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Why is tree drought mortality so hard to predict?

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Review

Why is Tree Drought Mortality so Hard to Predict?

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Widespread tree mortality following droughts has emerged as an environmentally and economically devastating 'ecological surprise'. It is well established that tree physiology is important in understanding drought-driven mortality; however, the accuracy of predictions based on physiology alone has been limited. We propose that complicating factors at two levels stymie predictions of drought-driven mortality: (i) organismal-level physiological and site factors that obscure understanding of drought exposure and vulnerability and (ii) community-level ecological interactions, particularly with biotic agents whose effects on tree mortality may reverse expectations based on stress physiology. We conclude with a path forward that emphasizes the need for an integrative approach to stress physiology and biotic agent dynamics when assessing forest risk to drought-driven morality in a changing climate.

Ecological and Physiological Complexity Mediate Tree Drought Responses

Understanding and predicting the drivers of tree mortality during and following drought is a long-standing scientific problem with wide-ranging ramifications from environmental conservation to climate change mitigation efforts [1–11]. Widespread observations of drought-driven mortality with anthropogenic climate change makes understanding tree mortality mechanisms particularly timely [12–14]. Though it has been well established that tree physiology is important in understanding tree **vulnerability** (see Glossary) to drought-driven mortality, so far, physiology alone has had limited success in predicting which trees will die and when (or drought **mortality risk**).

Over the past decade, considerable effort has been invested to mechanistically predict tree mortality based on physiology with mixed results [15]. In some systems, **plant functional traits** as simple as wood density or specific leaf area are statistically associated with mortality rates among species in a community [16–19]. Where known, more mechanistic plant **hydraulic traits** have proven useful for understanding mechanisms that underlie drought-induced tree mortality [17,20–22]. For example, species mean hydraulic traits, such as the **water potential** at which 50% of stem **xylem** conductivity is lost (**P50**) and **hydraulic safety margin** (**HSM**, the difference between P50 and minimum stem water potential), may imply a distinct ordering of co-occurring species' risks to drought mortality. However, our predictive ability remains relatively weak. For instance, with site-specific measurements, hydraulic traits predicted around 60% of the variation in local mortality rates among 53 species in a diverse tropical forest [23]. Yet, in a global meta-analysis of predominantly less diverse systems where species mean trait values were used, less than 30% of the variation in relative mortality risk was explained [17].

Moreover, plant hydraulic models of various complexity have had partial, but fairly limited, success explaining spatial mortality patterns within individual species on the landscape during

Highlights

Drought mortality has wide-ranging ramifications from environmental conservation to climate change mitigation efforts. Thus far, mortality prediction efforts using physiology alone have found limited success.

Physiological interactions, such as within-species trait variation, trait covariation, and trait–environment covariation, can reverse or confound mortality predictions.

Ecological complexity, particularly the degree to which biotic mortality agents are linked to stress physiology, is highly variable. Thus, the presence of biotic agents has strong potential to reverse or confound mortality predictions.

We present a framework to integrate our understanding of complex drought physiology and biotic mortality agents.

Future work is needed to understand where and when biotic mortality agents might amplify patterns of physiological stress and where and when the effects of biotic agents might be largely decoupled from physiological stress.

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drought [3,7,24–28]. For example, a combination of a soil hydraulic model and species HSMs predicted only 27% of the spatial variation in mortality of 44 European tree species [25], and a detailed site-level plant hydraulics model could explain <10% of the spatial mortality variation in eight North American tree species [27].

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The challenge of mechanistic mortality prediction arises in part from uncertainty in our understanding about basic plant hydraulic physiology, which makes it difficult to rank species or individuals at a site or populations within a species on a landscape in terms of their physiological vulnerability. For example, the actual anatomical drivers of xylem resistance to drought-induced damage remain contested [29–33]. Further, a strong understanding of **physiological drought vulnerability** at multiple spatial, temporal, ecological, and evolutionary scales will require both the resolution of long-standing methodological debates [34] and collaboration between functional and evolutionary xylem biologists and anatomists [35,36].

However, we argue that predictions of drought mortality are critically impeded by the fact that some key mortality drivers are not incorporated into hypothesized mortality mechanisms rooted in physiology alone. Indeed, an experiment explicitly designed to emulate forecasted end-of-century climate scenario conditions with co-occurring elevated temperature and severe drought resulted in no tree mortality, despite widespread observed climate-driven die-off of the same species in the same region several years earlier [37], likely due to local site conditions and the absence of drought interactions with bark beetle outbreaks [38]. In this review, we outline some prominent reasons for why tree mortality is so hard to predict from physiology alone and present key avenues for making progress.

Within-Species Trait Variation and Trait Covariation in Key Unmeasured Traits Can Confound Mortality Predictions

To date, information on individual hydraulic traits (such as P50) that are emerging mediators of drought mortality often comes from a single tissue (e.g., branches) from only a handful of trees that were selected as being ‘typical’ or ‘ideal’ and are often represented as a species mean despite the fact that a handful of trees is not a good population sample. Further, these species mean trait values yield no inference about the underlying selective pressures that reveal a trait’s ‘functionality’ (i.e., effect on fitness), which operate within a population [39]. If we attempt to predict the physiological vulnerability to mortality of various species in a plant community from a trait, limited trait sampling (often due to the extreme logistical challenges of hydraulic measurements) can result in a drought risk ordering that shows distinct, nonoverlapping peaks for the species (Figure 1A).

Two processes likely broaden the distribution of mortality risk among individuals of a species, variation in physiological vulnerability (i.e., variation in physiological traits linked to drought survival) and variation in drought **exposure** (i.e., within-site and between-site variation in water availability). When processes like natural variation in chronic stress are added to a population of trees [40], mortality risk distributions likely broaden and are biased toward higher mortality risk simply because most trees in natural populations experience some level of chronic stress (as indicated by suboptimal growth compared with the maximum potential growth at that site), whether due to competition, poor microsite conditions, biotic attack, past damage, or other mechanisms. Chronically growth-suppressed trees can have xylem architecture that reduces their hydraulic conductivity (such as narrow tracheids or pits in conifers), greater hydraulic failure (cavitation) preceding and during drought, greater diffusional resistance to gas exchange (limiting carbon fixation), and reduced defenses (such as fewer or narrower resin ducts) or a combination of compensating physiological factors [40–43].

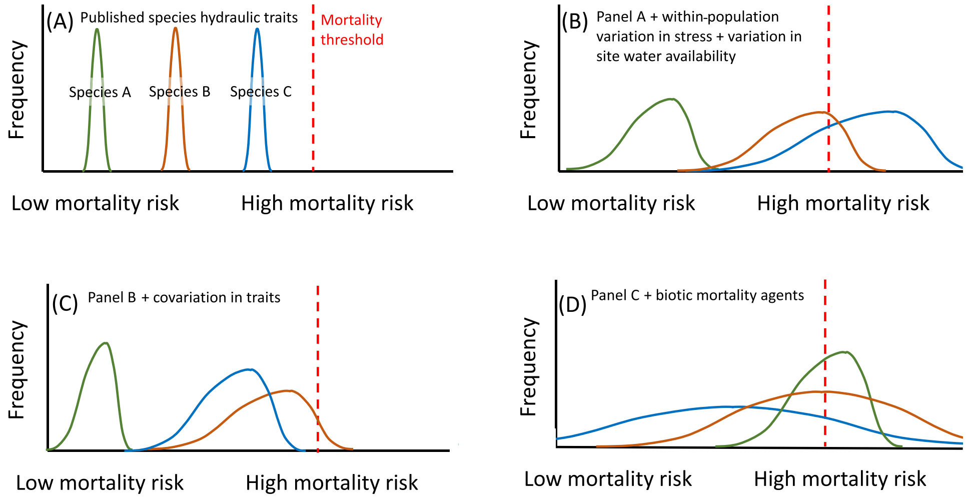

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Figure 1. Example Ordering of Mortality Risk and the Mortality Threshold During a Particular Drought for Three Different Tree Species with Different Hydraulic Traits. (A) At one extreme, species 'mean' hydraulic trait values for one trait determine physiological vulnerability and therefore mortality risk. (B) Curves broaden due to natural variation in chronic stress within a population of trees and site-to-site variation in tree water availability. Curves are likely dominated by higher mortality risk because most trees in natural populations experience some level of chronic stress, whether due to competition, poor site conditions, biotic attack, past damage, etc. (C) Covariation between multiple physiological traits (e.g., plant height and rooting depth) or between physiology and environment [the water potential at which 50% of stem xylem conductivity is lost (P50) and soil water-holding capacity] may tighten and/or reverse risk curves as multiple physiological dimensions have the potential to balance out or exacerbate vulnerability. (D) The prevalence and effects of biotic mortality agents vary broadly among tree species and can reverse ordering of species along the mortality risk axis.

Drought exposure also varies among individuals because there is heterogeneity in water access within and between sites. Thus, a chronically growth-suppressed tree, such as one that is shaded by a taller tree, might have access to adequate water if there is an upslope subsurface reservoir feeding its site [44,45]. By contrast, a chronically stressed tree on a dry site will suddenly have acute drought stress layered onto its chronic stress and will be at high risk of mortality [46]. The combination of variation in vulnerability due to chronic stress within a population and microsite variation in tree exposure may result in the broadening of vulnerability distributions within a species to the point of overlapping mortality risks among species (Figure 1B).

Covariation among multiple physiological traits or between traits and exposure may tighten and/or reverse risk distributions (Figure 1C). Some notable examples where covariation among physiological traits is fundamental to plant mortality risk include (i) **trait covariation** between tree height and P50, where xylem taper acts to mitigate the increase in hydraulic resistance as an individual grows taller at the cost of less **embolism**-resistant xylem [47,48]; (ii) covariation between factors such as plant height and rooting depth, which affect tree hydraulic vulnerability and water access, respectively; (iii) covariation between physiological traits and environment, such as covariation between P50 and soil water holding capacity or root water access [49]; and (iv) genetic tradeoffs between growth and biotic agent defense [50] versus vigor-related positive correlations between growth and defense [42,43]. These multiple physiological dimensions have the potential to either balance (as illustrated in Figure 1C by a net shift toward lower risk) or exacerbate risk as assessed by one plant trait.

Tree height presents an illuminating example, as there is a wealth of research on changes in traits with tree height, and height has been hypothesized to drive vulnerability to drought mortality

Glossary

Diameter at breast height (DBH): a standard method of expressing the diameter of the trunk or bole of a standing tree measured at ~1.3 m in height.
Disarraying agents: biotic agents disarray mortality predictions among species in a community that are based on our current understanding of physiology alone because the nature and magnitude of effects of biotic mortality agents vary broadly among tree species.
Embolism: xylem embolism occurs when the tension of water within the xylem causes the formation of air bubbles that expand and block a xylem vessel.
Endemic biotic agents: biotic agents, such as insects, fungi, bacterial and viral pathogens, and parasitic plants, that occur more in continuous background levels without strong spatial patterns.
Epidemic biotic agents: biotic agents that occur primarily in rare, large, spatially aggregated outbreaks.
Exposure: environmentally driven variation in drought mortality risk, either among microsites within a site or among sites on the landscape.
Hydraulic safety margin (HSM): the difference between P50 and either minimum stem water potential or **leaf turgor loss point**.
Hydraulic traits: plant functional traits describing a plant's ability to move water and withstand extreme **plant water potentials** that reflect a plant's evolutionary history and functional ability to respond to changes in water availability.
Leaf turgor loss point: point at which dehydration overcomes a plant's capacity to maintain leaf cell turgor.
Mortality risk: the integrated probability (including physiological vulnerability, exposure to drought stress, and the net result of biotic interactions) of an individual, population, or species dying as a result of a drought event.
P50: the water potential at which 50% of stem xylem conductivity is lost.
Physiological drought vulnerability: the drought mortality risk as predicted based on plant hydraulic traits.
Plant functional traits: plant characteristics at tissue-to-organismal scales that impact plant fitness.
Plant water potential: a physiological diagnostic of plant water status.
Physiological stress: factors that reduce a plant's growth below the

[51–54]. Considering the hydraulic costs of tree height in isolation (longer path length, larger hydrostatic gradient), larger trees would appear to be more vulnerable to water stress [55]. Based on our theoretical understanding of plant hydraulics, on the driest sites, trees of all sizes suffer hydraulic damage, but in wetter soils, only the tallest trees suffer hydraulic damage and thus suffer higher mortality risk if all other traits are held constant (Figure 2A). Thus, any species with no covariation between tree size and water access will be more vulnerable to hydraulic damage as it grows taller (blue line in Figure 2A incurs more hydraulic damage at large heights).

Crucially, however, trees tend to offset hydraulic limitations with height by accessing more and different water resources as they grow. For example, the allometry of root biomass, rooting volume, and rooting depth to aboveground biomass changes with plant size, particularly as trees progress from saplings to mature trees [56–58], and mature trees can exploit deep water in weathered bedrock and other reservoirs [59–66]. Moreover, deep water access or groundwater subsidies may be a precondition for the presence of tall trees on many landscapes, so that tall trees are actually indicators of low exposure because they grow on anomalously wet sites that are least likely to dry out during drought [44]. For example, *Sequoiadendron giganteum* (giant sequoia) in Sequoia and Kings Canyon national parks (CA, USA), which can reach heights >90 m, suffered extremely low overall mortality (<1%) compared with surrounding forests during a recent severe Californian drought (2012–2016), likely due in part to enhanced local water availability on the sites that they dominate [67]. Such deep water subsidies may prove to be stable buffers against future droughts, unless long-term changes to the hydroclimate alter these subsidies.

The belowground resource access afforded to large trees can easily decrease mortality risk with tree size due to a root-driven increases in water access. Thus, depending on the nature of this covariation, tall individuals of a certain species can be more vulnerable to drought stress (blue line, Figure 2A), less vulnerable to drought stress (red line, Figure 2B), or equally vulnerable due to equifinality between rooting depth/volume, height-driven hydraulic costs (turquoise line, Figure 2B), providing one explanation for the lack of consensus within and among observational studies of size-specific tree mortality [10,23,26,63,68–72]. Similarly, the tendency for large trees to only grow on wet sites with groundwater support and the effects of xylem tapering to counter increased hydraulic resistance with height would have the same outcome as the covariation between tree size and rooting depth [48,67].

Studies of landscape mortality patterns (such as those using remote sensing) that make no distinctions among tree species may conflate within- versus among-species mortality risk. Thus, it is critical to differentiate whether tall species experience the highest mortality, in which case mortality risk may be driven by species-specific drought and biotic mortality agent vulnerability, or whether tall individuals within a species experience high mortality, in which case stature may actually be driving vulnerability and ultimately mortality risk. For example, it is possible that across species, small individuals experience the most mortality (Figure 2C). However, at the landscape level, the wettest and tallest species is most vulnerable overall because of its combination of hydraulic traits. Even though the tallest individuals of this species are actually the least vulnerable, if no distinction is made among species, tree height appears to predict mortality purely because sites dominated by the tallest species show the most mortality [73]. In actuality, within-species patterns may be quite complex, where tall individuals are more vulnerable in some species and small individuals are more vulnerable in others [10,70]. Meanwhile, across species, the potential for compensating physiological variation along numerous environmental and/or physiological axes likely reduces the predictive ability of any one particular physiological trait in isolation [40,63,70]. Further, among-species differences, not only in absolute physiological trait values but also in the extent to which traits covary, may result in a reordering along the mortality

optimum that it would achieve in their absence.

Stress compounder: a biotic agent that primarily reinforces physiological patterns of stress within a given species during drought, either because they preferentially attack stressed individuals or only successfully overcome the defenses of stressed individuals. Stress compounders effectively lower the physiological stress threshold for mortality.

Stress confounder: a biotic agent that complicates or contradicts patterns of physiological drought stress within a species where factors other than stress, such as tree size independent of stress, determine attack dynamics and mediate which trees die.

Trait covariation: covariation among multiple functional traits.

Vulnerability: physiological risk of drought mortality determined by functional traits such as plant hydraulic traits.

Xylem: plant vascular tissue that conducts water and dissolved minerals from the roots to the rest of the plant.

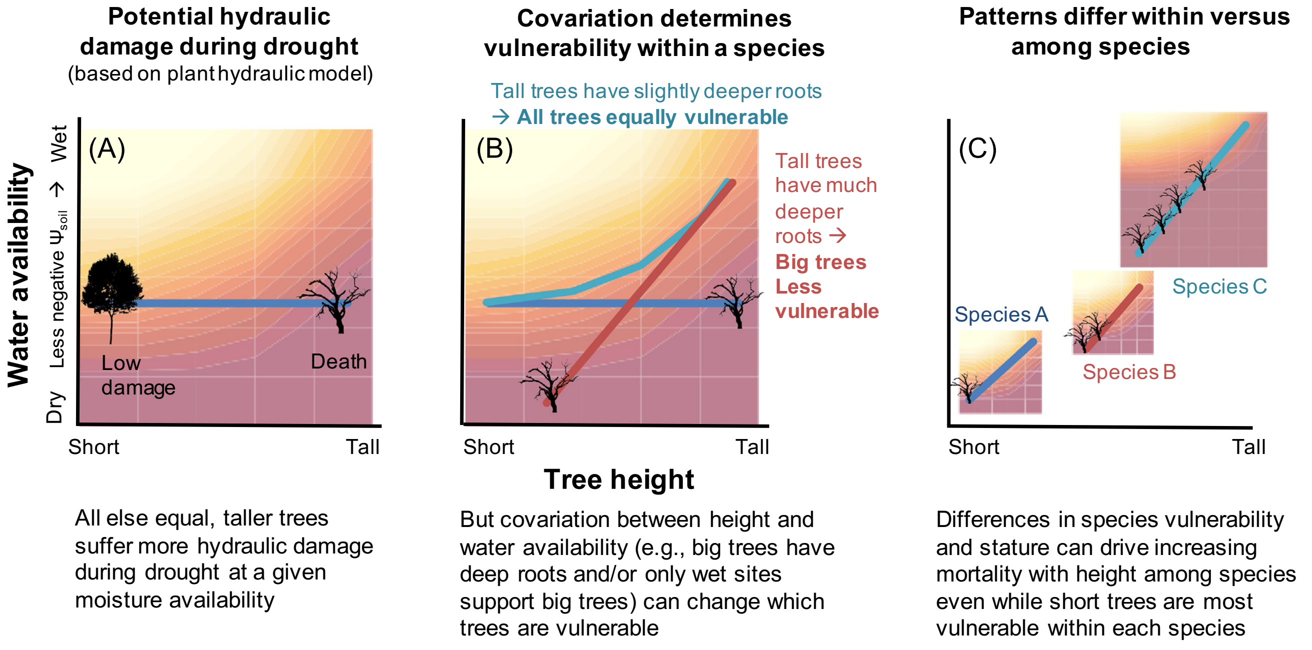

Figure 2. Covariation in Tree Height and Water Availability Can Lead to a Variety of Vulnerability Profiles with Tree Height. (A) Heat map of hydraulic damage during drought based on a plant hydraulic model [55] as a function of tree height and water availability. Darker colors indicate increased hydraulic damage, with the darkest red indicating mortality. Blue line illustrates a hypothetical species with constant microsite water availability across size classes (no covariation between height and rooting depth or site quality), whose tallest individuals experience mortality (dead tree icons indicate drought mortality). (B) Scenarios of covariation between height and water availability. Red line, water access increases substantially with size, either through increased root depth or microsite selection whereby only wetter sites support larger trees, and the smallest trees die during drought. Turquoise line, rooting depth increases slightly with size, primarily in the tallest trees, and all size classes experience similar sublethal damage. (C) Hypothetical patterns of mortality among species that inhabit different parts of a water availability gradient. Small trees of each species are most vulnerable to mortality; however, the wettest, tallest species (Species C) is most vulnerable overall (as indicated with dead tree icons) either due to its combination of physiological traits or susceptibility to biotic mortality agents. When examining trends across species on the landscape (across colors), tree height appears to be positively related to mortality because Species C is the most vulnerable to drought based on its other hydraulic traits, not because of the effect of height *per se*.

risk spectrum compared with predictions derived from vulnerability estimates using only one physiological trait (Figure 1C).

Empirically, predawn leaf water potential reflects soil water availability integrated across a plant's root system (assuming no nighttime transpiration) and provides a useful metric for examining whether larger trees are either less vulnerable or less exposed to water stress (Figure 2). In the literature, while it is not uncommon to find more negative water potentials in larger trees (beyond that expected from the hydrostatic gradient alone [74]), it is more common to find either no effect of tree size [75,76] or that larger trees have less negative predawn water potentials [77,78]. For example, within elevation bands of *Pinus ponderosa* (ponderosa pine) across an elevation gradient in southwest Colorado, taller trees have less negative predawn water potentials ($P = 0.01$ for low elevation and $P < 0.0001$ for mid; Figure 3A) and thus appear to be less vulnerable because they have access to more soil moisture during periods of peak water stress, presumably due to greater rooting depths. Meanwhile, *Populus tremuloides* (trembling aspen) growing higher up the same mountain in Colorado do not show strong height-related water potential patterns within any elevation but grow taller at wetter and cooler high-elevation sites ($P < 0.001$; Figure 3B). In this case, stable wet sites are a strong precondition for tall aspens on the landscape, and maximum height is negatively correlated with exposure, resulting in higher observed mortality at drier sites with shorter canopies) [24,40,79]. Thus, covariation among traits and between traits and environmental exposure can quite easily confound mortality risk predictions based on individual traits alone.

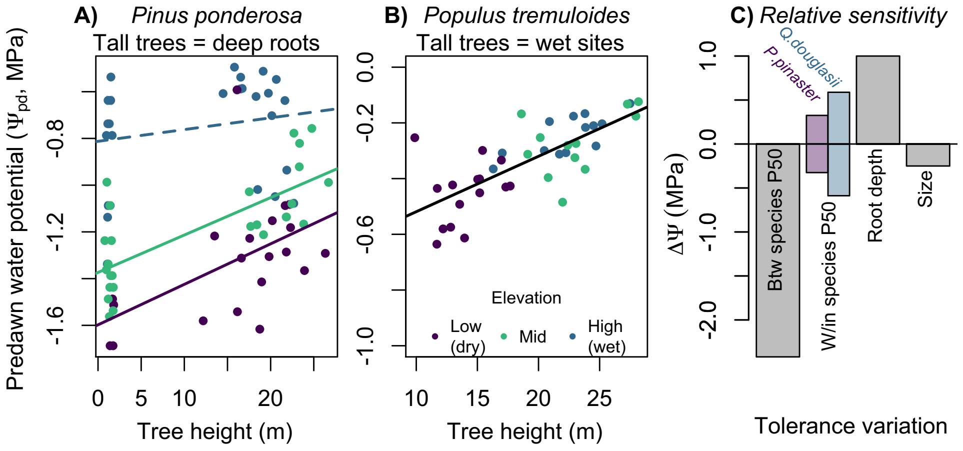

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Figure 3. Height-Corrected Predawn Water Potentials as a Function of Tree Size for a Gymnosperm Species (A) and an Angiosperm Species (B) across an Elevational Climate Gradient in the Western USA. Water potentials were corrected for the hydrostatic gradient with height to better indicate root water access, which increased with tree height within dry *Pinus ponderosa* sites except at the wettest high elevation (A) and across *Populus tremuloides* sites (B) [40]. (C) Potential variation in plant physiological stress tolerance as measured through (i) relative increases in physiological stress due to changes in the water potential at which 50% of stem xylem conductivity is lost (P50) between a more drought-resistant and a more drought-vulnerable species for colocated species in the western USA ['between (Btw)-species P50'] [109]; (ii) range of physiological stress due to literature-reported intraspecific variation in P50 relative to species' means for two well-studied species ['within (W/in)-species P50'], average of between-population range of *Pinus pinaster* [110] and between-individual interquartile range of *Quercus douglasii* [111]; (iii) change in physiological stress for a deep-rooted tree relative to a shallow-rooted tree ('Root depth'); (iv) relative changes in water potential stress required to cause irreparable damage in a mechanistic plant hydraulic model due to variations in tree size for a 32-m tall or an 80-cm **diameter at breast height (DBH)** tree relative to an 11-m tall or a 10-cm DBH tree with equal water access ('Size') [55].

We further used a tree model that couples carbon allocation to local environmental conditions through gas exchange and plant hydraulic transport [55] to understand the impacts of increasing size in determining a tree's ability to recover from drought damage and compared results with observed among- and within-species P50 variation and potential increases in root water access within a soil profile (Table 1 and Figure 3C). Though all factors influenced tree vulnerability to water stress, among-species variation in the P50 of colocated species has the largest impact on **physiological stress**, and the simulated impact of tree size had the smallest (Figure 3C), indicating that fairly minor changes in either rooting depth or P50 with height could offset height-related hydraulic burdens. Moving from a simplified, unidimensional understanding of plant mortality risk (e.g., based on vulnerability estimates from single traits such as height or P50; Figure 1A) to a whole-tree understanding of mortality risk requires new insights into the evolutionary, biophysical, and ecological mechanisms driving trait–trait covariation underpinning vulnerability and trait–environment covariation underpinning exposure. For example, understanding the evolutionary selection pressures maintaining constant leaf-specific hydraulic conductance with height or constant metabolic ratios of xylem volume and leaf area with height could broadly illuminate whether tall trees are inherently more vulnerable during drought and why [48].

Biotic Mortality Agent Population and Attack Dynamics Can Reverse or Confound Physiological Mortality Predictions

The difficulty in fully understanding stress physiologies also stems from the highly variable linkages between physiology and biotic agent dynamics in determining tree drought mortality [38,42]. For example, biotic agent outbreaks, such as bark beetle outbreaks, may start in the most stressed trees early in a drought, but beetle outbreaks often persist for many years after the drought has

Table 1. Summary of Comparisons Illustrated in Figure 3 with Physiological and Environmental Data

Comparison name	Summary	Details
Between-species traits	Illustration of the relative differences in drought vulnerability due to between-species differences in species mean water potential at which 50% of stem xylem conductivity is lost (P50) values of colocated species.	Relative differences in the ability to withstand physiological stress as quantified by differences in species mean P50 values of colocated species. Values plotted as relative difference between a more drought-resistant (more negative P50) and a more drought-vulnerable (less negative P50) species. Relative difference in species mean values calculated as the median within-plot variation in P50 for all plots within a latitude/longitude box spanning from 115°W to 105°W and from 35°N to 40°N based on [109].
Within-species traits	Illustration of the range in drought vulnerability due to within-species variation in P50.	Relative range within a species of the ability to withstand physiological stress as quantified by relative range in recorded P50 values. Values are literature-reported within-species variation in P50 relative to the species mean P50 for two well-studied species (total between-population P50 range for *Pinus pinaster* [110] and interquartile range of individuals of *Quercus douglasii* [112]).
Root depth	Illustration of the increase in soil water access with increased tree rooting depth.	Relative increase in soil water potential with a root depth increase of 1 m (as might be expected for a tree growing from a sapling to a mature tree). Water potential change was estimated using the GLDAS-2 monthly soil moisture product for the soil layer 100–200 cm in depth relative to the layer 10–40 cm in depth. Mean water potential difference was estimated for a climatology for the same spatial area as co-occurring P50 values (a latitude/longitude box spanning from 115°W to 105°W and from 35°N to 40°N). Allometric scaling in root depth with tree size was estimated from [56]. This represents a conservative estimate for increases in root depth and water access with size (for example, the increase in soil water potential was 1.6 MPa on average over the same region when comparing the soil layer 100–200 cm in depth with the layer 0–10 cm in depth, where seedlings likely have root water access).
Size	Illustration of the relative difference in physiological stress due to tree size.	Simulated changes in the physiological stress due to size-related hydraulic and carbon costs [55] for a 32-m tall or 80-cm diameter at breast height (DBH) tree relative to an 11-m tall or 10-m DBH tree (a size range that is inclusive of the majority of trees seen in North America) with equal root water access and the same P50.

ended, highlighting a subsequent disconnect between stress physiology and biotic agent mortality [50,80]. The stress physiology and biotic mortality agent disconnect is further illustrated by instances where, during drought, some beetle species attack and kill the most chronically stressed trees but others attack based on tree size and independent of chronic stress. The end result is that for some tree species, the least-stressed trees may die and the most-stressed trees might live [10].

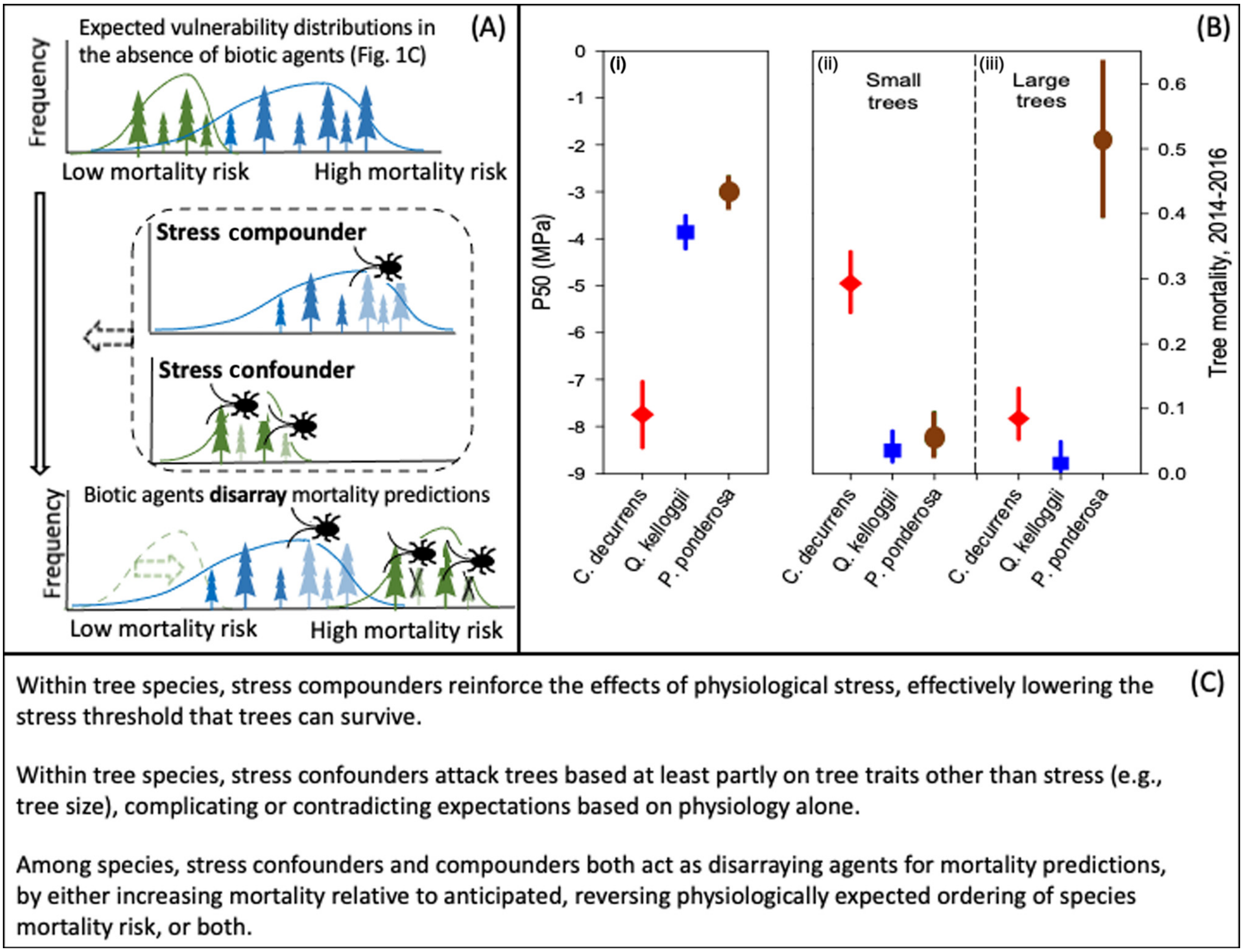
Pathogen and pest attacks have traditionally been characterized as either **epidemic** or **endemic** [81] and as either 'primary' (killing otherwise healthy trees) or 'secondary' (typically attacking already weakened/stressed trees and rarely killing trees in isolation) [11]. However, these categories do not fully address complexities of within- versus among-species effects and can even be misleading as the dynamics of biotic mortality agents change in the face of ongoing environmental changes.

We propose to categorize biotic attacks based on the way in which they interact with plant stress physiology such that **stress compounders** primarily reinforce physiological patterns of stress within a given species during drought (i.e., biotic agents effectively lower the physiological stress threshold for mortality, but tree stress still predicts which trees die) versus **stress confounders** that complicate or contradict patterns of physiological drought stress within a species (i.e., factors other than stress, such as tree size independent of stress, predict which trees die) (Figure 4). Importantly, both within-species compounders and confounders can be epidemic (or not). Forests in California's Sierra Nevada, USA, present an illustrative example of the complexity of biotic agent dynamics during drought. For example, attack dynamics of *Scolytus* bark beetles acted as stress compounders on *Abies concolor* (white fir), killing the most stressed trees with chronically low growth rates and reaching epidemic levels on the landscape. Meanwhile, *Dendroctonus* bark beetles acted as stress confounders on Pinus, targeting the largest individuals regardless of stress and reaching epidemic levels. At the same time, *Pseudopityophthorus* bark beetles acted as stress compounders on *Quercus kelloggii* (black oak), targeting the most stressed trees, although *Pseudopityophthorus* did not reach epidemic levels [10].

Among species, both stress compounders and stress confounders act as **disarraying agents** for mortality predictions, that is, they disarray mortality predictions that are based on physiology alone because the nature and magnitude of effects of biotic mortality agents vary broadly among tree species [10,82–84] and might even reverse physiologically derived ordering of species along the mortality risk axis (e.g., Figures 1D and 4A). Given that it is extremely unlikely that various biotic mortality agents will affect each species in a forest proportionately, preserving the same mortality patterns as one would expect based on physiology alone (e.g., Figure 1A) is improbable. Moreover, the confounder versus compounder behavior of a biotic mortality agent within a species can be context dependent, with some agents becoming stress confounders during extreme droughts, in novel climates, or outside of their native range.

Revisiting the Sierra Nevada example for three co-occurring tree species, based on P50 alone, one would expect *Calocedrus decurrens* (incense cedar) to be much less vulnerable to drought mortality than *P. ponderosa* and *Q. kelloggii* (as indicated through the extremely negative P50 of *C. decurrens*) and *P. ponderosa* to be the most vulnerable to drought mortality (as indicated through the less negative P50 of *P. ponderosa*). However, the observed mortality patterns do not correspond with the physiologically derived predictions, with mortality trends even diverging across tree size classes (Figure 4B). Among small trees, the tree mortality fraction of *C. decurrens* is larger than that of *P. ponderosa* and *Q. kelloggii* by an order of magnitude, despite predictions based on P50 indicating that mortality fraction should be lowest in *C. decurrens* (Figure 4B). Among large trees, the mortality fraction roughly corresponds with physiologically derived ordering of species for *C. decurrens* and *P. ponderosa*. However, *Q. kelloggii*, which has a species mean P50 that suggests moderate to high vulnerability, suffered almost no mortality. In the case of small *C. decurrens* and large *P. ponderosa*, mortality was driven by native bark beetles, with host selection differing strongly by size both within and among species [10].

Despite the critical role of biotic agents on tree mortality, anticipating and predicting biotic agent-driven mortality remains elusive [1,2,85] because host–pathogen interactions vary wildly among tree species and biotic agents (and agents often co-occur and are present at background levels in healthy ecosystems) [82–84,86] and because of the complexity and importance of indirect and interactive effects of biotic mortality agents with other environmental factors [87,88], which



Within tree species, stress compounders reinforce the effects of physiological stress, effectively lowering the stress threshold that trees can survive.

Within tree species, stress confounders attack trees based at least partly on tree traits other than stress (e.g., tree size), complicating or contradicting expectations based on physiology alone.

Among species, stress confounders and compounders both act as disarraying agents for mortality predictions, by either increasing mortality relative to anticipated, reversing physiologically expected ordering of species mortality risk, or both.

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Figure 4. Biotic Agents can Disarray Mortality Predictions by Amplifying or Reversing Expectations for Mortality Based on Physiology Alone. (A) Schematic of the diverse, context-dependent dynamics of biotic agents, ranging from agents that are primarily stress compounders, attacking already stressed individuals (blue distribution), to agents that are primarily stress confounders, attacking the least stressed individuals (green distribution). Faded individuals indicate trees attacked by biotic agents. Both stress compounders and confounders act as disarraying agents for mortality predictions because they increase mortality relative to anticipated levels based on physiology alone and can reverse physiologically derived ordering of species. In this scenario, blue and green distributions reverse because the attacked green trees die (black 'X'), while the attacked blue trees survive. The faint green distribution references the expected risk distributions in the absence of biotic agents. (B) Observed mortality fraction during drought for colocated species does not necessarily correspond with species that are more physiologically vulnerable to drought stress due to diverse biotic agents. (i) Drought vulnerability as measured through the water potential at which 50% of stem xylem conductivity is lost (P50) values (where less negative values correspond with more drought-vulnerable species) for three co-occurring species, including *Calocedrus decurrens* (red), *Quercus kelloggii* (blue), and *Pinus ponderosa* (brown). Fractional mortality (in number of trees) in Sierra Nevada, CA, USA, during an extreme drought for *C. decurrens*, *Q. kelloggii*, and *P. ponderosa* for small trees (<10-cm diameter at breast height, DBH; ii) and large trees (>50-cm DBH; iii). Mortality data are from [10]. Symbols represent means and error bars represent 95% confidence intervals for P50 [e.g., (i)] and 95% credible intervals for mortality [e.g., (ii and iii)] (see the supplemental information online). (C) Terminology for how biotic agents can influence mortality predictions.

amplifies the number of unknowns surrounding host–pathogen ecological interactions. Further, biotic agent mortality prediction has the potential to become increasingly difficult with the novel climate regimes expected to accompany anthropogenic climate change [89,90] because host–pathogen interactions may change compared with historical observations [91–97].

There are three primary mechanisms through which we expect climate change to affect biotic agent-based mortality predictions. First, climate change can increase the likelihood of epidemic-level eruptions of known biotic mortality agents, such as bark beetles, in known host species [84,93,98]. In particular, agents such as *Dendroctonus* beetles known to employ pheromone-based mass-attack strategies are most likely to drive stress, confounding mortality whenever they achieve an outbreak. Outbreaks are more likely either when climate conditions, such as warming winters, allow pest populations to more easily reach epidemic levels [99] or expand to attack previously naive populations [100] or when climate conditions stress hosts and suppress defense responses to allow major outbreaks [101]. Currently, a handful of agents that may increase their epidemic-level eruptions constitute 'known unknowns' of biotic agent-driven mortality and are good candidates for improved mortality predictions through better understanding of biotic agent biology (e.g., see [70]).

Second, formerly innocuous insects and pathogens can unexpectedly emerge as significant agents of tree mortality [10,94,95,102–104]. Recent examples include bark weevils of the genus *Pissodes,* which are not generally associated with host mortality [81] but became an important mortality agent of small pine trees in a recent California drought [10]. Likewise, *Dothistroma* needle blight went from a relatively mild endemic pathogen to an unprecedented tree mortality driver in its native range in British Columbia, Canada, likely due to an increase in warm, wet conditions [95]. This type of 'unknown unknown' may prove particularly challenging because it involves novel pest behavior, and there is often a dearth of data collection geared toward documenting formerly innocuous insect and pathogen prevalence and impact. Early detection and documentation of co-occurring agents present at locations of ongoing tree mortality is needed to better understand when formerly innocuous agents become deadly.

Third, the introduction of nonnative pests can increase biotic agent-driven mortality and act as strong disarraying mortality agents because they are a second variety of 'unknown unknowns'. Examples of nonnative pests include the emerald ash borer *Agrilus planipennis* in the eastern USA [105,106], acacia wilts of the genus *Ceratocystis* in Indonesia [107], the red turpentine beetle *Dendroctonus valens* in China [92], the bark beetle *Polygraphus proximus* in Russia [96], and pathogens such as *Sphaeropsis* canker (midwestern and northeastern USA) and red band needle blight *Dothistroma* (extensive pine defoliation worldwide, including in Africa, New Zealand, South America, and North America) [97]. The nonnative pest challenge presents a significant opportunity for collaboration among plant physiologists, entomologists, and pathologists to target appropriate measurements and increase data availability so that tree species and forest ecosystems at risk for nonnative invasion mortality events can be identified.

Concluding Remarks and Future Perspectives

Here, we present a path forward to open new avenues of measurements and exploration directed toward tree drought mortality and terrestrial carbon cycle predictions (see Outstanding Questions). We argue that treating plant physiology and biotic mortality agents as an integrated system rather than bifurcated disciplines in data collection and prediction is a critical step toward understanding the highly variable linkages between physiology and biotic agents in drought-driven tree mortality and forest responses to global change. An essential first step in advancing tree drought mortality predictions is constraining where and when tree mortality at the hands of biotic agents is linked to stress physiology and where and when biotic agents and physiology are decoupled. Such baseline data, particularly for tropical systems where data are extremely sparse [108], will be critical for understanding the baseline natural history and shifting dynamics of host–pathogen interactions. More existentially, most of the forest pest canon and theory behind biotic agent ecology derives from low diversity temperate forests, and it remains largely

Outstanding Questions

What trait covariation axes are key in mediating tree stress responses during drought both within and among species?

What key physiological attributes are most critical for mortality prediction either because they hold the most leverage over plant stress physiology or because they are most variable within a species?

Despite their diversity of effects, can biotic mortality agents be productively categorized and modeled as 'stress confounders', where effects of biotic agents are largely decoupled from tree stress physiology, and 'stress compounders', where effects of biotic agents amplify patterns of tree physiological stress?

Is it possible to predict which trees or tree species are most likely to be attacked by 'disarraying agents' (biotic agents that upend expected mortality patterns)?

Which biotic mortality agents are likely to be the most important disarrayers?

unknown how to generalize the role of biotic agents in tree mortality in the tropics [5]. Addressing these knowledge gaps requires a reframing of current field sampling and experimental design best practices such that forest demographic surveys and plant physiological measurements rigorously document both biotic agent presence in demographic surveys and the physiological consequences of trees actively fighting them.

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Declaration of Interests

No interests are declared.

Supplemental Information

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