1 **The effect of fungal pathogens on the water and carbon economy of trees:**

2 **implications for drought-induced mortality**

3 Jonàs Oliva[1,*], Jan Stenlid[1] and Jordi Martínez-Vilalta[2,3]

4 [1], Dept. Forest Mycology and Plant Pathology, Swedish University of Agricultural Sciences, Box 7026, S-750

5 07, Uppsala, Sweden.

6 [2], CREAF, Cerdanyola del Vallès 08193 (Barcelona), Spain

7 [3], Univ Autònoma Barcelona, Cerdanyola del Vallès 08193 (Barcelona), Spain

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9 *, corresponding author (e-mail: jonas.oliva@slu.se ; telephone: +46 (0) 18671602

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12 Drought-induced forest mortality is emerging as a widespread phenomenon with potentially large

13 implications for forest function and dynamics (Allen *et al.*, 2010; Anderegg *et al.*, 2012; Martínez-

14 Vilalta *et al.*, 2012). Although the physiological mechanisms underlying tree mortality are still not

15 completely understood, there is agreement that they involve the storage and transport systems of water

16 and carbohydrates (McDowell *et al.*, 2008; Sala *et al.*, 2010; McDowell, 2011). The xylem of plants is

17 susceptible to drought-induced embolism and severe water deficits may result in the complete loss of

18 xylem hydraulic conductivity and cause tree mortality (hydraulic failure; cf. Tyree & Sperry, 1988;

19 McDowell *et al.*, 2008; Choat *et al.*, 2012). Drought also has detrimental effects on the carbon

20 economy of plants, and it has been hypothesized that reduced assimilation due to stomatal closure

21 may lead to a depletion of stored carbon reserves and, eventually, to tree death due to carbon

22 starvation (Waring, 1987; Martínez-Vilalta *et al.*, 2002; Bréda *et al.*, 2006; McDowell *et al.*, 2008).

23 However, only in recent studies has a direct link between reduced carbon reserves and tree mortality

24 been established (Adams *et al.*, 2009; Galiano *et al.*, 2011; Adams *et al.*, 2013; Hartmann *et al.*, 2013;

25 Mitchell *et al.*, 2013; Quirk *et al.*, 2013; Sevanto *et al.*, 2014). Finally, phloem transport could also

26 become impaired due to the inability of plants to maintain phloem turgor under extremely low xylem

27 water potentials, limiting the local availability of carbohydrates for metabolic functions (Sala *et al.*,

28 2010; Sevanto *et al.*, 2014).

29 We postulate that tree mortality research has suffered from a false dichotomy of drought versus biotic

30 attack (McDowell *et al.*, 2013). Pests and pathogens cause tree mortality and it is well known that

31 drought may predispose forests to attacks by insects (Mattson & Haack, 1987; Gaylord *et al.*, 2013)

32 and fungal pathogens (Desprez-Loustau *et al.*, 2006; La Porta *et al.*, 2008). The interaction between

33 drought stress and the damage caused by forest pests and pathogens has been addressed in a recent

34   meta-analysis (Jactel *et al.*, 2012), and the connection between the physiological status of the tree and

35   disease development has motivated a number of reviews in the past (Schoeneweiss, 1975; Boyer,

36   1995). Biotic agents have also been included in theoretical models for drought induced mortality

37   (Martínez-Vilalta *et al.*, 2002; McDowell *et al.*, 2008; McDowell *et al.*, 2011). However, previous

38   reports have not fully acknowledged the diversity of trophic interactions that microorganisms

39   establish with the host trees and how this diversity has direct consequences in terms of the

40   physiological mechanisms leading to mortality. Tree mortality can result directly from a toxic effect

41   from metabolites produced by pathogens, but pathogens can also disrupt the xylem and phloem of the

42   infected hosts and affect their carbon economy through the consumption of carbon reserves and the

43   induction of carbon-expensive defences. Here, we develop a new framework that brings together the

44   effects of pathogens and drought on the water and carbon economy of trees, and explore the

45   implications for the process of drought-induced mortality.

46   **A new framework based on trophic interactions**

47   We argue that predictions of drought-induced mortality under pathogen attack can be improved by

48   taking into account the type of trophic interaction that the pathogen establishes with the host. Three

49   main types of trophic interactions can be distinguished amongst tree pathogens: biotrophs,

50   necrotrophs and vascular wilts (Deacon, 1997) (Fig. 1). In general terms, biotrophs drain carbon and

51   nutrients from living cells – the host response is based on recognition followed by programmed cell

52   death (Glazebrook, 2005). Necrotrophs instead interact with the host through the defence response

53   and get carbon and nutrients from dead cells – the host response is based on carbon-based constitutive

54   and induced responses from living cells surrounding the infection (Glazebrook, 2005). A third

55   category includes vascular fungi that colonize the vascular system systemically, often aided by toxins

56   (Yadeta & Thomma, 2013) – the host responses are based on blocking vertical and lateral spread in

57   the xylem.

58   In this letter, we describe how each of these pathogen types interact with the water and carbon

59   transport systems of trees, and by which mechanisms they may contribute to drought-induced

60   mortality (Fig. 2). As a basis for our rationale, we use the mechanistic model of McDowell *et al.*

61   (2011) to represent the mortality process of trees subjected to drought stress. We show that during

62   drought pathogens may disrupt the carbon balance of trees through three non-exclusive processes: (i)

63   by directly depleting non-structural carbohydrate (NSC) reserves, (ii) by forcing consumption of NSC

64   reserves by the host or (iii) by increasing repair costs (Fig. 2a). Our model makes explicit predictions

65   on the changes in photosynthesis, growth and respiration; as well as on the impacts on the NSC

66   budget, phloem, and xylem transport during a drought episode leading to tree death. The amount of

67   carbon allocated to defence and the impact on a biotic agent's biomass are also included. Tree death is

68 represented as the point in which no carbon for sustaining the basic metabolism is available (i.e., *zero*

69 *available carbon* point), regardless of the process leading to this point. Death occurs when carbon

70 available falls below (intersects) the amount of carbon needed for osmotic adjustment and

71 maintenance of phloem and xylem transport. By considering different trophic interactions, two novel

72 perspectives for current mortality models are put forward: (i) we show the fundamental differences

73 among the *mechanisms* leading to tree mortality between biotrophs, necrotrophs and vascular wilt

74 pathogens, and (ii) we predict how different type of pathogens affect the timing of the *zero available*

75 *carbon* point, and consequently whether they contribute or not to drought-induced mortality. We have

76 considered the timing of the interaction between drought and pathogens in two ways. Either the

77 pathogen acts simultaneously with drought, as an opportunistic agent taking advantage of the effects

78 of reduced water availability on the host (inciting or contributing factor following Manion's (1981)

79 theory of decline); or else acts prior to the drought episode, causing a long-term effect weakening the

80 tree (predisposing factor). Our framework focuses on drought as the stress condition of the host, and

81 we do not discuss drought as the weather phenomenon that could facilitate/impair the pathogen spore

82 dispersal or germination and competition with other microorganisms.

83 **Biotrophs and their direct dependence from the carbon in the infected tissue**

84 Biotrophic pathogens have evolved mechanisms to derive carbon directly from living cells with

85 specialised structures named haustoria, which tap into host cells and create a local carbon sink (Fig.

86 1). Some well-studied biotrophic pathogens are *Erysiphe alphitoides*, *Phaeocryptopus gaeumannii*,

87 and rust pathogens like for instance *Peridermium pini* or *Melampsora* spp. Trees have evolved

88 defence mechanisms that shut the flow of carbon towards the pathogen. The defence is based on a fast

89 recognition of the threatening agent that triggers a programmed cell death (PCD) that kills the infected

90 cells and withdraws the carbon and nutrients before they are assimilated by the pathogen (Fig. 1)

91 (Glazebrook, 2005). Together with PCD, trees also trigger salicylic-acid mediated defence responses

92 (Fig. 2d). With effector molecules, biotrophs manipulate the defence machinery of the host in order to

93 delay defence responses in order to gain enough time to multiply and spread into neighbouring cells

94 (Fig. 2e). Biotrophs mainly affect the carbon cycle by reducing assimilation and, compared with

95 hemi-biotrophs and necrotrophs, they produce little disruption of the water and carbon transport

96 systems of the host (Fig. 2c) (Bassanezi *et al.*, 2002). Known mechanisms of reduction of

97 photosynthetic capacity involve the reduction of stomatal conductance by physically occluding of

98 stomata with mycelia or fruiting bodies, as well as other not-yet-understood mechanisms of fungal

99 interference with RuBisCO activity (Manter *et al.*, 2000; Hajji *et al.*, 2009). During fungal

100 establishment and especially when fruiting bodies are produced, carbon is drained from the leaves,

101 which become carbon sinks (Hewitt & Ayres, 1976), hence early leaf-shedding is a common tree

102 reaction to reduce carbon losses (Manter *et al.*, 2003). In those cases in which stomatal functions are

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103   heavily impaired (Manter *et al.*, 2000), damages can be very severe, leading to significant growth

104   reductions of infected trees (Kimberley et al., 2010). Damages can also accumulate over several years

105   by, for instance, eliciting recurrent early leaf-shedding processes, reducing NSC reserves and

106   increasing the chances of death in the long run (Marcais & Bréda, 2006).

107   During acute drought carbon assimilation decreases and leaf cells may decrease their non-structural

108   carbon reserves (e.g., Adams et al. 2013). Increasing demands of sucrose by leaves decrease the influx

109   of carbon into the biotrophs, which cannot compete for sugars with living cells under drought

110   conditions (Wyness & Ayres, 1987). Low carbon accessibility during drought slows down fungal

111   multiplication, and deters further damages (Fig. 2e). Sporulation and mycelial growth, for example,

112   has been shown to be negatively affected by previous water stress (Ayres, 1977; Woolacott & Ayres,

113   1984), and the link between low disease levels of biotrophs and low NSC carbon availability has been

114   established in model plant systems (Engelsdorf *et al.*, 2013). The strong connection between the

115   nutritional status of the host and the pathogen makes us hypothesize that drought will negatively

116   affect biotrophs during pathogen attack and therefore no worsening effects on tree death are

117   anticipated. As shown in our framework, no significant changes on the timing of the *zero carbon*

118   *available point* are predicted (Fig. 2b). Our hypothesis is supported by the fact that biotrophs tend to

119   be more prevalent in well watered and fertilized sites (Toome *et al.*, 2010), and are expected to

120   decrease in current climate scenarios including increased drought conditions (Desprez-Loustau *et al.*,

121   2007; La Porta *et al.*, 2008; Sturrock *et al.*, 2011; Marçais & Desprez-Loustau, 2012). Still, we predict

122   worsening effects of drought on host survival in those cases in which biotrophs attain significant

123   population levels on the tree prior to the drought onset (Fig. 2b). Depleted carbohydrate reserves may

124   impair the subsequent capacity of trees to cope with water stress. Furthermore, if early leaf-shedding

125   has followed the biotroph attack, a carbon-expensive crown restoration may also accelerate tree death.

126   **Necrotrophic pathogens and the importance of carbon for defense and pathogenicity**

127   Necrotrophic pathogens obtain nutrients from dead cells and from structural carbon sources such as

128   cellulose and hemicellulose. Necrotrophs can attack leaves, twigs, branches, the stem or the root

129   system where they can destroy cambium and the vascular tissue and hence affect both carbon and

130   water transport systems. Tree defence is activated upon pathogen contact with living cells and is

131   mainly directed at compartmentalizing the pathogen within carbon-expensive barriers (Fig. 1).

132   Compartmentalization also implies the sacrificial conversion of vascular tissues in the sapwood (Oliva

133   *et al.*, 2012), and, in the case of pathogens causing cankers, in the cambial zone and the phloem.

134   Necrotrophs neutralize tree defences and kill living cells by secreting enzymes and toxins (Fig. 1).

135   Some well-known necrotrophic pathogens include many root rots such as *Heterobasidion annosum* or

136   *Armillaria sp.* and canker pathogens such as *Cryphonectria parasitica* or *Cytospora chrysosperma.*

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137 The accessibility to carbon by both the tree and the pathogen determines the outcome of the

138 interaction by simultaneously affecting the capacity of the pathogen to build up further inoculum and

139 counteract tree defences, and the capacity of the tree to build up a sufficiently strong response (Fig.

140 2h). Some necrotrophic root pathogens gain access by themselves to the carbon sources within the

141 host by degrading constitutive and induced defence barriers, like bark or lignin. In these cases

142 pathogens use carbon from external sources like neighbouring infected or dead trees (Stenlid, 1987;

143 Cleary *et al.*, 2012). In the case of necrotrophs affecting branches or the main stem, the pathogen must

144 gain access to carbon rich tissues of the phloem passively, either via airborne infection of wounded

145 tissues or by entering the tree as endophytes (Manion & French, 1967). In any case, the outcome of

146 the interaction depends on the host's carbon availability in order to react fast and compartmentalize

147 the pathogen (Guyon *et al.*, 1996). The magnitude of carbon needed for defence is large and it has

148 been shown to have a negative impact on tree radial growth (Bendz-Hellgren & Stenlid, 1995;

149 Krokene *et al.*, 2008; Cruickshank *et al.*, 2011; Oliva *et al.*, 2012). By forcing the tree to invest carbon

150 in defence, necrotrophs affect water transport and storage indirectly by inducing low growth, which

151 results in lowering the overall conductivity of diseased tissues (Joseph *et al.*, 1998) and reducing

152 sapwood storage (Oliva *et al.*, 2012). Necrotrophs can also destroy functional tissues in leaves, stem

153 and roots, which may require repair, and thus they can increase further the carbon needs from the

154 host. Under favourable conditions for the host, necrotrophic may persist for decades until

155 trees ultimately die (Cherubini *et al.*, 2002). Indeed, large cankers are often seen in trees and are the

156 result of many years of seasonal variations in the capacity of the tree to prevent the pathogen advance

157 (Manion, 1981; Solla *et al.*, 2006) (Fig. 2i).

158 The outcome of necrotrophic interactions is influenced by external stress factors such as drought

159 affecting carbon availability in the host. Severe and prolonged drought periods usually reduce carbon

160 reserves (Galiano *et al.*, 2011; McDowell, 2011; Galiano *et al.*, 2012), limiting the availability of

161 carbon to support defences and preventing the establishment or the expansion of previously

162 established necrotrophs [e.g., Kane and Kolb (2010), Gaylord *et al.* (2013), Anderegg and Anderegg

163 (2013)]. Decreased tree defences facilitate the access of necrotrophic pathogens to carbon sources,

164 from which they build up further inoculum and produce further damages (Fig. 2) (Manion & French,

165 1967; Lygis *et al.*, 2005; Marcais & Bréda, 2006). Defoliation frequently occurs during drought

166 periods, and degradation of starch into readily usable/transportable sugar compounds to restore the

167 crown can also facilitate carbon access to necrotrophic root pathogens (Wargo, 1972). As lesions

168 enlarge, the size of the front, where host and pathogen interact, increases and with it the carbon costs

169 to contain the pathogen's progression (Fig. 2h). As with biotrophs, carbon used prior to the drought

170 for repairing infected tissues or for building up defences can also contribute to accelerating tree

171 mortality (Fig. 2f). Overall, necrotrophs accelerate drought-induced mortality (Fig. 2f) either by

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172 depleting resources and creating repair needs in advance or by making trees run out of carbon at a
173 faster rate. Consistent with our framework, increased damages have often been observed/expected
174 under drought conditions by necrotrophic canker (Luque *et al.*, 2000; Desprez-Loustau *et al.*, 2006;
175 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

177 Vascular wilt pathogens thrive inside xylem conduits, releasing toxic compounds and disturbing water
178 transport (Fig. 1). Some examples of vascular wilt pathogens include some *Ophiostoma* species,
179 remarkably *O. novo-ulmi* and also several *Ceratocystis* and *Leptographium* species. These type of
180 pathogens feed on xylem sap sugars, carbon leakages, defence compounds and sugars from cell-wall
181 degradation processes (Hammerbacher *et al.*, 2013; Yadeta & Thomma, 2013). Trees block vertical
182 spread by clogging the conduits with tyloses, while lateral spread is prevented by *in situ* synthesis of
183 carbon compounds and barrier structures to compartmentalize the infection (Shigo & Tippett, 1981;
184 Bonsen *et al.*, 1985; Yadeta & Thomma, 2013). Defence can be carbon expensive (Guérard *et al.*,
185 2007) and result in a reduction of sugars in the vicinity of the lesion (Viiri *et al.*, 2001). Investment in
186 defence can be at the expense of radial growth (Krokene *et al.*, 2008) and also imply a sacrificial loss
187 of conductive tissue (Joseph *et al.*, 1998). In contrast to necrotrophs, vascular wilt pathogens have
188 significant direct effects on water transport and storage in trees (Fig. 2). Xylem disruption has
189 immediate effects and may cause sudden mortality on adult trees (Tyree & Zimmermann, 2002).
190 Conduit clogging results in foliage wilting that impacts current and future carbon reserves by cutting
191 downstream carbon supply and by reducing autumn re-assimilation of nutrients from leaves. Under
192 these conditions, xylem, phloem and foliage damage become very costly to repair (Fig. 2j). Wilt
193 diseases are often associated with bark beetles that feed on the phloem, increasing even further the
194 costs of repair and reducing the capacity to allocate carbon to the crown and restore foliage.
195 Nevertheless, insect phloem damage has been shown to be of lesser importance compared with xylem
196 dysfunction induced by insect-vectored wilt pathogens (Hubbard *et al.*, 2013), although in some cases
197 disruption of the water balance of the tree is not a pre-requisite for the success of the bark beetles
198 (Wullschleger *et al.*, 2004).

199 In contrast to carbon starvation-driven mortality in the case of necrotrophs, mortality in trees infected
200 by vascular wilt pathogens seems to be triggered by hydraulic failure (Fig. 2k). Disruption of the
201 vascular system is fast and permanent, hence rapid mortality of the corresponding areas of the crown
202 or the whole tree can be observed. Increased damages by insect bark beetles and their associated
203 vascular wilt pathogens are associated with dryer climatic conditions (Williams *et al.*, 2010), but,
204 contrarily to necrotrophs, drought during the infection/attack may be more important than previous
205 drought events (Croisé *et al.*, 2001). The availability of carbon for defence at the moment of attack is

206   also of a lesser importance in comparison with necrotrophs (Christiansen & Ericsson, 1986). We thus

207   postulate that vascular wilt pathogens accelerate drought-induced mortality under drought mostly by

208   damaging the xylem vascular system and subsequently causing phloem impairment and foliage

209   wilting. Of special importance is the rapid escalation of repair costs as the attack builds up (Fig. 2j).

210   While carbon reserves can be reasonably high at the onset of a drought event, they may still not be

211   enough for rebuilding a sufficient amount of foliage, phloem and xylem for tree survival. By

212   increasing repair costs, wilt pathogens can also accelerate drought induced mortality processes (Fig.

213   2j).

Concluding remarks

215   The presented framework sets the ground for predicting the role of pathogens on tree mortality under

216   drought based on the type of trophic interaction established with the host. Although most pathogens

217   fall within the three categories described in the previous sections, some might establish more than one

218   type of trophic interaction. This is the case of the so-called hemibiotrophs, a category that includes

219   many *Phytophthora* species that share characteristics with both biotrophs and necrotrophs. In these

220   cases, we suggest that the type of trophic interaction that contributes more to the pathogen's inoculum

221   build-up should be considered. Would pre-inoculation water stress (Marçais *et al.*, 1993) or carbon

222   starvation (Engelsdorf *et al.*, 2013) favour disease development, these pathogens should be considered

223   for their necrotrophic phase and thus be expected to accelerate drought-induced-mortality. Other

224   pathogens can display a behaviour in between a wilt pathogen and a necrotroph. These pathogens are

225   typically secondary pathogens affecting woody tissues, like shoots and twigs (Jactel *et al.*, 2012), and

226   while they can cause disease under negative water potentials, tree resistance is typically restored when

227   water stress is remediated (Crist & Schoeneweiss, 1975; Schoeneweiss, 1975; Johnson *et al.*, 1997).

228   The fact that the pathogenicity of these fungi is strongly dependent on xylem colonisation (Luchi *et*

229   *al.*, 2005), and that the necrotrophic phase precedes the wilting of the infected tissue, makes them

230   similar to the "vascular wilt pathogens" in our framework. The same reasoning can be applied to

231   similar pathogens for which pre-inoculation water stress and carbon limitation would contribute little

232   to host susceptibility (Madar *et al.*, 1989).

233   Future climate scenarios predict an impact on water and carbon balance of trees (Wang *et al.*, 2012).

234   At the same time, forest pathogens are pervasive in forest ecosystems all over the globe and are

235   known to cause tree mortality and have a major role in forest dynamics (Worrall *et al.*, 2005). Carbon

236   and water systems are inevitably connected and both are affected by drought and by pathogens.

237   Pathogens can accelerate drought-induced mortality by directly depleting NSC, accelerating NSC

238   consumption by the host or by increasing repairing costs (Fig. 2a). These three processes are tightly

239   connected with the type of trophic interactions established between the host and the pathogen. We

240 describe how these types of pathogens would interact with the host, and by which mechanisms would

241 cause the death of the tree. This theoretical framework allows us to predict that some pathogens such

242 as necrotrophs or vascular wilts can benefit from drought events, and thus contribute to drought

243 induced mortality; and that some, like biotrophs are very unlikely to cause significant damages under

244 drought. Considering their different effects on the host and the contrasted interaction with drought,

245 determining under what environmental conditions the previous trophic interactions will be favoured

246 (or disfavoured) is pivotal to predictions of how forests will respond to warmer and drier conditions in

247 the future. Future research needs to quantify the contribution of pathogens to direct drought effects in

248 the context of drought-induced tree mortality. Manipulative experiments controlling both drought and

249 pathogen inoculum can be used to assess the extent to which pathogens accelerate mortality by

250 comparing the time needed to kill trees under drought with and without specific pathogens (Fig. 2a).

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References

Adams HD, Germino MJ, Breshears DD, Barron-Gafford GA, Guardiola-Claramonte M, Zou CB, Huxman TE. 2013. Nonstructural leaf carbohydrate dynamics of *Pinus edulis* during drought-induced tree mortality reveal role for carbon metabolism in mortality mechanism. *New Phytologist* **197**(4): 1142-1151.

Adams HD, Guardiola-Claramonte M, Barron-Gafford GA, Villegas JC, Breshears DD, Zou CB, Troch PA, Huxman TE. 2009. Temperature sensitivity of drought-induced tree mortality portends increased regional die-off under global-change-type drought. *Proceedings of the National Academy of Sciences* **106**(17): 7063-7066.

Allen CD, Macalady AK, Chenchouni H, Bachelet D, McDowell N, Vennetier M, Kitzberger T, Rigling A, Breshears DD, Hogg EH, et al. 2010. A global overview of drought and heat-induced tree mortality reveals emerging climate change risks for forests. *Forest Ecology and Management* **259**(4): 660-684.

Anderegg WRL, Anderegg LDL. 2013. Hydraulic and carbohydrate changes in experimental drought-induced mortality of saplings in two conifer species. *Tree Physiology* **33**(3): 252-260.

Anderegg WRL, Anderegg LDL, Sherman C, Karp DS. 2012. Effects of widespread drought-induced aspen mortality on understory plants. *Conservation Biology* **26**(6): 1082-1090.

Ayres PG. 1977. Effect of water potential of pea leaves on spore production by *Erysiphe pisi* (powdery mildew). *Transactions of the British Mycological Society* **68**(1): 97-100.

Bassanezi RB, Amorim L, Filho AB, Berger RD. 2002. Gas exchange and emission of chlorophyll fluorescence during the monocycle of rust, angular leaf spot and anthracnose on bean leaves as a function of their trophic characteristics. *Journal of Phytopathology* **150**(1): 37-47.

Bendz-Hellgren M, Stenlid J. 1995. Long-term reduction in the diameter growth of butt rot affected Norway spruce, *Picea abies*. *Forest Ecology and Management* **74**(1-3): 239-243.

Bonsen KJM, Scheffer RJ, Elgersma DM. 1985. Barrier zone formation as a resistance mechanism of elms to dutch elm disease. *IAWA Bulletin* **6**: 71-76.

Boyer JS. 1995. Biochemical and biophysical aspects of water deficits and the predisposition to disease. *Annual Review of Phytopathology* **33**(1): 251-274.

Bréda N, Huc R, Granier A, Dreyer E. 2006. Temperate forest trees and stands under severe drought: a review of ecophysiological responses, adaptation processes and long-term consequences. *Ann. For. Sci.* **63**(6): 625-644.

Cherubini P, Fontana G, Rigling D, Dobbertin M, Brang P, Innes JL. 2002. Tree-life history prior to death: two fungal root pathogens affect tree-ring growth differently. *Journal of Ecology* **90**(5): 839-850.

Choat B, Jansen S, Brodribb TJ, Cochard H, Delzon S, Bhaskar R, Bucci SJ, Feild TS, Gleason SM, Hacke UG, et al. 2012. Global convergence in the vulnerability of forests to drought. *Nature* **491**(7426): 752-755.

Christiansen E, Ericsson A. 1986. Starch reserves in *Picea abies* in relation to defence reaction against a bark beetle transmitted blue-stain fungus, *Ceratocystis polonica*. *Canadian Journal of Forest Research* **16**(1): 78-83.

Cleary MR, van der Kamp BJ, Morrison DJ. 2012. Pathogenicity and virulence of *Armillaria sinapina* and host response to infection in Douglas-fir, western hemlock and western redcedar in the southern Interior of British Columbia. *Forest Pathology* **42**(6): 481-491.

Crist CR, Schoeneweiss DF. 1975. The influence of controlled stresses on susceptibility of European white birch stems to attack by *Botryosphaeria dothidea*. *Phytopathology* **65**: 369-373

Croisé L, Lieutier F, Cochard H, Dreyer E. 2001. Effects of drought stress and high density stem inoculations with *Leptographium wingfieldii* on hydraulic properties of young Scots pine trees. *Tree Physiology* **21**(7): 427-436.

303 **Cruickshank MG, Morrison DJ, Lalumière A. 2011.** Site, plot, and individual tree yield reduction of
304   interior Douglas-fir associated with non-lethal infection by Armillaria root disease in
305   southern British Columbia. *Forest Ecology and Management* **261**(2): 297-307.
306 **Deacon J. 1997.** *Modern mycology*: Blackwell Science Ltd.
307 **Desprez-Loustau M-L, Marçais B, Nageleisen L-M, Piou D, Vannini A. 2006.** Interactive effects of
308   drought and pathogens in forest trees. *Annals of Forest Science* **63**(6): 597-612.
309 **Desprez-Loustau M-L, Robin C, Reynaud G, Déqué M, Badeau V, Piou D, Husson C, Marçais B. 2007.**
310   Simulating the effects of a climate-change scenario on the geographical range and activity of
311   forest-pathogenic fungi. *Canadian Journal of Plant Pathology* **29**(2): 101-120.
312 **Engelsdorf T, Horst RJ, Pröls R, Pröschel M, Dietz F, Hückelhoven R, Voll LM. 2013.** Reduced
313   carbohydrate availability enhances the susceptibility of *Arabidopsis* toward *Colletotrichum*
314   *higginsianum*. *Plant Physiology* **162**(1): 225-238.
315 **Galiano L, Martínez-Vilalta J, Lloret F. 2011.** Carbon reserves and canopy defoliation determine the
316   recovery of Scots pine 4 yr after a drought episode. *New Phytologist* **190**(3): 750-759.
317 **Galiano L, Martínez-Vilalta J, Sabaté S, Lloret F. 2012.** Determinants of drought effects on crown
318   condition and their relationship with depletion of carbon reserves in a Mediterranean holm
319   oak forest. *Tree Physiology* **32**(4): 478-489.
320 **Gaylord ML, Kolb TE, Pockman WT, Plaut JA, Yepez EA, Macalady AK, Pangle RE, McDowell NG.**
321   **2013.** Drought predisposes piñon–juniper woodlands to insect attacks and mortality. *New*
322   *Phytologist* **198**(2): 567-578.
323 **Glazebrook J. 2005.** Contrasting mechanisms of defense against biotrophic and necrotrophic
324   pathogens. *Annual Review of Phytopathology* **43**(1): 205-227.
325 **Guérard N, Maillard P, Bréchet C, Lieutier F, Dreyer E. 2007.** Do trees use reserve or newly
326   assimilated carbon for their defense reactions? A 13C labeling approach with young Scots
327   pines inoculated with a bark-beetle-associated fungus (*Ophiostoma brunneo ciliatum*).
328   *Annals of Forest Science* **64**(6): 601-608.
329 **Guyon J, Jacobi W, McIntyre G. 1996.** Effects of environmental stress on the development of
330   Cytospora canker of aspen. *Plant Disease* **80**: 1320-1326.
331 **Hajji M, Dreyer E, Marçais B. 2009.** Impact of *Erysiphe alphitoides* on transpiration and
332   photosynthesis in *Quercus robur* leaves. *European Journal of Plant Pathology* **125**(1): 63-72.
333 **Hammerbacher A, Schmidt A, Wadke N, Wright LP, Schneider B, Bohlmann J, Brand WA, Fenning**
334   **TM, Gershenzon J, Paetz C. 2013.** A Common Fungal Associate of the Spruce Bark Beetle
335   Metabolizes the Stilbene Defenses of Norway Spruce. *Plant Physiology* **162**(3): 1324-1336.
336 **Hartmann H, Ziegler W, Trumbore S. 2013.** Lethal drought leads to reduction in nonstructural
337   carbohydrates in Norway spruce tree roots but not in the canopy. *Functional Ecology* **27**(2):
338   413-427.
339 **Hewitt HG, Ayres PG. 1976.** Effect of infection by *Microsphaera alphitoides* (powdery mildew) on
340   carbohybrate levels and translocation in seedlings of *Quercus robur*. *New Phytologist* **77**(2):
341   379-390.
342 **Hubbard RM, Rhoades CC, Elder K, Negron J. 2013.** Changes in transpiration and foliage growth in
343   lodgepole pine trees following mountain pine beetle attack and mechanical girdling. *Forest*
344   *Ecology and Management* **289**(0): 312-317.
345 **Jactel H, Petit J, Desprez-Loustau M-L, Delzon S, Piou D, Battisti A, Koricheva J. 2012.** Drought
346   effects on damage by forest insects and pathogens: a meta-analysis. *Global Change Biology*
347   **18**(1): 267-276.
348 **Johnson JW, Gleason ML, Parker SK, Provin EB, Iles JK. 1997.** Duration of water stress affects
349   development of *Sphaeropsis* canker on Scots pine. *Journal of Arboriculture* **23**: 73-76.
350 **Joseph G, Kelsey RG, Thies WG. 1998.** Hydraulic conductivity in roots of ponderosa pine infected
351   with black-stain (*Leptographium wageneri*) or annosus (*Heterobasidion annosum*) root
352   disease. *Tree Physiology* **18**(5): 333-339.

353   **Kane JM, Kolb TE. 2010.** Importance of resin ducts in reducing ponderosa pine mortality from bark
354   beetle attack. *Oecologia* **164**(3): 601-609.

355   **Kimberley MO, Hood IA, Knowles RL. 2010.** Impact of Swiss needle-cast on growth of Douglas-fir.
356   *Phytopathology* **101**(5): 583-593.

357   **Krokene P, Nagy NE, Solheim H. 2008.** Methyl jasmonate and oxalic acid treatment of Norway
358   spruce: anatomically based defense responses and increased resistance against fungal
359   infection. *Tree Physiology* **28**(1): 29-35.

360   **La Porta N, Capretti P, Thomsen IM, Kasanen R, Hietala AM, Von Weissenberg K. 2008.** Forest
361   pathogens with higher damage potential due to climate change in Europe. *Canadian Journal*
362   *of Plant Pathology* **30**(2): 177-195.

363   **Luchi N, Ma R, Capretti P, Bonello P. 2005.** Systemic induction of traumatic resin ducts and resin
364   flow in Austrian pine by wounding and inoculation with *Sphaeropsis sapinea* and *Diplodia*
365   *scrobiculata*. *Planta* **221**(1): 75-84.

366   **Luque J, Parlade J, Pera J. 2000.** Pathogenicity of fungi isolated from *Quercus suber* in Catalonia (NE
367   Spain). *Forest Pathology* **30**(5): 247-263.

368   **Lygis V, Vasiliauskas R, Larsson K-H, Stenlid J. 2005.** Wood-inhabiting fungi in stems of *Fraxinus*
369   *excelsior* in declining ash stands of northern Lithuania, with particular reference to *Armillaria*
370   *cepistipes*. *Scandinavian Journal of Forest Research* **20**(4): 337-346.

371   **Madar ZZ, Solel Z, Kimchi M. 1989.** Effect of water stress in cypress on the development of cankers
372   caused by *Diplodia pinea* f. sp. *cupressi* and *Seiridium cardinale*. *Plant Disease* **73**(484-486).

373   **Manion PD. 1981.** *Tree disease concepts*. Englewood Cliffs, NJ.: Prentice Hall.

374   **Manion PD, French DW. 1967.** *Nectria galligena* and *Ceratocystis fimbriata* cankers of aspen in
375   Minnesota. *Forest Science* **13**(1): 23-28.

376   **Manter DK, Bond BJ, Kavanagh KL, Rosso PH, Filip GM. 2000.** Pseudothecia of Swiss needle cast
377   fungus, *Phaeocryptopus gaeumannii*, physically block stomata of Douglas fir, reducing CO2
378   assimilation. *New Phytologist* **148**(3): 481-491.

379   **Manter DK, Bond BJ, Kavanagh KL, Stone JK, Filip GM. 2003.** Modelling the impacts of the foliar
380   pathogen, *Phaeocryptopus gaeumannii,* on Douglas-fir physiology: net canopy carbon
381   assimilation, needle abscission and growth. *Ecological Modelling* **164**(2–3): 211-226.

382   **Marcais B, Bréda N. 2006.** Role of an opportunistic pathogen in the decline of stressed oak trees.
383   *Journal of Ecology* **94**(6): 1214-1223.

384   **Marçais B, Desprez-Loustau M-L. 2012.** European oak powdery mildew: impact on trees, effects of
385   environmental factors, and potential effects of climate change. *Annals of Forest Science*: 1-
386   10.

387   **Marçais B, Dupuis F, Desprez-Loustau ML. 1993.** Influence of water stress on susceptibility of red
388   oak (*Quercus rubra*) to *Phytophthora cinnamomi*. *European Journal of Forest Pathology*
389   **23**(5): 295-305.

390   **Martínez-Vilalta J, Lloret F, Breshears DD. 2012.** Drought-induced forest decline: causes, scope and
391   implications. *Biology Letters* **8**(5): 689-691.

392   **Martínez-Vilalta J, Prat E, Oliveras I, Piñol J. 2002.** Xylem hydraulic properties of roots and stems of
393   nine Mediterranean woody species. *Oecologia* **133**(1): 19-29.

394   **Mattson WJ, Haack RA. 1987.** The role of drought in outbreaks of plant-eating insects. *BioScience*
395   **37**(2): 110-118.

396   **McDowell N, Pockman WT, Allen CD, Breshears DD, Cobb N, Kolb T, Plaut J, Sperry J, West A,**
397   **Williams DG, et al. 2008.** Mechanisms of plant survival and mortality during drought: why do
398   some plants survive while others succumb to drought? *New Phytologist* **178**(4): 719-739.

399   **McDowell NG. 2011.** Mechanisms linking drought, hydraulics, carbon metabolism, and vegetation
400   mortality. *Plant Physiology* **155**(3): 1051-1059.

401 **McDowell NG, Beerling DJ, Breshears DD, Fisher RA, Raffa KF, Stitt M. 2011.** The interdependence
402   of mechanisms underlying climate-driven vegetation mortality. *Trends in Ecology and*
403   *Evolution* **26**(10): 523-532.
404 **McDowell NG, Ryan MG, Zeppel MJB, Tissue DT. 2013.** Feature: Improving our knowledge of
405   drought-induced forest mortality through experiments, observations, and modeling. *New*
406   *Phytologist* **200**(2): 289-293.
407 **Mitchell PJ, O'Grady AP, Tissue DT, White DA, Ottenschlaeger ML, Pinkard EA. 2013.** Drought
408   response strategies define the relative contributions of hydraulic dysfunction and
409   carbohydrate depletion during tree mortality. *New Phytologist* **197**(3): 862-872.
410 **Oliva J, Camarero JJ, Stenlid J. 2012.** Understanding the role of sapwood loss and reaction zone
411   formation on radial growth of Norway spruce (*Picea abies*) trees decayed by *Heterobasidion*
412   *annosum s.l. Forest Ecology and Management* **274**(0): 201-209.
413 **Quirk J, McDowell NG, Leake JR, Hudson PJ, Beerling DJ. 2013.** Increased susceptibility to drought-
414   induced mortality in Sequoia sempervirens (Cupressaceae) trees under Cenozoic
415   atmospheric carbon dioxide starvation. *American Journal of Botany* **100**(3): 582-591.
416 **Sala A, Piper F, Hoch G. 2010.** Physiological mechanisms of drought-induced tree mortality are far
417   from being resolved. *New Phytologist* **186**(2): 274-281.
418 **Schoeneweiss DF. 1975.** Predisposition, stress, and plant disease. *Annual Review of Phytopathology*
419   **13**(1): 193-211.
420 **Sevanto S, McDowell NG, Dickman LT, Pangle R, Pockman WT. 2014.** How do trees die? A test of
421   the hydraulic failure and carbon starvation hypotheses. *Plant, Cell & Environment* **37**(1): 153-
422   161.
423 **Shigo A, Tippett JT. 1981.** Compartimentalization of American elm tissues infected by *Ceratocystis*
424   *ulmi*. *Plant Disease* **65**: 715-718.
425 **Solla A, Sánchez-Miranda Á, Camarero JJ. 2006.** Radial-growth and wood anatomical changes in
426   *Abies alba* infected by *Melampsorella caryophyllacearum*: a dendroecological assessment of
427   fungal damage. *Annals of Forest Science* **63**(3): 293- 300.
428 **Stenlid J. 1987.** Controlling and predicting the spread of *Heterobasidion annosum* from infected
429   stumps and trees of *Picea abies*. *Scandinavian Journal of Forest Research* **2**(1): 187 - 198.
430 **Sturrock RN, Frankel SJ, Brown AV, Hennon PE, Kliejunas JT, Lewis KJ, Worrall JJ, Woods AJ. 2011.**
431   Climate change and forest diseases. *Plant Pathology* **60**(1): 133-149.
432 **Toome M, Heinsoo K, Luik A. 2010.** Relation between leaf rust (*Melampsora epitea*) severity and the
433   specific leaf area in short rotation coppice willows. *European Journal of Plant Pathology*
434   **126**(4): 583-588.
435 **Tyree M, Zimmermann M. 2002.** *Xylem structure and the ascent of sap*. Berlin: Springer.
436 **Tyree MT, Sperry JS. 1988.** Do woody plants operate near the point of catastrophic xylem
437   dysfunction caused by dynamic water stress? *Plant Physiology* **88**(3): 574–580.
438 **Viiri H, Niemelä P, Kitunen V, Annila E. 2001.** Soluble carbohydrates, radial growth and vigour of
439   fertilized Norway spruce after inoculation with blue-stain fungus, *Ceratocystis polonica*.
440   *Trees - Structure and Function* **15**(6): 327-334.
441 **Waldboth M, Oberhuber W. 2009.** Synergistic effect of drought and chestnut blight (*Cryphonectria*
442   *parasitica*) on growth decline of European chestnut (*Castanea sativa*). *Forest Pathology*
443   **39**(1): 43-55.
444 **Wang W, Peng C, Kneeshaw DD, Larocque GR, Luo Z. 2012.** Drought-induced tree mortality:
445   ecological consequences, causes, and modeling. *Environmental Reviews* **20**(2): 109-121.
446 **Wargo PM. 1972.** Defoliation-induced chemical changes in sugar maple roots stimulate growth of
447   *Armillaria mellea*. *Phytopathology* **62**: 1278-1283.
448 **Waring RH. 1987.** Characteristics of trees predisposed to die. *Bioscience* **37**: 569-577.

449   **Williams AP, Allen CD, Millar CI, Swetnam TW, Michaelsen J, Still CJ, Leavitt SW. 2010.** Forest
450   responses to increasing aridity and warmth in the southwestern United States. *Proceedings*
451   *of the National Academy of Sciences*.
452   **Woolacott B, Ayres PG. 1984.** Effects of plant age and water stress on production of conidia by
453   *Erysiphe graminis* f.sp. *hordei* examined by non-destructive sampling. *Transactions of the*
454   *British Mycological Society* **82**(3): 449-454.
455   **Worrall JJ, Lee TD, Harrington TC. 2005.** Forest dynamics and agents that initiate and expand canopy
456   gaps in *Picea-Abies* forests of Crawford Notch, New Hampshire, USA. *J Ecology* **93**(1): 178-
457   190.
458   **Wullschleger SD, McLaughlin SB, Ayres MP. 2004.** High-resolution analysis of stem increment and
459   sap flow for loblolly pine trees attacked by southern pine beetle. *Canadian Journal Forest*
460   *Research* **34**: 2387–2393.
461   **Wyness LE, Ayres PG. 1987.** Plant-fungus water relations affect carbohydrate transport from pea
462   leaf to powdery mildew (*Erysiphe pisi*) mycelium. *Transactions of the British Mycological*
463   *Society* **88**(1): 97-104.
464   **Yadeta K, Thomma B. 2013.** The xylem as battleground for plant hosts and vascular wilt pathogens.
465   *Frontiers in Plant Science* **4**.
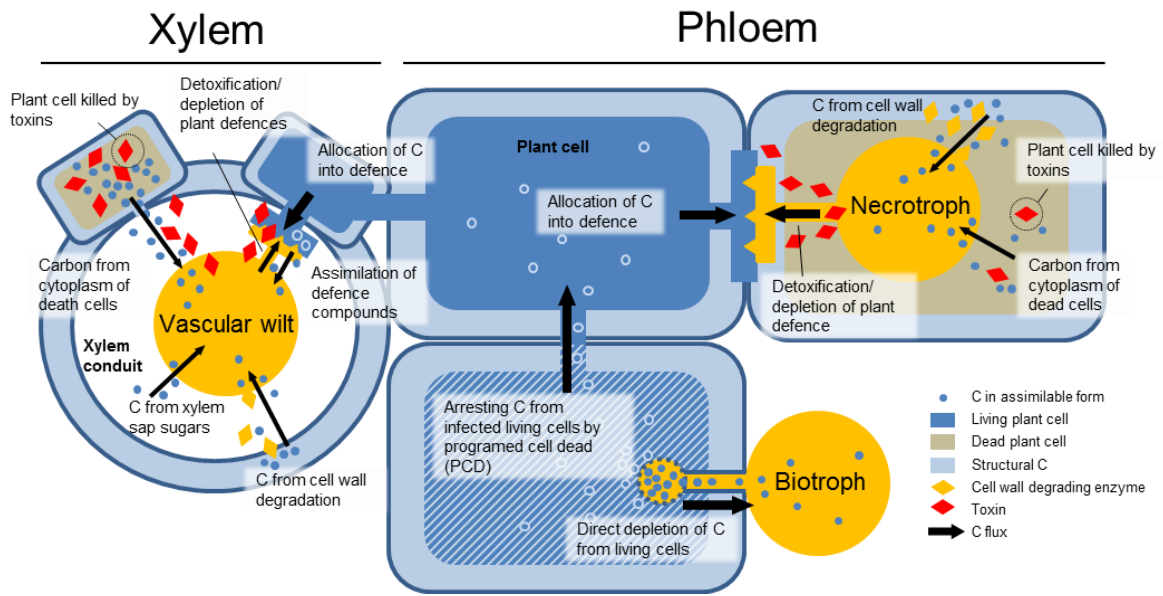
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Figure captions

468 **Figure 1. Carbon fluxes between host and pathogen depending on the type of trophic interaction**

469 **occurring in xylem and phloem.** Three types of pathogens are represented: biotrophs, necrotrophs

470 and vascular wilts. Biotrophic pathogens derive carbon directly from living cells. Although chemical

471 tree responses are triggered (not represented), the defence system of the tree is based on a

472 programmed cell death that removes the carbon from the infected cells and stops the flow of carbon to

473 the pathogen. Vascular wilt pathogens thrive inside xylem conduits where they feed on carbon leaking

474 from cells killed by means of toxins/enzymes and on carbon from xylem sap. They are also able to

475 metabolize defence compounds and can obtain sugars by degrading the xylem cell wall. Damages in

476 the xylem elicit carbon based defence responses. Necrotrophic pathogens kill living cells by secreting

477 enzymes and toxins and obtain nutrients from dead cells and from structural carbon sources such as

478 cellulose. Like vascular wilts, necrotrophs may also feed on tree defence compounds (not

479 represented). Trees defend themselves by compartmentalizing the pathogen within carbon-expensive

480 barriers.

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482 **Figure 2. Hypothetical mechanisms of mortality for biotrophic, necrotrophic and vascular wilt**

483 **pathogens under drought conditions.** Drought induced mortality is based on the model by

484 McDowell *et al.* (2011) and it is assumed to happen when the amount of NSC is smaller than the

485 amount of carbon needed to maintain osmotic regulation and xylem and phloem transport (marked as

486 *, in panels "**a**","**b**","**f**" and "**j**"). Pathogens affect NSC reserves differently depending on the type of

487 trophic interaction (Fig. 1), by directly depleting NSC reserves, accelerating NSC consumption and

488 increasing repair costs (**a**). The mechanism of mortality is shown for each type of pathogen and

489 separately depending on whether the attack occurs during drought (solid red line) or before the

490 drought period (dashed red line). For the sake of comparison, the process of mortality under drought

491 conditions without biotic agents is also shown (thick black line). In general, low water availability

492 causes stomatal closure and lower carbon assimilation. Under prolonged drought conditions the tree

493 may require using NSC reserves to maintain tissue growth and respiration. NSC may also be used to

494 restore the hydraulic system of the tree by refilling embolized conduits. If under pathogen attack, and

495 depending on the type of trophic interaction, trees elicit different responses impacting the acquisition,

496 storage and transport of water and carbon in the tree. The main effects of the drought pathogen

497 interaction on NSC, xylem and phloem transport and induced defence are represented. **Biotrophs** feed

498 directly on NSC reserves (**b**) without affecting phloem and xylem (**c**). The direct use of NSC by the

499 pathogen translates directly into pathogen population growth, which accelerates NSC depletion in a

500 positive feedback (**e**). In the case of drought, biotrophs cannot compete with plant tissues for carbon;

501 hence pathogen biomass decreases (**e**). Previous depletion of NSC by the biotroph can accelerate

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502 mortality due to lower reserves at the onset of the drought (**b**). A minimal biotroph establishment is
503 predicted during the drought owing the declining availability of carbon as water deficit intensifies (**e**),
504 and no interactive effects with drought are predicted for infections establishing during drought (**b**).
505 **Necrotrophs** find their main carbon source on phloem elements in leaves, stem, and roots (Fig. 1).
506 Under normal conditions they establish a long-term interaction requiring carbon investment from the
507 host into defence (**f**). Following phloem and xylem destruction, the host compartmentalizes the
508 pathogen further sacrificing more phloem and xylem (**g**). Eventual defence failures allow the
509 pathogen access to living cells and structural carbon sources (Fig. 1), from which it builds up further
510 biomass (**i**). Under drought, carbon for defence becomes scarce (**f**) allowing previous infections to
511 expand (**i**) causing further damage to phloem and xylem (**g**), increasing further the costs of
512 compartmentalization (**h**). As a result, we predict that necrotrophs exacerbate drought effects,
513 accelerating tree mortality (**f**). A similar outcome is expected for necrotroph infections occurring
514 during drought (**f**). Pathogen biomass remains after tree death (**h**) due to the capacity of this type of
515 pathogens to survive on dead tissues. **Vascular wilt** pathogens thrive in the xylem. Trees block
516 vertical pathogen spread by clogging the conduits (Fig. 1), with consequences for both xylem
517 transport (**k**) and NSC (**j**). Negative water potentials allow them to colonise large parts of the xylem
518 (**m,j**), causing foliage wilting and mortality of phloem tissues (**k**).
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