Mechanisms of woody-plant mortality under rising drought, CO_2 and vapour pressure deficit

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Abstract | Drought-associated woody-plant mortality has been increasing in most regions with multi-decadal records and is projected to increase in the future, impacting terrestrial climate forcing, biodiversity and resource availability. The mechanisms underlying such mortality, however, are debated, owing to complex interactions between the drivers and the processes. In this Review, we synthesize knowledge of drought-related tree mortality under a warming and drying atmosphere with rising atmospheric CO_2. Drought-associated mortality results from water and carbon depletion and declines in their fluxes relative to demand by living tissues. These pools and fluxes are interdependent and underlay plant defences against biotic agents. Death via failure to maintain a positive water balance is particularly dependent on soil-to-root conductance, capacitance, vulnerability to hydraulic failure, cuticular water losses and dehydration tolerance, all of which could be exacerbated by reduced carbon supply rates to support cellular survival or the carbon starvation process. The depletion of plant water and carbon pools is accelerated under rising vapour pressure deficit, but increasing CO_2 can mitigate these impacts. Advancing knowledge and reducing predictive uncertainties requires the integration of carbon, water and defensive processes, and the use of a range of experimental and modelling approaches.

Mortality
The irreversible cessation of metabolism and the associated inability to regenerate.

Die-off
Widespread and rapid mortality of a species or community.

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Woody-plant mortality results in the irreversible cessation of metabolism and the resultant inability to regenerate. Since widespread observations began in the 1960s, there has been evidence of increasing background tree mortality in many regions of the world[1–6] (FIG. 1a), including regional-scale die-off events[7–15] (widespread, rapid tree loss). Large mortality events have been recorded in dry tropical forests[15], tropical rainforests[16–18], temperate rainforests[19,20], semi-arid woodland and savannahs[8,13,21], boreal forests[22–24] and temperate deciduous to evergreen forests[12,25]. Such mortality is insidious in the case of the slowly but steadily increasing background mortality and dramatic in the case of die-off events.

These contemporary increases in background tree mortality and extreme regional die-off events are associated with atmospheric warming and a corresponding increase in vapour pressure deficit (VPD) and evapotranspiration[26–29] (FIG. 1b). As a result of greater water loss from foliage and soil surfaces, historically, non-lethal soil droughts have become lethal[29,30]. With anthropogenic forcing anticipated to further increase warming, as well as the frequency and duration of heatwaves and soil drought[10,30–33] (FIG. 1c), tree mortality is also expected to increase in the future[34,35]. However, rising CO_2 (FIG. 1b) can mitigate the negative impacts of increased VPD through higher carbon uptake and reduced water loss, affording potential water savings[36,37]. Contemporary observations suggest that rising VPD could be increasingly offsetting CO_2 benefits[38–40], and, so, the net balance might be in favour of either greater survival or mortality[37,41–44].

Background mortality
Mortality rates in the absence of disturbances.

Droughts
Periods of anomalously low precipitation.

Hydraulic failure
The accumulation of emboli within the sapwood past a threshold after which water transport is irrecoverable.

Threshold
The magnitude or intensity that must be exceeded to cause a reaction or change.

Mechanistic understanding of such drought-related and heat-related mortality is limited. Hydraulic failure, the accumulation of sapwood emboli past a threshold after which water transport is irrecoverable, and carbon starvation, the process by which a limited supply of carbohydrate impairs maintenance of carbon-dependent metabolic, defence or hydraulic functions, have both been proposed as key processes[45–47]. These processes are both challenged by a lack of clear definitions and hypotheses and the large range of experimental conditions under which they have been studied, leading to a wide range of results regarding their occurrence[48]. Though recent models have incorporated these potential processes of mortality[49–54], their interdependent contributions to drought-induced mortality of woody plants remains uncertain.

Given that tree mortality leads to substantial changes in the structure and function of ecosystems, understanding drought-related mortality is fundamental to basic biology, ecosystem management and climate-feedback predictions[55,56]. Changes in ecosystem structure and function due to mortality lead to large ecohydrologic shifts, with abrupt and potentially sustained changes in streamflow[57], as well as downstream water quality, quantity and timing[58,59]. Long-term shifts in forest demographics might also result from shifts in tree mortality rates[60], with corresponding limitations to net terrestrial carbon storage[11]; a doubling of mortality halves forest carbon storage over 50 years if net primary production doesn't equally increase. Resulting impacts on biodiversity might be large and surprisingly unpredictable at regional and global scales, and likely depend on disturbance type, biome and species, among other factors[61,62]. Moreover, the economic impacts of forest loss, particularly in regions where wood production is vital to societal well-being, could be substantial[63].

In this Review, we synthesize understanding of woody-plant mortality under rising VPD and CO_2. We begin with a discussion of contemporary, observed drought-associated tree mortality. We follow with consideration of three general, interdependent mechanisms of tree mortality — water relations, carbon relations and defensive failure — and propose an integrative, predictive framework for mortality under a changing global climate. We subsequently examine how such processes might interact to promote vulnerability and influence future projections. We end with recommendations for future research. Throughout the Review, all mechanisms are treated as part of the mortality process: that is, from failure of root water uptake through to hydraulic failure, carbon starvation and irreversible cell dehydration. We consider a plant in the 'dying' phase to have passed a point of no return or a threshold[64–70], beyond which mortality of the organ (branch dieback)[71] or the entire plant is certain. Processes that occur before the dying phase can be critical in promoting or delaying mortality, whereas those that occur thereafter can be considered consequences, not causes.

Background mortality and regional die-off

The regionally distributed trends of increasing background tree mortality[1–6] (FIG. 1a) — as observed throughout western and boreal North America[1,2], the Amazon basin[6] and Europe[11] — indicate that a common driver underlies changes in woody-plant mortality. The degree of increase in mortality rates varies with region, including a non-significant change in the Congo, the underlying climatic and physiological processes of which remain relatively unknown[72]. Elsewhere, these background rate increases could reflect increasing VPD impacts[26,29,73], and, under wetter conditions, might also reflect elevated productivity and turnover[74,75]. However, there is little evidence for increasing competition-induced mortality[18,37]. Additionally, when water is ample, rising CO_2 and warming could provide conditions for structural overshoot, where forests rapidly gain biomass and leaf area (at the individual plant and the stand scales) to levels not hydraulically sustainable during the eventual hotter droughts[76,77], which could, likewise, promote increasing mortality.

Regional die-off events are now also being observed across both warm and dry and wet and cool biomes[7–25],

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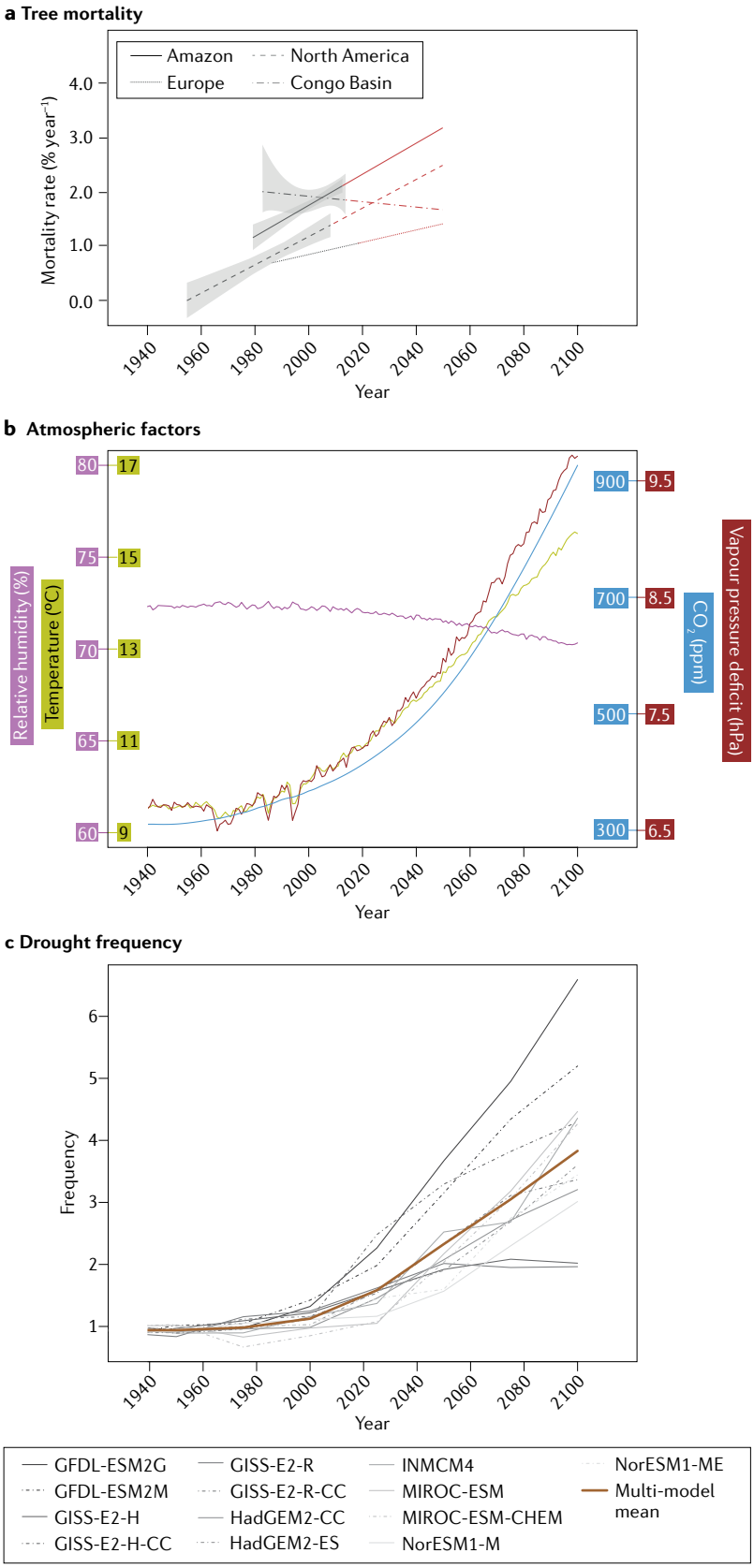


Fig. 1 | **Changing tree mortality and climate variables.** **a** | Observed (grey) and linearly projected (red) tree mortality rates for North America[1,2], the Amazon[6], Europe[11] and the Congo Basin[6]. Grey shading represents 95% confidence intervals. **b** | Average simulated CO_2, surface temperature, relative humidity and vapour pressure deficit for grid points only over global vegetated land. Projections are based on RCP8.5. **c** | Simulated drought frequencies relative to the historical mean (1850–1999); values <1 indicate lower frequency compared with historical and those >1 indicate greater frequency compared with historical. Drought frequency is based on extreme plant-available soil water as <2 percentile of the 1850–1999 period. Projections follow RCP8.5 using 13 models from CMIP5. See REF.[34] for details of calculations. Increasing atmospheric drivers of mortality are consistent with increasing mortality rates, except in the Congo.

even following decades of productive growth[78], indicating that no biome is invulnerable. Die-off events are regional in scale, can kill one or more species and occur rapidly[14]. This global distribution of die-off events is associated with global increases in temperature and VPD[14]. Droughts eventually occur everywhere, but now with warmer temperature and higher VPD than historically[28].

Drivers and mechanisms of mortality

Understanding and predicting mortality under future climate requires a framework that provides unambiguous definitions, generates testable hypotheses and identifies uncertainties. In the case of drought-associated woody-plant mortality, this framework is expected to also identify pools and fluxes of critical resources and their potential lethal thresholds, and to be relevant across different biomes and environmental conditions. Previous frameworks have advanced knowledge of drought-related mortality[45–47,79]. However, existing frameworks grapple with the complexity of the interdependent processes that occur while trees are dying[45–47,80], including hydraulic failure, carbon starvation and attack by biotic agents[81,82] (FIG. 2). Overall, there remains a lack of consensus on the appropriate terminology, mechanisms and, ultimately, a clear set of hypotheses.

Owing to consistent evidence of deteriorating water status during death[45–47,80] and widespread prevalence of hydraulic dysfunction in drought-associated mortality[13,14,22,23], the mechanisms underlying woody-plant mortality originate from a whole-plant water-relations backbone. Examining mortality can start from a basic end point in the process: the rupture of cell membranes owing to water content falling below a critical threshold, preventing plant recovery[65,68] (FIGS 2,3). Crossing this critical threshold depends, in part, on the absolute water content below which the cells cannot survive, the osmoregulation potential of the cells and the capacity to provide substrates and energy for continuous membrane maintenance and osmolyte production[68]. Such cellular death must manifest across tissues at the whole-plant scale and eventually impact all meristematic cells needed for growth and reproduction before organismal mortality has occurred. Thus, while plant water relations are the keystone process for drought-associated mortality, hydraulic function and associated failure might also depend on starch and sugar availability, thus, water and carbon supply, and their respective cellular reservoir size, might be critical for survival[83].

Carbon starvation
The process whereby a limited carbohydrate supply rate impairs maintenance of carbon-dependent metabolic, defence or hydraulic functions.

Process
A series of mechanisms that leads to an end point.

Mechanisms
Systems of parts working together within a process; pieces of the machinery.

Dying
Committed to death; beyond the point of no return; to have passed a threshold beyond which mortality is certain.

Biotic agents
Living organisms — especially fungi, bacteria and insects — that interdependently impact the water and carbon economies of plants.

Meristematic cells
Undifferentiated cells capable of division and formation into new tissues.

Cytorrhysis
Irreparable damage to cell walls after cellular collapse from the loss of internal positive pressure.

Likewise, biotic attack is also highly likely to interact with physiological declines given defensive dependency on water and carbon relations[47,81]. The mortality process is now discussed in terms of whole-plant water relations, water–carbon dependencies and water–carbon–biotic dependencies.

Whole-plant water relations preceding mortality. Failure of whole-plant water relations and subsequent mortality occurs through a series of sequential and interdependent mechanisms (FIG. 3). First, a severe decline in root water uptake, whole-plant hydraulic conductance and associated stomatal closure[84,85] occurs, causing the water and carbon pools to become finite and exhaustible. The depletion of water pools is followed by continued loss of water through evaporation from plant surfaces and, subsequently, increasing occurrence of sapwood embolism as water pools are depleted[86,87]. Ultimately, hydraulic failure occurs and subsequent downstream mortality ensues as water pools deplete below the threshold for irreversible cell death[46,64–68], known as cell wall rupture or cytorrhysis[88,89]. In this view, hydraulic failure is a mechanism within the larger process of mortality, rather than a sole cause in itself. Hydraulic failure in woody plants, strictly defined, is the accumulation of emboli within conduits causing a decline in conductance and an increase in tension of the remaining conduits, creating a feedback loop of runaway cavitation and, subsequently, insufficient distal water supply relative to water loss. By definition, hydraulic failure includes a threshold resulting from the whole-plant processes of water flow, that is, a threshold beyond which recovery of flow is impossible due to emboli accumulation within conduits. Critically, the importance of fluxes and pools of water to survival increases as drought progresses[83] (FIG. 3). The sequence of events that promotes a constraint upon survival through failure of whole-plant water relations and associated hydraulic failure is now discussed.

Drying soil demands a steep increase in the amount of tension, or water potential gradient, that plants must withstand to extract soil water[90]. The soil–root interface becomes a primary limitation on whole-plant

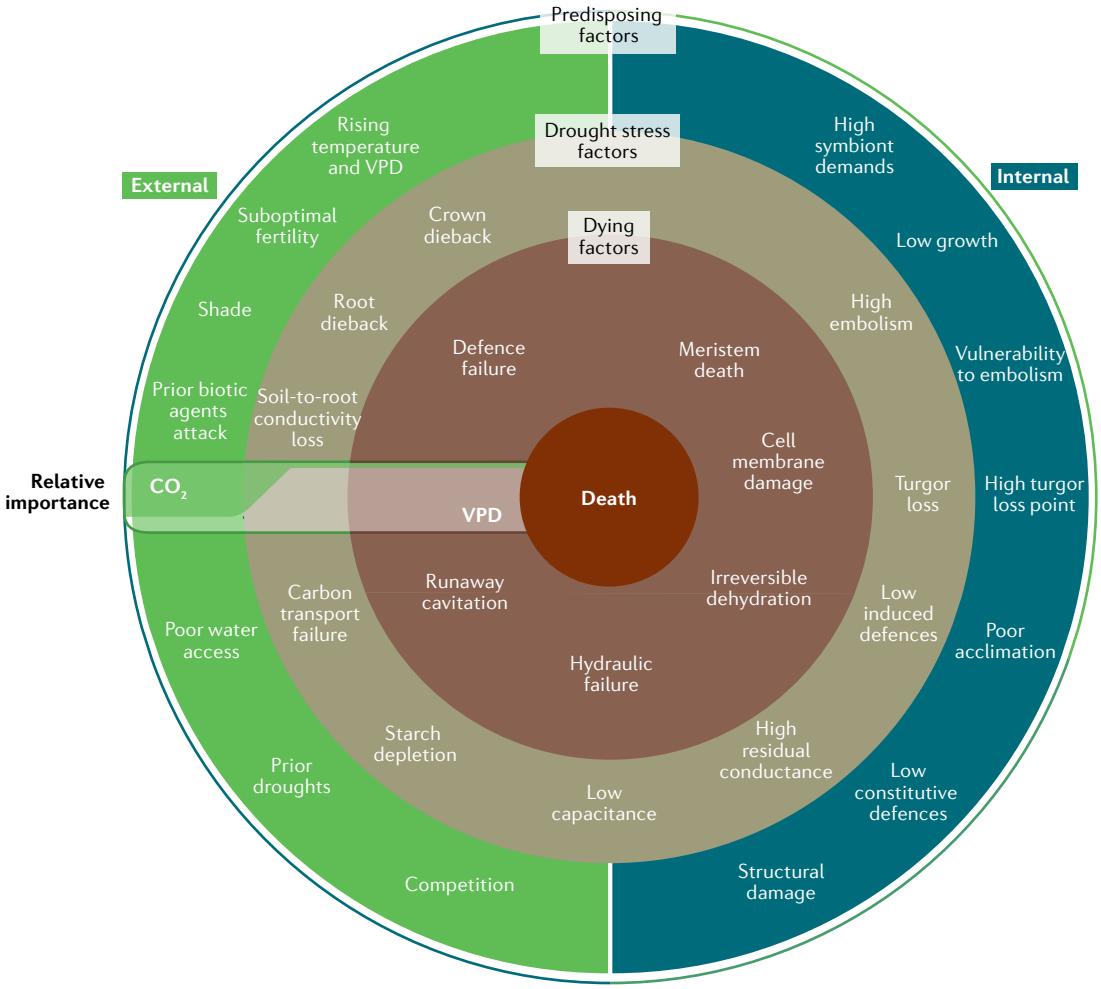


Fig. 2 | **The interconnected mortality process.** A hypothetical representation of the mortality processes from predisposing factors to death. Predisposing factors are linked to mortality via the mechanisms in the second innermost ring, which subsequently cause a plant to pass a threshold beyond which mortality is inevitable. The death spiral results from the interaction of external drivers, the processes of hydraulic failure and carbon starvation, and their underlying, interdependent mechanisms. VPD, vapour pressure deficit. Figure inspired by REF.[80].

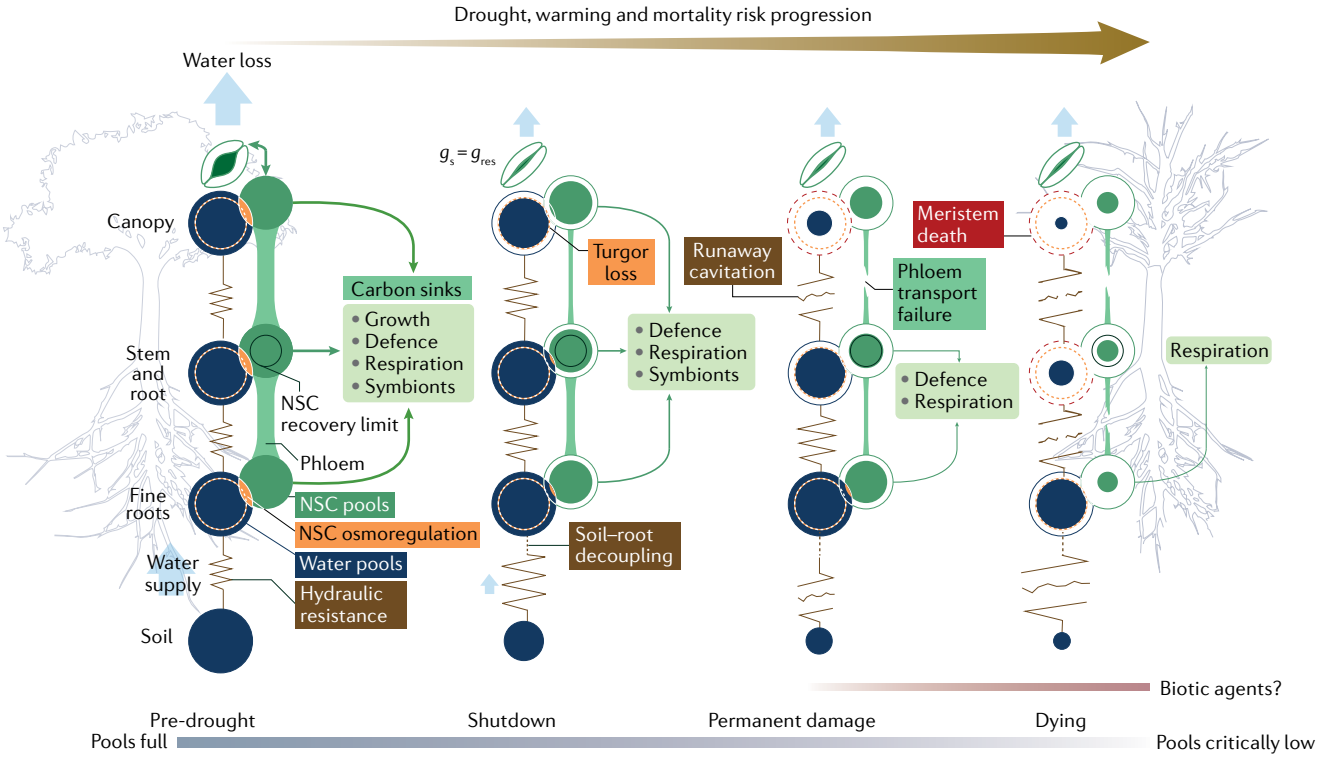


Fig. 3 | **Mechanisms that lead towards mortality.** Hypothesized mechanisms, including pools and fluxes, that influence mortality as drought progresses. Primary water and carbohydrate pools are in blue and green circles, respectively, with their fluxes as resistors and green arrows. Interactions between water and carbon pools are in orange. As drought progresses, stomatal and belowground conductance decline to near zero. g_{res} (residual conductance from foliage and bark post-stomatal closure) then dominates water loss and plant survival depends on the finite, stored carbon and water pools. Ultimately, dehydration and depletion of these pools promotes sustained negative turgor (dashed orange circles), followed by meristem death (dashed red circles) from the cellular water content falling below a threshold for cellular rupture. The black circle within the stem and root carbohydrate pools indicates the point at which there is insufficient carbohydrates to recover via regrowth. g_s, stomatal conductance; NSC, non-structural carbohydrate.

conductance under these conditions and can constitute >95% of whole-plant resistance[91,92]. Complete hydraulic disconnection between the soil and roots has been demonstrated in mature woody plants, including during drought-associated mortality[93,94], although the frequency of such disconnection during drought is unknown. Under these conditions, stomatal conductance declines to near zero[91], reducing water loss and photosynthesis[95]. Near-zero belowground (soil–root) conductance and stomatal conductance mark a transition point[56,58] when the individual plant becomes solely reliant on its limited water stores[96,97]. The size of these stores, their net depletion rate and the tissue-level and cell-level tolerances for depletion define the likelihood of mortality[98] (FIG. 3).

As hydraulic failure and cytorrhysis ensue for some tissues, crown dieback (foliage and branch loss) and root loss will advance, and, if the damage proceeds to destroy all meristematic tissues, individual mortality occurs (FIG. 3). Such cellular failure can occur at organ (dieback) or individual (mortality) levels, depending on its extent and the degree of hydraulic segmentation within the plant. Variation in cellular thresholds for survival is likely substantial and could be important in regulating where and when woody-plant mortality occurs. The threshold for cellular cytorrhysis is above 0% water content[89] and might vary with species, genotypes, phenotypes, tissue and cell types, but remains relatively unexplored in woody plants.

Evaporative losses after stomatal closure are dominated by residual vapour fluxes through leaky stomata, cuticle, bark and possibly roots[99–103] (residual conductance (g_{res})). The evaporative flux through g_{res} becomes the dominant path of whole-plant water loss during a severe drought and is exacerbated at high air temperatures[86,87]. Continued water loss (which is not replaced) results in declining water pools and increasing embolism (FIG. 3). Even under situations where the soil–root connection is maintained, if the fluxes of water to the plant are smaller than those lost through g_{res}, the stored pools of water will deplete and embolisms will develop.

The bidirectional feedbacks between the depletion of water pools and irreparable hydraulic failure is a logical and testable hypothesis for mortality progression. However, a water-only perspective ignores the potentially critical roles of carbon and defence against biotic agents during drought, both of which could promote failure of water relations if they individually fail. During short and hotter droughts, failure to maintain the critical water supply can dominate the mortality process because of the rapid rate of water loss relative to the rate of carbohydrate and defensive losses[104–107]. In longer droughts, or in cases of mortality years after drought, the role of carbon supply to critical survival processes should increase[104–107] due to the far longer residence time and slower changes in carbohydrates than in water pools[108,109].

Dieback
The partial loss of canopy or root biomass, without whole-plant mortality.

Failure of water relations
Impairment of the interacting water and carbon processes that forces declines in water supply and subsequent dehydration.

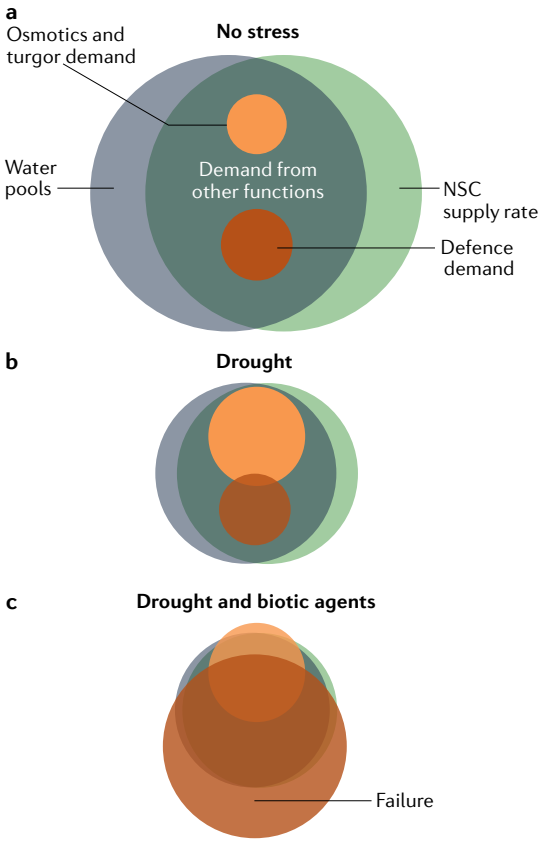


Fig. 4 | **The linkage between woody plant's defence systems and biotic attack. a** | Pre-drought, water and carbon pools and supply rates are sufficient to support the demand for defence, osmotic regulation, metabolism and other functions. **b** | During drought, the demand for osmotic regulation increases as water and carbon pools and supply rates decline. **c** | Once biotic agents attack, the demand for defence increases, potentially beyond that which can be supported by the water and carbon pools and fluxes. This viewpoint suggests that, if any component of the system fails, it can lead to the cessation of the interdependent distal processes critical to survival. NSC, non-structural carbohydrate.

The water–carbon interdependency. The process by which plants' carbon economy has a role in mortality before, during and after lethal droughts involves multiple potential mechanisms. These include those associated with decreased photosynthetic gain[45], reduced xylem and constitutive defensive compound production[110–113], initial carbohydrate storage increases and then decreases prior to death[11,47–114], a critical increasing role of carbohydrates in osmoregulation and cellular maintenance[115–122], and possible carbohydrate feedbacks upon hydraulic and defensive function[47]. In this view, carbon starvation, or the steps by which metabolic functions are impaired by limitations in the supply rate of carbohydrates, is a mechanism within the larger process of carbon limitations upon mortality. These mechanisms are described chronologically from pre-drought through the dying phases (FIGS 2,3).

There is evidence that water–carbon-related factors that occur prior to drought can promote mortality (FIG. 2). Decades-long reductions in carbon allocation to stem wood and resin ducts within dying conifer species indicate potential roles of pre-drought allocation to mortality[105–107,110–113]. Low growth preceding death can be a lingering consequence of prior climate or injuries that predispose trees to carbon constraints[79] (FIG. 2). By contrast, shifts in allocation towards greater pre-drought growth can also influence mortality owing to reduced allocation to defence at the expense of growth[123–127] or from over-allocation to aboveground biomass (in particular, leaf area at the individual and stand levels) during favourable conditions, which cannot be sustained during subsequent hotter droughts (structural overshoot)[76]. A feedback loop can be created, in which pre-drought factors can predispose plants to mortality during drought (FIG. 2). Indeed, declines in growth, hydraulic function and defence can be a function of reductions in crown leaf area owing to crown dieback or root loss from prior drought, lightning, wind damage and defoliating or root-feeding insects or pathogens[128–133]. However, declining leaf area can also promote survival by reducing water loss during drought[134,135]. The net consequence of pre-drought shifts in carbon and water pools and fluxes remains a research challenge.

Once drought has become sufficiently severe and/or prolonged such that photosynthesis has declined to near zero, the starvation process can promote mortality through water relations or defensive failure (FIG. 3). Carbon starvation is the process by which carbon-dependent metabolism, defence and possibly hydraulic maintenance are shifted owing to limited carbon supply rate relative to demand (FIG. 4). This definition is consistent with the literature on starvation across global animal and plant taxa[136–138], which characterize starvation as causing significant shifts in metabolism, which are reversible, until a threshold is passed, after which the interdependent processes required for survival are not met. Carbon starvation is considered to occur nightly in plants[139], though for the purposes of drought-associated mortality, this process becomes relevant when stomatal closure precludes photosynthesis relative to carbon demand for abnormally prolonged periods (months to years). Starvation manifests at the cellular scale but can occur widely throughout an organism. As starvation progresses, the supply rate and/or pool of carbohydrates (sugars derived from starch, lipid and hemicellulose breakdown, as well as products derived from autophagy)[140–142] could possibly decline below the threshold at which cellular to whole-tree mortality is promoted. Such thresholds can include the minimum metabolism required for survival, failure to maintain membrane stability, inability to maintain the osmotic functions for the hydraulic system or failure to maintain the defence system (FIGS 3,4).

It is the interplay between the available carbon pools, their fluxes and the demands for survival that cause carbohydrate supply rates to not meet the requirements to avoid hydraulic failure[105,143–145] or insect and/or pathogen defence failure[79,146,147] (FIGS 3,4). If an insufficient supply rate of carbon substrates to required metabolic processes[115] results from short-distance or long-distance transport constraints, for example, through source

strength impacts upon phloem loading and viscosity challenges[148–151], failure of carbon-dependent processes could be promoted. Declining resistance to xylem embolism has been associated with low carbohydrate concentrations[145]. Reduced carbon supply rates can promote osmoregulation failure, loss of protein and membrane stability, failure to scavenge free radicals and reductions in cross-membrane transport of ions and amino acids[115–122]. Simultaneously, the carbon requirements for these processes increase with drought stress[45,152–157], thus, the carbon safety margin, or the difference between carbon availability and demand, decreases with drought. Therefore, failure to maintain hydraulic integrity and cellular water content appear directly linked to the carbon economy of plants (FIG. 3), particularly under longer droughts.

The water–carbon–defence interdependency. Attacks by biotic agents such as insects or pathogens are frequently concomitant with drought-associated mortality prior to, during or shortly after the drought event[81,158–160], and are likely interdependently associated with impacts on carbon and water relations (FIGS 3,4). When a biotic attack occurs prior to drought, it can impact plants through defoliation or root loss, predisposing plants to subsequent mortality if the attack impaired their water and carbon economies (FIGS 3,4). When drought stress predisposes trees to attack, it can lead to hydraulic failure if the vascular system is infected[161–163]. Biotic agents can disrupt carbon uptake and transport through leaf loss[164] or depletion of carbohydrate reserves via direct consumption[165,166], and they can stimulate a plant's induced defence response[167,168].

The initiation of regional-scale outbreaks typically occurs during or after drought when the defensive capacity of host trees is constrained, and a critical number of vulnerable trees becomes susceptible across the landscape[169–171]. The level of stress that limits defensive function remains unknown owing to a lack of empirical evidence linking carbohydrates, hydraulics and defences in field experiments with mature trees[172]. Experiments that preclude biotic attack, however, such as caging, insecticide/fungicide and anti-aggregation pheromones, have demonstrated that some insects, fungi and other pathogens have the potential to directly move drought-weakened plants into the dying phase, while those free of biotic attack survive[47] (FIG. 3). In these cases, the roles of the plant water and carbon economies in defensive failure can be substantial.

The water and carbon economies directly impact the defensive system, such that failure of one component can lead to failure of another (FIG. 4). The relative water content and water potential of cells directly impacts turgor pressure and the substrate transport critical to defence[173,174]. Synthesis of secondary metabolites and metabolic transport costs of the defensive system depend directly on labile carbon availability for induced defences[173–176] (FIG. 4). Higher sugar concentrations are associated with reduced attack by insects and fungi, demonstrating the potential role of carbohydrates in defence, perhaps through fuelling induced responses[174,176]. Defensive compounds have a particularly high carbon concentration[177] and tend to be upregulated at the transcriptional level during drought[178,179]. Similarly, a rapid increase in defensive allocation upon attack has been associated with declining local carbohydrates[168]. Thus, defence incurs a substantial carbon cost[180] (FIG. 4), suggesting that reduced carbohydrate supply rates could render the plant susceptible to biotic attack[181,182]. For example, conifers with fewer resin ducts formed in years prior to drought are frequently more likely to die during beetle attack[111,167,183–186], consistent with a carbon constraint inducing defensive failure. When insects and pathogens reach epidemic levels, the attacks might switch from the poorly defended to the faster-growing, well-defended plants due to the higher availability of resources available to the attacking agents[127]. Thus, defensive failure can be both a cause and a consequence of impaired water and carbon economies during drought (FIG. 3).

The interdependent framework of mechanisms. This framework (FIGS 2–4) outlines the individual mechanisms within the larger processes that lead plants into the dying phase. As a plant enters a hotter drought with higher VPD, it must manage limited water resources amidst the edaphic and structural conditions that it enters the drought with (FIG. 2), including defensive dependence and hydraulic dependence on carbon allocation to sapwood, roots and foliage prior to the drought. As drought ensues and belowground and stomatal conductance declines to near zero, embolisms can accumulate, thus, reducing whole-plant conductance and photosynthesis, setting up a feedback cycle. Once water fluxes approach zero, the finite pools and fluxes of water and carbon become exhaustible, while demands for allocation of carbohydrates to osmoregulation and maintenance of cellular, defensive and, potentially, hydraulic metabolism rise (FIG. 3). Thus, allocation to sapwood, roots, foliage and carbohydrates prior to drought, and to osmoregulation, cytorrhysis tolerance and defence during drought, can all feed back directly upon the likelihood of hydraulic failure, with rising VPD causing a smaller threshold between survival and mortality. However, rising CO_2 might influence such mortality either through promoting structural overshoot or water use benefits, either promoting or delaying mortality as VPD rises (BOX 1).

Potential thresholds also emerge from the mortality mechanisms framework. A decline in cellular water pools below the threshold for cytorrhysis is a clear point of no return of lethal dehydration, which results from the chain of hydraulic events that leads to a threshold amount of hydraulic failure, after which the cytorrhysis threshold was exceeded. The maintenance of the hydraulic system, avoidance of hydraulic failure and minimization and tolerance of cellular dehydration might all depend directly on carbon pool sizes and fluxes to sites of demand. Likewise, induced defensive responses depend on localized fluxes of both water and carbohydrates. These are all possible key failure points within the system — the loss of one can trigger a cascade of losses in the others. These individual or interdependent

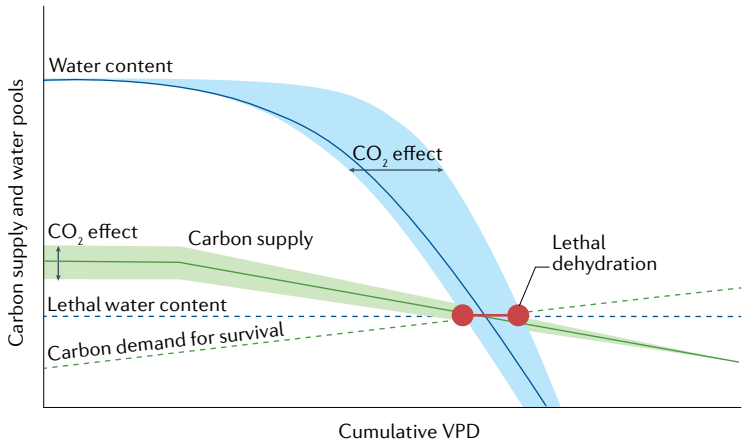
failures at small scales (tissue to organ), with VPD and CO_2 dependencies, will reduce survival likelihood at the whole-plant level.

Box 1 | The impact of rising VPD and CO_2 on mortality risk

Increasing cumulative vapour pressure deficit (VPD) exposure (such as temperature degree days)[228] ultimately causes plant water content (see the figure, blue line) and carbon supply (see the figure, green line) to decrease towards a threshold of lethal dehydration during drought. The mortality thresholds (see the figure, blue and green dashed lines) vary with cellular resistance to cytorrhysis from water and carbon supply limitations. The mortality threshold for water content is assumed to be unchanging with accumulating VPD and, for carbon supply, is assumed to increase with VPD due to increased maintenance requirements[229]. There is a wide range of functional spaces that the curves and mortality thresholds could occupy, with edaphic, taxa, VPD and CO_2 all having a regulatory role. While not represented, temperature causes a small range of negative responses due to its impacts on water and carbon demand (residual conductance (g_{res}) and respiration rates, respectively). The hypothesized responses shown here have been rarely tested, owing to limited research that has manipulated either VPD or CO_2 under drought.

The figure, built upon the proposed mechanistic framework (FIGS 2–4), presents hypotheses regarding the interdependency of the carbon-related and water-related processes, their thresholds and their responses to VPD and CO_2.

- Hypothesis 1: accumulating greater exposure to VPD, particularly during drought, reduces plant water content because it increases the demand for transpiration relative to supply.
- Hypothesis 2: as water content declines, the risk of hydraulic failure increases, leading to a feedback loop of increasing dehydration.
- Hypothesis 3: greater VPD exposure reduces whole-plant carbon supply through reduced stomatal conductance to CO_2.
- Hypothesis 4: rising cumulative VPD forces a decline in photosynthesis to near zero as transpiration increases to an upper threshold, exacerbating both carbon supply and water pool declines. However, under particularly high temperature and VPD, transpiration can decline as hydraulic failure progresses.
- Hypothesis 5: reduced CO_2 supply at plant and tissue levels pushes the plant towards the lethal threshold.
- Hypothesis 6: elevated CO_2 shifts the trajectory of the response of water content and carbon supply to accumulating VPD exposure.
- Hypothesis 7: water pools could increase if rising CO_2 reduces transpiration.
- Hypothesis 8: plant-level carbon supply rate increases via CO_2-induced photosynthetic stimulation.
- Hypothesis 9: if structural overshoot[73] occurs such that the shoot-to-root ratio increases, larger biomass requires water and carbon during droughts and heatwaves, accelerating progression towards the lethal thresholds. Higher leaf area can increase carbon uptake prior to drought however, shifting the carbon supply rate above the assumed trajectory.
- Hypothesis 10: overshoot at the stand level through increasing plant density should also reduce water and carbon supply demand through increased competition for finite resources.



Environmental change impacts on mortality

The observed increase in tree mortality (FIG. 1a) makes it imperative to further understand the potential impacts of rising CO_2 and VPD. Projections of future woody plant loss are now discussed, highlighting the potential roles of a changing environment on mortality and outlining the strengths and challenges facing the current generation of models for simulating mortality.

Modelling climate impacts on drought-induced mortality. Existing predictions of future forest mortality are limited, but all point to a common threat of increasing background mortality and die-off events. For example, according to an empirical model, regional-scale conifer loss is predicted across the Southwestern USA by 2050 owing to increases in VPD, along with periodic droughts[29]. These findings were confirmed using multiple tree-scale and ecosystem-scale process models, with Earth system models predicting widespread conifer mortality throughout the Northern Hemisphere, again due to drying[35]. Predictions of increasing future mortality also exist for aspen, eucalypts, conifers and a broad suite of ecosystems under climate change[187–189]. While these models point to threats of woody-plant mortality under warming and droughts, they are challenged in simulation of the combined impacts of rising VPD and CO_2.

To highlight how some potential mortality mechanisms respond to changing climate, a multi-model analysis of mortality likelihood under the independent and combined impacts of rising VPD and CO_2 is provided (FIG. 5; Supplementary Information). Trees are simulated in a Swiss forest where there were physiological observations of both dying and surviving Norway spruce (*Picea abies*) during a severe drought in 2018 (REF.[104]). Model-predicted percentage loss of whole-tree conductance (PLC_{plant}) is used as an index of the risk of mortality as prognostic of drought-associated mortality[48,80,189]; higher PLC_{plant} indicates greater loss of conductance and a greater likelihood of hydraulic failure. Predicting mortality from PLC assumes that PLC_{plant} captures any changes in carbon metabolism that might drive variation in PLC (FIG. 3). Model-data evaluation suggests that models simulated surviving trees better than dying trees (FIG. 5a), particularly late in the mortality process, and, so, estimates of PLC in dying trees are conservative.

The ensemble-mean results indicate several key roles of the changing atmosphere on drought-associated mortality. Firstly, surviving trees with deeper root systems and/or more resistant xylem exhibit similar responses to dying trees but with one distinction: surviving trees consistently have lower PLC_{plant} than dying trees (FIG. 5b,c). Secondly, relative to the baseline 2018 simulations, elevated CO_2 consistently alleviates mortality risk (FIG. 5) by reducing water loss through stomatal closure, prolonging maintenance of belowground conductance, increasing xylem water potentials and, hence, reducing PLC_{plant} (REF.[189]) (FIGS 3,5). Simulated water savings are consistent with observations of decreased stomatal conductance with increasing CO_2 (REFS[190–192]), potentially reducing water loss and slowing the depletion of both plant and soil water pools. However, such maintenance of soil water pools arising from reduced transpiration is

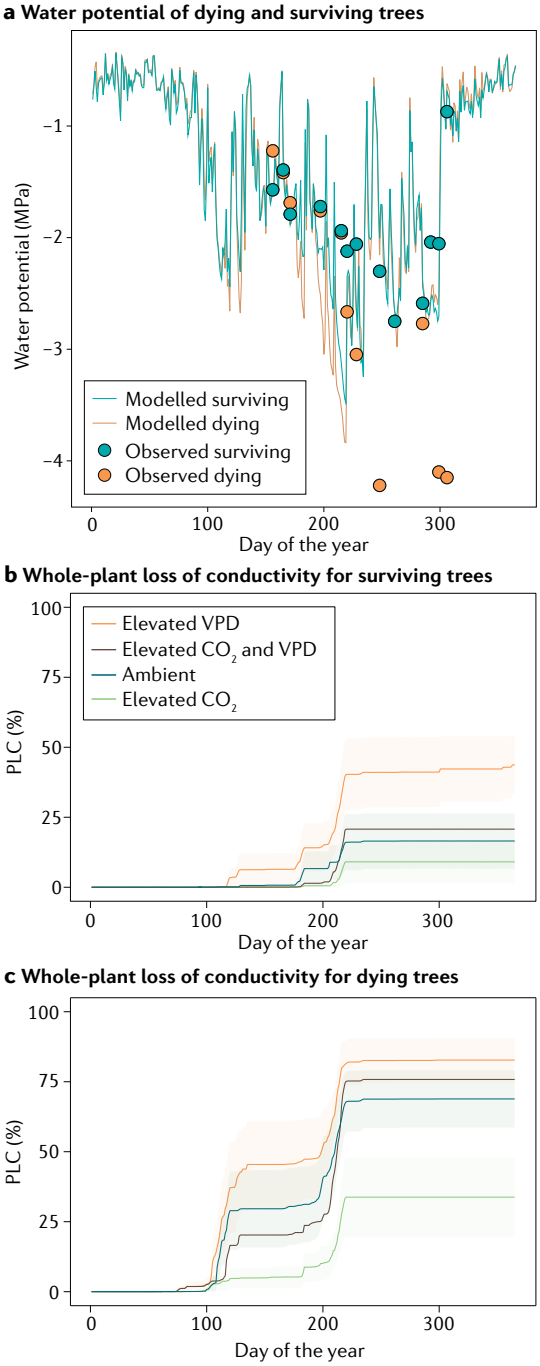


Fig. 5 | **Simulated whole-plant hydraulic failure during drought-associated mortality. a** | Multi-model ensemble-mean predictions of plant water potential compared with observed values in a mature Norway spruce forest during a drought-associated mortality event separated by surviving and dying trees. **b** | Model predictions of mortality via elevated percentage loss of conductance (PLC) under varying CO_2 and vapour pressure deficit (VPD) for those trees that survived the 2018 drought. Lines represent the ensemble-mean values for each climate scenario, where ambient represents the control simulation using 2018 observed climate. The shaded areas represent the standard error around the ensemble mean. Future CO_2, VPD, temperature and relative humidity are derived for the region using downscaled simulations from EURO-CORDEX[227]. **c** | As in panel **b** but for dying trees (those that did not survive the 2018 drought). The models and modelling approach are described in the Supplemental Information. Elevated CO_2 could alleviate mortality risk, while elevated VPD can increase it.

not frequently observed[193–196] due to the compensating effects of increased crown leaf area on water use. Beyond what was modelled, rising CO_2 also increases photosynthetic rates[37] and defensive allocation[197], and influences hydraulic architecture[198], mitigating the risk of carbon limitations and defensive failure. Carbohydrate pools are generally reduced by drought and increased by elevated CO_2 (REF.[199]), thus, potentially balancing each other.

VPD can act in the opposite direction of CO_2 in its impact on mortality likelihood. The model simulations indicate that elevated VPD induces higher PLC_{plant} compared with the 2018 scenario (FIG. 5) owing to increased evaporation from foliage and soils[32], and, thus, decreased belowground conductance. When belowground conductance approaches zero and stomata are closed (FIG. 3), the benefits of CO_2 are manifest to a lesser extent. Furthermore, once the plant is relatively disconnected from external resources and is dependent on its internal resource pools, elevated VPD acts to accelerate the depletion of internal water reserves through evaporation via g_{res}. This evaporative loss is accentuated during heatwaves owing to the temperature sensitivity of g_{res}, in which the cuticular permeability can dramatically increase above 40 °C (REFS[86,87]).

The ensemble-mean simulation results for the elevated CO_2 and elevated VPD combined scenario suggest a slight increase in mortality likelihood (FIG. 5). However, given the inter-model variability, such a slight increase might be insignificant and within the variation across the models. Nonetheless, these results suggest that warming-based VPD increases could balance the ameliorating gains from elevated CO_2. This balancing of the benefits of elevated CO_2 with the consequences of elevated VPD is consistent with the shared time to death of elevated CO_2 and ambient CO_2 drought experiments[200,201], but contrasts with the increases in mortality observed globally (FIG. 1a). Given that the model-data evaluation revealed an overestimation of the water potential of dying trees (FIG. 5a), it is possible that the predicted increases in mortality under the elevated CO_2 and VPD scenario are underestimated.

While not modelled in the simulations, it is important to note that the impact of rising CO_2 on photosynthesis and carbohydrate pools also has implications for defence against biotic attack. Defensive failure can occur via shifts in total nonstructural carbohydrates, specific leaf area and reallocation of leaf nitrogen, all of which alter plant host quality, and thus, suitability for herbivores[202]. Elevated CO_2 also affects host susceptibility and resistance to biotic attack by altering the synthesis or downregulation of phytochemical defence compounds[203]. However, complete resource budgets that document the fluxes and pools of carbon to and from the defence system has not been done, potentially limiting the capacity of models to forecast future mortality.

Mortality mechanisms under rising VPD and CO_2. When examined within the context of rising VPD and CO_2, several clear hypotheses originate from the proposed drought-associated mortality framework (FIGS 2–4) and the model results (FIG. 5). Perhaps the most critical hypotheses are that drought-associated mortality is triggered physiologically by severe declines in belowground hydraulic conductance, with the concurrent water loss, and the water-based and carbon-based thresholds for cytorrhysis, all linking together to drive plants into the dying stage (FIGS 2–4). Further, the logical emergent hypothesis is that rising VPD negatively impacts plant survival during drought, whereas rising CO_2 can have both positive and negative impacts on mortality likelihood (BOX 1). These hypotheses are well supported by non-mortality research but have rarely been examined in relation to drought-induced death[200,201].

Hypothesized mechanisms underlying these responses also emerge. As exposure to VPD accumulates, both through prolonged chronic rises and extreme events, water loss increases, which, if not met by increased belowground supply, forces plant water pools to decline. Furthermore, rising VPD promotes stomatal closure, which reduces carbon supply at the whole-plant scale. Rising CO_2, by contrast, can reduce water loss through stomatal closure and increase photosynthetic rates, leading to higher water content and carbon supply, subsequently reducing mortality likelihood (BOX 1). However, structural overshoot leading to reduced root allocation relative to shoot allocation can predispose trees to mortality through reductions in both water and carbon supply demand during drought. Structural overshoot could also potentially happen at the stand level, in which larger biomass leads to more competition for finite resources (including water and nutrients)[204] during drought.

The interdependency between carbon-related and water-related mechanisms of drought-associated mortality suggest that cytorrhysis occurs when the carbon supply rate needed to maintain hydraulic function exceeds a minimum threshold for survival (BOX 1). An equally likely hypothesis is that water content can fall below the threshold for cytorrhysis before the minimum carbon supply rate is surpassed. Both scenarios could occur depending on the length of the drought and the accumulated exposure to VPD. For example, particularly severe droughts with higher cumulative VPD might promote more rapid drops in water content than in carbon supply rate, thus, exceeding the water content threshold for mortality prior to that for carbon supply rate. Therefore, an associated critical test is to determine the water content and carbon supply rate thresholds and how they respond to increasing cumulative VPD and CO_2.

Challenges to modelling mortality

Models provide useful hypothesis-generating tools for understanding the impacts of a changing environment on mortality (FIG. 5). Models have improved considerably in the representation of water and carbohydrate dynamics at the organ to whole-plant level, and implemented at scales from individual plants to the terrestrial biosphere[49–54,86,205–207]. The coupling between carbon and water at both long and short timescales can now be represented[205,206], although many of the carbon–water interdependencies in the proposed framework (FIG. 3) are either not yet developed or remain untested. Furthermore, gas exchange is represented ever more elegantly[189]. Some models also represent cuticular fluxes and their temperature sensitivity and the subsequent drawdown of internal water pools[86] (FIG. 3). Many models are also trait based and incorporate parameters that are potentially critical to mortality (FIG. 3), some of which can be empirically measured a priori, allowing mechanistically constrained parameterization and large scaling potential[188].

Nevertheless, there remain numerous modelling challenges for those processes that are represented and those that are not, owing, in part, to the interactive nature of mortality drivers and mechanisms. These challenges are highlighted by the model analyses, which, in some cases, failed to capture the particularly negative water potentials of dying trees (FIG. 5a). For those processes captured by models, there can be difficulty in constraining the response functions. For example, belowground conductance is poorly constrained, owing to a lack of empirical measurements. Furthermore, some processes that might be critical to mortality are not yet represented by ecosystem-process models, notably, biotic agents, the attacks of which often coincide with drought[167]. Few models additionally represent the starvation-related mechanisms that could promote the mortality process, including the carbohydrate dependency of metabolism, osmoregulation and hydraulic and defensive functions. Failure of phloem transport can exacerbate localized carbon starvation[130,144,208] but is rarely modelled (but see REF.[149]). Representation of these processes will require more empirical and numerical testing to justify their inclusion in already complex modelling schemes.

A further challenge in modelling woody-plant mortality is the absence of acclimation, partly related to a lack of knowledge of what parameters acclimate and at what rate. If acclimation keeps pace with changes in climate and CO_2, multiple traits could enhance survival likelihood. Some traits that could acclimate to reduced water loss under elevated CO_2 and VPD include reductions in maximum stomatal conductance (g_{max}), g_{res} and individual plant leaf area, along with increases in embolism resistance (estimated as the pressure at which 50% of conductance is lost (P50)).

The impacts of these trait shifts on mortality likelihood were assessed using the SurEau model, representing a spruce tree in the year 2100 (FIG. 6). In this set of scenarios, if g_{res} and P50 decline by 10%, PLC declines (within distal branches) from >90% to ~35%. Similar 10% declines in g_{max} and individual leaf area push PLC likelihood from >90% all the way to 0%. These declines in PLC likelihood translate into declining risk of mortality. Accordingly, reductions in these traits are beneficial to survival under a changing environment (though reductions in all but g_{res} will constrain carbon uptake). g_{max} might acclimate[190,191,209], while P50 has exhibited acclimation potential in angiosperms[210] but less so in gymnosperms[211]. Acclimation to elevated CO_2 results in larger leaf area[200,201,212], which would increase the likelihood of elevated mortality. Acclimation of g_{res} to

Acclimation
Structural or physiological shifts in response to external drivers.

long-term warming is unlikely to buffer plants from hydraulic risks during punctuated heatwaves[87]. These shifts can all reduce the likelihood of critical dehydration through reductions in water demand per unit leaf area, but they have potential negative consequences on the carbon economy through reductions in whole-plant photosynthesis (via reduced leaf area and g_{max}).

Ultimately, acclimation could be important in survival over longer time periods. However, punctuated droughts and heatwaves can occur much too rapidly for acclimation to manifest (except in the case of leaf loss, which occurs in angiosperms during droughts and heatwaves)[212,213] and exacerbate mortality[33]. Heatwaves are of particular concern because they dramatically increase VPD and leaf temperature when water availability is limited, with foliage typically dying at temperatures above 54 °C (REF.[214]). Beyond acclimation, the extension of the phenological cycle through elevated temperature in temperate and boreal regions could, however, mitigate some of the above-mentioned challenges to the potential of acclimation to mitigate mortality likelihood[215–217], with complicated impacts on mortality–reproduction relationships in masting species[218]. A longer growing season can also promote greater water loss, so the net impacts remain unknown.

Summary and future perspectives

Increasing background mortality rates (FIG. 1a) are alarming in the context of the strong current and future increases in their environmental drivers (FIG. 1b,c). The broad geographic distribution of rising mortality suggests a globally distributed driver, which is consistent with physiological theory (FIGS 2–4) and model results (FIG. 5) that suggest elevated CO_2 and VPD could be critical drivers (BOX 1). Mortality appears to be initiated by severe reductions in belowground conductance and subsequent increased risk of hydraulic failure as embolism increases with continued water loss from g_{res} and carbon starvation as stomata shut and, thus, curtail photosynthesis (FIG. 3). Lost hydraulic conductance most immediately increases the risk of hydraulic failure, but if drought is sufficiently prolonged, can deplete carbohydrate stores and fluxes required to maintain metabolism and defence (FIG. 4). Acclimation to drought, CO_2 and VPD could provide some buffering of mortality against environmental change, if such acclimation is of sufficient magnitude and speed to accommodate the rapid rate of climate change (FIGS 1b,c,5). The emergent framework generates a set of hypotheses that require testing if understanding and simulation of woody-plant mortality is to be improved under a changing climate.

The proposed framework identifies considerable experimental challenges, particularly for quantifying the key mortality mechanisms and thresholds, and the carbon–water–defence interdependencies that matter most to survival. For example, we must identify the point-of-no-return value for each critical pool and flux (FIGS 2–4), their timing of achieving the threshold and their dependence upon interacting mechanisms. Identifying these thresholds and their underlying mechanisms is achievable through detailed experimentation that investigates the dynamics and interdependencies of the hydraulic, carbon and defensive mechanisms, pools and fluxes, during the dying process.

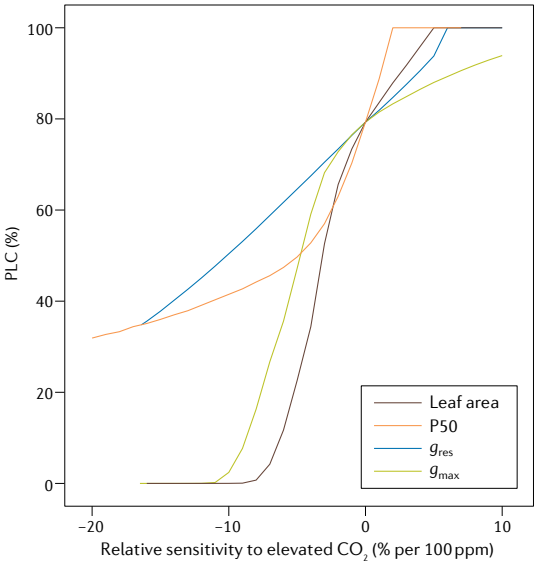


Fig. 6 | **Trait acclimation can reduce mortality likelihood.** Sensitivity analysis, using the SurEau model, in which a spruce tree under elevated CO_2 and vapour pressure deficit (from FIG. 5) is allowed to shift traits as CO_2 rises. The individually shifted parameters are leaf area, P50, g_{res} and g_{max}. If any of these parameters decline as CO_2 rises, they are predicted to reduce the likelihood of mortality, indexed as percentage loss of conductance (PLC), through the influences upon water loss (leaf area, g_{res} and g_{max}) and tolerance to water loss (P50).

In addition to experimental challenges, multiple steps can be taken to test the hypotheses regarding mortality mechanisms (FIGS 2–4). Understanding the predisposing factors, such as differences in carbon allocation to water conduction traits and how they predispose or protect plants from drought-associated death, will require long-term observational and experimental studies. Examining the timing and magnitudes of hydraulic limitations (FIG. 3) and the hydraulic point of no return is feasible[66]. However, a substantial challenge remains to quantify belowground hydraulic dynamics accurately[90], including xylary and extra-xylary components of the pathway. Identifying when and where belowground conductance approaches zero is critical, as it sets the rest of the mortality mechanisms into motion[98] (FIG. 3) and is likely essential to accurate modelling (FIG. 5). Likewise, identifying dehydration thresholds and quantifying how long plants can survive on water stores, while losing water to g_{res} under a warming atmosphere, is a large but critical challenge.

Many components of the proposed carbon failure process are also possible to measure. However, identifying the key carbon-based mechanisms leading to mortality will require substantial investment in developing detailed carbon budgets at both the whole-plant and the cellular scales, as has been done for *Arabidopsis* under mild drought[219]. Such carbon budgets must integrate and quantify the fluctuating carbon demands for metabolic and defensive maintenance, osmoregulation and hydraulic function, and can use metabolomic, transcriptomic and proteomic approaches.

The role of biotic agents is also crucial to test but is challenged, in particular, by the lack of knowledge on the exact role of specific defence compounds[220–222]. Nonetheless, manipulative exclusion and inclusion experiments with biotic agents coupled to detailed defensive-carbon budgets could be used to advance understanding of failure to defend against biotic attack. Ultimately, all of these thresholds can be used to identify the mechanism underlying the mortality threshold, or the point of no return.

The role of temporal and spatial shifts in mechanisms that lead to mortality (FIGS 2–4) are a largely unexplored frontier that must be addressed to reduce model uncertainty. We must determine to what degree can acclimation promote survival and to what degree does a lack of acclimation promote mortality. For plants that can adjust rooting depths, g_{res}, g_{smax}, P50, leaf area (FIG. 6) and other critical variables prior to severe droughts, survival likelihood is increased. If they shift at all[212], the rate of shifts in these variables must be quantified to enable improved predictions. However, non-adaptive acclimation is also a threat, in which extended periods of mesic conditions could promote carbon allocation to increased leaf area and decreased root area (or decreased sapwood area or a myriad of other shifts) that predispose the plant to death when water limitations are severe[76,213]. Scaling of predictions to the ecosystem, region and globe requires consideration of the distinct allometries and allocation patterns of different species/plant functional types[134] and on the distribution of plant roots relative to ephemeral versus constant water sources[206,223,224]. Furthermore, the role of stand density remains a large question[18,225]. If rising CO_2 promotes increasing stand density during periods of abundant precipitation, then when a drought occurs, the stand might be overstocked relative to the site's ability to provide water, thus, promoting mortality. This process, however, is not observed in all regional situations[18,225].

Ultimately, the model predictions of tree mortality are critical to estimates of the future terrestrial carbon sink, land–atmosphere interactions and, hence, the rate of climate warming[226]. Maximizing predictive accuracy requires understanding the mechanistic basis for mortality and simultaneously identifying the simplest and most parsimonious approach to modelling mortality at broad scales. Models should be validated at each temporal and spatial scale and applied to aid understanding of future mortality risks. Understanding and predicting the interdependent mechanisms of mortality under climate warming is a critical research priority for disciplines ranging from tissue-level physiology to global-scale prediction.

Data availability

All data from the simulations can be obtained from the lead author.

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N.G.M. led the effort to generate this manuscript. G.S. generated the figures. A.P., H.C., M.D.C., M.G.D.K. and D.S.M. conducted the modelling simulations. The authors contributed equally to the generation of ideas and writing of the article.

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