



## Mechanisms of woody-plant mortality under rising drought, CO<sub>2</sub> and vapour pressure deficit

Nate G. McDowell<sup>1,2</sup>✉, Gerard Sapes<sup>3</sup>, Alexandria Pivovarov<sup>1</sup>, Henry D. Adams<sup>4</sup>, Craig D. Allen<sup>5</sup>, William R. L. Anderegg<sup>6</sup>, Matthias Arend<sup>7</sup>, David D. Breshears<sup>8</sup>, Tim Brodribb<sup>9</sup>, Brendan Choat<sup>10</sup>, Hervé Cochard<sup>11</sup>, Miquel De Cáceres<sup>12</sup>, Martin G. De Kauwe<sup>13,14,15</sup>, Charlotte Grossiord<sup>16,17</sup>, William M. Hammond<sup>18</sup>, Henrik Hartmann<sup>19</sup>, Günter Hoch<sup>7</sup>, Ansgar Kahmen<sup>7</sup>, Tamir Klein<sup>20</sup>, D. Scott Mackay<sup>21,22</sup>, Marylou Mantova<sup>11</sup>, Jordi Martínez-Vilalta<sup>12,23</sup>, Belinda E. Medlyn<sup>10</sup>, Maurizio Mencuccini<sup>12,24</sup>, Andrea Nardini<sup>25</sup>, Rafael S. Oliveira<sup>26</sup>, Anna Sala<sup>27</sup>, David T. Tissue<sup>10</sup>, José M. Torres-Ruiz<sup>11</sup>, Amy M. Trowbridge<sup>28</sup>, Anna T. Trugman<sup>29</sup>, Erin Wiley<sup>30</sup> and Chonggang Xu<sup>31</sup>

**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<sub>2</sub>. 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<sub>2</sub> 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.

✉e-mail: [nate.mcdowell@pnnl.gov](mailto:nate.mcdowell@pnnl.gov)

<https://doi.org/10.1038/s43017-022-00272-1>

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<sup>1–6</sup> (FIG. 1a), including regional-scale die-off events<sup>7–15</sup> (widespread, rapid tree loss). Large mortality events have been recorded in dry tropical forests<sup>15</sup>, tropical rainforests<sup>16–18</sup>, temperate rainforests<sup>19,20</sup>, semi-arid woodland and savannahs<sup>8,13,21</sup>, boreal forests<sup>22–24</sup> and temperate deciduous to evergreen forests<sup>12,25</sup>. 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<sup>26–29</sup> (FIG. 1b). As a result of greater water loss from foliage and soil surfaces, historically, non-lethal soil droughts have become lethal<sup>29,30</sup>. With anthropogenic forcing anticipated to further increase warming, as well as the frequency and duration of heatwaves and soil drought<sup>10,30–33</sup> (FIG. 1c), tree mortality is also expected to increase in the future<sup>34,35</sup>. However, rising CO<sub>2</sub> (FIG. 1b) can mitigate the negative impacts of increased VPD through higher carbon uptake and reduced water loss, affording potential water savings<sup>36,37</sup>. Contemporary observations suggest that rising VPD could be increasingly offsetting CO<sub>2</sub> benefits<sup>38–40</sup>, and, so, the net balance might be in favour of either greater survival or mortality<sup>37,41–44</sup>.

## 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<sup>45–47</sup>. 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<sup>48</sup>. Though recent models have incorporated these potential processes of mortality<sup>49–54</sup>, 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<sup>55,56</sup>. Changes in ecosystem structure and function due to mortality lead to large ecohydrologic shifts, with abrupt and potentially sustained changes in streamflow<sup>57</sup>, as well as downstream water quality, quantity and timing<sup>58,59</sup>. Long-term shifts in forest demographics might also result from shifts in tree mortality rates<sup>60</sup>, with corresponding limitations to net terrestrial carbon storage<sup>11</sup>; 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<sup>61,62</sup>. Moreover, the economic impacts of forest loss, particularly in regions where wood production is vital to societal well-being, could be substantial<sup>63</sup>.

In this Review, we synthesize understanding of woody-plant mortality under rising VPD and CO<sub>2</sub>. 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<sup>64–70</sup>, beyond which mortality of the organ (branch dieback)<sup>71</sup> 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<sup>1–6</sup> (FIG. 1a) — as observed throughout western and boreal North America<sup>1,2</sup>, the Amazon basin<sup>6</sup> and Europe<sup>11</sup> — 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<sup>72</sup>. Elsewhere, these background rate increases could reflect increasing VPD impacts<sup>26,29,73</sup>, and, under wetter conditions, might also reflect elevated productivity and turnover<sup>74,75</sup>. However, there is little evidence for increasing competition-induced mortality<sup>18,37</sup>. Additionally, when water is ample, rising CO<sub>2</sub> 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<sup>76,77</sup>, 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<sup>7–25</sup>,

## Author addresses

<sup>1</sup>Atmospheric Sciences and Global Change Division, Pacific Northwest National Laboratory, Richland, WA, USA.

<sup>2</sup>School of Biological Sciences, Washington State University, Pullman, WA, USA.

<sup>3</sup>Department of Ecology, Evolution, and Behavior, University of Minnesota, St. Paul, MN, USA.

<sup>4</sup>School of the Environment, Washington State University, Pullman, WA, USA.

<sup>5</sup>Department of Geography and Environmental Studies, University of New Mexico, Albuquerque, NM, USA.

<sup>6</sup>School of Biological Sciences, University of Utah, Salt Lake City, UT, USA.

<sup>7</sup>Department of Environmental Sciences — Botany, University of Basel, Basel, Switzerland.

<sup>8</sup>School of Natural Resources and the Environment, University of Arizona, Tucson, AZ, USA.

<sup>9</sup>School of Natural Sciences, University of Tasmania, Hobart, Tasmania, Australia.

<sup>10</sup>Hawkesbury Institute for the Environment, Western Sydney University, Richmond, New South Wales, Australia.

<sup>11</sup>Université Clermont-Auvergne, INRAE, PIAF, Clermont-Ferrand, France.

<sup>12</sup>Centre for Research on Ecology and Forestry Applications (CREAF), Bellaterra, Spain.

<sup>13</sup>School of Biological Sciences, University of Bristol, Bristol, UK.

<sup>14</sup>ARC Centre of Excellence for Climate Extremes, Sydney, New South Wales, Australia.

<sup>15</sup>Climate Change Research Centre, University of New South Wales, Sydney, New South Wales, Australia.

<sup>16</sup>Plant Ecology Research Laboratory (PERL), École Polytechnique Fédérale de Lausanne (EPFL), Lausanne, Switzerland.

<sup>17</sup>Community Ecology Unit, Swiss Federal Institute for Forest, Snow and Landscape Research (WSL), Lausanne, Switzerland.

<sup>18</sup>Agronomy Department, University of Florida, Gainesville, FL, USA.

<sup>19</sup>Department of Biogeochemical Processes, Max Planck Institute for Biogeochemistry, Jena, Germany.

<sup>20</sup>Department of Plant and Environmental Sciences, Weizmann Institute of Science, Rehovot, Israel.

<sup>21</sup>Department of Geography, University at Buffalo, Buffalo, NY, USA.

<sup>22</sup>Department of Environment and Sustainability, University at Buffalo, Buffalo, NY, USA.

<sup>23</sup>Universitat Autònoma de Barcelona, Bellaterra, Spain.

<sup>24</sup>Institució Catalana de Recerca i Estudis Avançats (ICREA), Barcelona, Spain.

<sup>25</sup>Department of Life Sciences, University of Trieste, Trieste, Italy.

<sup>26</sup>Department of Plant Biology, University of Campinas — UNICAMP, Campinas, Brazil.

<sup>27</sup>Division of Biological Sciences, University of Montana, Missoula, MT, USA.

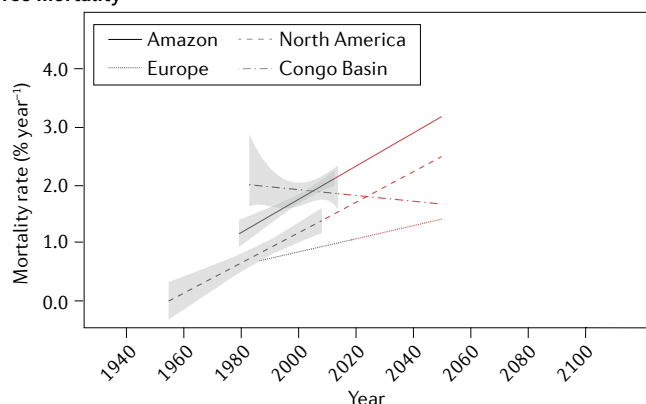
<sup>28</sup>Department of Entomology, University of Wisconsin-Madison, Madison, WI, USA.

<sup>29</sup>Department of Geography, University of California, Santa Barbara, Santa Barbara, CA, USA.

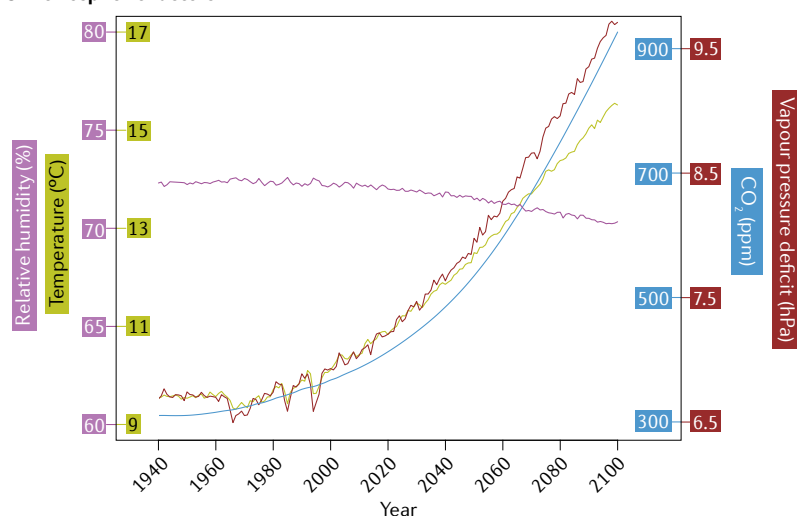
<sup>30</sup>Department of Biology, University of Central Arkansas, Conway, AR, USA.

<sup>31</sup>Earth and Environmental Sciences Division, Los Alamos National Laboratory, Los Alamos, NM, USA.

### a Tree mortality



### b Atmospheric factors



### c Drought frequency

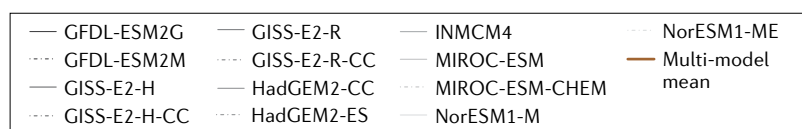
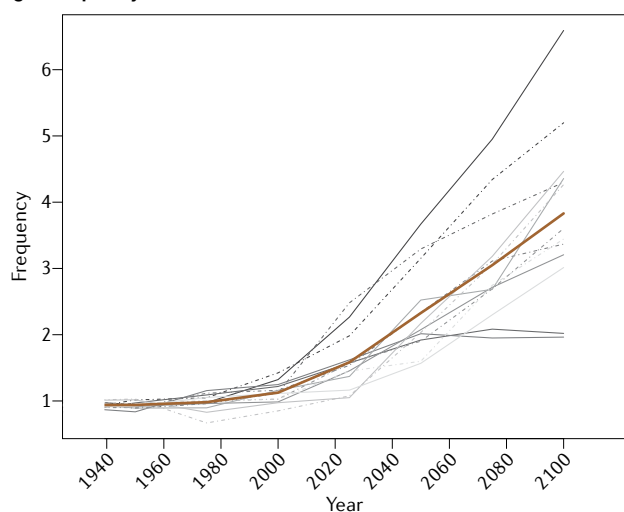


**Fig. 1 | Changing tree mortality and climate variables.**

**a** | Observed (grey) and linearly projected (red) tree mortality rates for North America<sup>1,2</sup>, the Amazon<sup>6</sup>, Europe<sup>11</sup> and the Congo Basin<sup>6</sup>. Grey shading represents 95% confidence intervals. **b** | Average simulated CO<sub>2</sub>, 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.<sup>34</sup> for details of calculations. Increasing atmospheric drivers of mortality are consistent with increasing mortality rates, except in the Congo.

is associated with global increases in temperature and VPD<sup>14</sup>. Droughts eventually occur everywhere, but now with warmer temperature and higher VPD than historically<sup>28</sup>.

### 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<sup>45–47,79</sup>. However, existing frameworks grapple with the complexity of the interdependent processes that occur while trees are dying<sup>45–47,80</sup>, including hydraulic failure, carbon starvation and attack by biotic agents<sup>81,82</sup> (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<sup>45–47,80</sup> and widespread prevalence of hydraulic dysfunction in drought-associated mortality<sup>13,14,22,23</sup>, 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<sup>65,68</sup> (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<sup>68</sup>. 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<sup>83</sup>.

even following decades of productive growth<sup>78</sup>, indicating that no biome is invulnerable. Die-off events are regional in scale, can kill one or more species and occur rapidly<sup>14</sup>. This global distribution of die-off events

## 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<sup>47,81</sup>. 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<sup>84,85</sup> 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<sup>86,87</sup>. Ultimately, hydraulic failure occurs and subsequent downstream mortality ensues as water pools deplete below the threshold for irreversible cell death<sup>46,64–68</sup>, known as cell wall rupture

or cytorrhysis<sup>88,89</sup>. 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<sup>83</sup> (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<sup>90</sup>. The soil–root interface becomes a primary limitation on whole-plant

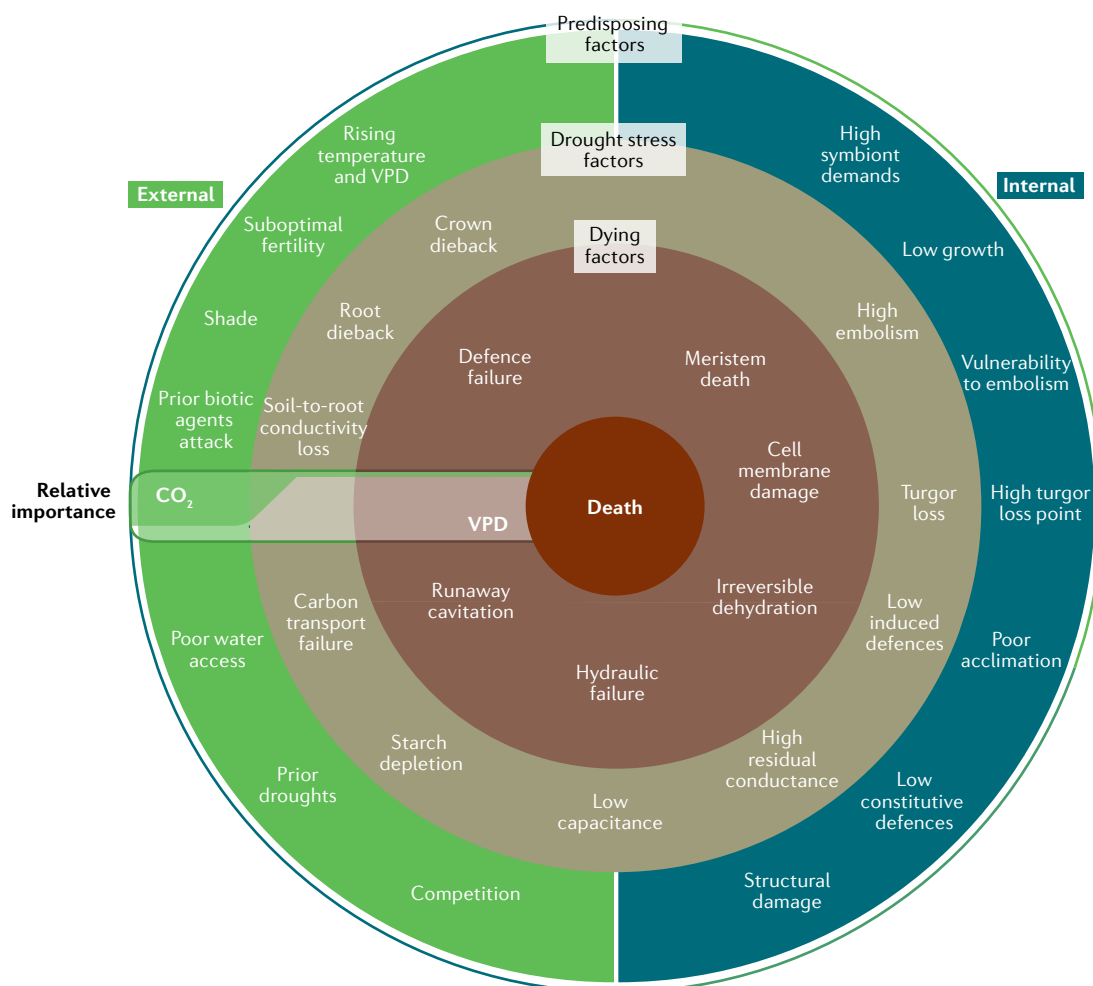


Fig. 2 | **The interconnected mortality process.** A hypothetical representation of the mortality processes from predisposing factors to death. Predisposing factors are linked to mortality via the mechanisms in the second innermost ring, which subsequently cause a plant to pass a threshold beyond which 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.<sup>80</sup>.

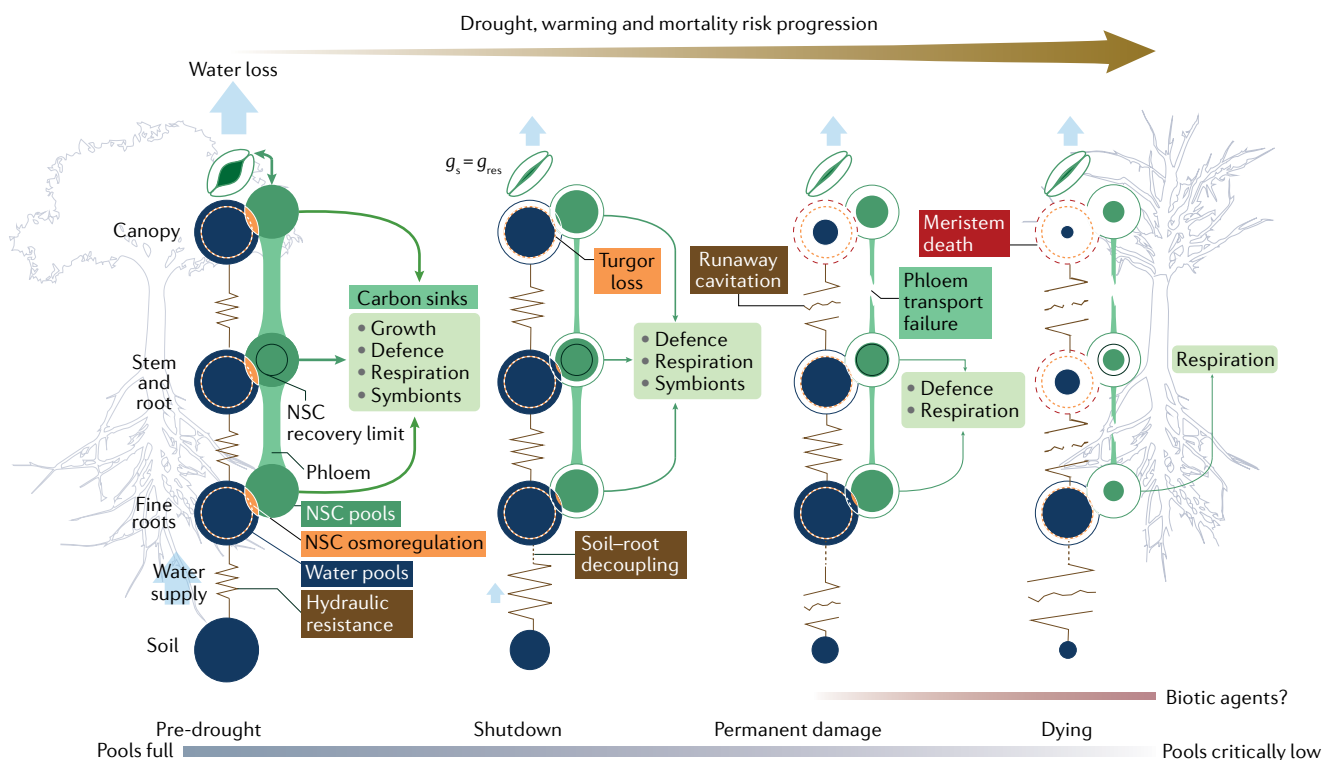


**Fig. 3 | Mechanisms that lead towards mortality.** Hypothesized mechanisms, including pools and fluxes, that influence mortality as drought progresses. Primary water and carbohydrate pools are in blue and green circles, respectively, with their fluxes as resistors and green arrows. Interactions between 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<sup>91,92</sup>. Complete hydraulic disconnection between the soil and roots has been demonstrated in mature woody plants, including during drought-associated mortality<sup>93,94</sup>, although the frequency of such disconnection during drought is unknown. Under these conditions, stomatal conductance declines to near zero<sup>91</sup>, reducing water loss and photosynthesis<sup>95</sup>. Near-zero belowground (soil–root) conductance and stomatal conductance mark a transition point<sup>45,58</sup> when the individual plant becomes solely reliant on its limited water stores<sup>96,97</sup>. The size of these stores, their net depletion rate and the tissue-level and cell-level tolerances for depletion define the likelihood of mortality<sup>98</sup> (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<sup>89</sup> 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<sup>99–103</sup> (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<sup>86,87</sup>. 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<sup>104–107</sup>. In longer droughts, or in cases of mortality years after drought, the role of carbon supply to critical survival processes should increase<sup>104–107</sup> due to the far longer residence time and slower changes in carbohydrates than in water pools<sup>108,109</sup>.

#### 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.

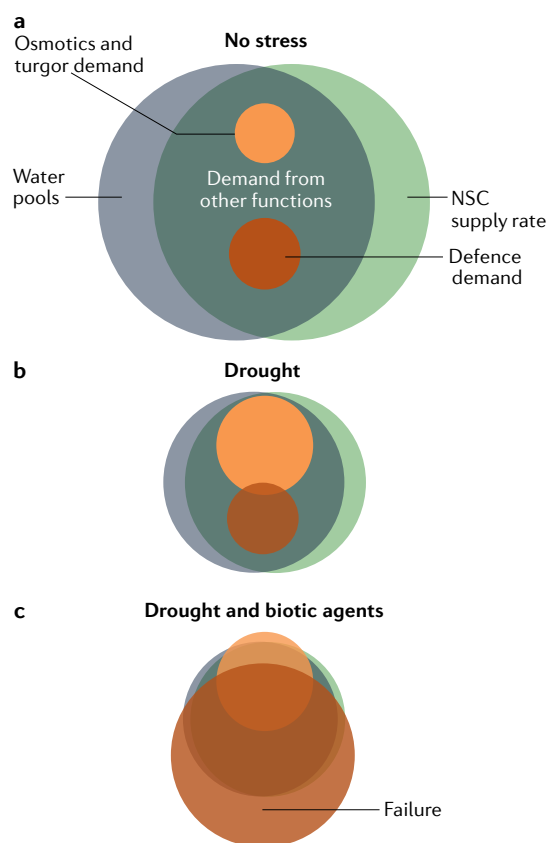


**Fig. 4 | The linkage between woody plant's defence systems and biotic attack.** **a** | Pre-drought, water and carbon pools and supply rates are sufficient to support the demand for defence, osmotic regulation, metabolism and other functions. **b** | During drought, the demand for osmotic regulation 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<sup>45</sup>, reduced xylem and constitutive defensive compound production<sup>110–113</sup>, initial carbohydrate storage increases and then decreases prior to death<sup>11,47–114</sup>, a critical increasing role of carbohydrates in osmoregulation and cellular maintenance<sup>115–122</sup>, and possible carbohydrate feedbacks upon hydraulic and defensive function<sup>47</sup>. 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<sup>105–107,110–113</sup>. Low growth preceding death can be a lingering consequence of prior climate or injuries that predispose trees to carbon constraints<sup>79</sup> (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<sup>123–127</sup> 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)<sup>76</sup>. 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<sup>128–133</sup>. However, declining leaf area can also promote survival by reducing water loss during drought<sup>134,135</sup>. 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<sup>136–138</sup>, 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<sup>139</sup>, 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)<sup>140–142</sup> 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<sup>105,143–145</sup> or insect and/or pathogen defence failure<sup>79,146,147</sup> (FIGS 3,4). If an insufficient supply rate of carbon substrates to required metabolic processes<sup>115</sup> results from short-distance or long-distance transport constraints, for example, through source

strength impacts upon phloem loading and viscosity challenges<sup>148–151</sup>, failure of carbon-dependent processes could be promoted. Declining resistance to xylem embolism has been associated with low carbohydrate concentrations<sup>145</sup>. 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<sup>115–122</sup>. Simultaneously, the carbon requirements for these processes increase with drought stress<sup>45,152–157</sup>, 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<sup>81,158–160</sup>, 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<sup>161–163</sup>. Biotic agents can disrupt carbon uptake and transport through leaf loss<sup>164</sup> or depletion of carbohydrate reserves via direct consumption<sup>165,166</sup>, and they can stimulate a plant's induced defence response<sup>167,168</sup>.

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<sup>169–171</sup>. 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<sup>172</sup>. 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<sup>47</sup> (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<sup>173,174</sup>. Synthesis of secondary metabolites and metabolic transport costs of the defensive system depend directly on labile carbon availability for induced defences<sup>173–176</sup> (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<sup>174,176</sup>. Defensive compounds have a particularly

high carbon concentration<sup>177</sup> and tend to be upregulated at the transcriptional level during drought<sup>178,179</sup>. Similarly, a rapid increase in defensive allocation upon attack has been associated with declining local carbohydrates<sup>168</sup>. Thus, defence incurs a substantial carbon cost<sup>180</sup> (FIG. 4), suggesting that reduced carbohydrate supply rates could render the plant susceptible to biotic attack<sup>181,182</sup>. For example, conifers with fewer resin ducts formed in years prior to drought are frequently more likely to die during beetle attack<sup>111,167,183–186</sup>, 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<sup>127</sup>. 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<sub>2</sub> 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

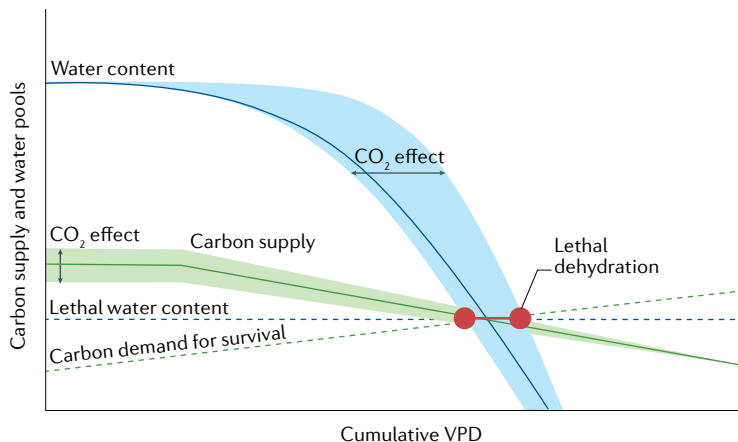
failures at small scales (tissue to organ), with VPD and CO<sub>2</sub> dependencies, will reduce survival likelihood at the whole-plant level.

#### Box 1 | The impact of rising VPD and CO<sub>2</sub> on mortality risk

Increasing cumulative vapour pressure deficit (VPD) exposure (such as temperature degree days)<sup>228</sup> 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<sup>229</sup>. There is a wide range of functional spaces that the curves and mortality thresholds could occupy, with edaphic, taxa, VPD and CO<sub>2</sub> 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<sub>2</sub> 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<sub>2</sub>.

- 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<sub>2</sub>.
- 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<sub>2</sub> supply at plant and tissue levels pushes the plant towards the lethal threshold.
- Hypothesis 6: elevated CO<sub>2</sub> 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<sub>2</sub> reduces transpiration.
- Hypothesis 8: plant-level carbon supply rate increases via CO<sub>2</sub>-induced photosynthetic stimulation.
- Hypothesis 9: if structural overshoot<sup>73</sup> 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

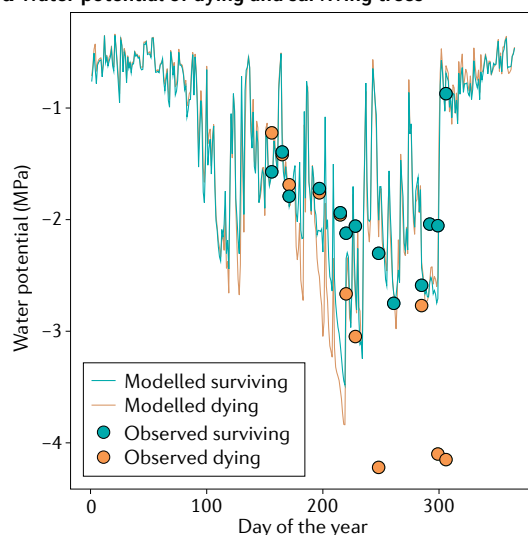
The observed increase in tree mortality (FIG. 1a) makes it imperative to further understand the potential impacts of rising CO<sub>2</sub> and VPD. Projections of future woody plant loss are now discussed, highlighting the potential roles of a changing environment on mortality and outlining the strengths 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<sup>29</sup>. 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<sup>35</sup>. Predictions of increasing future mortality also exist for aspen, eucalypts, conifers and a broad suite of ecosystems under climate change<sup>187–189</sup>. 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<sub>2</sub>.

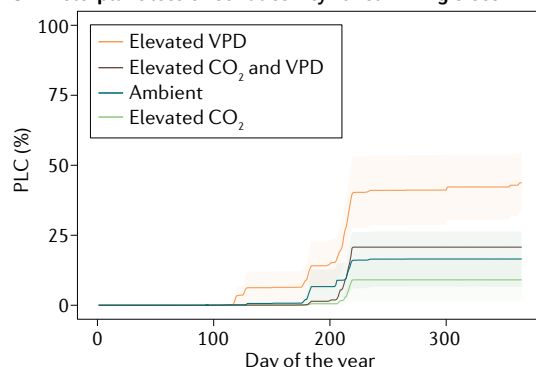
To highlight how some potential mortality mechanisms respond to changing climate, a multi-model analysis of mortality likelihood under the independent and combined impacts of rising VPD and CO<sub>2</sub> is provided (FIG. 5; Supplementary Information). Trees are simulated in a Swiss forest where there were physiological observations of both dying and surviving Norway spruce (*Picea abies*) during a severe drought in 2018 (REF.<sup>104</sup>). Model-predicted percentage loss of whole-tree conductance (PLC<sub>plant</sub>) is used as an index of the risk of mortality as prognostic of drought-associated mortality<sup>48,80,189</sup>; higher PLC<sub>plant</sub> indicates greater loss of conductance and a greater likelihood of hydraulic failure. Predicting mortality from PLC assumes that PLC<sub>plant</sub> 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<sub>plant</sub> than dying trees (FIG. 5b,c). Secondly, relative to the baseline 2018 simulations, elevated CO<sub>2</sub> 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<sub>plant</sub> (REF.<sup>189</sup>) (FIGS 3,5). Simulated water savings are consistent with observations of decreased stomatal conductance with increasing CO<sub>2</sub> (REFS<sup>190–192</sup>), 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

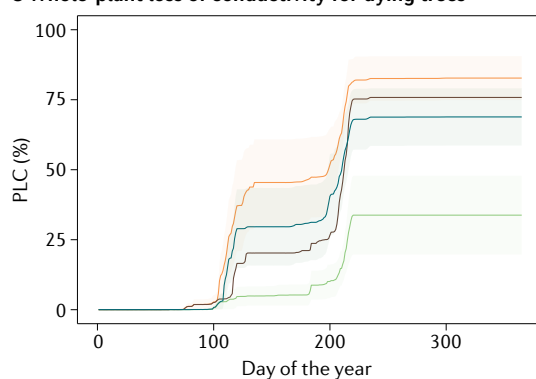
**a** Water potential of dying and surviving trees



**b** Whole-plant loss of conductivity for surviving trees



**c** Whole-plant loss of conductivity for dying trees



not frequently observed<sup>193–196</sup> due to the compensating effects of increased crown leaf area on water use. Beyond what was modelled, rising CO<sub>2</sub> also increases photosynthetic rates<sup>37</sup> and defensive allocation<sup>197</sup>, and influences hydraulic architecture<sup>198</sup>, mitigating the risk of carbon limitations and defensive failure. Carbohydrate pools are generally reduced by drought and increased by elevated CO<sub>2</sub> (REF.<sup>199</sup>), thus, potentially balancing each other.

VPD can act in the opposite direction of CO<sub>2</sub> in its impact on mortality likelihood. The model simulations indicate that elevated VPD induces higher PLC<sub>plant</sub> compared with the 2018 scenario (FIG. 5) owing to increased evaporation from foliage and soils<sup>32</sup>, and, thus, decreased belowground conductance. When belowground

**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<sub>2</sub> 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<sub>2</sub>, VPD, temperature and relative humidity are derived for the region using downscaled simulations from EURO-CORDEX<sup>227</sup>. **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<sub>2</sub> could alleviate mortality risk, while elevated VPD can increase it.

conductance approaches zero and stomata are closed (FIG. 3), the benefits of CO<sub>2</sub> 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<sup>86,87</sup>).

The ensemble-mean simulation results for the elevated CO<sub>2</sub> 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<sub>2</sub>. This balancing of the benefits of elevated CO<sub>2</sub> with the consequences of elevated VPD is consistent with the shared time to death of elevated CO<sub>2</sub> and ambient CO<sub>2</sub> drought experiments<sup>200,201</sup>, 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<sub>2</sub> and VPD scenario are underestimated.

While not modelled in the simulations, it is important to note that the impact of rising CO<sub>2</sub> 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<sup>202</sup>. Elevated CO<sub>2</sub> also affects host susceptibility and resistance to biotic attack by altering the synthesis or downregulation of phytochemical defence compounds<sup>203</sup>. 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<sub>2</sub>.** When examined within the context of rising VPD and CO<sub>2</sub>, 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<sub>2</sub> 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<sup>200,201</sup>.

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<sub>2</sub>, 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)<sup>204</sup> 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<sub>2</sub>.

### 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<sup>49–54,86,205–207</sup>. The coupling between carbon

and water at both long and short timescales can now be represented<sup>205,206</sup>, 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<sup>189</sup>. Some models also represent cuticular fluxes and their temperature sensitivity and the subsequent drawdown of internal water pools<sup>86</sup> (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<sup>188</sup>.

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<sup>167</sup>. 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<sup>130,144,208</sup> but is rarely modelled (but see REF.<sup>149</sup>). 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<sub>2</sub>, multiple traits could enhance survival likelihood. Some traits that could acclimate to reduced water loss under elevated CO<sub>2</sub> and VPD include reductions in maximum stomatal conductance ( $g_{\max}$ ),  $g_{\text{res}}$  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  $g_{\text{res}}$  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  $g_{\text{res}}$  will constrain carbon uptake).  $g_{\max}$  might acclimate<sup>190,191,209</sup>, while P50 has exhibited acclimation potential in angiosperms<sup>210</sup> but less so in gymnosperms<sup>211</sup>. Acclimation to elevated CO<sub>2</sub> results in larger leaf area<sup>200,201,212</sup>, which would increase the likelihood of elevated mortality. Acclimation of  $g_{\text{res}}$  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<sup>87</sup>. 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)<sup>212,213</sup> and exacerbate mortality<sup>33</sup>. 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.<sup>214</sup>). 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<sup>215–217</sup>, with complicated impacts on mortality–reproduction relationships in masting species<sup>218</sup>. 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  $\text{CO}_2$  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  $g_{\text{res}}$  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,  $\text{CO}_2$  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

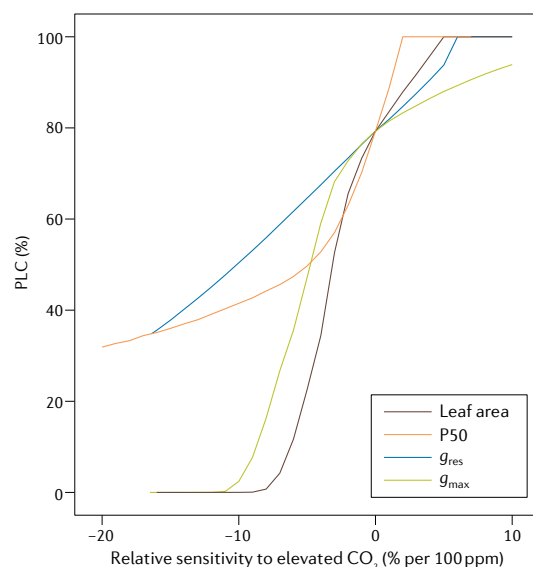


**Fig. 6 | Trait acclimation can reduce mortality likelihood.** Sensitivity analysis, using the SurEau model, in which a spruce tree under elevated  $\text{CO}_2$  and vapour pressure deficit (from FIG. 5) is allowed to shift traits as  $\text{CO}_2$  rises. The individually shifted parameters are leaf area, P50,  $g_{\text{res}}$  and  $g_{\max}$ . If any of these parameters decline as  $\text{CO}_2$  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,  $g_{\text{res}}$  and  $g_{\max}$ ) and tolerance to water loss (P50).

the hydraulic, carbon and defensive mechanisms, pools and fluxes, during the dying process.

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<sup>66</sup>. However, a substantial challenge remains to quantify belowground hydraulic dynamics accurately<sup>90</sup>, 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<sup>98</sup> (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  $g_{\text{res}}$  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<sup>219</sup>. 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<sup>220–222</sup>. 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<sup>212</sup>, 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<sup>76,213</sup>. Scaling of predictions to the ecosystem, region and globe requires consideration of the distinct allometries and allocation patterns

of different species/plant functional types<sup>134</sup> and on the distribution of plant roots relative to ephemeral versus constant water sources<sup>206,223,224</sup>. Furthermore, the role of stand density remains a large question<sup>18,225</sup>. If rising CO<sub>2</sub> 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<sup>18,225</sup>.

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<sup>226</sup>. 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.

Published online: 29 March 2022

1. van Mantgem, P. J. et al. Widespread increase of tree mortality rates in the western United States. *Science* **323**, 521–524 (2009).
2. Peng, C. et al. A drought-induced pervasive increase in tree mortality across Canada's boreal forests. *Nat. Clim. Chang.* **1**, 467–471 (2011).
3. Brien, R. J. et al. Long-term decline of the Amazon carbon sink. *Nature* **519**, 344–348 (2015).
4. Klein, T., Cahanovitch, R., Sprints, M., Herr, N. & Schiller, G. A nation-wide analysis of tree mortality under climate change: forest loss and its causes in Israel 1948–2017. *For. Ecol. Manag.* **432**, 840–849 (2019).
5. Yu, K. et al. Pervasive decreases in living vegetation carbon turnover time across forest climate zones. *Proc. Natl Acad. Sci. USA* **116**, 24662–24667 (2019).
6. Hubau, W. et al. Asynchronous carbon sink saturation in African and Amazonian tropical forests. *Nature* **579**, 80–87 (2020).
7. Kharuk, V. I. et al. Climate-driven conifer mortality in Siberia. *Glob. Ecol. Biogeogr.* **30**, 543–556 (2021).
8. Breshears, D. D. et al. Regional vegetation die-off in response to global-change-type drought. *Proc. Natl Acad. Sci. USA* **102**, 15144–15148 (2005).
9. Lewis, S. L., Brando, P. M., Phillips, O. L., van der Heijden, G. M. & Nepstad, D. The 2010 Amazon drought. *Science* **331**, 554 (2011).
10. Ruthrof, K. X. et al. Subcontinental heat wave triggers terrestrial and marine, multi-taxa responses. *Sci. Rep.* **8**, 13094 (2018).
11. Senf, C. et al. Canopy mortality has doubled in Europe's temperate forests over the last three decades. *Nat. Commun.* **9**, 4978 (2018).
12. Schuldt, B. et al. A first assessment of the impact of the extreme 2018 summer drought on Central European forests. *Basic Appl. Ecol.* **45**, 86–103 (2020).
13. Kannenberg, S. A., Driscoll, A. W., Malesky, D. & Anderegg, W. R. Rapid and surprising dieback of Utah juniper in the southwestern USA due to acute drought stress. *For. Ecol. Manag.* **480**, 118639 (2021).
14. Allen, C. D., Breshears, D. D. & McDowell, N. G. On underestimation of global vulnerability to tree mortality and forest die-off from hotter drought in the Anthropocene. *Ecosphere* **6**, 1–55 (2015).
15. Powers, J. S. et al. A catastrophic tropical drought kills hydraulically vulnerable tree species. *Glob. Change Biol.* **26**, 3122–3133 (2020).
16. Werner, W. L. Canopy dieback in the upper montane rain forests of Sri Lanka. *Geojournal* **17**, 245–248 (1988).
17. Feldpausch, T. R. et al. Amazon forest response to repeated droughts. *Glob. Biogeochem. Cycles* **30**, 964–982 (2016).
18. Esquivel-Muelbert, A. et al. Tree mode of death and mortality risk factors across Amazon forests. *Nat. Commun.* **11**, 5515 (2020).
19. Werner, R. A. & Holsten, E. H. Mortality of white spruce during a spruce beetle outbreak on the Kenai Peninsula in Alaska. *Can. J. For. Res.* **13**, 96–101 (1983).
20. Suarez, M. L., Ghermandi, L. & Kitzberger, T. Factors predisposing episodic drought-induced tree mortality in Nothofagus: site, climatic sensitivity and growth trends. *J. Ecol.* **92**, 954–966 (2004).
21. Swemmer, A. M. Locally high, but regionally low: the impact of the 2014–2016 drought on the trees of semi-arid savannas, South Africa. *Afr. J. Range Forage Sci.* **37**, 31–42 (2020).
22. Michaelian, M., Hogg, E. H., Hall, R. J. & Arsenault, E. Massive mortality of aspen following severe drought along the southern edge of the Canadian boreal forest. *Glob. Chang. Biol.* **17**, 2084–2094 (2011).
23. Kharuk, V. I. et al. Climate-induced mortality of Siberian pine and fir in the Lake Baikal Watershed, Siberia. *For. Ecol. Manag.* **384**, 191–199 (2017).
24. Kharuk, V. I., Ranson, K. J., Oskorbin, P. A., Im, S. T. & Dvinskaya, M. L. Climate induced birch mortality in Trans-Baikal lake region, Siberia. *For. Ecol. Manag.* **289**, 385–392 (2013).
25. Crouchet, S. E., Jensen, J., Schwartz, B. F. & Schwinning, S. Tree mortality after a hot drought: distinguishing density-dependent and -independent drivers and why it matters. *Front. For. Glob. Change* **2**, 21 (2019).
26. Breshears, D. D. et al. The critical amplifying role of increasing atmospheric moisture demand on tree mortality and associated regional die-off. *Front. Plant Sci.* **4**, 266 (2013).
27. Grossiord, C. et al. Plant responses to rising vapor pressure deficit. *New Phytol.* **226**, 1550–1566 (2020).
28. Trenberth, K. E. et al. Global warming and changes in drought. *Nat. Clim. Chang.* **4**, 17–22 (2014).
29. Williams, A. P. et al. Temperature as a potent driver of regional forest drought stress and tree mortality. *Nat. Clim. Chang.* **3**, 292–297 (2013).
30. Xu, C. et al. Increasing impacts of extreme droughts on vegetation productivity under climate change. *Nat. Clim. Chang.* **9**, 948–953 (2019).
31. Dore, M. H. Climate change and changes in global precipitation patterns: what do we know? *Environ. Int.* **31**, 1167–1181 (2005).
32. Ukkola, A. M., De Kauwe, M. G., Roderick, M. L., Abramowitz, G. & Pitman, A. J. Robust future changes in meteorological drought in CMIP6 projections despite uncertainty in precipitation. *Geophys. Res. Lett.* **31**, e2020GL087820 (2020).
33. Breshears, D. D. et al. Underappreciated plant vulnerabilities to heat waves. *New Phytol.* **231**, 32–39 (2021).
34. Adams, H. D. et al. Temperature response surfaces for mortality risk of tree species with future drought. *Environ. Res. Lett.* **12**, 115014 (2017).
35. McDowell, N. G. et al. Multi-scale predictions of massive conifer mortality due to chronic temperature rise. *Nat. Clim. Chang.* **6**, 295–300 (2016).
36. Keenan, T. F. et al. Increase in forest water-use efficiency as atmospheric carbon dioxide concentrations rise. *Nature* **499**, 324–327 (2013).
37. Walker, A. P. et al. Integrating the evidence for a terrestrial carbon sink caused by increasing atmospheric CO<sub>2</sub>. *New Phytol.* **229**, 2413–2445 (2020).
38. Long, S. P. Modification of the response of photosynthetic productivity to rising temperature by atmospheric CO<sub>2</sub> concentrations: has its importance been underestimated? *Plant Cell Environ.* **14**, 729–739 (1991).
39. Hickler, T. et al. CO<sub>2</sub> fertilization in temperate FACE experiments not representative of boreal and tropical forests. *Glob. Change Biol.* **14**, 1531–1542 (2008).
40. Baig, S., Medlyn, B. E., Mercado, L. & Zaehle, S. Does the growth response of woody plants to elevated CO<sub>2</sub> increase with temperature? A model-oriented meta-analysis. *Glob. Change Biol.* **21**, 4303–4319 (2015).
41. Peñuelas, J. et al. Shifting from a fertilization-dominated to a warming-dominated period. *Nat. Ecol. Evol.* **1**, 1438–1445 (2017).
42. Belmecheri, S. et al. Precipitation alters the CO<sub>2</sub> effect on water-use efficiency of temperate forests. *Glob. Change Biol.* **27**, 1560–1571 (2021).

43. Duffy, K. A. et al. How close are we to the temperature tipping point of the terrestrial biosphere? *Sci. Adv.* **7**, eay1052 (2021).
44. De Kauwe, M. G., Medlyn, B. E. & Tissue, D. T. To what extent can rising [CO<sub>2</sub>] ameliorate plant drought stress? *New Phytol.* **231**, 2118–2124 (2021).
45. Martínez-Vilalta, J., Piñol, J. & Beven, K. A hydraulic model to predict drought-induced mortality in woody plants: an application to climate change in the Mediterranean. *Ecol. Model.* **155**, 127–147 (2002).
46. McDowell, N. et al. Mechanisms of plant survival and mortality during drought: why do some plants survive while others succumb to drought? *New Phytol.* **178**, 719–739 (2008).
47. McDowell, N. G. et al. The interdependence of mechanisms underlying climate-driven vegetation mortality. *Trends Ecol. Evol.* **26**, 523–532 (2011).
48. Adams, H. D. et al. A multi-species synthesis of physiological mechanisms in drought-induced tree mortality. *Nat. Ecol. Evol.* **1**, 1285–1291 (2017).
49. Fisher, R. et al. Assessing uncertainties in a second-generation dynamic vegetation model caused by ecological scale limitations. *New Phytol.* **187**, 666–681 (2010).
50. McDowell, N. G. et al. Evaluating theories of drought-induced vegetation mortality using a multimodel–experiment framework. *New Phytol.* **200**, 304–321 (2013).
51. Anderegg, W. R. L. et al. Hydraulic diversity of forests regulates ecosystem resilience during drought. *Nature* **561**, 538–541 (2018).
52. Christoffersen, B. O. et al. Linking hydraulic traits to tropical forest function in a size-structured and trait-driven model (TFS v. 1-Hydro). *Geosci. Model Dev.* **9**, 4227–4255 (2016).
53. Kennedy, D. et al. Implementing plant hydraulics in the community land model, version 5. *J. Adv. Model. Earth Syst.* **11**, 485–513 (2019).
54. Koven, C. D. et al. Benchmarking and parameter sensitivity of physiological and vegetation dynamics using the Functionally Assembled Terrestrial Ecosystem Simulator (FATES) at Barro Colorado Island, Panama. *Biogeosciences* **17**, 3017–3044 (2020).
55. Anderegg, W. R., Kane, J. M. & Anderegg, L. D. Consequences of widespread tree mortality triggered by drought and temperature stress. *Nat. Clim. Chang.* **3**, 30–36 (2013).
56. Hartmann, H. et al. Research frontiers for improving our understanding of drought-induced tree and forest mortality. *New Phytol.* **218**, 15–28 (2018).
57. Adams, H. D. et al. Ecophysiological consequences of drought- and infestation-triggered tree die-off: insights and hypotheses. *Ecophysiology* **5**, 145–159 (2012).
58. Bearup, L. A., Maxwell, R. M., Clow, D. W. & McCray, J. E. Hydrological effects of forest transpiration loss in bark beetle-impacted watersheds. *Nat. Clim. Chang.* **4**, 481–486 (2014).
59. Bennett, K. E. et al. Climate-driven disturbances in the San Juan River sub-basin of the Colorado River. *Hydrol. Earth Syst. Sci.* **22**, 709–725 (2018).
60. Lutz, J. A. & Halpern, C. B. Tree mortality during early forest development: a long-term study of rates, causes, and consequences. *Ecol. Monogr.* **76**, 257–275 (2006).
61. Clark, J. S. et al. The impacts of increasing drought on forest dynamics, structure, and biodiversity in the United States. *Glob. Change Biol.* **22**, 2329–2352 (2016).
62. McDowell, N. G. et al. Pervasive shifts in forest dynamics in a changing world. *Science* **368**, eaaz9463 (2020).
63. Waring, K. M. et al. Modeling the impacts of two bark beetle species under a warming climate in the southwestern USA: ecological and economic consequences. *Environ. Manag.* **44**, 824–835 (2009).
64. Barigah, T. S. et al. Water stress-induced xylem hydraulic failure is a causal factor of tree mortality in beech and poplar. *Ann. Bot.* **112**, 1431–1437 (2013).
65. Guadagno, C. R. et al. Dead or alive? Using membrane failure and chlorophyll a fluorescence to predict plant mortality from drought. *Plant Physiol.* **175**, 223–234 (2017).
66. Hammond, W. M. et al. Dead or dying? Quantifying the point of no return from hydraulic failure in drought-induced tree mortality. *New Phytol.* **223**, 1834–1843 (2019).
67. Sapes, G. et al. Plant water content integrates hydraulics and carbon depletion to predict drought-induced seedling mortality. *Tree Physiol.* **39**, 1300–1312 (2019).
68. Mantova, M., Menezes-Silva, P. E., Badel, E., Cochard, H. & Torres-Ruiz, J. M. The interplay of hydraulic failure and cell vitality explains tree capacity to recover from drought. *Physiol. Plant.* **172**, 247–257 (2021).
69. Kono, Y. et al. Initial hydraulic failure followed by late-stage carbon starvation leads to drought-induced death in the tree *Trema orientalis*. *Commun. Biol.* **2**, 8 (2019).
70. Preisler, Y. et al. Seeking the “point of no return” in the sequence of events leading to mortality of mature trees. *Plant Cell Environ.* **44**, 1315–1328 (2020).
71. Allen, C. D. et al. A global overview of drought and heat-induced tree mortality reveals emerging climate change risks for forests. *For. Ecol. Manag.* **259**, 660–684 (2010).
72. Bennett, A. C. et al. Resistance of African tropical forests to an extreme climate anomaly. *Proc. Natl Acad. Sci. USA* **118**, e2003169118 (2021).
73. McDowell, N. G. & Allen, C. D. Darcy’s law predicts widespread forest mortality under climate warming. *Nat. Clim. Chang.* **5**, 669–672 (2015).
74. Stephenson, N. L. & van Mantgem, P. J. Forest turnover rates follow global and regional patterns of productivity. *Ecol. Lett.* **8**, 524–531 (2005).
75. Zhu, K. C. et al. Dual impacts of climate change: forest migration and turnover through life history. *Glob. Change Biol.* **20**, 251–264 (2014).
76. Jump, A. S. et al. Structural overshoot of tree growth with climate variability and the global spectrum of drought-induced forest dieback. *Glob. Change Biol.* **23**, 3742–3757 (2017).
77. Trugman, A. T. et al. Tree carbon allocation explains forest drought-kill and recovery patterns. *Ecol. Lett.* **21**, 1552–1560 (2018).
78. Hartmann, H. et al. Climate change risks to global forest health – emergence of unexpected events of elevated tree mortality world-wide. *Annu. Rev. Plant Biol.* <https://doi.org/10.1146/annurev-arplant-102820-012804> (2022).
79. Manion, P. D. *Tree Disease Concepts* (Prentice-Hall, 1981).
80. Brodribb, T. J. Learning from a century of droughts. *Nat. Ecol. Evol.* **4**, 1007–1008 (2020).
81. Anderegg, W. R. et al. Tree mortality from drought, insects, and their interactions in a changing climate. *New Phytol.* **208**, 674–683 (2015).
82. Huang, J. et al. Tree defence and bark beetles in a drying world: carbon partitioning, functioning and modelling. *New Phytol.* **225**, 26–36 (2019).
83. Martínez-Vilalta, J., Anderegg, W. R., Sapes, G. & Sala, A. Greater focus on water pools may improve our ability to understand and anticipate drought-induced mortality in plants. *New Phytol.* **223**, 22–32 (2019).
84. Cuneo, I. F., Knipfer, T., Brodersen, C. R. & McElrone, A. J. Mechanical failure of fine root cortical cells initiates plant hydraulic decline during drought. *Plant Physiol.* **172**, 1669–1678 (2016).
85. Johnson, D. M. et al. Co-occurring woody species have diverse hydraulic strategies and mortality rates during an extreme drought. *Plant Cell Environ.* **41**, 576–588 (2018).
86. Cochard, H. A new mechanism for tree mortality due to drought and heatwaves. *Peer Community J.* **1**, e36 (2021).
87. Duursma, R. A. et al. On the minimum leaf conductance: its role in models of plant water use, and ecological and environmental controls. *New Phytol.* **221**, 693–705 (2019).
88. Beckett, R. P. Pressure–volume analysis of a range of poikilohydric plants implies the existence of negative turgor in vegetative cells. *Ann. Bot.* **79**, 145–152 (1997).
89. Ding, Y., Zhang, Y., Zheng, Q. S. & Tyree, M. T. Pressure–volume curves: revisiting the impact of negative turgor during cell collapse by literature review and simulations of cell micromechanics. *New Phytol.* **203**, 378–387 (2014).
90. Sperry, J. S., Adler, F. R., Campbell, G. S. & Comstock, J. P. Limitation of plant water use by rhizosphere and xylem conductance: results from a model. *Plant Cell Environ.* **21**, 347–359 (1998).
91. Rodríguez-Domínguez, C. M. & Brodribb, T. J. Declining root water transport drives stomatal closure in olive under moderate water stress. *New Phytol.* **225**, 126–134 (2020).
92. Carminati, A. & Javaux, M. Soil rather than xylem vulnerability controls stomatal response to drought. *Trends Plant Sci.* **25**, 868–880 (2020).
93. Maseda, P. H. & Fernandez, R. J. Stay wet or else: three ways in which plants can adjust hydraulically to their environment. *J. Exp. Bot.* **57**, 3963–3977 (2006).
94. Plaut, J. A. et al. Hydraulic limits preceding mortality in a piñon–juniper woodland under experimental drought. *Plant Cell Environ.* **35**, 1601–1617 (2012).
95. Creek, D. et al. Xylem embolism in leaves does not occur with open stomata: evidence from direct observations using the optical visualization technique. *J. Exp. Bot.* **71**, 1151–1159 (2020).
96. Choat, B. et al. Triggers of tree mortality under drought. *Nature* **558**, 531–539 (2018).
97. Hammond, W. M. & Adams, H. D. Dying on time: traits influencing the dynamics of tree mortality risk from drought. *Tree Physiol.* **39**, 906–909 (2019).
98. Körner, C. No need for pipes when the well is dry — a comment on hydraulic failure in trees. *Tree Physiol.* **39**, 695–700 (2019).
99. Machado, R. et al. Where do leaf water leaks come from? Trade-offs underlying the variability in minimum conductance across tropical savanna species with contrasting growth strategies. *New Phytol.* **229**, 1415–1430 (2021).
100. Burghardt, M. & Riederer, M. in *Biology of the Plant Cuticle* (eds Riederer, M. & Müller, C.) 292–311 (Blackwell, 2006).
101. Billon, L. M. et al. The DroughtBox: a new tool for phenotyping residual branch conductance and its temperature dependence during drought. *Plant Cell Environ.* **43**, 1584–1594 (2020).
102. Wolfe, B. T. Bark water vapour conductance is associated with drought performance in tropical trees. *Biol. Lett.* **16**, 20200263 (2020).
103. Martín-Gómez, P., Serrano, L. & Ferrio, J. P. Short-term dynamics of evaporative enrichment of xylem water in woody stems: implications for ecohydrology. *Tree Physiol.* **37**, 511–522 (2017).
104. Arend, M. et al. Rapid hydraulic collapse as cause of drought-induced mortality in conifers. *Proc. Natl Acad. Sci. USA* **118**, e2025251118 (2021).
105. Wang, W. et al. Mortality predispositions of conifers across western USA. *New Phytol.* **229**, 831–844 (2020).
106. Christiansen, E., Waring, R. H. & Berryman, A. A. Resistance of conifers to bark beetle attack: searching for general relationships. *For. Ecol. Manag.* **22**, 89–106 (1987).
107. Bigler, C., Bräker, O. U., Bugmann, H., Döbertin, M. & Rigling, A. Drought as an inciting mortality factor in Scots pine stands of the Valais, Switzerland. *Ecosystems* **9**, 330–343 (2006).
108. Richardson, A. D. et al. Seasonal dynamics and age of stemwood nonstructural carbohydrates in temperate forest trees. *New Phytol.* **197**, 850–861 (2013).
109. Meinzer, F. C. et al. Dynamics of water transport and storage in conifers studied with deuterium and heat tracing techniques. *Plant Cell Environ.* **29**, 105–114 (2006).
110. McDowell, N. G., Allen, C. D. & Marshall, L. Growth, carbon-isotope discrimination, and drought-associated mortality across a *Pinus ponderosa* elevational transect. *Glob. Change Biol.* **16**, 399–415 (2010).
111. Kane, J. M. & Kolb, T. E. Importance of resin ducts in reducing ponderosa pine mortality from bark beetle attack. *Oecologia* **164**, 601–609 (2010).
112. Ferrenberg, S., Kane, J. M. & Mitton, J. B. Resin duct characteristics associated with tree resistance to bark beetles across lodgepole and limber pines. *Oecologia* **174**, 1283–1292 (2014).
113. Cailleret, M. et al. A synthesis of radial growth patterns preceding tree mortality. *Glob. Change Biol.* **23**, 1675–1690 (2017).
114. Muller, B., Pantin, F., Génard, M., Turc, O., Freixes, S., Piques, M. & Gibon, Y. Water deficits uncouple growth from photosynthesis, increase C content, and modify the relationships between C and growth in sink organs. *J. Exp. Bot.* **62**, 1715–1729 (2011).
115. Yu, S. Cellular and genetic responses of plants to sugar starvation. *Plant Physiol.* **121**, 687–693 (1999).
116. Koster, K. L. & Leopold, A. C. Sugars and desiccation tolerance in seeds. *Plant Physiol.* **88**, 829–832 (1988).
117. Sapes, G., Demaree, P., Lekberg, Y. & Sala, A. Plant carbohydrate depletion impairs water relations and spreads via ectomycorrhizal networks. *New Phytol.* **229**, 3172–3183 (2021).
118. Hoekstra, F. A., Golovina, E. A. & Buitink, J. Mechanisms of plant desiccation tolerance. *Trends Plant Sci.* **6**, 431–438 (2001).
119. Van den Ende, W. & Valluru, R. Sucrose, sucrosyl oligosaccharides, and oxidative stress: scavenging and salvaging? *J. Exp. Bot.* **60**, 9–18 (2009).

120. Matros, A., Peshev, D., Peukert, M., Mock, H.-P. & Ende, W. Vden Sugars as hydroxyl radical scavengers: proof-of-concept by studying the fate of sucralose in *Arabidopsis*. *Plant J.* **82**, 822–839 (2015).
121. Rolland, F., Baena-González, E. & Sheen, J. Sugar sensing and signaling in plants: conserved and novel mechanisms. *Annu. Rev. Plant Biol.* **57**, 675–709 (2006).
122. Ramel, F., Sulmon, C., Bogard, M., Couée, I. & Gouesbet, G. Differential patterns of reactive oxygen species and antioxidative mechanisms during atrazine injury and sucrose-induced tolerance in *Arabidopsis thaliana* plantlets. *BMC Plant Biol.* **9**, 28 (2009).
123. Fine, P. V. A. et al. The growth–defense trade-off and habitat specialization by plants in Amazonian forests. *Ecology* **87**, S150–S162 (2006).
124. Huot, B., Yao, J., Montgomery, B. L. & He, S. Y. Growth–defense tradeoffs in plants: a balancing act to optimize fitness. *Mol. Plant* **7**, 1267–1287 (2014).
125. Ouédraogo, D.-Y., Mortier, F., Gourlet-Fleury, S., Freycon, V. & Picard, N. Slow-growing species cope best with drought: evidence from long-term measurements in a tropical semi-deciduous moist forest of Central Africa. *J. Ecol.* **101**, 1459–1470 (2013).
126. de la Mata, R., Hood, S. & Sala, A. Insect outbreak shifts the direction of selection from fast to slow growth rates in the long-lived conifer *Pinus ponderosa*. *Proc. Natl Acad. Sci. USA* **114**, 7391–7396 (2017).
127. Roskilly, B., Keeling, E., Hood, S., Giugliola, A. & Sala, A. Conflicting functional effects of xylem pit structure relate to the growth-longevity trade-off in a conifer species. *Proc. Natl Acad. Sci. USA* **116**, 15282–15287 (2019).
128. Snyder, K. A. & Williams, D. G. Defoliation alters water uptake by deep and shallow roots of *Prosopis velutina* (Velvet Mesquite). *Funct. Ecol.* **17**, 363–374 (2003).
129. Eyles, A., Pinkard, E. A. & Mohammed, C. Shifts in biomass and resource allocation patterns following defoliation in *Eucalyptus globulus* growing with varying water and nutrient supplies. *Tree Physiol.* **29**, 753–764 (2009).
130. Hillbrand, R. M., Hacke, U. G. & Liefers, V. J. Defoliation constrains xylem and phloem functionality. *Tree Physiol.* **39**, 1099–1108 (2019).
131. Landhäuser, S. M. & Liefers, V. J. Defoliation increases risk of carbon starvation in root systems of mature aspen. *Trees* **26**, 653–661 (2012).
132. Poyatos, R., Aguadé, D., Galiano, L., Mencuccini, M. & Martínez-Vilalta, J. Drought-induced defoliation and long periods of near-zero gas exchange play a key role in accentuating metabolic decline of Scots pine. *New Phytol.* **200**, 388–401 (2013).
133. Cardoso, A. A., Batz, T. A. & McAdam, S. A. Xylem embolism resistance determines leaf mortality during drought in *Persea americana*. *Plant Physiol.* **182**, 547–554 (2020).
134. Mencuccini, M. et al. Leaf economics and plant hydraulics drive leaf:wood area ratios. *New Phytol.* **224**, 1544–1556 (2019).
135. Cochard, H., Pimont, F., Ruffault, J. & Martin-St Paul, N. SurEau: a mechanistic model of plant water relations under extreme drought. *Ann. Forest Sci.* **78**, 1–23 (2021).
136. Yin, M. C. & Blaxter, J. H. S. Temperature, salinity tolerance, and buoyancy during early development and starvation of Clyde and North Sea herring, cod, and flounder larvae. *J. Exp. Mar. Biol. Ecol.* **107**, 279–290 (1987).
137. Cahill, G. F. Jr. Fuel metabolism in starvation. *Annu. Rev. Nutr.* **26**, 1–22 (2006).
138. Yandi, I. & Altinok, I. Irreversible starvation using RNA/DNA on lab-grown larval anchovy, *Engraulis encrasicolus*, and evaluating starvation in the field-caught larval cohort. *Fish. Res.* **201**, 32–37 (2018).
139. Smith, A. M. & Stitt, M. Coordination of carbon supply and plant growth. *Plant Cell Environ.* **30**, 1126–1149 (2007).
140. Schädel, C., Richter, A., Blöchl, A. & Hoch, G. Hemicellulose concentration and composition in plant cell walls under extreme carbon source–sink imbalances. *Physiol. Plant.* **139**, 241–255 (2010).
141. Tsamir-Rimon, M. et al. Rapid starch degradation in the wood of olive trees under heat and drought is permitted by three stress-specific beta amylases. *New Phytol.* **229**, 1398–1414 (2020).
142. McLoughlin, F. et al. Autophagy plays prominent roles in amino acid, nucleotide, and carbohydrate metabolism during fixed-carbon starvation in maize. *Plant Cell* **32**, 2699–2724 (2020).
143. Quirk, J., McDowell, N. G., Leake, J. R., Hudson, P. J. & Beerling, D. J. Increased susceptibility to drought-induced mortality in *Sequoia sempervirens* (Cupressaceae) trees under Cenozoic atmospheric carbon dioxide starvation. *Am. J. Bot.* **100**, 582–591 (2013).
144. Sevanto, S., McDowell, N. G., Dickman, L. T., Pangle, R. & Pockman, W. T. How do trees die? A test of the hydraulic failure and carbon starvation hypotheses. *Plant Cell Environ.* **37**, 153–161 (2014).
145. Tomasella, M., Petrucci, E., Petruzzelli, F., Nardini, A. & Casolo, V. The possible role of non-structural carbohydrates in the regulation of tree hydraulics. *Int. J. Mol. Sci.* **21**, 144 (2020).
146. Gaylord, M. L. et al. Drought predisposes piñon–juniper woodlands to insect attacks and mortality. *New Phytol.* **198**, 567–578 (2013).
147. Dickman, L. T., McDowell, N. G., Sevanto, S., Pangle, R. E. & Pockman, W. T. Carbohydrate dynamics and mortality in a piñon–juniper woodland under three future precipitation scenarios. *Plant Cell Environ.* **38**, 729–739 (2015).
148. Ruehr, N. K. et al. Drought effects on allocation of recent carbon: from beech leaves to soil CO<sub>2</sub> efflux. *New Phytol.* **184**, 950–961 (2009).
149. Mencuccini, M., Minunno, F., Salmon, Y., Martínez-Vilalta, J. & Hölttä, T. Coordination of physiological traits involved in drought-induced mortality of woody plants. *New Phytol.* **208**, 396–409 (2015).
150. Hagedorn, F. et al. Recovery of trees from drought depends on belowground sink control. *Nat. Plants* **2**, 16111 (2016).
151. Hesse, B. D., Goisser, M., Hartmann, H. & Grams, T. E. E. Repeated summer drought delays sugar export from the leaf and impairs phloem transport in mature beech. *Tree Physiol.* **39**, 192–200 (2019).
152. Wiley, E., Hoch, G. & Landhäuser, S. M. Dying piece by piece: carbohydrate dynamics in aspen (*Populus tremuloides*) seedlings under severe carbon stress. *J. Exp. Bot.* **68**, 5221–5232 (2017).
153. Weber, R. et al. Living on next to nothing: tree seedlings can survive weeks with very low carbohydrate concentrations. *New Phytol.* **218**, 107–118 (2018).
154. Hasanuzzaman, M. & Tanveer, M. (eds) *Salt and Drought Stress Tolerance in Plants: Signaling Networks and Adaptive Mechanisms* (Springer, 2020).
155. O'Brien, M. J., Leuzinger, S., Philipson, C. D., Tay, J. & Hector, A. Drought survival of tropical tree seedlings enhanced by non-structural carbohydrate levels. *Nat. Clim. Chang.* **4**, 710–714 (2014).
156. Nardini, A. et al. Rooting depth, water relations and non-structural carbohydrate dynamics in three woody angiosperms differentially affected by an extreme summer drought. *Plant Cell Environ.* **39**, 618–627 (2016).
157. Zinselmeyer, C., Westgate, M. E., Schussler, J. R. & Jones, R. J. Low water potential disrupts carbohydrate metabolism in maize (*Zea mays* L.) ovaries. *Plant Physiol.* **107**, 385–391 (1995).
158. Desprez-Loustau, M.-L., Marçais, B., Nageleisen, L.-M., Piou, D. & Vannini, A. Interactive effects of drought and pathogens in forest trees. *Ann. For. Sci.* **63**, 597–612 (2006).
159. Oliva, J., Stenlid, J. & Martínez-Vilalta, J. The effect of fungal pathogens on the water and carbon economy of trees: implications for drought-induced mortality. *New Phytol.* **203**, 1028–1035 (2014).
160. Kolb, T. et al. Drought-mediated changes in tree physiological processes weaken tree defenses to bark beetle attack. *J. Chem. Ecol.* **45**, 888–900 (2019).
161. Croize, L., Lieutier, F., Cochard, H. & Dreyer, E. Effects of drought stress and high density stem inoculations with *Leptographium windfieldii* on hydraulic properties of young Scots pine trees. *Tree Physiol.* **21**, 427–436 (2001).
162. Wullschlegel, S. D., McLaughlin, S. B. & Ayres, M. P. High-resolution analysis of stem increment and sap flow for loblolly pine trees attacked by southern pine beetle. *Can. J. For. Res.* **34**, 387–393 (2004).
163. Hubbard, R. M., Rhoades, C. C., Elder, K. & Negron, J. Changes in transpiration and foliage growth in lodgepole pine trees following mountain pine beetle attack and mechanical girdling. *For. Ecol. Manag.* **289**, 312–317 (2013).
164. Manter, D. K. & Kavanagh, K. L. Stomatal regulation in Douglas fir following a fungal-mediated chronic reduction in leaf area. *Trees* **17**, 485–491 (2003).
165. Lahr, E. L. & Sala, A. Sapwood stored resources decline in whitebark and lodgepole pines attacked by mountain pine beetles (Coleoptera: Curculionidae). *Environ. Entomol.* **45**, 1463–1475 (2016).
166. Marler, T. E. & Cascasan, A. N. Carbohydrate depletion during lethal infestation of *Aulacaspis yasumatsui* on *Cycas revoluta*. *Int. J. Plant Sci.* **179**, 497–504 (2018).
167. Hood, S. & Sala, A. Ponderosa pine resin defenses and growth: metrics matter. *Tree Physiol.* **35**, 1223–1235 (2015).
168. Roth, M., Hussain, A., Cale, J. A. & Erbilgin, N. Successful colonization of lodgepole pine trees by mountain pine beetle increased monoterpenes production and exhausted carbohydrate reserves. *J. Chem. Ecol.* **44**, 209–214 (2018).
169. Raffa, K. F. et al. Cross-scale drivers of natural disturbances prone to anthropogenic amplification: the dynamics of bark beetle eruptions. *Bioscience* **58**, 501–517 (2008).
170. Seidl, R., Schelhaas, M. J., Rammer, W. & Verkerk, P. J. Increasing forest disturbances in Europe and their impact on carbon storage. *Nat. Clim. Chang.* **4**, 806–810 (2014).
171. Ryan, M. G., Sapes, G., Sala, A. & Hood, S. M. Tree physiology and bark beetles. *New Phytol.* **205**, 955–957 (2015).
172. Huang, J. et al. Tree defence and bark beetles in a drying world: carbon partitioning, functioning and modelling. *New Phytol.* **225**, 26–36 (2020).
173. Goodman, D. W., Lusebrink, I., Landhäuser, S. M., Erbilgin, N. & Liefers, V. J. Variation in carbon availability, defense chemistry and susceptibility to fungal invasion along the stems of mature trees. *New Phytol.* **197**, 586–594 (2013).
174. Wiley, E., Rogers, B. J., Hodgkinson, R. & Landhäuser, S. M. Nonstructural carbohydrate dynamics of lodgepole pine dying from mountain pine beetle attack. *New Phytol.* **209**, 550–562 (2016).
175. Netherer, S. et al. Do water-limiting conditions predispose Norway spruce to bark beetle attack? *New Phytol.* **205**, 1128–1141 (2015).
176. Rissanen, K. et al. Drought effects on carbon allocation to resin defences and on resin dynamics in old-grown Scots pine. *Environ. Exp. Bot.* **185**, 104410 (2021).
177. Gershenzon, J. Metabolic costs of terpenoid accumulation in higher plants. *J. Chem. Ecol.* **20**, 1281–1328 (1994).
178. Navarro, L. et al. DELLAs control plant immune responses by modulating the balance of jasmonic acid and salicylic acid signaling. *Curr. Biol.* **1**, 650–655 (2008).
179. Fox, H. et al. Transcriptome analysis of *Pinus halepensis* under drought stress and during recovery. *Tree Physiol.* **38**, 423–441 (2018).
180. Caretto, S., Linsalata, V., Colella, G., Mita, G. & Lattanzio, V. Carbon fluxes between primary metabolism and phenolic pathway in plant tissues under stress. *Int. J. Mol. Sci.* **16**, 26378–26394 (2015).
181. Franceschi, V. R., Krokene, P., Christiansen, E. & Krekling, T. Anatomical and chemical defenses of conifer bark against bark beetles and other pests. *New Phytol.* **167**, 353–376 (2005).
182. Suárez-Vidal, E. et al. Drought stress modifies early effective resistance and induced chemical defenses of Aleppo pine against a chewing insect herbivore. *Environ. Exp. Bot.* **162**, 550–559 (2019).
183. Hood, S., Sala, A., Heyerdahl, E. K. & Boutin, M. Low-severity fire increases tree defense against bark beetle attacks. *Ecology* **96**, 1846–1855 (2015).
184. Zhao, S. & Erbilgin, N. Larger resin ducts are linked to the survival of lodgepole pine trees during mountain pine beetle outbreak. *Front. Plant Sci.* **10**, 1459 (2019).
185. Kichas, N. E., Hood, S. M., Pederson, G. T., Everett, R. G. & McWethy, D. B. Whitebark pine (*Pinus albicaulis*) growth and defense in response to mountain pine beetle outbreaks. *For. Ecol. Manag.* **457**, 117736 (2020).
186. Gaylord, M. L., Kolb, T. E. & McDowell, N. G. Mechanisms of piñon pine mortality after severe drought: a retrospective study of mature trees. *Tree Physiol.* **35**, 806–816 (2015).
187. Anderegg, W. et al. Tree mortality predicted from drought-induced vascular damage. *Nat. Geosci.* **8**, 367–371 (2015).
188. De Kauwe, M. G. et al. Identifying areas at risk of drought-induced tree mortality across South-Eastern Australia. *Glob. Change Biol.* **26**, 5716–5733 (2020).
189. Sperry, J. S. et al. The impact of rising CO<sub>2</sub> and acclimation on the response of US forests to global warming. *Proc. Natl Acad. Sci. USA* **116**, 25734–25744 (2019).

190. Medlyn, B. E. et al. Stomatal conductance of forest species after long-term exposure to elevated CO<sub>2</sub> concentration: a synthesis. *New Phytol.* **149**, 247–264 (2001).
191. Klein, T. & Ramon, U. Stomatal sensitivity to CO<sub>2</sub> diverges between angiosperm and gymnosperm tree species. *Funct. Ecol.* **33**, 1411–1424 (2019).
192. Paudel, I. et al. Elevated CO<sub>2</sub> compensates for drought effects in lemon saplings via stomatal downregulation, increased soil moisture, and increased wood carbon storage. *Environ. Exp. Bot.* **148**, 117–127 (2018).
193. Bobich, E. G., Barron-Gafford, G. A., Rascher, K. G. & Murthy, R. Effects of drought and changes in vapour pressure deficit on water relations of *Populus deltoides* growing in ambient and elevated CO<sub>2</sub>. *Tree Physiol.* **30**, 866–875 (2010).
194. Gimeno, T. E., McVicar, T. R., O'Grady, A. P., Tissue, D. T. & Ellsworth, D. S. Elevated CO<sub>2</sub> did not affect the hydrological balance of a mature native *Eucalyptus* woodland. *Glob. Change Biol.* **24**, 3010–3024 (2018).
195. Nowak, R. S. et al. Elevated atmospheric CO<sub>2</sub> does not conserve soil water in the mojave desert. *Ecology* **85**, 93–99 (2004).
196. Schäfer, K. V., Oren, R., Lai, C. T. & Katul, G. G. Hydrologic balance in an intact temperate forest ecosystem under ambient and elevated atmospheric CO<sub>2</sub> concentration. *Glob. Change Biol.* **8**, 895–911 (2002).
197. Novick, K. A., Katul, G. G., McCarthy, H. R. & Oren, R. Increased resin flow in mature pine trees growing under elevated CO<sub>2</sub> and moderate soil fertility. *Tree Physiol.* **32**, 752–763 (2012).
198. Li, X. M. et al. Temperature alters the response of hydraulic architecture to CO<sub>2</sub> in cotton plants (*Gossypium hirsutum*). *Environ. Exp. Bot.* **172**, 104004 (2020).
199. Li, W. et al. The sweet side of global change—dynamic responses of non-structural carbohydrates to drought, elevated CO<sub>2</sub> and nitrogen fertilization in tree species. *Tree Physiol.* **38**, 1706–1723 (2018).
200. Duan, H. et al. Elevated [CO<sub>2</sub>] does not ameliorate the negative effects of elevated temperature on drought-induced mortality in *Eucalyptus radiata* seedlings. *Plant Cell Environ.* **37**, 1598–1613 (2014).
201. Duan, H. et al. CO<sub>2</sub> and temperature effects on morphological and physiological traits affecting risk of drought-induced mortality. *Tree Physiol.* **38**, 1138–1151 (2018).
202. Zavala, J. A., Nabity, P. D. & DeLucia, E. H. An emerging understanding of mechanisms governing insect herbivory under elevated CO<sub>2</sub>. *Annu. Rev. Entomol.* **58**, 79–97 (2013).
203. Kazan, K. Plant-biotic interactions under elevated CO<sub>2</sub>: a molecular perspective. *Environ. Exp. Bot.* **153**, 249–261 (2018).
204. Gessler, A., Schaub, M. & McDowell, N. G. The role of nutrients in drought-induced tree mortality and recovery. *New Phytol.* **214**, 513–520 (2017).
205. Mackay, D. S. et al. Interdependence of chronic hydraulic dysfunction and canopy processes can improve integrated models of tree response to drought. *Water Resour. Res.* **51**, 6156–6176 (2015).
206. Mackay, D. S. et al. Conifers depend on established roots during drought: results from a coupled model of carbon allocation and hydraulics. *New Phytol.* **225**, 679–692 (2020).
207. Tai, X. et al. Plant hydraulic stress explained tree mortality and tree size explained beetle attack in a mixed conifer forest. *J. Geophys. Res. Biogeosci.* **124**, 3555–3568 (2019).
208. Sala, A., Piper, F. & Hoch, G. Physiological mechanisms of drought-induced tree mortality are far from being resolved. *New Phytol.* **186**, 274–281 (2010).
209. Limousin, J. M. et al. Regulation and acclimation of leaf gas exchange in a piñon–juniper woodland exposed to three different precipitation regimes. *Plant Cell Environ.* **36**, 1812–1825 (2013).
210. Sorek, Y. et al. An increase in xylem embolism resistance of grapevine leaves during the growing season is coordinated with stomatal regulation, turgor loss point and intervessel pit membranes. *New Phytol.* **229**, 1955–1969 (2021).
211. Hudson, P. J. et al. Impacts of long-term precipitation manipulation on hydraulic architecture and xylem anatomy of piñon and juniper in Southwest USA. *Plant Cell Environ.* **41**, 421–435 (2018).
212. Warren, J. M., Norby, R. J. & Wullschlegel, S. D. Elevated CO<sub>2</sub> enhances leaf senescence during extreme drought in a temperate forest. *Tree Physiol.* **31**, 117–130 (2011).
213. Matusick, G. et al. Chronic historical drought legacy exacerbates tree mortality and crown dieback during acute heatwave-compounded drought. *Environ. Res. Lett.* **13**, 095002 (2018).
214. Shirley, H. L. Lethal high temperatures for conifers, and the cooling effect of transpiration. *J. Agric. Res.* **53**, 239–258 (1936).
215. Fisher, R. A. & Koven, C. D. Perspectives on the future of land surface models and the challenges of representing complex terrestrial systems. *J. Adv. Model. Earth Syst.* **12**, e2018MS001453 (2020).
216. Menzel, A., Sparks, T. H., Estrella, N. & Roy, D. B. Altered geographic and temporal variability in phenology in response to climate change. *Glob. Ecol. Biogeogr.* **15**, 498–504 (2006).
217. Keenan, T. F. et al. Net carbon uptake has increased through warming-induced changes in temperate forest phenology. *Nat. Clim. Chang.* **4**, 598–604 (2014).
218. Nakamura, T. et al. Tree hazards compounded by successive climate extremes after masting in a small endemic tree, *Distylium lepidotum*, on subtropical islands in Japan. *Glob. Change Biol.* **27**, 5094–5108 (2021).
219. Hummel, I. et al. Arabidopsis plants acclimate to water deficit at low cost through changes of carbon usage: an integrated perspective using growth, metabolite, enzyme, and gene expression analysis. *Plant Physiol.* **154**, 357–372 (2010).
220. Jamieson, M. A., Trowbridge, A. M., Raffa, K. F. & Lindroth, R. L. Consequences of climate warming and altered precipitation patterns for plant–insect and multitrophic interactions. *Plant Physiol.* **160**, 1719–1727 (2012).
221. Mithöfer, A. & Boland, W. Plant defense against herbivores: chemical aspects. *Annu. Rev. Plant Biol.* **63**, 431–450 (2012).
222. Netherer, S. et al. Interactions among Norway spruce, the bark beetle *Ips typographus* and its fungal symbionts in times of drought. *J. Pest Sci.* **94**, 591–614 (2021).
223. Love, D. M. et al. Dependence of aspen stands on a subsurface water subsidy: implications for climate change impacts. *Water Resour. Res.* **55**, 1833–1848 (2019).
224. McDowell, N. G. et al. Mechanisms of a coniferous woodland persistence under drought and heat. *Environ. Res. Lett.* **14**, 045014 (2019).
225. Rozendaal, D. M. et al. Competition influences tree growth, but not mortality, across environmental gradients in Amazonia and tropical Africa. *Ecology* **101**, e03052 (2020).
226. Friedlingstein, P. et al. Uncertainties in CMIP5 climate projections due to carbon cycle feedbacks. *J. Clim.* **27**, 511–526 (2014).
227. CH2018 Project Team. CH2018 — climate scenarios for Switzerland. NCCS <https://doi.org/10.18751/Climate/Scenarios/CH2018/1.0> (2018).
228. McMaster, G. S. & Wilhelm, W. W. Growing degree-days: one equation, two interpretations. *Agric. For. Meteorol.* **87**, 291–300 (1997).
229. McDowell, N. G. Mechanisms linking drought, hydraulics, carbon metabolism, and vegetation mortality. *Plant Physiol.* **155**, 1051–1059 (2011).

# Acknowledgements

The authors thank C. Körner for thoughtful advice, D. Basler for providing the CH2018 data on future climate projections for Switzerland and B. Roskilly, the Montgomery Laboratory and A. Castillo for feedback on figures. N.G.M. and C.X. were supported by the Department of Energy, Office of Science project Next Generation Ecosystem Experiment—Tropics (NGEE-Tropics). G.S. was supported by the NSFBI-Implementation (2021898). D.T.T. acknowledges support from the Australian Research Council (ARC) (DP0879531, DP110105102, LP0989881, LP140100232). M.G.D.K. acknowledges support from the ARC Centre of Excellence for Climate Extremes (CE170100023), the ARC Discovery Grant (DP190101823) and the NSW Research Attraction and Acceleration Program. C.G. was supported by the Swiss National Science Foundation (PZ00P3\_174068). M. Mencuccini and J.M.-V. were supported by the Spanish Ministry of Science and Innovation (MICINN, CGL2017-89149-C2-1-R). A.T.T. acknowledges funding from NSF grant 2003205, the USDA National Institute of Food and Agriculture, Agriculture and Food Research Initiative Competitive Grants Program no. 2018-67012-31496 and the University of California Laboratory Fees Research Program award no. LFR-20-652467. W.M.H. was supported by the NSF GRFP (1-746055). A.M.T. and H.D.A. were supported by the NSF Division of Integrative Organismal Systems, Integrative Ecological Physiology Program (IOS-1755345, IOS-1755346). H.D.A. also received support from the USDA National Institute of Food and Agriculture (NIFA), McIntire-Stennis Project WNP00009 and Agriculture and Food Research Initiative award 2021-67013-33716. D.D.B. was supported by NSF (DEB-1550756, DEB-1824796, DEB-1925837), USGS SW Climate Adaptation Science Center (G18AC00320), USDA NIFA McIntire-Stennis ARZT 1390130-M12-222 and a Murdoch University Distinguished Visiting Scholar award. D.S.M. was supported by NSF (IOS-1444571, IOS-1547796). R.S.O. acknowledges funding from NERC-FAPESP 19/07773-1. W.R.L.A. was supported by the David and Lucille Packard Foundation, NSF grants 1714972, 1802880 and 2003017, and USDA NIFA AFRI grant no. 2018-67019-27850. R.S.O. acknowledges funding from NERC-FAPESP 19/07773-1. B.E.M. is supported by an Australian Research Council Laureate Fellowship (FL190100003). A.S. was supported by a Bullard Fellowship (Harvard University) and the University of Montana.

# Author contributions

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.

# Competing interests

The authors declare no competing interests.

# Peer review information

*Nature Reviews Earth and Environment* thanks Amanda Cardoso, Teresa Gimeno and Atsushi Ishida, who co-reviewed with Shin-Taro Saiki, for their contribution to the peer review of this work.

# Publisher's note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

# Supplementary information

The online version contains supplementary material available at <https://doi.org/10.1038/s43017-022-00272-1>.

This is a US Government work and not under copyright protection in the US; foreign copyright protection may apply 2022