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A multi-species synthesis of physiological mechanisms in drought-induced tree mortality

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Widespread tree mortality associated with drought has been observed on all forested continents and global change is expected to exacerbate vegetation vulnerability. Forest mortality has implications for future biosphere–atmosphere interactions of carbon, water and energy balance, and is poorly represented in dynamic vegetation models. Reducing uncertainty requires improved mortality projections founded on robust physiological processes. However, the proposed mechanisms of drought-induced mortality, including hydraulic failure and carbon starvation, are unresolved. A growing number of empirical studies have investigated these mechanisms, but data have not been consistently analysed across species and biomes using a standardized physiological framework. Here, we show that xylem hydraulic failure was ubiquitous across multiple tree taxa at drought-induced mortality. All species assessed had 60% or higher loss of xylem hydraulic conductivity, consistent with proposed theoretical and modelled survival thresholds. We found diverse responses in non-structural carbohydrate reserves at mortality, indicating that evidence supporting carbon starvation was not universal. Reduced non-structural carbohydrates were more common for gymnosperms than angiosperms, associated with xylem hydraulic vulnerability, and may have a role in reducing hydraulic function. Our finding that hydraulic failure at drought-induced mortality was persistent across species indicates that substantial improvement in vegetation modelling can be achieved using thresholds in hydraulic function.

Increasing forest mortality from global change has been observed in all forested biomes[1,2] and will have profound implications for future energy and element fluxes[3–5]. Predictions of vegetation responses to future climate are uncertain due to the lack of realistic mortality mechanisms in vegetation models[3,6–9]. Recent research supports at least two tightly inter-related physiological mechanisms associated with tree mortality by drought: (1) hydraulic failure through partial or complete loss of xylem function from embolism that inhibits water transport through the vasculature, leading to tissue desiccation; and (2) carbon starvation via imbalance between carbohydrate demand and supply that may lead to an inability to meet osmotic, metabolic and defensive carbon requirements[3,6,7,10–15]. Hydraulic failure is most typically assessed via per cent loss of xylem conductivity (PLC) and carbon starvation via changes in tissue non-structural carbohydrate (NSC) concentrations[12–16]. There has been significant debate over these co-occurring mechanisms of mortality, particularly regarding the prevalence of carbon starvation and whether reduced carbohydrate reserves can be lethal during drought[11,17–22].

Although a number of studies on the mechanism of drought-induced mortality in trees have been conducted for a variety of tree species over the past decade, the prevalence of these mechanisms on a global scale remains uncertain. Differences in approach, variables measured, and species and life stage studied have limited global assessment of drought-induced tree mortality mechanisms. Here, we provide the first cross-species synthesis of tree drought mortality mechanisms. We used a standardized physiological framework to analyse drought-induced tree mortality across species and assessed hydraulic function as PLC, and carbohydrate status as NSC normalized relative to controls. We examined data from 19 recent experimental and observational studies on 26 species from around the globe. Most tree species were assessed in only one study, but for several species data were available from more than one study, resulting in

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34 cases (species–study combinations). However, data were not available for all analyses from all cases: more cases had NSC data (31 cases from 24 species) than PLC data (14 cases from 9 species), which could be used to compare NSC and PLC at mortality with that of surviving control trees (see Methods below, Supplementary Table 1). To make our synthesis comprehensive, we worked with all of the data that were available, including data from studies on a range of tree sizes and ontogenetic life stages (that is, seedlings, saplings and large trees), conducted in a variety of settings, including potted plants in greenhouses or growth chambers and trees grown in the field (Supplementary Tables 1 and 2). Given the diversity of studies synthesized, these data were not ideal for a statistical meta-analysis; therefore, we limited our analyses to a standard comparison within each case between plants that died and plants that remained healthy (Supplementary Methods). We also compared differences in degree of embolism and carbohydrate concentrations between plants at mortality and control plants with differences in functional traits[3,23,24]. For each species, we obtained available data for traits that are easily measured, widely available and likely to be relevant for drought tolerance, including wood density and specific leaf area (SLA)[25]. We also obtained data for hydraulic traits that are directly related to drought tolerance, but harder to measure, including xylem water potential at 50% loss of hydraulic conductivity (Ψ_{50}), point of embolism entry (Ψ_e) and corresponding hydraulic safety margins[23,26] (Supplementary Methods). We used this dataset to address the following hypotheses: (1) given the potential role of NSC in the maintenance of water transport during drought[6,27], both high PLC and reduced NSC reserves are common at tree death from drought; and (2) among species, species-level functional traits that have been positively related to drought tolerance (for example, low xylem vulnerability to embolism, low SLA, high wood density) are associated with high NSC at tree death. According to this hypothesis, we expect that for species with greater xylem vulnerability (quantified by Ψ_{50}, Ψ_e and hydraulic safety margin), NSC at death will be relatively lower. This hypothesis is based on previous proposals that drought-sensitive trees that close their stomata earlier during drought would be more likely to show a reduction in NSC associated with carbon starvation[3,24,28,29].

Results

For the cases where PLC data at mortality were available (Supplementary Methods), PLC was 60% or higher (Fig. 1a), demonstrating that a high degree of xylem embolism at drought-induced death was a universal aspect of mortality physiology in these species. Mean PLC was 84.3% at mortality and PLC was significantly higher at mortality than for control trees in every case ($P<0.05$, Student's t-test). For NSC, we focused our analysis on differences in NSC concentration between trees that died from drought and controls that did not die, measured at the same point in time for both groups, although we also considered differences over time for trees that died (Supplementary Discussion). Reductions in NSC at mortality were common among species, but not universal, and no common NSC threshold for mortality was identified. For 48% of cases and 38% of species with available data, NSCs were significantly lower at mortality in dying trees compared with surviving or control trees (for observational and experimental studies, respectively) in at least one tissue ($P<0.05$, ANOVA; Fig. 1b–d). Among all species, mean NSCs at mortality for leaves, aboveground woody tissues (bole, branch, stem, or twig) and roots were 13, 17 and 35% lower in dying trees than control measurements, respectively.

For boreal and temperate angiosperms, lower NSCs at mortality relative to control trees were observed in 56% of cases and 63% of the species for at least one tissue, and NSC reductions exceeded 50% in approximately 33% of these cases and 38% of these species (Fig. 1b). Higher NSCs at mortality relative to controls were common for tropical angiosperm seedlings[30], more than 100% higher in some cases, and reduced NSCs were not observed in this group, suggesting different physiological responses to severe drought in non-tropical and tropical tree species (Fig. 1b,c). In a similar seedling study with the same tropical species, however, lower pre-drought NSCs were consistently correlated with a shorter time to mortality, although NSCs did not decline during drought[14]. Lower NSCs at mortality relative to controls were most common in root tissues[31] and typically resulted from lower starch concentrations, consistent with a starch to sugar conversion to meet metabolic and osmoregulatory demands during drought stress[6] (Supplementary Fig. 1). Notably, only a few cases exhibited the hypothesized time series trend in NSCs of initial small increase and then a more pronounced decrease over time[27] (Supplementary Figs. 2–4).

Reductions in NSCs at mortality were more prevalent for gymnosperms than angiosperms (Fig. 1; Supplementary Figs. 2–4). Among gymnosperms, 83% of cases and 67% of species had lower NSC at mortality relative to controls for at least one tissue (Fig. 1d). This occurred in at least one tissue for all four species of the Pinaceae, but not for the two species in the Cupressaceae, which is consistent with divergent evolutionary pathways for stomatal control between these families[32]. Relative reductions in NSCs were also generally greater in gymnosperms than angiosperms, for example *Pinus sylvestris* had NSC reductions of >80% in some tissues prior to mortality (Fig. 1d).

Functional traits related to xylem embolism resistance and stomatal control have been suggested as useful predictors of the physiological causes of drought-induced mortality[3,24,29,33]. For all species, the deviation of NSCs in trees at mortality from their controls was not significantly associated with wood density or SLA ($P>0.05$, linear regression), regardless of whether the relationships were assessed for angiosperms, gymnosperms, or all species together. For gymnosperms, reduced NSCs at mortality in aboveground woody tissues (bole, branch, stem, or twig) were associated with lower resistance to xylem embolism (that is, higher Ψ_{50} and Ψ_e; $r^2=0.88$ and 0.91, respectively, $P<0.001$, linear regression; Fig. 2), indicating that hydraulic features in gymnosperms associated with drought resistance were related to NSC dynamics during lethal drought. Normalized NSCs in other tissues were positively correlated with embolism resistance at mortality (leaf NSC with Ψ_{50}, root NSC with Ψ_e; $P<0.05$, linear regression), and normalized NSCs in aboveground woody tissue and roots at mortality were also positively correlated with the Ψ_{50} hydraulic safety margin for gymnosperms ($P<0.001$, linear regression; Supplementary Fig. 5), but these relationships were strongly influenced by one species, *Callitris rhomboidea* (Supplementary Methods). Variation in PLC at mortality was not related to any functional traits assessed ($P>0.05$, linear regression).

Discussion

We found that tree mortality from drought was always associated with substantial loss of hydraulic function and that lower NSCs at mortality were common but not universal (Fig. 1). Our findings for PLC at mortality (Fig. 1a) are close to modelling and theoretical predictions of a stem PLC mortality threshold near or above 60%[7,10,34–36]. In all cases, we found that PLC at mortality was at least 60%, but values were much higher in a number of cases. The studies in our synthesis were not designed to quantify lethal PLC thresholds, which deserve future investigation to determine the duration and intensity of drought required to trigger mortality and the mechanisms underlying such a threshold. The physiological effects of a particular level of PLC are likely to vary among species, mediated by traits such as the capacity to refill embolism and replace conducting area via new growth[6,37]. Nonetheless, a sustained stem PLC at or above 60% provides a generally supported starting point for modelling vegetation response across spatial scales, a point beyond which the probability of mortality increases[7,10,35,36].

For the cases where both NSC and PLC data were available at mortality, all trees died with high PLC (100% of cases), but only 62% of cases also had low NSCs at mortality relative to controls (Fig. 3).

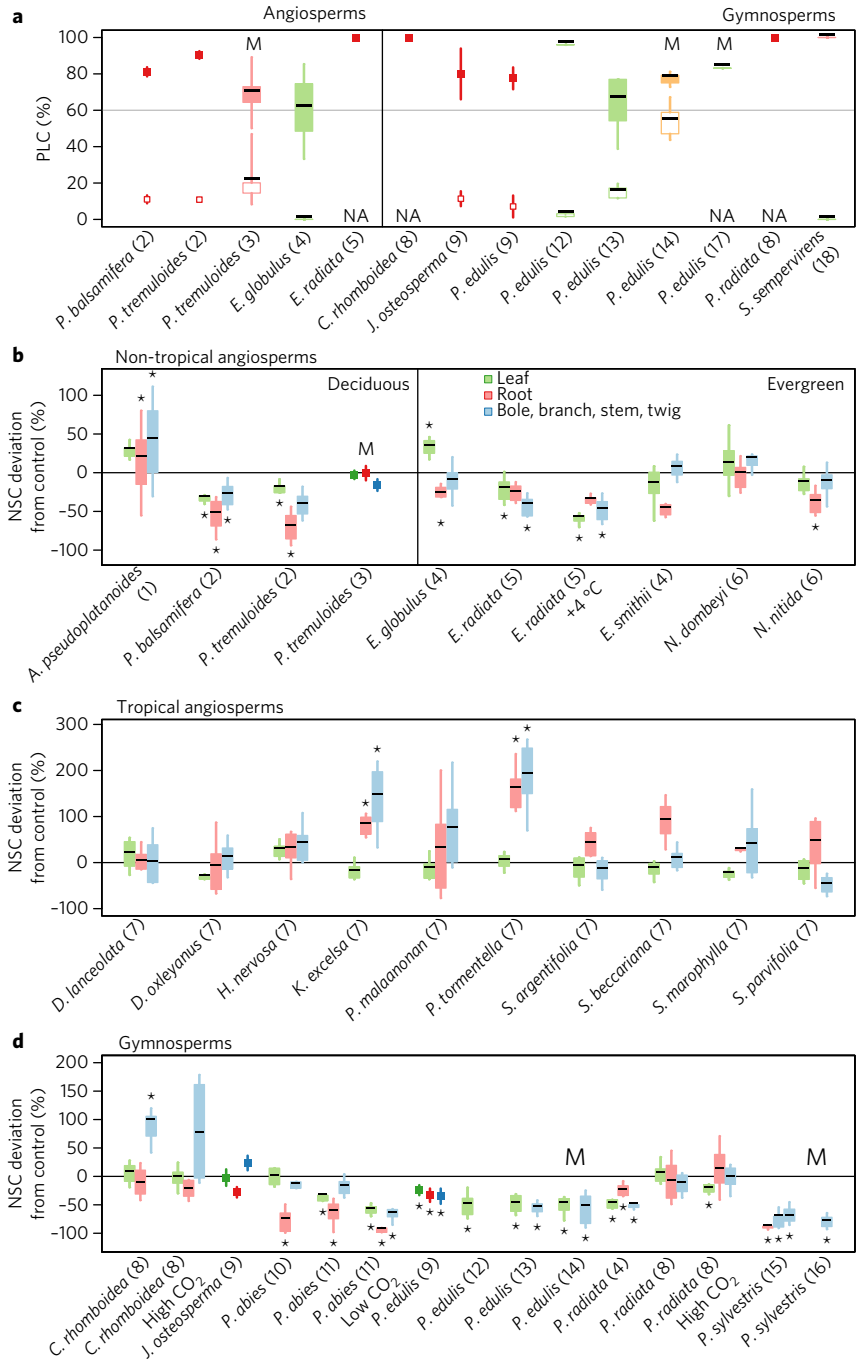

Fig. 1 | Physiological responses at, or prior to, mortality from drought for multiple tree species. a, Per cent loss of conductivity (PLC) for ambient moisture, control, or surviving trees and concurrently at mortality from drought is shown for both angiosperm and gymnosperm species. PLC was either measured directly (red) for control (open symbols) and dying (filled symbols) trees, or estimated from either water potential with a hydraulic vulnerability curve (green) for control (open) and dying (filled) trees, or modelled from hydraulic conductance (orange) for control (open) and dying (filled) trees. NA, control PLC data were not available. In all panels for cases where individual data were available, boxes indicate the 25% and 75% quartiles, whiskers indicate the extent of data, and black bars indicate the mean. For cases where only means and a measure of variability were available, means are indicated with squares and error bars are one standard error. For each case in **a** where control and dying tree data were available, PLC was significantly higher at mortality than for controls concurrently ($P < 0.05$, Student's t-test). A potential threshold for hydraulic failure is indicated by a line at 60%. **b**–**d**, Non-structural carbohydrate (NSC) concentration at mortality, normalized as the per cent deviation from concurrent measurements of ambient, control, or unaffected trees in each study for each plant tissue, is shown for deciduous and evergreen non-tropical angiosperm (**b**), evergreen tropical angiosperm (**c**) and evergreen gymnosperm (**d**) species. Significant differences for each tree tissue between drought trees at mortality (black bar or square) and ambient, control, or surviving trees (0% line) are indicated with an asterisk ($P < 0.05$, ANOVA). Note that the absolute values in NSC concentration used in statistical analysis varied for each tissue in each case, such that distances between the mean and zero in **b**–**d** are not a consistent indicator of statistical significance among cases or for tissues within a case. M, data from a study on mature trees; all other data are from studies of seedlings, saplings and small trees (Supplementary Tables 1 and 2). Numbers after species names in all panels designate original studies (Supplementary Table 1). Sample size for all data analysed for Fig. 1 are shown in Supplementary Table 4.

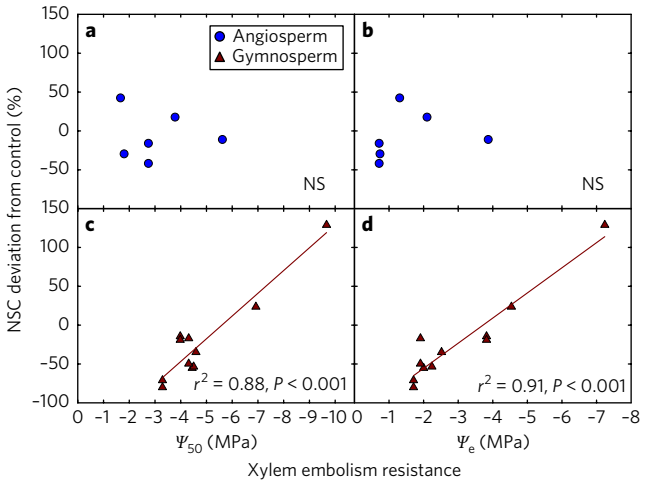

Fig. 2 | Relationship between the tree hydraulic traits related to xylem embolism resistance and normalized NSC in aboveground woody tissue at, or prior to, mortality from drought. a–d, Expressed as a deviation from concurrent measurements of surviving control trees, for angiosperm (blue circles; **a**,**b**) and gymnosperm (red triangles; **c**,**d**) species. Tree hydraulic traits related to embolism resistance are the water potential at 50% loss of hydraulic conductivity (Ψ_{50}; **a**,**c**) and point of xylem embolism entry (Ψ_e; **b**,**d**). Xylem embolism resistance increases to the right. NSC data shown are means for aboveground woody tissue (bole, branch, stem, or twig), normalized as a per cent of ambient moisture, control, or surviving trees in each case. Significant linear regressions were found for gymnosperms (**c**,**d**) but not angiosperms (**a**,**b**). Values for *C. rhomboidea* (upper right in **c**,**d**) were identified as potential outliers, but both relationships remain statistically significant ($P < 0.01$, linear regression) for the remaining data with the removal of these points (Supplementary Methods). NS, not significant.

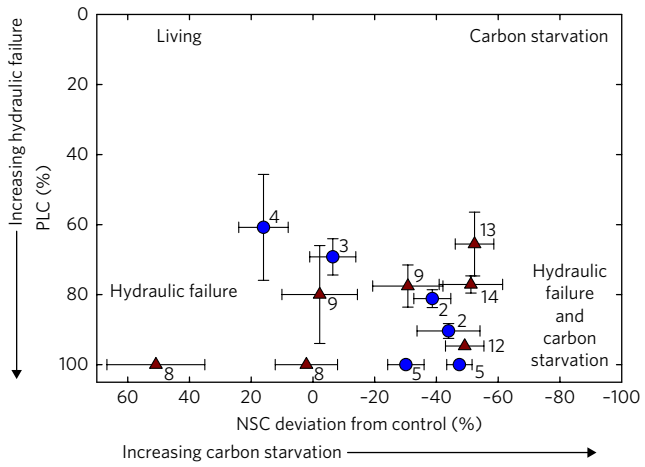

Fig. 3 | Physiological responses associated with hydraulic failure and carbon starvation, as defined by PLC and NSC deviation from control in 13 cases (study × species combinations) for which both data were available. Among these cases, trees either died with high PLC and low NSCs (8/13 cases), or with only high PLC (5/13 cases). NSC data are means for all sampled tissues available for each case and normalized as a per cent of difference from concurrent measurements of control trees. PLC data are those shown in Fig. 1a. NSC and PLC at mortality for angiosperm (blue circles) and gymnosperm (red triangles) species are shown relative to hypothesized drought mortality mechanisms. Numbers near points designate original studies (Supplementary Table 1). Error bars are one standard error.

This suggests that trees died from either hydraulic failure alone, or hydraulic failure in combination with reduced NSCs. This finding should help lay to rest the misconception of a dichotomy between hydraulic failure and carbon starvation, which are often mistakenly thought to represent mutually exclusive mechanisms[15]. Clearly, our results underscore the importance of maintaining a functional plant hydraulic system for survival, while suggesting a relationship between hydraulic failure and carbon starvation mechanisms in this process. The majority of studies included in our analysis were not designed to distinguish the drivers of mortality from the non-causative symptoms of dying. Thus, it is not possible with our data to determine conclusively if changes in either NSC or PLC facilitated death or were the result of the mortality process (Supplementary Discussion). Results from studies in which light and CO_2 concentration were manipulated to regulate carbon fixation do suggest a role for NSC as a survival mechanism against mortality via hydraulic failure during drought, even when NSC does not decline during drought or is not reduced below control values[12–16] (Supplementary Discussion).

Given the diversity of NSC responses found at mortality, there is an obvious need to develop frameworks for the sensitivity of plant metabolism to changes in NSC levels, including the potential for lethal thresholds[22,38]. Specific NSC thresholds for survival or mortality during drought are not well-resolved in our data, nor yet in the literature. Such survival thresholds are likely to vary with factors including tree species, ontogeny, tree tissue, canopy position, seasonality, environmental conditions and interactions with other organisms, but empirical investigation of these thresholds is needed[22,39,40]. Determination of these thresholds is hampered by an incomplete understanding of the role of NSC storage in plant function and its regulatory mechanism[22,38]. However, significantly lower NSCs at mortality were relatively common for a variety of species in our analysis, such that reduced NSCs can no longer be considered a rare or atypical response during tree death.

Our finding that reduced NSCs at mortality were more common for gymnosperms than for angiosperms (Fig. 1; Supplementary Figs. 2–4) is consistent with the wider hydraulic safety margins of gymnosperms relative to angiosperms[23,41]. For gymnosperms, our functional trait analysis revealed that species with greater xylem embolism resistance had higher NSC at mortality in boles, branches, stems, or twigs than surviving controls, indicating that species' hydraulic traits can affect carbon balance during lethal drought (Fig. 2). As embolism resistance is often associated with an ability to keep stomata open at lower water potentials[29,42], our results suggest that tree species that can maintain stomatal conductance and photosynthesis at higher xylem tension during drought are less likely to have reduced NSC at mortality[28]. These resistant tree species would be more likely to die from hydraulic failure alone without reduced NSC—consistent with hypotheses that stomatal regulation and hydraulic transport strategies influence the contribution of carbon starvation and hydraulic failure to mortality mechanisms among species[3,24,29]. Caution, however, should be used in assuming stomatal regulation is highly coupled with water potential regulation and hydraulic strategy[43]. Importantly, we did not find a relationship between NSC reduction and embolism resistance for angiosperms, nor did any other trait predict mortality physiology in these species.

Our synthesis of data from multiple studies on the physiology of drought-induced tree mortality exposes several key knowledge gaps in the field. Our dataset of only 26 species under-represents the enormous diversity of tree species found in forests globally, particularly so for tropical forests, where drought-induced mortality can have substantial implications for the global carbon cycle[9]. *Pinus* was relatively over-represented in this synthesis (nine cases from three species), although it is widely distributed and has been widely affected by forest die-off on multiple continents[1]. Also, our dataset is dominated by data from seedlings and saplings, often from studies conducted with potted plants, which may be predisposed

to die quickly from hydraulic failure due to limited rooting volume and lack of access to deeper soil water pools (Supplementary Tables 1 and 2). Data at mortality for more than one life stage were available for only three species (Fig. 1), and the consistency of NSC and PLC responses at mortality across a gradient of size and ontogeny varied in these species. Clearly, more research is needed on the physiology of mortality in large trees in the field, and the effect of size and ontogeny on the mortality process. Nonetheless, our overall observation that hydraulic failure was universal, and NSC reduction was not, does not change if we only consider data for each life stage separately. In all cases for which PLC data were available, mean PLC was 60% or greater at mortality, irrespective of life stage (Fig. 1a). Our finding that normalized NSC at mortality varied among cases and species also holds when seedlings, saplings and trees are considered separately (Fig. 1b–d; Supplementary Table 1).

Determining whether forests will continue to act as a global carbon sink or transition to a carbon source is a critical uncertainty for the carbon cycle, with large ramifications for society and climate policy[8,9,44]. Such a shift largely depends on tree mortality responses that could be anticipated by resolving the relative roles of hydraulic and carbohydrate mechanisms in causing tree death[7,10,45]. We found that hydraulic failure was ubiquitous among the studies we compared and that PLC at mortality in all cases with such data was at least 60%. These results affirm that simulating hydraulic function should be a first priority for development of mechanistic tree mortality algorithms in climate–vegetation models to improve projections of the future terrestrial carbon budget. Hydraulic models that capture drought damage on tree and landscape scales are rapidly developing[7,10,35,36,45–47] and substantial improvement in vegetation model projections may be possible with simulation of hydraulic-driven mortality, whether tree carbohydrate status is represented or not. Reduced NSC in tree species dying from drought was common in gymnosperms, but not angiosperms, suggesting an influence of NSC on hydraulic deterioration in some trees that requires further investigation. Yet, the diversity of NSC responses among only 26 species and the design limitations of past studies in determining causality demonstrate that we need to further assess the influence of carbon metabolism and storage on mortality[38]. Ultimately, an improved representation of the physiology of drought-induced tree mortality that includes both water and carbon relations will be crucial for forecasting the fate of forests in a changing climate.

Methods

Data synthesis. We used literature search and extensive discussion with colleagues to identify data from 19 experimental and observational studies on 26 species, for a total of 34 cases (study and species combinations). Literature search terms included 'non-structural carbohydrates', 'water potential', 'tree mortality' and 'drought'. Our synthesis was not limited to an objective literature search, as we sought to include all published data that fit our criteria for inclusion. Criteria for inclusion were that studies included data on: (1) tree mortality from drought; (2) NSC concentrations of at least one tissue, and/or PLC of aboveground woody tissue, either measured directly, or estimated from plant water potential (Ψ_p) measured at mortality, or modelled from hydraulic conductance[48,49] (Supplementary Methods); and (3) that data were either concurrently collected for trees that died (either at or near mortality) and from trees that either survived the drought or were in a paired control treatment; and/or available prior to drought or pre-treatment from the same trees that later died. We obtained data from each study directly from contributors. Details on the specific studies synthesized can be found in Supplementary Table 1. Determination of the point of mortality in dying trees was defined in each original study, as detailed in Supplementary Table 3, and we relied on data contributors to provide the appropriate data for at- (or near-) mortality assessments.

NSC measurements are methodologically challenging and comparisons of absolute concentrations can be problematic across studies due to issues of standards, NSC technique and lab protocol disagreement[50,51]. However, relative differences (treatment versus control and changes over time assessed with the same technique in the same laboratory) provide robust estimates of NSC dynamics within studies[50,51]. We limited all statistical analyses of absolute NSC data to within each case (detailed below) and we only present relative differences in NSC in figures. For studies where data were concurrently available for trees that died and control or surviving trees, we calculated a normalized NSC deviation from the difference between values at or near mortality and those for control or surviving trees divided by the control or surviving tree value. For studies where data were available prior to the drought for the same trees that later died (or seedlings in the same treatment harvested at measurement), normalized values were also calculated as the difference between values at or near mortality and initial pre-treatment or pre-drought values divided by the initial or pre-drought values. In both cases, normalized values were expressed as a per cent. For comparison of time series trends in NSC, we also calculated normalized, proportional NSCs in trees that died by scaling values relative to the maximum value in each time series, which was defined as a normalized value of 1. When possible, normalizations were calculated for individual trees, and specifically for each tissue sampled. For studies 3 and 9 (Supplementary Table 1), only means and standard errors for species and tissues were available, so normalized values were calculated from these metrics.

Note that all types of data were not available for all cases in our synthesis. Among the 34 cases in our dataset, PLC measured at mortality was available for nine cases (eight species), PLC was estimated in five cases (two species), NSC deviation from control/surviving trees at mortality was available for 31 cases (24 species) and per cent change in NSC was available for 28 cases (22 species). Sample sizes for PLC and NSC data are available in Supplementary Tables 4 and 5. Because PLC values are already normalized to the maximum conductivity per sample, no further normalization was conducted with these data. We also acknowledge that direct measurements of PLC and generation of hydraulic vulnerability curves can be challenging, and that method artefacts can affect results[52,53]. Although the majority of hydraulic data we report were collected following recommended practices (Supplementary Methods, Supplementary Table 5), we cannot rule out the possibility of such artefacts influencing our data.

To compare physiological mortality indicators with tree species traits, we obtained trait data for the species in this synthesis from a variety of sources. We investigated the relationships between physiology at mortality and traits related to drought tolerance that are easily measured and widely available, such as wood density and SLA. We also included hydraulic traits more directly related to drought tolerance that were measured with more-challenging hydraulic vulnerability curve methods. Wood density data for most species were obtained from the Global Wood Density Database[54,55] available through the Dryad digital repository (www.datadryad.org). We obtained SLA data from the TRY database (www.try-db.org)[56–58] for nearly all non-tropical species. We calculated species means for SLA from all data available for each species of interest for our analysis. Data for *Acer pseudoplatanus* were available from the mortality study population[16]. For *Callitris rhomboidea* and *Eucalyptus smithii*, SLA data were not available. Additional sources of wood density data are detailed in Supplementary Methods. Hydraulic trait data for the stem water potential at 50 PLC (Ψ_{50}) and hydraulic safety margin (Ψ_{50} − minimum Ψ)[23,26] were obtained from multiple sources (Supplementary Table 5, Supplementary Methods). Data for the embolism entry point (Ψ_e) were not available in the literature, so we calculated Ψ_e from the relevant hydraulic vulnerability curve for each case by applying a Weibull fit to the data and determining the x intercept of the line tangent to Ψ_{50} (Supplementary Table 5)[26,59]. Hydraulic trait data were unavailable for *Eucalyptus radiata*, *E. smithii* and *Nothofagus nitida*. No trait data were available for the tropical angiosperm species from study 7 (Supplementary Table 1) for any of the traits we assessed[30].

Statistical analyses. We used MATLAB R2012a (Mathworks, Natick, MA, USA) for all statistical analyses, with $\alpha = 0.05$. All NSC and PLC comparisons were performed using ANOVA or Student's t-test individually for each case, between dead (or dying) and control/surviving trees or between post-drought dead and corresponding pre-drought values, with tissue as a factor for analysis of NSC. As our NSC normalization could affect tissue comparisons within the same case, these analyses were performed on non-normalized NSC data to maintain the correct ratio among tissues, a conservative approach. Our within-individual case analysis on relative differences in non-normalized NSC does not bear the risk of error introduced by different NSC techniques or labs, or uncertainty in standards for determining absolute NSC, and furthermore the inferences are based on large effect sizes compared with possible measurement error[50,51]. In experimental cases that included temperature or CO_2 concentration treatments in addition to drought, we included these factors in ANOVA tests to determine if PLC and NSC should be pooled or split among levels of these factors (Supplementary Table 1). For NSC, these analyses also included tissue as a factor. Functional trait relationships with normalized NSC data at mortality were analysed with linear regression. Cook's distance was calculated for all points in significant linear regressions and a value greater than three times the mean of the Cook's distance was used to identify outliers for exclusion.

Data availability. The majority of datasets generated and analysed during the current study are available from the corresponding author on reasonable request. Trait data obtained for the current study from the TRY database were used under licence and, as restrictions apply to the availability of these data, these are not available from the corresponding author, but can be requested from the TRY database (www.try-db.org).

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Competing interests

The authors declare no competing financial interests.

Additional information

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