Letter

The effect of fungal pathogens on the water and carbon economy of trees: implications for drought-induced mortality

Introduction

Drought-induced forest mortality is emerging as a widespread phenomenon with potentially large implications for forest function and dynamics (Allen *et al.*, 2010; Anderegg *et al.*, 2012; Martínez-Vilalta *et al.*, 2012). Although the physiological mechanisms underlying tree mortality are still not completely understood, there is agreement that they involve the storage and transport systems of water and carbohydrates (McDowell *et al.*, 2008; Sala *et al.*, 2010; McDowell, 2011). The xylem of plants is susceptible to drought-induced embolism and severe water deficits may result in the complete loss of xylem hydraulic conductivity and cause tree mortality (hydraulic failure; cf. Tyree & Sperry, 1988; McDowell *et al.*, 2008; Choat *et al.*, 2012). Drought also has detrimental effects on the carbon (C) economy of plants, and it has been hypothesized that reduced assimilation due to stomatal closure may lead to a depletion of stored C reserves and, eventually, to tree death due to C starvation (Waring, 1987; Martínez-Vilalta *et al.*, 2002; Bréda *et al.*, 2006; McDowell *et al.*, 2008). However, only in recent studies has a direct link between reduced C reserves and tree mortality been established (Adams *et al.*, 2009, 2013; Galiano *et al.*, 2011; Hartmann *et al.*, 2013; Mitchell *et al.*, 2013; Quirk *et al.*, 2013; Sevanto *et al.*, 2014). Finally, phloem transport could also become impaired due to the inability of plants to maintain phloem turgor under extremely low xylem water potentials, limiting the local availability of carbohydrates for metabolic functions (Sala *et al.*, 2010; Sevanto *et al.*, 2014).

We postulate that tree mortality research has suffered from a false dichotomy of drought vs biotic attack (McDowell *et al.*, 2013). Pests and pathogens cause tree mortality and it is well known that drought may predispose forests to attacks by insects (Mattson & Haack, 1987; Gaylord *et al.*, 2013) and fungal pathogens (Desprez-Loustau *et al.*, 2006; La Porta *et al.*, 2008). The interaction between drought stress and the damage caused by forest pests and pathogens has been addressed in a recent meta-analysis (Jactel *et al.*, 2012), and the connection between the physiological status of the tree and disease development has motivated a number of reviews in the past (Schoeneweiss, 1975; Boyer, 1995). Biotic agents have also been included in theoretical models for drought induced mortality (Martínez-Vilalta *et al.*, 2002; McDowell *et al.*, 2008, 2011). However, previous reports have not fully acknowledged the diversity of trophic interactions that microorganisms establish with the host trees and how this diversity has direct consequences in terms of the physiological mechanisms leading to mortality. Tree mortality can result directly from a toxic effect from metabolites produced by pathogens, but pathogens can also disrupt the xylem and phloem of the infected hosts and affect their C economy through the consumption of C reserves and the induction of C-expensive defences. Here, we develop a new framework that brings together the effects of pathogens and drought on the water and C economy of trees, and explore the implications for the process of drought-induced mortality.

A new framework based on trophic interactions

We argue that predictions of drought-induced mortality under pathogen attack can be improved by taking into account the type of trophic interaction that the pathogen establishes with the host. Three main types of trophic interactions can be distinguished amongst tree pathogens: biotrophs, necrotrophs and vascular wilts (Deacon, 1997) (Fig. 1). In general terms, biotrophs drain C and nutrients from living cells – the host response is based on recognition followed by programmed cell death (Glazebrook, 2005). Necrotrophs instead interact with the host through the defence response and get C and nutrients from dead cells – the host response is based on C-based constitutive and induced responses from living cells surrounding the infection (Glazebrook, 2005). A third category includes vascular fungi that colonize the vascular system systemically, often aided by toxins (Yadeta & Thomma, 2013) – the host responses are based on blocking vertical and lateral spread in the xylem.

In this letter, we describe how each of these pathogen types interact with the water and C transport systems of trees, and by which mechanisms they may contribute to drought-induced mortality (Fig. 2). As a basis for our rationale, we use the mechanistic model of McDowell *et al.* (2011) to represent the mortality process of trees subjected to drought stress. We show that during drought pathogens may disrupt the C balance of trees through three non-exclusive processes: (1) by directly depleting non-structural carbohydrate (NSC) reserves, (2) by forcing consumption of NSC reserves by the host or (3) by increasing repair costs (Fig. 2a). Our model makes explicit predictions on the changes in photosynthesis, growth and respiration; as well as on the impacts on the NSC budget, phloem, and xylem transport during a drought episode leading to tree death. The amount of C allocated to defence and the impact on a biotic agent's biomass are also included. Tree death is represented as the point in which no C for sustaining the basic metabolism is available (i.e. *zero C available* point), regardless of the process leading to this point. Death occurs

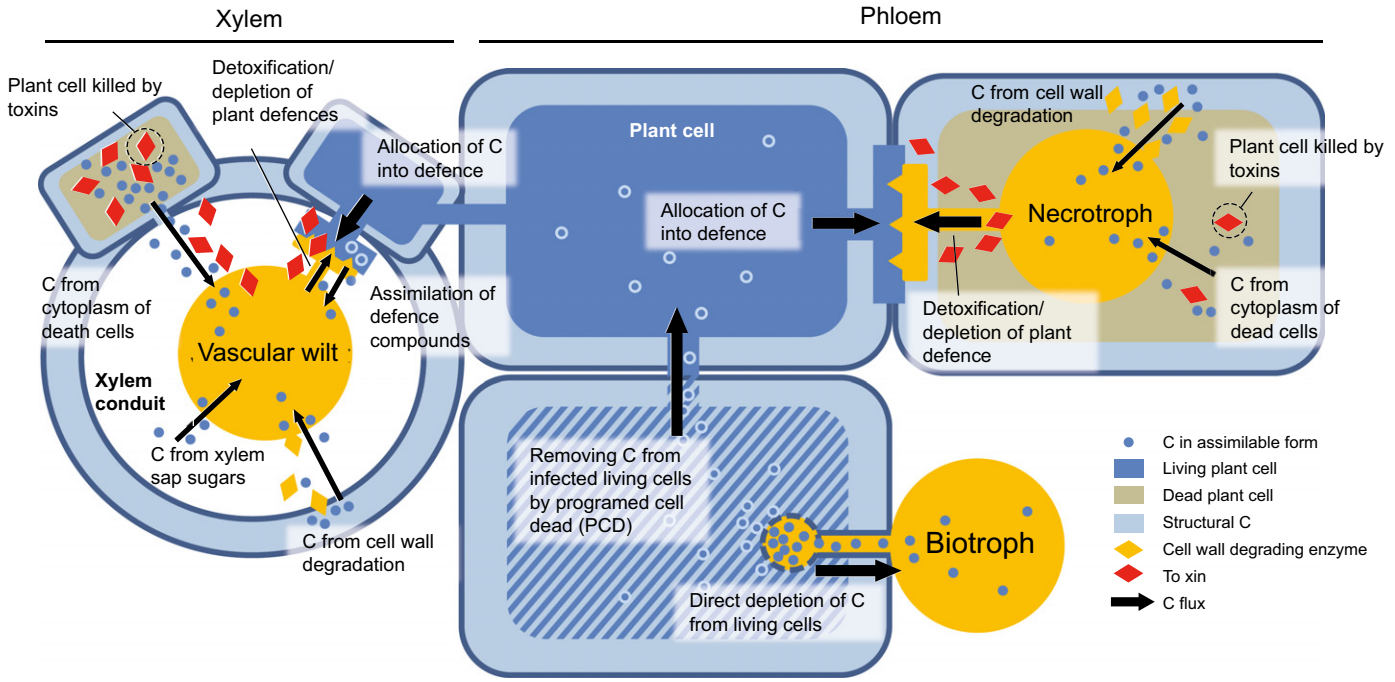
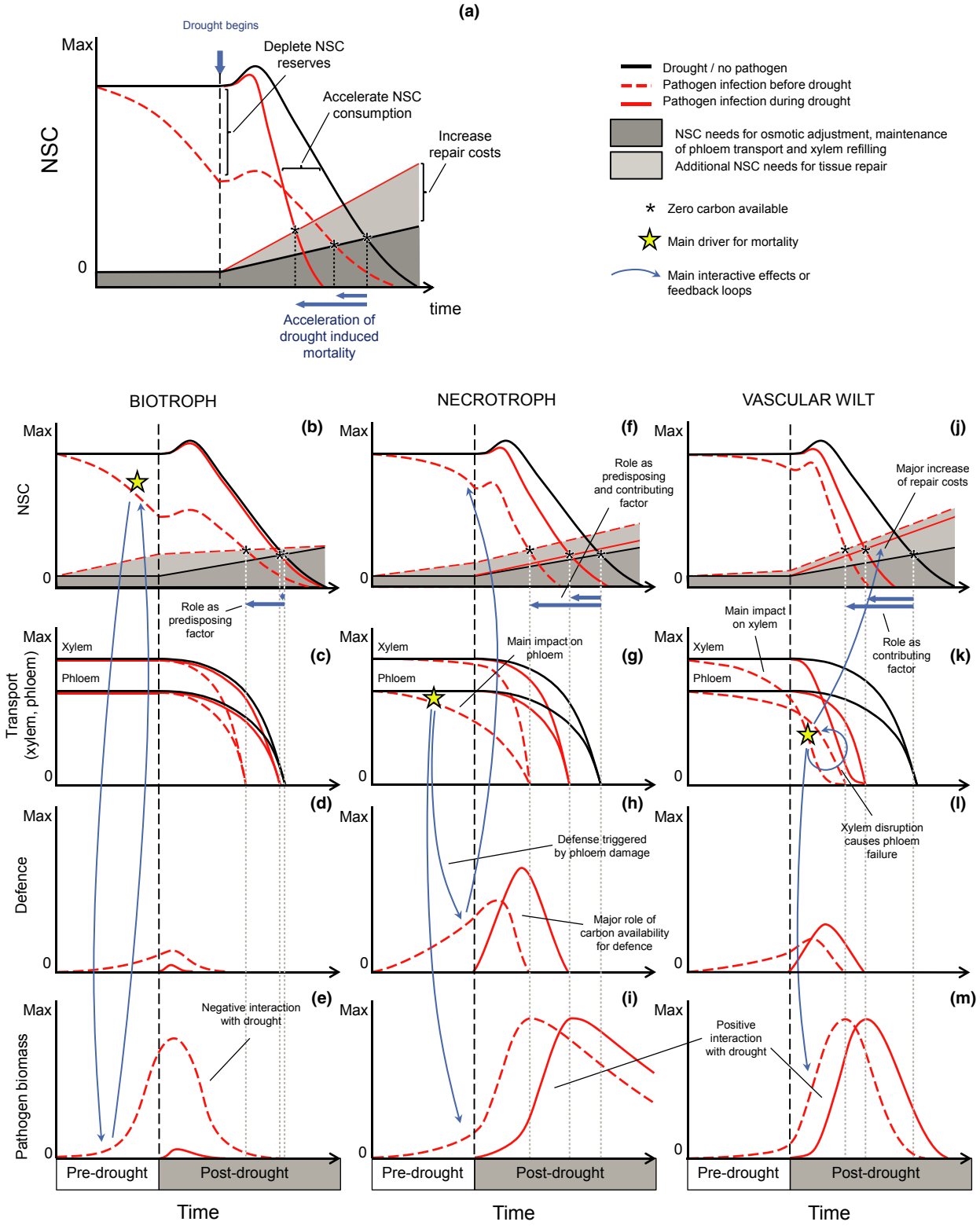


Fig. 1 Carbon (C) fluxes between host and pathogen depending on the type of trophic interaction occurring in xylem and phloem. Three types of pathogens are represented: biotrophs, necrotrophs and vascular wilts. Biotrophic pathogens derive C directly from living cells. Although chemical tree responses are triggered (not represented), the defence system of the tree is based on a programmed cell death (PCD) that removes the C from the infected cells and stops the flow of C to the pathogen. Vascular wilt pathogens thrive inside xylem conduits where they feed on C leaking from cells killed by means of toxins/enzymes and on C from xylem sap. They are also able to metabolize defence compounds and can obtain sugars by degrading the xylem cell wall. Damages in the xylem elicit C based defence responses. Necrotrophic pathogens kill living cells by secreting enzymes and toxins and obtain nutrients from dead cells and from structural C sources such as cellulose. Like vascular wilts, necrotrophs may also feed on tree defence compounds (not represented). Trees defend themselves by compartmentalizing the pathogen within C-expensive barriers.

when C available falls below (intersects) the amount of C needed for osmotic adjustment and maintenance of phloem and xylem transport. By considering different trophic interactions, two novel perspectives for current mortality models are put forward: (1) we show the fundamental differences among the *mechanisms* leading to tree mortality between biotrophs, necrotrophs and vascular wilt pathogens, and (2) we predict how different type of pathogens affect the timing of the *zero C available* point, and consequently whether they contribute or not to drought-induced mortality. We have considered the timing of the interaction between drought and pathogens in two ways. Either the pathogen acts simultaneously with

Fig. 2 Hypothetical mechanisms of mortality for biotrophic, necrotrophic and vascular wilt pathogens under drought conditions. Drought induced mortality is based on the model by McDowell *et al.* (2011) and it is assumed to happen when the amount of non-structural carbohydrate (NSC) is smaller than the amount of carbon (C) needed to maintain osmotic regulation and xylem and phloem transport (marked as an asterisk in panels a, b, f and j). Pathogens affect NSC reserves differently depending on the type of trophic interaction (Fig. 1), by directly depleting NSC reserves, accelerating NSC consumption and increasing repair costs (a). The mechanism of mortality is shown for each type of pathogen and separately depending on whether the attack occurs during drought (solid red line) or before the drought period (dashed red line). For the sake of comparison, the process of mortality under drought conditions without biotic agents is also shown (thick black line). In general, low water availability causes stomatal closure and lower C assimilation. Under prolonged drought conditions the tree may require using NSC reserves to maintain tissue growth and respiration. NSC may also be used to restore the hydraulic system of the tree by refilling embolized conduits. If under pathogen attack, and depending on the type of trophic interaction, trees elicit different responses impacting the acquisition, storage and transport of water and C in the tree. The main effects of the drought pathogen interaction on NSC (b, f, j), xylem and phloem transport (c, g, k) and induced defence (d, h, l) are represented. Biotrophs feed directly on NSC reserves (b) without affecting phloem and xylem (c). The direct use of NSC by the pathogen translates directly into pathogen population growth, which accelerates NSC depletion in a positive feedback (e). In the case of drought, biotrophs cannot compete with plant tissues for C; hence pathogen biomass decreases (e). Previous depletion of NSC by the biotroph can accelerate mortality due to lower reserves at the onset of the drought (b). A minimal biotroph establishment is predicted during the drought owing to the declining availability of C as water deficit intensifies (e), and no interactive effects with drought are predicted for infections establishing during drought (b). Necrotrophs find their main C source on phloem elements in leaves, stem, and roots (Fig. 1). Under normal conditions they establish a long-term interaction requiring C investment from the host into defence (f). Following phloem and xylem destruction, the host compartmentalizes the pathogen further sacrificing more phloem and xylem (g). Eventual defence failures allow the pathogen access to living cells and structural C sources (Fig. 1), from which it builds up further biomass (i). Under drought, C for defence becomes scarce (f) allowing previous infections to expand (i) causing further damage to phloem and xylem (g), increasing further the costs of compartmentalization (h). As a result, we predict that necrotrophs exacerbate drought effects, accelerating tree mortality (f). A similar outcome is expected for necrotroph infections occurring during drought (f). Pathogen biomass remains after tree death (h) due to the capacity of this type of pathogens to survive on dead tissues. Vascular wilt pathogens thrive in the xylem. Trees block vertical pathogen spread by clogging the conduits (Fig. 1), with consequences for both xylem transport (k) and NSC (j). Negative water potentials allow them to colonize large parts of the xylem (m, j), causing foliage wilting and mortality of phloem tissues (k).



drought, as an opportunistic agent taking advantage of the effects of reduced water availability on the host (inciting or contributing factor following Manion's (1981) theory of decline); or else acts before the drought episode, causing a long-term effect weakening the tree (predisposing factor). Our framework focuses on drought as the stress condition of the host, and we do not discuss drought as the weather phenomenon that could facilitate/impair the pathogen spore

dispersal or germination and competition with other microorganisms.

Biotrophs and their direct dependence from the C in the infected tissue

Biotrophic pathogens have evolved mechanisms to derive C directly from living cells with specialized structures named haustoria, which tap into host cells and create a local C sink (Fig. 1). Some well-studied biotrophic pathogens are *Erysiphe alphitoides*, *Phaeocryptopus gaeumannii*, and rust fungi of the genus *Melampsora*. Trees have evolved defence mechanisms that shut the flow of C towards the pathogen. The defence is based on a fast recognition of the threatening agent that triggers a programmed cell death (PCD) that kills the infected cells and withdraws the C and nutrients before they are assimilated by the pathogen (Fig. 1) (Glazebrook, 2005). Together with PCD, trees also trigger salicylic-acid mediated defence responses (Fig. 2d). With effector molecules, biotrophs manipulate the defence machinery of the host in order to delay defence responses in order to gain enough time to multiply and spread into neighbouring cells (Fig. 2e). Biotrophs mainly affect the C cycle by reducing assimilation and, compared with hemi-biotrophs and necrotrophs, they produce little disruption of the water and C transport systems of the host (Fig. 2c) (Bassanezi *et al.*, 2002). Known mechanisms of reduction of photosynthetic capacity involve the reduction of stomatal conductance by physically occluding of stomata with mycelia or fruiting bodies, as well as other not-yet-understood mechanisms of fungal interference with RuBisCO activity (Manter *et al.*, 2000; Hajji *et al.*, 2009). During fungal establishment and especially when fruiting bodies are produced, C is drained from the leaves, which become C sinks (Hewitt & Ayres, 1976), hence early leaf-shedding is a common tree reaction to reduce C losses (Manter *et al.*, 2003). In those cases in which stomatal functions are heavily impaired (Manter *et al.*, 2000), damages can be very severe, leading to significant growth reductions of infected trees (Kimberley *et al.*, 2010). Damages can also accumulate over several years by, for instance, eliciting recurrent early leaf-shedding processes, reducing NSC reserves and increasing the chances of death in the long run (Marcais & Bréda, 2006).

During acute drought C assimilation decreases and leaf cells may decrease their non-structural C reserves (e.g. Adams *et al.*, 2013). Increasing demands of sucrose by leaves decrease the influx of C into the biotrophs, which cannot compete for sugars with living cells under drought conditions (Wyness & Ayres, 1987). Low C accessibility during drought slows down fungal multiplication, and deters further damages (Fig. 2e). Sporulation and mycelial growth, for example, has been shown to be negatively affected by previous water stress (Ayres, 1977; Woolacott & Ayres, 1984), and the link between low disease levels of biotrophs and low NSC availability has been established in model plant systems (Engelsdorf *et al.*, 2013). The strong connection between the nutritional status of the host and the pathogen makes us hypothesize that drought will negatively affect biotrophs during pathogen attack and therefore no worsening effects on tree death are anticipated. As shown in our framework, no significant changes on the timing of the *zero C available* point are predicted (Fig. 2b). Our hypothesis is supported by the fact that biotrophs tend to be more prevalent in well watered and fertilized sites (Toome *et al.*, 2010), and are expected to decrease in current climate scenarios including increased drought conditions (Desprez-Loustau *et al.*, 2007; La Porta *et al.*, 2008; Sturrock *et al.*, 2011; Marçais & Desprez-Loustau, 2012). Still, we predict worsening effects of drought on host survival in those cases in which biotrophs attain significant population levels on the tree before the drought onset (Fig. 2b). Depleted carbohydrate reserves may impair the subsequent capacity of trees to cope with water stress. Furthermore, if early leaf-shedding has followed the biotroph attack, a C-expensive crown restoration may also accelerate tree death.

Necrotrophic pathogens and the importance of C for defense and pathogenicity

Necrotrophic pathogens obtain nutrients from dead cells and from structural C sources such as cellulose and hemicellulose. Necrotrophs can attack leaves, twigs, branches, the stem or the root system where they can destroy cambium and the vascular tissue and hence affect both C and water transport systems. Tree defence is activated upon pathogen contact with living cells and is mainly directed at compartmentalizing the pathogen within C-expensive barriers (Fig. 1). Compartmentalization also implies the sacrificial conversion of vascular tissues in the sapwood (Oliva *et al.*, 2012), and, in the case of pathogens causing cankers, in the cambial zone and the phloem. Necrotrophs neutralize tree defences and kill living cells by secreting enzymes and toxins (Fig. 1). Some well-known necrotrophic pathogens include many root rots such as *Heterobasidion annosum* or *Armillaria* sp. and canker pathogens such as *Cryphonectria parasitica* or *Cytospora chrysosperma.*

The accessibility to C by both the tree and the pathogen determines the outcome of the interaction by simultaneously affecting the capacity of the pathogen to build up further inoculum and counteract tree defences, and the capacity of the tree to build up a sufficiently strong response (Fig. 2h). Some necrotrophic root pathogens gain access to the C sources within the host by degrading constitutive and induced defence barriers, like bark or lignin. In these cases pathogens use C from external sources like neighbouring infected or dead trees (Stenlid, 1987; Cleary *et al.*, 2012). In the case of necrotrophs affecting branches or the main stem, the pathogen must gain access to C rich tissues of the phloem passively, either via airborne infection of wounded tissues or by entering the tree as endophytes (Manion & French, 1967). In any case, the outcome of the interaction depends on the host's C availability in order to react fast and compartmentalize the pathogen (Guyon *et al.*, 1996). The magnitude of C needed for defence is large and it has been shown to have a negative impact on tree radial growth (Bendz-Hellgren & Stenlid, 1995; Krokene *et al.*, 2008; Cruickshank *et al.*, 2011; Oliva *et al.*, 2012). By forcing the tree to invest C in defence, necrotrophs affect water transport and storage indirectly by inducing low growth, which results in lowering the overall conductivity of diseased tissues (Joseph *et al.*, 1998) and reducing sapwood storage (Oliva *et al.*, 2012). Necrotrophs can also destroy functional tissues in leaves, stem and roots, which may require

repair, and thus they can increase further the C needs from the host. Under favourable conditions for the host, necrotrophic interactions may persist for decades until trees ultimately die (Cherubini *et al.*, 2002). Indeed, large cankers are often seen in trees and are the result of many years of seasonal variations in the capacity of the tree to prevent the pathogen advance (Manion, 1981; Solla *et al.*, 2006).

The outcome of necrotrophic interactions is influenced by external stress factors such as drought affecting C availability in the host. Severe and prolonged drought periods usually reduce C reserves (Galiano *et al.*, 2011, 2012; McDowell, 2011), limiting the availability of C to support defences and preventing the establishment or the expansion of previously established necrotrophs (Kane & Kolb, 2010; Anderegg & Anderegg, 2013; Gaylord *et al.*, 2013). Decreased tree defences facilitate the access of necrotrophic pathogens to C sources, from which they build up further inoculum and produce further damages (Fig. 2) (Manion & French, 1967; Lygis *et al.*, 2005; Marcais & Bréda, 2006). Defoliation frequently occurs during drought periods, and degradation of starch into readily usable/transportable sugar compounds to restore the crown can also facilitate C access to necrotrophic root pathogens (Wargo, 1972). As lesions enlarge, the size of the front, where host and pathogen interact, increases and with it the C costs to contain the pathogen's progression (Fig. 2h). As with biotrophs, C used before the drought for repairing infected tissues or for building up defences can also contribute to accelerating tree mortality (Fig. 2f). Overall, necrotrophs accelerate drought-induced mortality either by depleting resources and creating repair needs in advance or by making trees run out of C at a faster rate (Fig. 2f) . Consistent with our framework, increased damages have often been observed/expected under drought conditions by necrotrophic canker (Luque *et al.*, 2000; Desprez-Loustau *et al.*, 2006; Waldboth & Oberhuber, 2009) and root rot pathogens (La Porta *et al.*, 2008; Sturrock *et al.*, 2011).

Vascular wilts and the destruction of the water transport system

Vascular wilt pathogens thrive inside xylem conduits, releasing toxic compounds and disturbing water transport (Fig. 1). Some examples of vascular wilt pathogens include some *Ophiostoma* species, remarkably *O. novo-ulmi* and also several *Ceratocystis* and *Leptographium* species. These type of pathogens feed on xylem sap sugars, C leakages, defence compounds and sugars from cell-wall degradation processes (Hammerbacher *et al.*, 2013; Yadeta & Thomma, 2013). Trees block vertical spread by clogging the conduits with tyloses, while lateral spread is prevented by *in situ* synthesis of C compounds and barrier structures to compartmentalize the infection (Shigo & Tippett, 1981; Bonsen *et al.*, 1985; Yadeta & Thomma, 2013). Defence can be C expensive (Guérard *et al.*, 2007) and result in a reduction of sugars in the vicinity of the lesion (Viiri *et al.*, 2001). Investment in defence can be at the expense of radial growth (Krokene *et al.*, 2008) and also imply a sacrificial loss of conductive tissue (Joseph *et al.*, 1998). In contrast to necrotrophs, vascular wilt pathogens have significant direct effects on water transport and storage in trees (Fig. 2). Xylem disruption has immediate effects and may cause sudden mortality on adult trees (Tyree & Zimmermann, 2002). Conduit clogging results in foliage wilting that impacts current and future C reserves by cutting downstream C supply and by reducing autumn re-assimilation of nutrients from leaves. Under these conditions, xylem, phloem and foliage damage become very costly to repair (Fig. 2j). Wilt diseases are often associated with bark beetles that feed on the phloem, increasing even further the costs of repair and reducing the capacity to allocate C to the crown and restore foliage. Nevertheless, insect phloem damage has been shown to be of lesser importance compared with xylem dysfunction induced by insect-vectored wilt pathogens (Hubbard *et al.*, 2013), although in some cases disruption of the water balance of the tree is not a pre-requisite for the success of the bark beetles (Wullschleger *et al.*, 2004).

In contrast to C starvation-driven mortality in the case of necrotrophs, mortality in trees infected by vascular wilt pathogens seems to be triggered by hydraulic failure (Fig. 2k). Disruption of the vascular system is fast and permanent, hence rapid mortality of the corresponding areas of the crown or the whole tree can be observed. Increased damages by insect bark beetles and their associated vascular wilt pathogens are associated with dryer climatic conditions (Williams *et al.*, 2010), but, contrarily to necrotrophs, drought during the infection/attack may be more important than previous drought events (Croisé *et al.*, 2001). The availability of C for defence at the moment of attack is also of a lesser importance in comparison with necrotrophs (Christiansen & Ericsson, 1986). We thus postulate that vascular wilt pathogens accelerate drought-induced mortality under drought mostly by damaging the xylem vascular system and subsequently causing phloem impairment and foliage wilting. Of special importance is the rapid escalation of repair costs as the attack builds up (Fig. 2j). While C reserves can be reasonably high at the onset of a drought event, they may still not be enough for rebuilding a sufficient amount of foliage, phloem and xylem for tree survival. By increasing repair costs, wilt pathogens can also accelerate drought induced mortality processes (Fig. 2j).

Concluding remarks

The presented framework sets the ground for predicting the role of pathogens on tree mortality under drought based on the type of trophic interaction established with the host. Although most pathogens fall within the three categories described in the previous sections, some might establish more than one type of trophic interaction. This is the case of the so-called hemibiotrophs, a category that includes many *Phytophthora* species that share characteristics with both biotrophs and necrotrophs. In these cases, we suggest that the type of trophic interaction that contributes more to the pathogen's inoculum build-up should be considered. Would pre-inoculation water stress (Marçais *et al.*, 1993) or C starvation (Engelsdorf *et al.*, 2013) favour disease development, these pathogens should be considered for their necrotrophic phase and thus be expected to accelerate drought-induced-mortality. Other pathogens can display a behaviour in between a wilt pathogen and a necrotroph. These pathogens are typically secondary pathogens affecting woody tissues, like shoots and twigs (Jactel *et al.*, 2012), and while they can cause disease under negative water potentials,

tree resistance is typically restored when water stress is remediated (Crist & Schoeneweiss, 1975; Schoeneweiss, 1975; Johnson *et al.*, 1997). The fact that the pathogenicity of these fungi is strongly dependent on xylem colonization (Luchi *et al.*, 2005), and that the necrotrophic phase precedes the wilting of the infected tissue, makes them similar to the 'vascular wilt pathogens' in our framework. The same reasoning can be applied to similar pathogens for which pre-inoculation water stress and C limitation would contribute little to host susceptibility (Madar *et al.*, 1989).

Future climate scenarios predict an impact on water and C balance of trees (Wang *et al.*, 2012). At the same time, forest pathogens are pervasive in forest ecosystems all over the globe and are known to cause tree mortality and have a major role in forest dynamics (Worrall *et al.*, 2005). Carbon and water systems are inevitably connected and both are affected by drought and by pathogens. Pathogens can accelerate drought-induced mortality by directly depleting NSC, accelerating NSC consumption by the host or by increasing repairing costs (Fig. 2a). These three processes are tightly connected with the type of trophic interactions established between the host and the pathogen. We describe how these types of pathogens would interact with the host, and by which mechanisms would cause the death of the tree. This theoretical framework allows us to predict that some pathogens such as necrotrophs or vascular wilts can benefit from drought events, and thus contribute to drought induced mortality; and that some, like biotrophs are very unlikely to cause significant damages under drought. Considering their different effects on the host and the contrasted interaction with drought, determining under what environmental conditions the previous trophic interactions will be favoured (or disfavoured) is pivotal to predictions of how forests will respond to warmer and drier conditions in the future. Future research needs to quantify the contribution of pathogens to direct drought effects in the context of drought-induced tree mortality. Manipulative experiments controlling both drought and pathogen inoculum can be used to assess the extent to which pathogens accelerate mortality by comparing the time needed to kill trees under drought with and without specific pathogens (Fig. 2a).

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Key words: carbon economy, carbon starvation, drought, embolism, hydraulic failure, non-structural carbohydrates (NSCs), tree mortality, trophic interaction.