Review

Cell PRESS

The interdependence of mechanisms underlying climate-driven vegetation mortality

Nate G. McDowell[1], David J. Beerling[2], David D. Breshears[3], Rosie A. Fisher[4], Kenneth F. Raffa[5] and Mark Stitt[6]

[1] Los Alamos National Laboratory, Los Alamos, NM, USA
[2] Department of Animal and Plant Sciences, University of Sheffield, Sheffield, UK
[3] School of Natural Resources and the Environment, and Department of Ecology and Evolutionary Biology, University of Arizona, Tucson, AZ, USA
[4] National Center for Atmospheric Research, Boulder, CO, USA
[5] Department of Entomology, University of Wisconsin, Madison WI, USA
[6] Max Planck Institute for Molecular Plant Physiology, Potsdam, Germany

Climate-driven vegetation mortality is occurring globally and is predicted to increase in the near future. The expected climate feedbacks of regional-scale mortality events have intensified the need to improve the simple mortality algorithms used for future predictions, but uncertainty regarding mortality processes precludes mechanistic modeling. By integrating new evidence from a wide range of fields, we conclude that hydraulic function and carbohydrate and defense metabolism have numerous potential failure points, and that these processes are strongly interdependent, both with each other and with destructive pathogen and insect populations. Crucially, most of these mechanisms and their interdependencies are likely to become amplified under a warmer, drier climate. Here, we outline the observations and experiments needed to test this interdependence and to improve simulations of this emergent global phenomenon.

The need for an improved understanding of vegetation mortality

Vegetation mortality in association with a higher frequency and intensity of droughts and increased temperature has recently been documented on all six vegetated continents and for most biomes across the Earth [1] (Figure 1). When these observations are combined with forecasts of rising global temperature, declining regional precipitation and more extreme droughts [2], a scenario emerges in which many vegetation communities could be pushed past their mortality thresholds in coming decades [2–4]. This threat of increased vegetation mortality is already influencing governmental policies [2,5] because of both its implications for ecosystem services, and feedbacks between regional-scale mortality, carbon storage and climate [2,6–9].

The potential climatic feedback of globally distributed mortality events has increased the need to predict when, where and how vegetation will change in the future [2,7].

Glossary

Autonecrotic lesion: a defensive response in which the plant kills its tissue in advance of the invading organism, thereby confining it.
Capacitance: the change in water content per unit change in water potential. Functionally, xylem capacitance is the release of water into the transpiration stream via embolism, which enables transient maintenance of downstream water potential and stomatal conductance.
Carbon starvation: the process by which maintenance of cellular and defensive metabolism is not met owing to low carbohydrate supply from photosynthesis and available stores (see [16] for additional definitions). Carbon starvation results from the law of conservation of mass and energy at the individual level (mols carbon plant^{-1}): respiration [maintenance of live cells] + turgor preservation + defense = photosynthesis + available carbohydrate storage + autophagy - growth. Studies of carbon starvation-caused mortality have indirectly tested the hypothesis and support exists for components of the process.
Cavitation: xylem embolism formation by vaporization of water, occurring via air seeding from the porous connections between embolized and water-filled conduits.
DELLA proteins: a family of gibberellin-regulated proteins thought to act as transcriptional regulators in an 'anticipatory' mode that facilitates growth reductions during early stages of various stresses that are greater than expected owing to the direct effect of the stress itself. DELLA proteins are present in gymnosperms, monocots and dicots.
Dynamic Global Vegetation Models (DGVM): process models that simulate growth, death and reproduction of the major vegetation communities on Earth in relation to climate and environmental changes.
Embolism: blockage of xylem conduits by an air bubble.
Gibberellic acid: a plant hormone, more precisely termed gibberellin 3 of GA3, which promotes cell expansion and growth by controlling the stability of DELLA proteins.
Hydraulic and symplastic failure: cellular desiccation, cessation of symplastic biochemical and hydraulic function, and subsequent mortality after prolonged, whole-plant failure to transport water to the canopy. Hydraulic failure was associated with mortality in several cases; however, existing data do not exclude other interdependent mechanisms.
Mechanism (n): '1: a system of parts working together…; 2: a natural or established process by which something takes place or is brought about' [87].
Metabolic and phloem transport limitation: constraints on the ability of cells to use available carbohydrates, and the ability of phloem to transport carbohydrates. If these limitations occur, they could inhibit use of carbohydrates for survival and hasten carbon starvation. Indirect tests of these hypotheses exist.
Mortality: at the individual level, the irreversible cessation of maintenance metabolism and subsequent failure to regenerate.
Overwhelming biotic attack: occurs when biotic agent populations reach threshold levels at which the number of attacks exhausts plant defenses. Landscape- or regional-scale mortality ensues owing to the runaway feedback between attack success and reproduction. Tests linking attack to plant defense, carbon and water balance are needed.

Corresponding author: McDowell, N.G. (mcdowell@lanl.gov).

  doi:10.1016/j.tree.2011.06.003

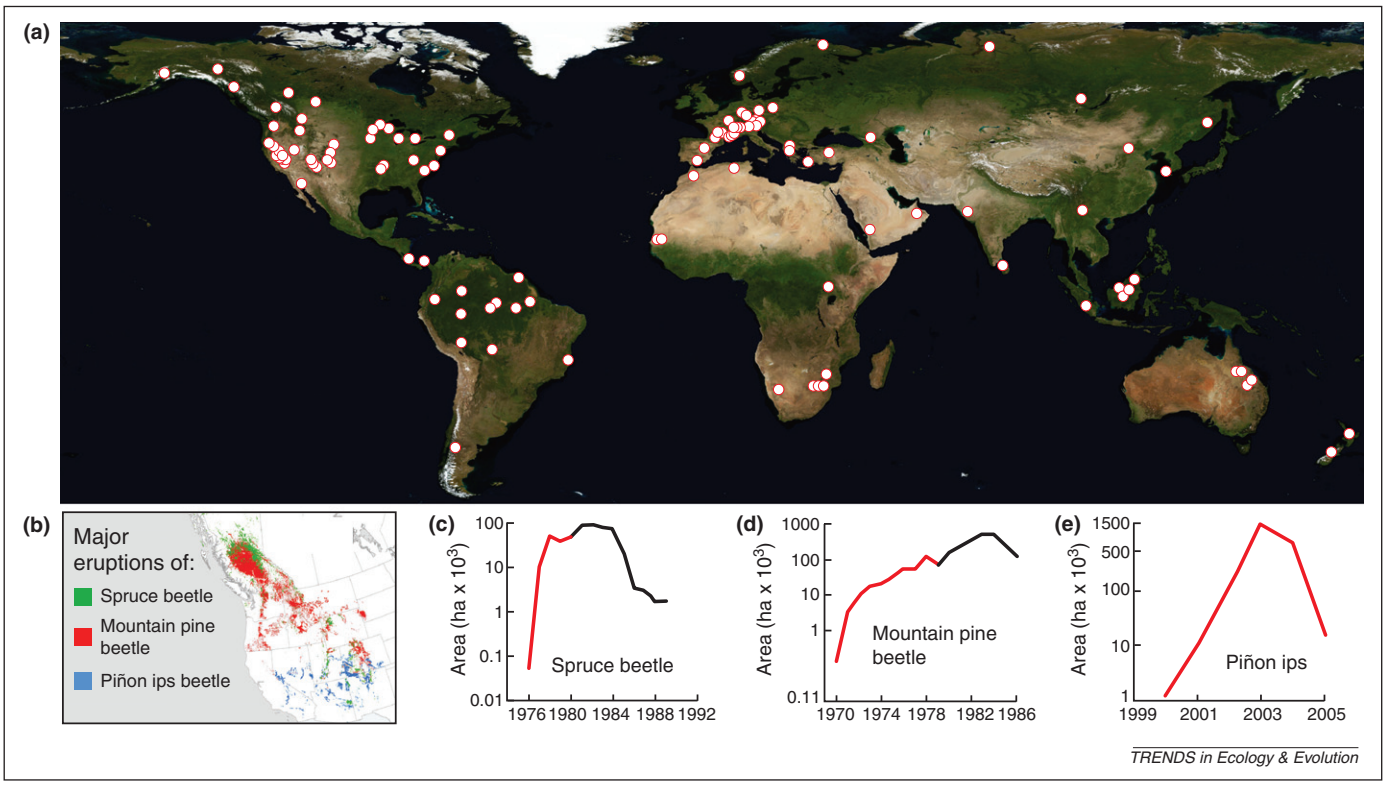

Figure 1. Global occurrence of vegetation mortality. **(a)** Dots show locations of documented mortality that have been associated with drought since 1970 [1]. **(b)** Regions of western North America where massive bark beetle outbreaks have resulted in widespread mortality since 1998 [30]. The annual area (hectares) of forest impacted by spruce beetle **(c)**, mountain pine beetle **(d)** and piñon ips **(e)** for the areas represented in **(b)**. The portion of the outbreak period that was significantly related to climate deviation from the 30-year mean is shown in red. The climate variable **(c–e)** is summer temperature anomaly, percentage of spring precipitation deviation, and percentage of annual precipitation deviation, respectively [30]. Although the onset of all three outbreaks can be attributed to temperature or precipitation anomalies, the extent to which the continuation of each outbreak relies on predisposing weather varies among insect species. This illustrates that integrative models of plant mortality must account for variation owing to insect and pathogen species, plant species, region and type of physiological stress.

Dynamic Global Vegetation Models (DGVMs; see Glossary and Box 1) are the key tool used to predict future vegetation dynamics and associated influences on atmospheric CO_2 and climate in major assessments such as that carried out by the International Panel for Climate Change [2,7]. Although results from DGVM all show that vegetation mortality will increase with future climate change, there is a large degree of uncertainty in predictions of vegetation dynamics [7,10–12] (Box 1, Figure I). This is partly because fundamental understanding of vegetation mortality remains speculative [13,14], preventing mechanistic algorithm development and parameterization of DGVMs. This forces modelers to use a variety of logical but untested algorithms to simulate mortality (Box 1).

Recent reviews that built upon the research legacy linking plant physiology with mortality have suggested that mortality results from photosynthetic and respiratory impacts on carbohydrate storage and hydraulic function [13,14] and the potential limitations of phloem transport and carbohydrate metabolism [15,16]. However, these reviews were not able to include new understanding of vegetation stress and mortality that has emerged from a range of disparate scientific fields. Integration across disciplines is needed to bring the full breadth of existing knowledge to bear on the question of mortality mechanisms and subsequent DGVM improvement. The current review differs from [13–16] in that it (i) integrates new evidence from a broader set of disciplines (i.e. molecular biology, paleobotany and entomology); (ii) considers potential mortality mechanisms as being interdependent rather than independent processes, in the hope of minimizing the creation of false dichotomies; iii) to maximize inferences regarding mortality mechanisms, we have cautiously included results on plant function during mortality or severe drought from relevant observations and experiments, regardless of studied taxa, (inherent variation in reproductive strategies and other life-history aspects might alter the rates or importance of processes proposed here); iv) specifically assesses sensitivities to warmer, drier climate; and (v) specifically enumerates current DGVM assumptions and relates them to insights into mortality mechanisms. We aim to clarify existing confusion regarding definitions and concepts of mortality mechanisms, determine consistency between theories and available results from molecular to global scales, and identify future research that could improve DGVMs.

Mechanistic insights from historical and contemporary mortality events

Fossil evidence indicates that rapid environmental changes have driven vegetation mortality events throughout the history of the Earth, and that physiological mechanisms have mediated many of these events [17]. During periods of low atmospheric CO_2, plants experienced minimum chloroplast CO_2 concentrations that promoted carbon starvation [18,19]. Widespread mortality is particularly evident during periods of rapidly increasing temperature [20]. Two of the largest floral extinction events, the Permian [ca 250 million

Box 1. Common mortality algorithms within dynamic global vegetation models.

The most commonly used, global-scale DGVMs use a wide array of mechanisms to kill vegetation in response to climate and competition. It is useful for models to use a diversity of mechanisms to avoid theoretical constraints from sharing common paradigms, but this also results in a range of mortality predictions (Figure I). Table I isummarizes some of the most common mortality algorithms used in global-scale simulations of the major vegetation communities on Earth, excluding mortality from fire, wind disturbance or harvest.

Table I. Plant mortality algorithms from a selection of the most commonly used DGVMs, listed approximately in order of progressive increase in mechanistic detail, with example models cited in the references

Mortality algorithms	Description
Productivity dependence	No explicit concept of mortality; plant biomass reduced via declining productivity [88]
Background rate	Mortality is set at a constant, invariant rate (approximately 1–2% yr^{-1}). This does not allow climate to drive variation in mortality [89–91]. In [12,92], background mortality increases as wood density decreases relative to the community maximum
Climate tolerance	Death occurs if the 20-year average climate exceeds predefined monthly climatic tolerances [93–96]
Size threshold	Death occurs if trunk diameter > 1.0 m [96].
Age threshold	Death increases as stand age approaches the plant functional type-specific maximum [84]
Heat stress threshold	Mortality is a function of the number of days per year in which the average temperature exceeds a threshold temperature, and the number of degrees (°C) by which this threshold is exceeded [84,92–97]
Negative productivity	Death occurs if annual net productivity < 0.0 g [93–96]
Shading/competition	Mortality increases as a function of canopy cover [12,92–97]
Growth efficiency threshold	Mortality occurs when biomass increment per unit leaf area falls below a quantitative threshold that varies between models [86,93–96,98]
Carbon starvation	Mortality is a function of carbohydrate storage per unit leaf biomass [12]

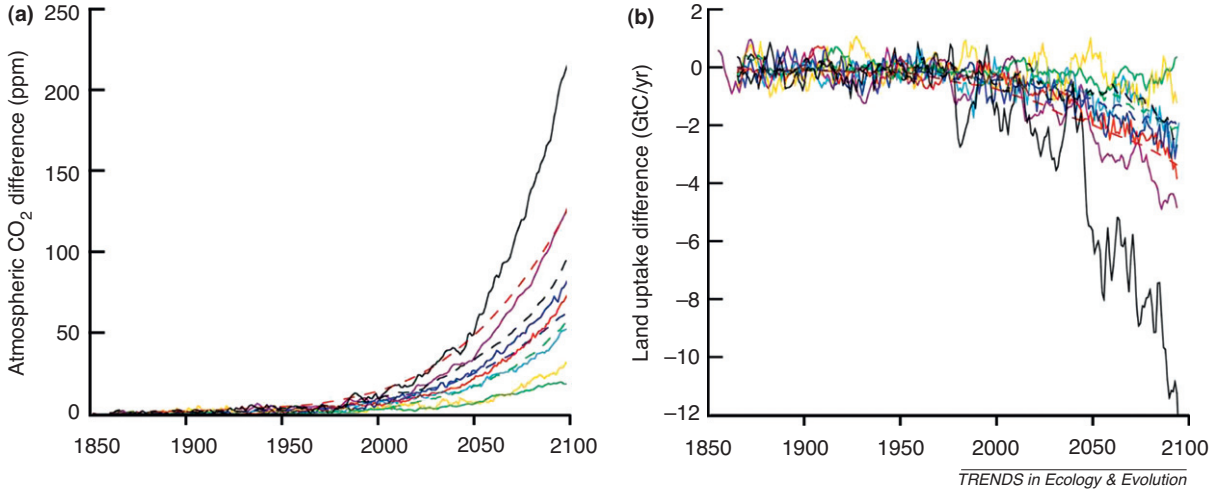

Figure I. Dynamic global vegetation models all simulate a net rise in future atmospheric CO_2 **(a)**, owing to a decline in land carbon uptake **(b)**. The models have wide divergence owing, in part, to the variety of mortality algorithms used [10–12]; see Table I for details on algorithms. Adapted from [10]).

years ago (My)] and the Triassic–Jurassic boundary (ca 200 My), were associated with rapid rises in temperature and imposition of semi-arid conditions, resulting in species-level, regional extinctions of up to 95% [21,22].

Recent observations include findings of increases in background mortality correlated with increased temperature [23], regional events associated with severe droughts [1,4,13,24–28] (Figure 1) and frequent association of mortality with attack by native or non-native insects and pathogens [1,28–30]. Drought-associated mortality has recently occurred not only in forests and woodlands, but also in chaparral, grassland and cacti species [13,25,31]. Plant physiology is a probable underlying driver because: (i) mortality sometimes occurs without evidence of biotic attack [1]; (ii) plants that die typically have lower growth, tissue density and canopy size than do those that survive [13,24,28]; and (iii) even biotic agents that attack apparently healthy trees during eruptive outbreaks usually require stressed trees for initial population growth [30,32] (Figure 1). The few experimental manipulations that have induced mortality indicate cause-and-effect relationships between mortality and reduced precipitation [33,34] and elevated temperature [35].

A proposed integration of mortality mechanisms

Various drought-related mortality mechanisms have been suggested. We propose that these mechanisms, rather than being mutually inclusive but independent processes, as previously posited [13], can be integrated as an interdependent set of processes (Box 2, Figure I). Prolonged stomatal closure eventually depletes carbohydrate stores owing to inhibition of photosynthesis as maintenance respiration continues (Box 2, Figure Ia,b). Carbohydrate availability for maintenance respiration is reduced further because carbohydrates are required to drive phloem transport, maintain turgor and refill embolized xylem during

Box 2. Hypothetical integration and feedbacks of mortality mechanisms.

There are multiple potential interactions between drought (low water availability and high temperature), plant hydraulics, carbohydrate and defense metabolism, and population dynamics of biotic agents (summarized in Figure I). Low water availability and high evaporative demand can induce partial hydraulic failure, which subsequently promotes carbon starvation by reducing: (1) stomatal conductance to CO_2; (2) metabolic repair owing to severely negative water potential, or (3) phloem transport. Likewise, carbon starvation promotes hydraulic failure if carbon is not available for (4) refilling of xylem; (5) maintenance of repair processes crucial to water transport (e.g. aquaporins, roots and foliage); and (6) turgor maintenance. Subsequently, both carbon starvation and hydraulic failure promote defense failure if: (7) carbon and water are not available for defense metabolism; and (8) water is not available for sap pressure. Defense failure feeds back to promote hydraulic failure and carbon starvation by: (9) facilitating population growth of attack agents, enabling pathogens to (10) impair host physiology, such as fungal blockage of xylem or foliage and root loss to herbivory.

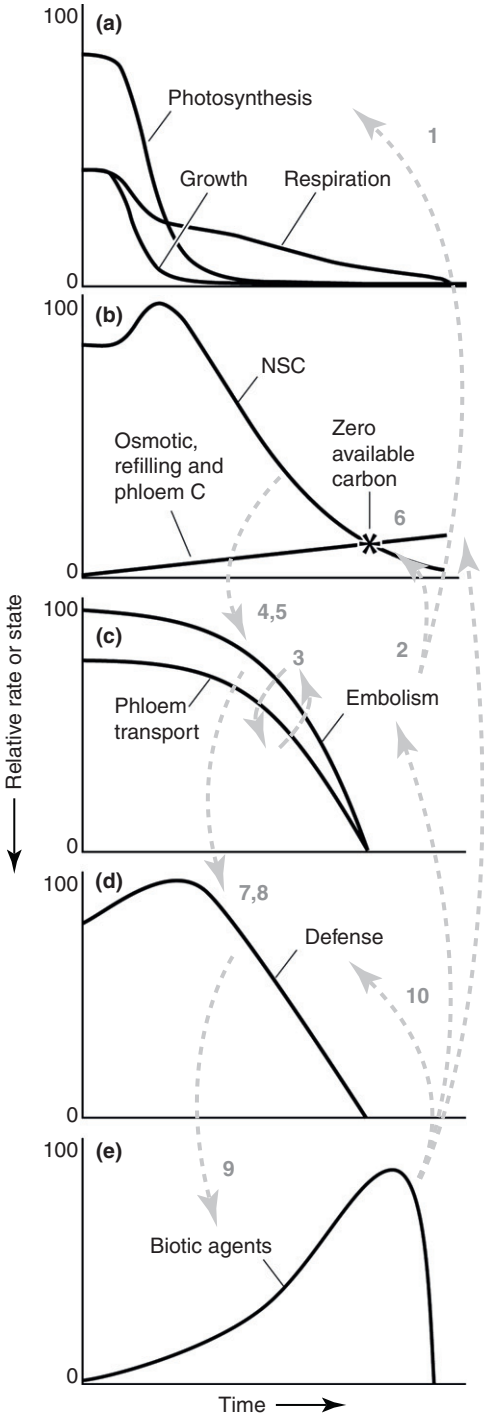

Figure I. An integrated hypothesis of vegetation mortality during drought. The solid lines indicate hypothesized trajectories of crucial parameters, and dashed lines indicate hypothesized, testable, coupling of parameters across the carbon–water–biotic agent continuum. Drought starts at time zero for all graphs. **(a)** Modeled patterns of photosynthesis, growth and respiration (see [14] for model details). **(b)** Non-structural carbohydrates (NSC) are simulated based on the carbon budget shown in **(a)**. Carbohydrates used for osmotic adjustment, maintenance of phloem transport and xylem refilling are assumed to increase linearly over time owing to a lack of data to parameterize the curve mechanistically. The intercept (*) represents the point at which there is no carbon available to maintain essential metabolism. **(c)** The proportion of embolized xylem conduits that are not refilled increases over time (based on representative curves from [59]), with an associated decline in phloem transport rates (simulated as a function of the embolism curve). **(d)** Predicted defense capacity against biotic attack, shown as a single curve encompassing both constitutive and induced responses (based on [63]). **(e)** Predicted patterns of the population growth of some biotic agents, such as bark beetles, as host plants become progressively more vulnerable and the reproduction of biotic agents increases, until the available host population is lost and the biotic agents return to original levels (based on [30]). All panels, including **(e)**, can manifest on both the individual plant to regional scales. The absolute values, rates and temporal dynamics are likely to vary with factors such as region, species and climate (i.e. Figure 1, main text).

drought (Box 2, Figure Ib,c,). Water and carbon limitation progressively increase the proportion of xylem embolism that is not refilled because soil water becomes unavailable and refilling is an energy (carbon)-requiring process (Box 2, Figure Ic). Feedbacks ensue, with reduced hydraulic conductance driving decreased photosynthesis and increased reliance on stored carbohydrates for turgor and refilling. Together, low carbohydrate and water availability reduce defensive capacity (Box 2, Figure Id,). Therefore, mortality could occur when one or more of these processes reach a threshold, and feedbacks across processes probably hasten the process. Reproduction of biotic agents can promote a positive feedback on population growth until the number of attacks exceeds the threshold at which even plants with strong defenses are killed (Box 2, Figure Ie). We next evaluate the key mechanisms underpinning this integrated theory.

The dependence of carbohydrate metabolism on drought

During the early phases of environmental stress, growth declines more than does photosynthesis, resulting in conservation, and even an increase, of carbohydrate reserves (Box 1, Figure Ia,b; reviewed in [14,36,37]). Novel molecular and genomics research in *Arabidopsis* and other model species provides useful regulatory information about carbon metabolism during stress and before mortality. During carbon-limited periods, signaling mechanisms triggered by low photosynthate and storage pools act to downregulate carbon partitioning to growth and respiration and upregulate carbon storage [38–41]. Specifically, carbon deficit leads to (i) rapid and widespread decreases in transcripts encoding enzymes involved in starch degradation, respiration and protein synthesis; (ii) increases in

transcripts encoding enzymes involved in starch synthesis and catabolism; and (iii) maintenance of enzymes involved in photosynthesis [38–41]. Maintenance respiration is prolonged through increased partitioning of carbohydrates to storage (e.g. in the form of starch), but is decreased by the downregulation of processes with high respiration costs, such as protein synthesis, turnover and growth [38–42]. A range of respiratory responses to drought have been documented in field-grown plants; thus, the net effect of respiratory down- and upregulation on carbohydrate consumption remains unclear [43,44]. Carbohydrate reserves decline only after a prolonged period of low carbon assimilation (Box 2, Figure Ia,b) [14]). Analogous responses in woody plants probably occur over longer time frames than in annuals (i.e. seasonal and interannual), owing to their larger carbohydrate storage [13,14,37,45–49].

Notably, observed transcriptional and post-translational regulatory changes respond to carbohydrate availability *per se*, indicating that carbohydrate availability is a resource crucial for survival [38–40,44]. These 'anticipatory' responses are facilitated by a signaling network in which DELLA proteins act to inhibit growth through interactions with the growth-promoting hormone gibberellic acid. Plants with mutated DELLA proteins show higher growth rates than do wild-type plants under stress conditions [50,51].

Despite the numerous regulatory adjustments to prolong survival during stress, the mass and energy balance still dictates how much starvation plants can tolerate. The starvation threshold is constrained further by multiple demands on stored carbohydrates for survival. For example, widespread turgor loss is fatal (in non-sprouting species); thus, increased reliance on carbohydrates and their metabolic products to maintain turgor and minimize oxidative stress and macromolecular destabilization within the symplast could preclude the use of some carbohydrates for respiration (Box 2, Figure Ia) [14,38,44,46,52]. When stored carbohydrates are exhausted, catabolism provides short-term energy sources (e.g. [53]), but serves only as a temporary bridge to more favorable conditions.

Interdependency between hydraulic failure and carbohydrate metabolism

Although drought impacts on photosynthesis are widely recognized (e.g. [54]), recent research reveals that the interdependence of hydraulics and carbon utilization and transport can impact survival more strongly than previously appreciated (e.g. [13]). The tolerance and regulation of xylem tension during drought exists as a continuum, from species that maintain relatively open stomata during drought and have associated adaptations to tolerate particularly negative xylem tensions (labeled 'anisohydric'), to species with vulnerable xylem that avoid highly negative xylem tensions through stomatal closure (labeled 'isohydric'). A common paradigm is that isohydric species are conservative during drought and avoid cavitation and conduit embolism, whereas anisohydric species take larger hydraulic risks [13]. However, new evidence shows cavitation occurs more routinely in isohydric species [55]. The threshold for complete stomatal closure in isohydric species frequently exceeds the point of embolism entry, whereas anisohydric species maintain a larger safety margin via xylem and rooting adaptations that enable higher stomatal conductance during drought while maintaining xylem tension above those that cause embolism entry [55]. Exceeding the embolism entry point provides greater daily capacitance [55,56] and potentially lowers the risk of xylem wall implosion as conduits fill with gas rather than experience more negative water tensions [56].

The strategy of using capacitance for transpiration requires nocturnal refilling of embolized conduits. Refilling can be limited during drought owing to drying soil, effectively isolating the plant from its ultimate water source. This constraint is exacerbated because refilling is an energetic process that consumes carbohydrates [57]. Thus, dry soil and carbohydrate limitation can both drive a feedback process whereby the proportion of non-refilled embolized conduits increases (Box 2, Figure Ic), photosynthesis declines further owing to reduced hydraulic conductivity (Box 2, Figure Ia) and carbohydrate transport to starving tissues is constrained by a reduced supply of water and photosynthate to the phloem column [58] (Box 2, Figure Ic). A negative spiral progresses until plants die from one or multiple causes, including outright starvation [14], desiccation [59] or biotic attack [29,32]. These interdependent feedback mechanisms might first occur in species that depend upon xylem cavitation as a transpiration supply mechanism, but should occur in all species if drought is sufficiently severe (i.e. [58]).

The interdependence of plant physiology and attack agents

There is evidence that biotic attack agents are sometimes involved in vegetation mortality, as shown through their abundance during regional mortality events [5,27–30] and increased plant survival via protection from attack by caging, insecticide, or anti-aggregant pheromone applications [60–62]. Not all species of insects, fungi and bacteria benefit from drought, but those that do include agents that cause the most extensive mortality [30]. Bark beetles (Curculionidae: Scolytinae), which are the major mortality agents in the northern hemisphere, are restricted to rare, highly stressed trees during environmentally benign periods because they lack sufficient numbers to conduct mass attacks that can overcome the defenses of vigorous trees [32,60]. The large growth in observations of 'destructive' insects and pathogens [29] and the recent doubling of background mortality in North America [23] might be associated with increased abundance of these stressed trees. Eruptive population growth (Box 2, Figure Ie) occurs when warm temperatures favor reproduction, host availability increases when environmental stress compromises plant defense (Box 2, Figure Id) across contiguous landscapes of mature plants, and populations of interacting agents, such as microbial symbionts, competitors and predators, are conducive for beetle reproduction [29,30]. Under these circumstances, population growth of the biotic agents can generate positive feedbacks through synchronized attacks that overwhelm the defenses of otherwise healthy trees, rendering host physiology less crucial to survival (Box 2, Figure Ie [32]).

Defensive responses to biotic agents include physical blockage via resin extrusion, confinement in autonecrotic

Table 1. Current advances and key questions regarding mortality mechanisms[a]

Advance	Refs	Critical questions
Carbon (C) starvation		**Threshold of functional carbohydrate availability?**
Minimized by pre-emptive C storage	[14,35–41]	What controls pre-drought C storage?
Available C limited by osmotic adjustment	[14,36–38,46,52]	How much C is required for osmotic maintenance?
Impacts phloem transport	[58]	How much C is required to maintain phloem transport?
Constrains defense response	[63–67]	How much C is required for defense? What is the source?
Exacerbated by warm temperature	[35,42–44,73]	How much can respiration acclimate to temperature?
Molecular control is becoming understood	[38–41,44,50,51]	What controls allocation to defense and metabolism?
Hydraulic failure		**Threshold of functional internal water availability?**
A significant role in isohydric species	[14,55,56,99]	Role in anisohydric species?
Lethal at 50–100% conductivity loss	[14,59]	What controls the lethal threshold?
Exacerbated by refilling limitations	[57]	How does refilling occur? How much C does it require?
Limits attack defense	[68]	How much is defense constrained by cavitation?
Biotic attack		**Threshold defense function relative to attack rate?**
Feedback process; strong threshold function	[30,32,75]	What are the tipping points of regional-scale attacks?
Final step in regional-scale mortality events	[30,75]	What controls the spatial patterns of biotic attack?
Native species moving into novel regions	[30,75,79]	What controls range shifts?
Non-native species bypass plant defenses	[29,77,78]	What controls non-native invasions?
Correlated with elevated temperature	[74,75,79]	Role of plant physiology verses biotic metabolism?
DGVM		**What critical mechanisms and feedbacks are necessary?**
Validation data sets/framework improving	[8,80,85,86]	What are the spatiotemporal and taxa mortality patterns?
C storage and dependence can be modeled	[12]	What is the C starvation threshold? Can defense be modeled?
Hydraulic failure can be modeled	[55,59,100]	What is the crucial level of failure leading to mortality?
Phloem transport can be modeled	[58]	What regulates phloem transport; variation with drought?
Age dependence can be simulated	[12]	When is mortality age dependent? Is competition important?
Biotic agent populations can be simulated	[75,79]	Can biotic population growth and range shifts be predicted?

[a]Includes cross-cutting questions; for example: will novel future climate expose new mechanisms, and what is the variation with taxa, plant size, and age?

lesions, and the production of toxic concentration of terpenoids and phenolics [63]. Allocation of photosynthate to expensive (high [C]) defensive compounds typically increases with mild stress [14,64] (Box 2, Figure Ic), owing in part to transcriptional responses that upregulate defense at the cost of growth [65]. Severe or prolonged drought eventually reduces defense [64,66] (Box 2, Figure Ic) owing to carbon starvation [67]. The final steps in biotic-driven mortality can be pathogenic toxicity, hydraulic failure associated with fungal occlusion of xylem [68] or destruction of resource-acquiring tissues, such as foliage or roots [69].

The integrated framework (Box 2, Figure I) provides a testable set of hypotheses regarding mortality mechanisms. We suggest that the evidence available from molecular to ecological levels is consistent with the following: (i) observations that tissue carbohydrates increase at the same time that growth, photosynthesis and respiration decline [16]; (ii) observations of carbon–water dependency of phloem transport and xylem refilling during drought [57,58] that could promote mortality, particularly of isohydric species; and (iii) observations of trade-offs between growth and defense [64,66].

Crucial challenges to understanding and predicting future mortality

This review identifies key questions that should be addressed to improve future mortality predictions (Table 1). In particular, understanding mortality thresholds and interdependencies of the functional metabolism of carbohydrates, water and defense (Box 2, Figure I) is essential to enable mechanistic yet simple algorithm development of DGVMs (Box 1, Table I; Figure 2) [7,10–12]. Although beyond the scope of this review, factors such as fire, wind destruction and competition also pose similar modeling challenges to DGVMs [70]. Here, we identify crucial uncertainties regarding the response of plant mortality to global changes, highlight the importance of a global mortality observation framework and more mechanistic experiments, and discuss crucial needs for DGVMs.

Predicted global changes are expected to exacerbate mortality rates

The interdependent mechanisms identified here are likely to be amplified by current and future changes in either temperature (both chronic and extreme) or precipitation (increasing frequency, severity, or duration of drought [71,72]; Figure 2b). The threat of mortality via changing temperature and drought is greatly magnified because these drivers are often spatiotemporally correlated, are forecast to increase both chronically and through greater extreme events across much of the Earth [2,71], and they accelerate mortality when they co-occur [1,4,24–28,35]. Thus, plants adapted to historic climates might be exposed to novel, extreme conditions that overwhelm their acclimatory responses (Figure 2b). For example, rising temperatures are likely to increase carbohydrate consumption owing to the temperature dependence of respiration (despite acclimation), particularly during extreme high-temperature events [13,14,34,42–44,54,73]. Extreme temperatures damage photosynthetic apparatus, reducing photosynthesis and increasing carbohydrate use for repair [13,38,43,44,46,73]. Temperature rise can increase insect population growth owing to reduced over-winter mortality,

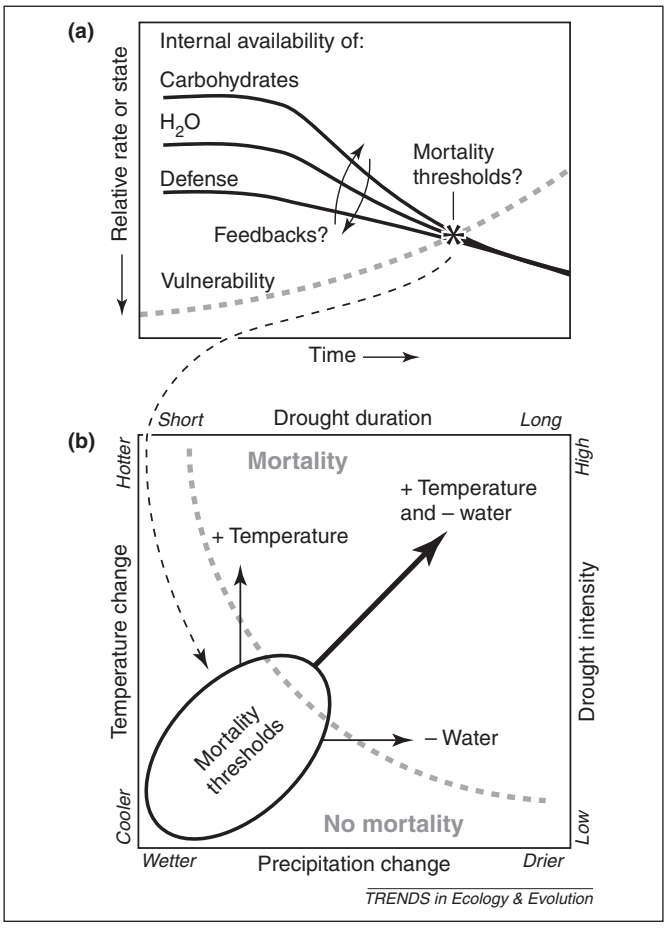

Figure 2. Hypothetical metabolic mortality thresholds that could be targets for DGVM parameterization. **(a)** Internal availability of carbohydrate, water and defense function are integrative, potentially testable parameters that each could have a role in failure as drought progresses. Vulnerability increases as the safety margin between available and zero functional carbohydrate, water and defense narrows, and as attack rates from biotic agents increase. Testing of the rates and interdependencies of critical thresholds is needed. **(b)** Future regional climates are likely to accelerate the rate of each process shown in **(a)** and interdependency shown in Figure I (Box 2), resulting in increased mortality for plants that fail to acclimate. Warming and reduced precipitation alone should accelerate mortality mechanisms in many regions, but are likely to have compounding effects when experienced simultaneously. Exceptions can exist for some plant–biotic agent interactions owing to feedback-driven changes in plant or insect pathogen physiology (see main text). Modified from [1].

decreased generation times, greater host vulnerability and access to vulnerable hosts following range expansion [30,74,75]. Decreased water availability will compound temperature effects, by increasing cavitation and reducing xylem refilling, photosynthesis and phloem transport (Box 2, Figure I). Rising temperature increases evaporative demand, forcing greater stomatal closure and higher ecosystem evaporation, thus accelerating progression of, and feedbacks between, mortality mechanisms [13] (Figure 2a,b). These mechanisms and feedbacks can vary with local climate owing to balancing or compounding influences of changing temperature and precipitation on plant physiology and biotic agent life cycles [28–30,74–76].

Additional challenges to DGVMs result from the unpredictable invasion of nonindigenous biotic agents [29]. Invasive biotic agents cause widespread mortality by responding to the same stressed hosts as native species, when hosts lack coevolved defenses, or when coevolved predators are absent [77,78]. Similar to rising temperature and declining precipitation, changing biotic attack populations can push plants closer to mortality thresholds (Figure 2). The mechanisms of dispersal and attack by indigenous and nonindigenous biota during changing climate are crucial research arenas [74–76,79].

Crucial future observations

Spatially and temporally comprehensive observations around the Earth are necessary to determine where, when, which taxa and under what climatic circumstances plant mortality occurs [1,26], to test the hypothesis posed in Figure 2b, and to inform the development and validation of DGVMs (Table 1; Box 1, Table I). Unfortunately, comprehensive observations do not yet exist (e.g. Figure 1 [1]). Current preferential sampling in regions of affluence might miss increases and decreases in mortality rates (Figure 1) [26]. Rapid technical advances in remote sensing of mortality and disturbance are occurring (e.g. [8,80]), but development is needed to improve spatial resolution and pattern interpretation for attributing causes to observed vegetation changes (e.g. fire, wind destruction, harvest, drought with and without biotic attacks) and for detecting spectral signatures of plant stress before mortality [81].

Crucial future experiments

Understanding mortality causes requires manipulative experiments to tease apart the interdependent roles of various mechanisms. Future experiments should adequately test each potential mortality mechanism (including, but not limited to, those in Box 2), to quantify interdependencies and exclude alternative hypotheses. Multifactor experiments are required to determine individual and combined impacts of environmental changes on mortality mechanisms, including realistic manipulations of CO_2, temperature, precipitation and exclusion or addition of biotic agents (e.g. [60–62,74,82,83]). Experiments must push vegetation to mortality [15]. Failure to achieve mortality can yield misleading information (e.g., carbohydrate and defensive allocation increases under moderate drought but declines under severe drought [14,64–67]; Box 2, Figure I).

Application of new tools could facilitate breakthroughs in understanding of vegetation mortality, including determination of which parameters and interdependencies are valuable targets for DGVM incorporation (Figure 2a). For example, carbohydrate augmentation to herbaceous species demonstrated that carbon starvation rather than low plant water potential *per se* causes tissue death (reviewed in [14]). Application of analogous approaches to woody plants could be revolutionary for testing carbon starvation. New techniques are required to continuously observe patterns and mechanisms of embolism and phloem transport *in situ*. Genomics and next-generation sequencing technologies are now sufficiently mature for them to be applied to woody species to generate and test hypotheses regarding metabolic adaptations and vulnerabilities. Results of these studies could be integrated with bioinformatics and modeling to reach mechanistic conclusions that feed back into DGVMs [82]. Better understanding of how novel future climates drive adaptations is particularly

crucial for models, because changes in communities caused by adaptation and selection to novel future climates are rarely accounted for in current DGVMs [12].

Crucial future modeling

Future DGVM efforts will benefit from direct testing against observational and experimental studies. The representation of mechanisms within the plant functional types typically used by DGVMs should be increased [84]; however, until the models can be tested against data, it is impossible to conclude what mechanisms and level of detail are needed. For example, carbon starvation was added to a DGVM with the premise that the more carbohydrates plants store relative to photosynthesis, the slower they will grow and the more likely they are to be outcompeted in stable environments [12]. The resulting dominance by faster growing trees that store less carbohydrate created a simulated ecosystem that was more prone to carbon starvation during drought. This hypothesis cannot be tested without data on plant carbohydrate storage, and requires better understanding of the processes that control the coexistence of fast- and slow-growing species. Terrestrial biogeochemistry models are now being tested against common datasets [85,86]; a similar approach to testing DGVMs will be valuable.

Concluding remarks

Here, we propose a synthetic theory of climate-driven vegetation mortality that is consistent with available evidence from molecular to global scales, and that provides a foundation for the development of more mechanistic experiments and DGVMs. Rather than considering mechanisms as mutually inclusive but independent (e.g. [13]), we interpret available data as indicating that mechanisms are highly interdependent. Drought-associated vegetation mortality is likely to occur: (i) via failure of the carbon–water interdependency for turgor maintenance, hydraulic conductance, phloem transport, or defense; (ii) across the iso–anisohydry continuum of plant species; and (iii) via strong feedbacks between biotic attack populations and host plant physiology. Many mortality mechanisms and interdependencies are likely to become amplified under a warmer, drier climate, supporting the growing concern that future mortality events are likely to be larger and more frequent. Many challenges remain, including understanding regional and taxon-based variation in mortality and associated mechanisms, and identifying mechanisms that are transferable to DGVMs. Further observations, experiments and modeling tests are needed so that DGVMs can be used to assess more confidently how the surface of the Earth will respond to projected climate change and to determine the extent of associated land surface–atmosphere feedbacks.

Acknowledgments

We appreciate the assistance of Craig Allen, Jeff Amthor, Rick Meinzer, numerous other colleagues and three excellent reviewers. Funding is acknowledged as follows: NGM, DOE Office of Science (BER), LANL-LDRD, Fulbright Scholar Program; DJB, Royal Society-Wolfson Research Merit Award; DDB, DOE NICCR (DE-FCO2-06ER64159), NSF (DEB-0443526, EAR-0724958), Biosphere 2 Philecology; RAF, DOE Office of Science (BER), LANL-LDRD; KFR, USDA NRI 2008-02438, NSF DEB-0816541, UW College of Agricultural and Life Sciences; MS, Max Planck Society.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.tree.2011.06.003.

References

1 Allen, C.D. *et al.* (2010) A global overview of drought and heat-induced tree mortality reveals emerging climate change risks for forests. *For. Ecol. Manage.* 259, 660–684
2 Allison, N.L. *et al.* (2009) *The Copenhagen Diagnosis, 2009: Updating the World on the Latest Climate Science*, The University of New South Wales Climate Change Research Centre
3 Malhi, Y. *et al.* (2009) Exploring the likelihood and mechanism of a climate-change-induced dieback of the Amazon rainforest. *Proc. Natl. Acad. Sci. U.S.A.* 106, 20610
4 Breshears, D.D. *et al.* (2005) Regional vegetation die-off in response to global-change-type drought. *Proc. Natl. Acad. Sci. U.S.A.* 102, 15144–15148
5 Kurz, W.A. *et al.* (2008) Risk of natural disturbances makes future contribution of Canada's forests to the global carbon cycle highly uncertain. *Proc. Natl. Acad. Sci. U.S.A.* 105, 1551–1555
6 Kurz, W.A. *et al.* (2008) Mountain pine beetle and forest carbon feedback to climate change. *Nature* 452, 987–990
7 Bonan, G.B. (2008) Forests and climate change: forcings, feedbacks, and the climate benefits of forests. *Science* 320, 1444–1449
8 Zhao, M. and Running, S.W. (2010) Drought-induced reduction in global terrestrial net primary production from 2000 through 2009. *Science* 329, 940–943
9 Huang, C. *et al.* (2010) Regional aboveground live carbon losses due to drought-induced tree dieback in piñon–juniper ecosystems. *Rem. Sens. Environ.* 114, 1471–1479
10 Friedlingstein, P. *et al.* (2006) Climate-carbon cycle feedback analysis, results from the C4MIP model intercomparison. *J. Clim.* 19, 3337–3353
11 Sitch, S. *et al.* (2008) Evaluation of the terrestrial carbon cycle, future plant geography and climate-carbon cycle feedbacks using five Dynamic Global Vegetation Models (DGVMs). *Global Change Biol.* 14, 1–25
12 Fisher, R. *et al.* (2010) Assessing uncertainties in a second-generation dynamic vegetation model due to ecological scale limitations. *New Phytologist.* 187, 666–681
13 McDowell, N.G. *et al.* (2008) Mechanisms of plant survival and mortality during drought: why do some plants survive while others succumb? *New Phytol.* 178, 719–739
14 McDowell, N.G. (2011) Mechanisms linking drought, hydraulics, carbon metabolism, and mortality. *Plant Phys.* 155, 1051–1059
15 McDowell, N.G. and Sevanto, S. (2010) The mechanisms of carbon starvation: how, when, or does it even occur at all? *New Phytol.* 186, 264–266
16 Sala, A. *et al.* (2010) Physiological mechanisms of drought-induced tree mortality are far from being resolved. *New Phytol.* 186, 274–281
17 Beerling, D.J. (2007) *The Emerald Planet. How Plants Changed Earth's History*, Oxford University Press
18 Ehleringer, J.R. and Cerling, T.E. (1995) Atmospheric CO_2 and the ratio of intercellular to ambient CO_2 concentrations in plants. *Tree Physiol.* 15, 105
19 Ward, J.K. *et al.* (2005) Carbon starvation in glacial trees recovered from La Brea tar pits. *Proc. Natl. Acad. Sci. U.S.A.* 102, 690–694
20 McElwain, J.C. *et al.* (2009) Fossil plant relative abundances indicate sudden loss of late Triassic biodiversity in East Greenland. *Science* 324, 1554–1556
21 McElwain, J.C. and Punyasena, S.W. (2007) Mass extinction events and the plant fossil record. *Trends Ecol. Evol.* 22, 548–557
22 Van de Schootbrugge, B. *et al.* (2009) Floral changes across the Triassic–Jurassic boundary linked to flood basalt volcanism. *Nat. Geosci.* 2, 589–594
23 van Mantgem, P.J. *et al.* (2009) Widespread increase of tree mortality rates in the western United States. *Science* 323, 521–524
24 Phillips, O.L. *et al.* (2009) Drought sensitivity of the Amazon Rainforest. *Science* 323, 1344–1347
25 Swetnam, T.W. and Betancourt, J.L. (1997) Mesoscale disturbance and ecological response to decadal climatic variability in the American Southwest. *J. Clim.* 11, 3128–3147

26 Jump, A.S. *et al.* (2009) The altitude-for-latitude disparity in the range retractions of woody species. *Trends Ecol. Evol.* 24, 694–701
27 Williams, A.P. *et al.* (2010) Forest response to increasing aridity and warmth in the southwestern United States. *Proc. Natl. Acad. Sci. U.S.A.* 107, 21289–21294
28 Carnicer, J. *et al.* (2011) Widespread crown condition decline, food web disruption, and amplified tree mortality with increased climate change-type drought. *Proc. Natl. Acad. Sci. U.S.A.* 108, 1474–1478
29 Aukema, J.E. *et al.* (2010) Historical accumulation of nonindigenous forest pests in the continental United States. *Bioscience* 60, 886–897
30 Raffa, K.F. *et al.* (2008) Cross-scale drivers of natural disturbances prone to anthropogenic amplification: the dynamics of bark beetle eruptions. *BioScience* 58, 501–517
31 Lajtha, K. *et al.* (1997) Ecophysiology of the saguaro cactus (*Carnegiea gigantea*) in the Saguaro National Monument: relationship to symptoms of decline. *J. Arid Environ.* 36, 579–590
32 Boone, C.K. *et al.* (2011) Efficacy of tree defense physiology varies with bark beetle population density: a basis for positive feedback in eruptive species. *Can. J. For. Res.* 41, 1174–1188
33 Nepstad, D.C. *et al.* (2007) Mortality of large trees and lianas following experimental drought in an Amazon forest. *Ecology* 88, 2259–2269
34 da Costa, A.C.L. *et al.* (2010) Effects of 7 years of experimental drought on the aboveground biomass storage of an eastern Amazonian rainforest. *New Phytologist.* 30, 579–591
35 Adams, H.D. *et al.* (2009) Temperature sensitivity of drought-induced tree mortality portends increased regional die-off under global-change-type drought. *Proc. Natl. Acad. Sci. U.S.A.* 106, 7063–7066
36 Muller, B. *et al.* (2011) 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
37 Tardieu, F. *et al.* (2011) Water deficit and growth. Coordinating processes without an orchestrator? *Curr. Opin. Plant Biol.* 14, 1–7
38 Hummel, I. *et al.* (2010) *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
39 Smith, A.M. and Stitt, M. (2007) Coordination of carbon supply and plant growth. *Plant Cell Environ.* 30, 1126–1149
40 Stitt, M. *et al.* (2010) Primary photosynthetic metabolism-more than the icing on the cake. *Plant J.* 61, 1067–1091
41 Gibon, Y. *et al.* (2009) Adjustment of growth, starch turnover, protein content and central metabolism to a decrease of the carbon supply when *Arabidopsis* is grown in very short photoperiods. *Plant Cell Environ.* 32, 859–874
42 Amthor, J.S. (1994) Plant respiratory responses to the environment and their effects on carbon balance. In *Plant–Environment Interactions* (Wilkinson, R.E., ed.), pp. 501–554, Marcel Dekker
43 Atkin, O.K. and Macherel, D. (2009) The crucial role of plant mitochondria in orchestrating drought tolerance. *Ann. Bot.* 103, 581–597
44 Pinheiro, C. and Chaves, M.M. (2010) Photosynthesis and drought: can we make metabolic connections from available data? *J. Exp. Bot.* 62, 869–882
45 Körner, C. (2003) Carbon limitation in trees. *J. Ecol.* 91, 4–17
46 Kozlowski, T.T. and Pallardy, S.G. (2002) Acclimation and adaptive responses of woody plants to environmental stresses. *Bot. Rev.* 68, 270–334
47 Hoch, G. *et al.* (2003) Non-structural carbon compounds in temperate forest trees. *Plant Cell Environ.* 26, 1067–1081
48 Würth, M.K.R. *et al.* (2005) Non-structural carbohydrate pools in a tropical forest. *Oecologia* 143, 11–24
49 Galiano, L. *et al.* (2011) Carbon reservers and canopy defoliation determine the recovery of Scots pine 4 yr after a drought episode. *New Phytol.* 190, 750–759
50 Harberd, N.P. *et al.* (2009) The angiosperm gibberellin-GID1-DELLA growth regulatory mechanism: how an 'inhibitor of an inhibitor' enables flexible response to fluctuating environments. *Plant Cell* 21, 1328–1339
51 Achard, P. *et al.* (2006) Integration of plant responses to environmentally activated phytohormonal signals. *Science* 311, 91–94
52 Bartels, D. and Sunkar, R. (2005) Drought and salt tolerance in plants. *Crit. Rev. Plant. Sci.* 24, 23–58
53 Lundgren-Rose, T. *et al.* (2006) Starvation-induced expression of autophagy-related genes in *Arabidopsis*. *Biol. Cell* 98, 53–67
54 Flexas, J. *et al.* (2006) Keeping a positive carbon balance under adverse conditions: responses of photosynthesis and respiration to water stress. *Physiol. Plant.* 127, 343–352
55 Meinzer, F.C. *et al.* (2009) Xylem hydraulic safety margins in woody plants: coordination of stomatal control of xylem tension with hydraulic capacitance. *Funct. Ecol.* 23, 922–930
56 Hölttä, T. *et al.* (2009) Capacitive effect of cavitation in xylem conduits: results from a dynamic model. *Plant Cell Environ.* 32, 10–21
57 Zwieniecki, M.A. and Holbrook, N.M. (2009) Confronting Maxwell's demon: biophysics of xylem embolism repair. *Trends Plant Sci.* 14, 530–534
58 Hölttä, T. *et al.* (2009) Linking phloem function to structure: analyses with a coupled xylem–phloem transport model. *J. Theor. Biol.* 259, 325–337
59 Brodribb, T.J. and Cochard, H. (2009) Hydraulic failure defines the recovery and point of death in water-stressed conifers. *Plant Physiol.* 149, 575–584
60 Raffa, K.F. and Berryman, A.A. (1983) The role of host plant resistance in the colonization behavior and ecology of bark beetles. *Ecol. Monogr.* 53, 27–49
61 Carroll, A.L. *et al.* (2006) Direct control: theory and practice. In *The Mountain Pine Beetle: A Synthesis of its Biology, Management and Impacts on Lodgepole Pine* (Safranyik, L. and Wilson, B., eds), pp. 155–172, Natural Resources Canada
62 Gillette, N.E. *et al.* (2009) Aerially applied verbenone-releasing laminated flakes protect *Pinus contorta* stands from attack by *Dendroctonus ponderosae* in California and Idaho. *For. Ecol. Manage.* 257, 1405–1412
63 Keeling, C.I. and Bohlmann, J. (2006) Genes, enzymes, and chemicals of terpenoids diversity in the constitutive and induced defense of conifers against insects and pathogens. *New Phytol.* 170, 657–675
64 Herms, D.A. and Mattson, W.J. (1992) The dilemma of plants: to grow or defend. *Q. Rev. Biol.* 67, 282–335
65 Navarro, L. *et al.* (2008) DELLAs control plant immune responses by modulating the balance and salicylic acid signaling. *Curr. Biol.* 1, 650–655
66 Bonello, P. *et al.* (2006) Nature and ecological implications of pathogen-induced systemic resistance in conifers: a novel hypothesis. *Physiol. Mol. Plant Pathol.* 68, 95–104
67 Guérard, N. *et al.* (2007) Do trees use reserve or newly assimilated carbon for their defense reactions?. A ^{13}C labeling approach with young Scots pines inoculated with a bark-beetle-associated fungus (*Ophiostoma brunneo ciliatum*). *Ann. For. Sci.* 64, 601–608
68 Fukuda, K. *et al.* (2007) Correlation between acoustic emission, water status and xylem embolism in pine wilt disease. *Tree Physiol.* 27, 969–976
69 Aukema, B.H. *et al.* (2010) Interactions between below- and above-ground herbivores drive a forest decline and gap-forming syndrome. *For. Ecol. Manage.* 259, 374–382
70 Seidl, R. *et al.* (2011) Modelling natural disturbances in forest ecosystems: a review. *Ecol. Model.* 222, 903–924
71 Huntington, T.G. (2006) Evidence for intensification of the global water cycle: review and synthesis. *J. Hydrol.* 319, 83–95
72 Sheffield, J. and Wood, E.F. (2008) Global trends and variability in soil moisture and drought characteristics, 1950-2000, from observation-driven simulations of the terrestrial hydrologic cycle. *J. Clim.* 21, 432–458
73 Atkin, O.K. *et al.* (2007) Respiration as a percentage of daily photosynthesis in whole plants is homeostatic at moderate, but not high, growth temperatures. *New Phytol.* 174, 367–380
74 Zvereva, E.L. and Kozlov, M.V. (2006) Consequences of simultaneous elevation of carbon dioxide and temperature for plant-herbivore interactions: a meta-analysis. *Global Change Biol.* 12, 27–41
75 Bentz, B.J. *et al.* (2010) Global climate change and bark beetles of the Western United States and Canada: direct and indirect effects. *Bioscience* 60, 602–613
76 Johnson, D.M. *et al.* (2010) Climatic warming disrupts recurrent Alpine insect outbreaks. *Proc. Natl. Acad. Sci.* 107, 20576–20581

77 Havill, N.P. *et al.* (2006) Mitochondrial DNA from hemlock woolly adelgid (Hemiptera: Adelgidae) suggests cryptic speciation and pinpoints the source of the introduction to eastern North America. *Ann. Entomol. Soc. Am.* 99, 195–203
78 Rebek, E.J. *et al.* (2008) Interspecific variation in resistance to emerald ash borer (Coleoptera: Buprestidae) among North American and Asian ash (*Fraxinus* spp.). *Environ. Entomol.* 37, 242–246
79 Régnière, J. *et al.* (2009) Predicting insect distributions under climate change from physiological responses: spruce budworm as an example. *Biol. Invasions* DOI: 10.1007/s10530-010-9918-1
80 Coops, N. *et al.* (2006) Assessment of Quickbird high spatial resolution imagery to detect red-attack damage due to mountain pine beetle infestation. *Remote Sens. Environ.* 103, 67–80
81 Scheffer, M. *et al.* (2009) Early-warning signals for critical transitions. *Nature* 461, 53–59
82 Calfapietra, C. *et al.* (2009) Challenges in elevated CO_2 experiments on forests. *Trends Plant Sci.* 15, 5–10
83 Wu, Z. *et al.* (2011) Responses of terrestrial ecosystems to temperature and precipitation change: a meta-analysis of experimental manipulation. *Glob. Change Biol.* 17, 927–942
84 Arora, V.K. and Boer, G.J. (2006) Simulating competition and coexistence between plant functional types in a dynamic vegetation model. *Earth Interact.* 10, 1–30
85 Stöckli, R. *et al.* (2008) Use of FLUXNET in the Community Land Model development. *J. Geophys. Res.* 113, DOI: 10.1029/2007JG000562 G01025
86 Randerson, J.T. *et al.* (2009) Systematic assessment of terrestrial biogeochemistry in coupled climate–carbon models. *Global Change Biol.* 15, 2462–2484
87 Anon (2009) *Oxford English Dictionary*, Oxford University Press
88 Cox P.M. (2001) Description of the 'TRIFFID' Dynamic Global Vegetation Model, Hadley Centre Technical Note No. 24
89 Thornton, P.E. *et al.* (2007) Influence of carbon–nitrogen cycle coupling on land model response to CO_2 fertilization and climate variability. *Global Biogeosci. Cycles* DOI: 10.1029/2006GB002868
90 Delbart, N. *et al.* (2010) Mortality as a key driver of the spatial distribution of aboveground biomass in Amazonian forests: results from a Dynamic Vegetation Model. *Biogeosci. Disc.* 7, 3095–3123
91 White, M.A. *et al.* (2000) Parameterization and sensitivity analysis of the BIOME-BGC terrestrial ecosystem model: net primary production controls. *Earth Interact.* 4, 1–82
92 Moorcroft, P.R. *et al.* (2001) A method for scaling vegetation dynamics: the ecosystem demography model (ED). *Ecol. Monogr.* 71, 557–586
93 Sitch, S. *et al.* (2003) Evaluation of ecosystem dynamics, plant geography and terrestrial carbon cycling in the LPJ dynamic global vegetation model. *Global Change Biol.* 9, 161–185
94 Levis, S. et al. (2004) *The Community Land Model's Dynamic Global Vegetation Model (CLM-DGVM): Technical Description and User's Guide,* NCAR Tech. Note TN-4591IA.
95 Krinner, G. *et al.* (2005) A dynamic global vegetation model for studies of the coupled atmosphere–biosphere system. *Global Biogeosci. Cycles* DOI: 10.1029.2003GB002199
96 Sato, H. *et al.* (2007) SEIB–DGVM: A new Dynamic Global Vegetation Model using a spatially explicit individual-based approach. *Ecol. Model.* 200, 279–307
97 Medvigy, D. *et al.* (2009) Mechanistic scaling of ecosystem function and dynamics in space and time: the Ecosystem Demography model version 2. *J. Geophys. Res. Biogeosci.* 114, (G1)
98 Woodward, F.I. and Lomas, M.R. (2004) Vegetation-dynamics-simulating responses to climate change. *Biol. Rev.* 79, 643–670
99 Pratt, R.B. *et al.* (2008) Plant hydraulics: new discoveries in the pipeline. *New Phytol.* 179, 590–593
100 Sperry, J.S. *et al.* (1998) Limitation of plant water use by rhizosphere and xylem conductance: results from a model. *Plant Cell Environ.* 21, 47–359