought-induced mortality in the near future. Furthermore, the prevalence of certain proximate mechanisms (e.g., hydraulic failure) may be influenced substantially by the nature of the drought (e.g., severe versus sustained) and the tree's preexisting condition (e.g., root to leaf ratio) and may thus be hard to generalize. Therefore, we suggest a resource-based approach that focuses on capturing the broader processes that occur in a tree's water fluxes and reserves (such as accumulated water debt over multiple timescales), rather than specific thresholds in single-organ subsystems (such as a minimal non-structural carbohydrate pool) that themselves depend on accurate characterization of soil and plant water dynamics. This approach provides a framework within which to examine the contingent nature of tree death over multiple years/droughts and the ultimate consequences of pathogen/insect attack and other stresses (e.g., [67,74]), and a potential to scale from individual physiology to regional or global models.

This framework also suggests several promising avenues for future research. Quantifying water uptake, evapotranspiration loss of tree water reserves, and water potential dynamics, especially in extreme drought stress (Figure 2c), would greatly improve our understanding of tree water relations during drought. Quantification of water loss through other organs, bark, and cuticular conductance in severe drought is especially needed. Because these fluxes are naturally quite small, new technology and methods are needed to quantify them. This may be particularly important to pursue in field situations, because tree root distributions, fine root networks, root to leaf ratios, and soil texture are all likely to be important in the shapes of plant water uptake and water loss curves. Determining the minimal water debt of a tree in a given environment beyond which system breakdown begins is an important research question (Figure 3). Carefully controlled water additions to drought-stressed trees could illuminate the daily amount of water required to keep transport functional without accumulation of damage. In addition, examining the continuum of lethal drought stress that maps to mortality will greatly improve our knowledge of how trees die during drought (Figure 3c).

The framework for drought–mortality articulated here provides guidance for modeling tree mortality in climate–vegetation models (see [18] for an overview of current vegetation model mortality functions). For future research, rather than simulating increased mortality rates after a defined threshold in carbon/growth [75–78] or hydraulic systems [79], this approach suggests that a distribution of stresses based on accumulated whole-plant water deficit (e.g., [80] for an initial step) could be related to plot-level or grid cell-level mortality in global models (Figure 3c). This would emphasize a stochastic and contingent (awareness

of past droughts) rather than time-independent/deterministic drought–mortality response. This probability distribution function of mortality could thus be altered within models by previous drought stresses (similar to other carry-over effects of drought [75,81]). Furthermore, the W_p necessary to calculate a water debt (Equation 2) could be incorporated in vegetation models, drawing on experimental evidence or first-principles approaches to determining curves for U and ET (with species/community-specific parameters), with relatively little alteration of model structure. This accumulated water debt, with the possibilities of repair over certain timescales, could then be directly incorporated into mortality functions from water stress.

Complete understanding of the physiological pathways that occur during drought-induced mortality may not be necessary to model mortality with reasonable accuracy. By no means do we wish to discourage further research into the proximate mechanisms and their interactions during tree mortality, but instead wish to highlight that an adequate understanding of some key processes may be enough to capture the coarse mortality necessary for simulating forest response and mortality with climate change. In particular, we suspect that processes such as a tree's relative water debt may be able to capture mortality with some fidelity in the absence of complete knowledge of all the processes that occur.

Even though tree death is ubiquitous, it remains poorly understood, which hinders our ability to make projections in the future. We believe that a holistic approach allows consideration of physiological mechanisms of drought-induced mortality in a broader context. Such an approach involves recognition of: (i) the fundamental interdependencies between water and carbon within plants leading to system-level failure during death; (ii) the critical role of the ability to repair damage across multiple timescales; (iii) carbon allocation in the context of the whole organism's life cycle and risk-avoidance strategy; (iv) acclimation and the plasticity of response to multiple and/or extended droughts; and (v) framing of definitions of death in a manner appropriate to a tree as a life form.

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