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**Commentary**

# Mechanisms of fire-caused tree death are far from resolved

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This scientific commentary refers to 'Stem heating results in hydraulic dysfunction in *Symplocos tinctoria*: implications for post-fire tree death' by Hoffmann et al. (https://doi.org/10.1093/treephys/tpae023).

How does fire kill trees? This is a fundamental question to which we surprisingly do not have the answer. The capacity to predict when and where trees will die due to fire is important for addressing a range of applied research problems, such as modelling fire effects on vegetation and dependent fauna, carbon emissions and modelling future fire regimes. As fire regimes continue to change due to climate change and other anthropogenic factors (Jones et al. 2022), gaps in our understanding of how trees respond to fire and other stressors are laid bare.

Several hypotheses have been proposed to explain fire-caused tree mortality. These hypotheses correspond to differential timing of death and have mixed levels of experimental support (Fig. 1). Immediate or direct causes of fire-induced mortality can be attributed to necrosis or consumption of leaves and meristems (Hood et al. 2018) or physical consumption of roots or the main stem leading to structural failure and tree collapse (Nolan et al. 2021). These mechanisms enjoy the strongest support, with widespread agreement about how fire can directly cause immediate mortality (Hood et al. 2018). For those species that resprout following fire, depletion of the bud bank may trigger tree death following complete canopy scorch or consumption; however, there is limited evidence for this (Nolan et al. 2021). Sub-lethal fire can also initiate complex physiological processes leading to delayed tree mortality in the weeks to years following fire. These mechanisms can be broadly grouped into those that cause carbon imbalance/starvation and those that cause hydraulic dysfunction (Michaletz et al. 2012; Bär et al. 2018; Dickman et al. 2023). Carbohydrate imbalance/starvation may take months or years to ultimately cause tree death (Reed and Hood 2024), while hydraulic dysfunction is likely to trigger tree death much more rapidly, as has been suggested for tree death due to drought (Choat et al. 2018).

In the recent paper by Hoffmann et al. (2024), the authors focus on how fire can cause tree hydraulic dysfunction. There are two main hypothesized mechanisms by which fire-catalyzed hydraulic dysfunction can result in tree death. The first mechanism is the heat plume hypothesis, which proposes that the extreme temperatures and dry air that occur during fire cause a sudden loss of water, triggering extensive cavitation, which may cause hydraulic failure or limit the capacity of the hydraulic system to withstand further stressors (Michaletz and Johnson 2007; Kavanagh et al. 2010; Midgley et al. 2011; Bär et al. 2018; Lodge et al. 2018; Salladay and Pittermann 2023). Research from the drought literature indicates that tree mortality occurs when >50% of xylem is embolized in gymnosperms and >88% of xylem is embolized in angiosperms (Choat et al. 2018). The second mechanism is the xylem deformation hypothesis, which proposes that heat causes deformation of xylem cell walls, thereby restricting water flow and potentially making branches or stems more vulnerable to cavitation (Michaletz et al. 2012; West et al. 2016; Bär et al. 2018). While experimental heating of branches has been demonstrated to cause hydraulic dysfunction, possibly via both mechanisms (Michaletz et al. 2012; Lodge et al. 2018), there is mixed evidence that xylem deformation is the ultimate cause of postfire tree mortality if phloem and cambium necrosis occur under lower temperatures. For example, in a heating experiment on *Sequoia sempervirens*, Salladay and Pittermann (2023) found that a greater 'dose' of heat was required to damage xylem function than to trigger cambium necrosis, suggesting that heat-induced xylem deformation may not be a relevant mechanism of mortality, and damage to the cambium and phloem via heat-caused necrosis may be the more common cause of tree death.

To investigate the impact of fire on hydraulic dysfunction via either hypothesis, Hoffmann et al. (2024) examine the temporal progression of plant hydraulic status following a field-based stem-heating experiment on an understorey tree, *Symplocos tinctoria*. One of the main advances provided by Hoffmann et al. (2024) is their quantification of the timeframe over which stem heating causes hydraulic dysfunction and, ultimately, tree death. Importantly, the study finds that there was an immediate, but gradual and progressive decline in sap flow, stomatal conductance and whole-plant hydraulic conductivity following stem heating over 1–2 weeks, with tree death occurring over 2–3 weeks. Yet, water stress, as

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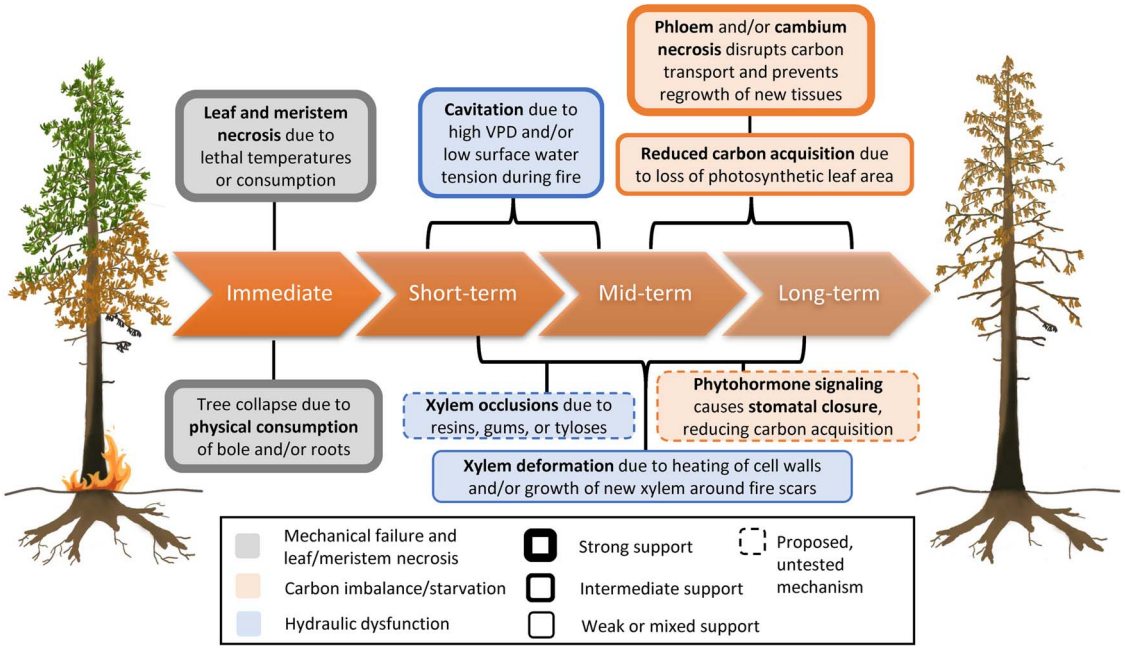


**Figure 1.** Overview of the hypothesized mechanisms of fire-caused tree death illustrating the differential timing of the mechanisms and their level of support in the literature. The dashed boxes are new potential mechanisms that may contribute to tree mortality based on the results of Hoffmann et al. (2024) (VPD, vapor pressure deficit).

measured by leaf water potential, remained steady before rapidly increasing (i.e. leaf water potential became more negative) about 10 days after the heating treatment. These results provide valuable insight into the timeframe within which fire-caused hydraulic dysfunction can lead to tree mortality, and useful guidance for future studies on the timing of measurements of xylem function and coordination of within-plant water status. Specifically, measurements made in the days immediately following fire or stem heating experiments are unlikely to detect the full impacts of heat on hydraulic function.

In contrast to previous studies, Hoffmann et al. (2024) found no direct evidence that fire causes xylem cavitation. However, this may be due in part to their methods of simulating fire, which involved directly applying heat to a stem section. This method does not simulate the extreme vapor pressure deficit that could cause a sudden loss of water from foliage, triggering cavitation (Kavanagh et al. 2010), although stem heating could still potentially cause cavitation via the reduction of the surface tension of water (Michaletz et al. 2012). Hoffmann et al. (2024) also found no evidence of xylem deformation. While deformation of existing xylem has been observed in studies using water baths (Michaletz et al. 2012, West et al. 2016, Bär et al. 2018), it has not been observed following fire-based or externally applied heat experiments (Bär et al. 2018, Partelli-Feltrin et al. 2021). However, Partelli-Feltrin et al. (2021) did show deformation of new xylem formed after fire, which increased xylem vulnerability 14–21 months posttreatment. The hydraulic dysfunction observed by Hoffmann et al. (2024) was instead attributed to a new potential mechanism, namely physical occlusions in the xylem, although these were not directly observed when xylem was examined under scanning electron microscope. The formation of occlusions is a plausible explanation for the decline in conductance and sapflow, as repeated attempts to flush stems and recover conductivity were unsuccessful. That previous studies find evidence for differing mechanisms causing hydraulic dysfunction may reflect the timing of observations or may be an artefact of the different methods used to simulate fire effects, which points to the need for standardized protocols for experimental pyro-ecophysiology studies (Hood 2021). Importantly, all of the studies that have shown fire impacts on hydraulic function apply doses of heat that would also cause necrosis of the cambium and phloem, which, even if complete heat-caused girdling does not occur, may have consequences for photosynthate transport and whole-tree carbon balance.

Tissue necrosis, whether to phloem and cambium in branches, roots and stems, or to tree foliage, is well understood to play an important role in tree mortality following fire, primarily via its impact on plant carbon balance (Michaletz and Johnson 2007; Bär et al. 2018). Although Hoffmann et al. (2024) do not address impacts to carbon balance initiated by phloem and cambium death in their study, they do highlight their potential importance in their discussion. Reduction of photosynthetic leaf area via foliage necrosis (e.g. crown scorch or consumption) can deplete stored nonstructural carbohydrates (NSCs) and reduce NSC acquisition (Varner et al. 2021; Dickman et al. 2023), which has been linked to postfire tree mortality (Reed and Hood 2024). This mechanism of delayed postfire tree mortality is particularly important for non resprouting species, although repeated fires and those that cause extensive meristematic damage may potentially lead to resprouting exhaustion due to depletion of stored NSCs in resprouters as well (Clarke et al. 2013; Smith et al. 2018; Nolan et al. 2021). Heat transfer through the bark can also cause necrosis, first of the phloem and then to the cambium as heat is conducted inward. If the entire stem circumference is affected (as in the Hoffmann et al. (2024) experiment), heat-caused girdling can occur. If

cambial death prevents phloem regeneration, photosynthate translocation is disrupted, resulting in downstream depletion of NSCs (Partelli-Feltrin et al. 2023). Whether carbohydrate imbalance is due to cambium and phloem necrosis, reduced photosynthetic leaf area or some combination of the two mechanisms, depletion of accessible carbohydrate reserves can take months or years to cause mortality (Noel 1970; Reed and Hood 2024). Depletion or translocation impairment of NSCs also likely increases vulnerability to postfire drought, insects and disease, as NSCs are important for recovery and regrowth of fire-injured tissues, as well as postfire drought tolerance and defence against insects and disease (Hood et al. 2018). While Hoffmann et al. (2024) do not quantify NSCs, numerous studies highlight their likely important role in postfire tree recovery, maintenance of hydraulic function, and survival, and future studies should consider examining concurrent impacts to both carbon balance and hydraulic function.

Hoffmann et al. (2024) provide a valuable discussion on the relative importance of these different mechanisms: 'It is not entirely clear that this [hydraulic failure] is an important mechanism of tree death, given that most stems killed by heating of xylem are already doomed to die from necrosis of phloem and cambium . . . ' Hoffmann et al. (2024) go on to argue that it is important to understand the role of xylem dysfunction in postfire tree death when the timing of tree death matters: 'The much quicker mortality caused by xylem dysfunction predisposes post-fire landscapes to more rapid vegetation change and more abrupt changes in ecosystem services . . . ' Such thoughtful discussion about how mechanisms leading to fire-caused tree mortality influences timing of death, and the larger ecological implications, serves as a good reminder that there is unlikely one single mechanism, but rather multiple, interacting causes that require integrative experiments to resolve.

Another key advance provided by Hoffmann et al. (2024) is the presentation of a novel hypothesis that phytohormones, such as abscisic acid (ABA), may contribute to tree death following fire. Abscisic acid is synthesized in response to plant stress and triggers stomatal closure (Chen et al. 2020). In the Hoffmann et al. (2024) study, stomatal closure occurred rapidly following stem heating, with water potential slightly increasing (i.e. becoming less negative) for about 10 days post-treatment. If hormonal signalling from stem heating closed stomates, a concomitant decrease in NSC acquisition would occur. The ability of fire to inhibit photosynthesis through signalling-induced stomatal closure deserves further study.

While Hoffmann et al. (2024) do not explicitly determine the mechanism by which heating causes hydraulic dysfunction in their study, they propose novel hypotheses regarding links between hydraulic dysfunction and fire-induced wound responses such as physical occlusions in the xylem or ABA signalling. Perhaps most importantly, they acknowledge the complexities of understanding tree physiological responses to fire, highlighting the multiple interacting mechanisms likely involved. Hoffmann et al. (2024) and other studies increasingly find little support for heat-caused hydraulic dysfunction via xylem cavitation or deformation to directly cause mortality in the first few weeks after fire. Proposed mechanisms of fire-caused tree death—tissue necrosis, physical consumption, carbon imbalance/starvation, and hydraulic dysfunction via xylem cavitation, deformation, occlusions and/or hormone signalling—require further investigation to resolve this fundamental question and to determine the relative importance and timing of each mechanism across ecosystems (Figure 1). Field observations following wildfire may be confounded by other effects on tree function, such as drought, while experimental studies on cut branches or saplings may fail to adequately simulate realistic fire effects. Planning experiments around prescribed fires may overcome some of the disadvantages of laboratory experiments and provides an opportunity to collect pre- and postfire observations (Cawson and Duff 2018; Reed and Hood 2024). As highlighted by Hoffmann et al. (2024), studies should ideally monitor tree physiology closely in the initial weeks following fire, and also over months to years, to fully quantify the physiological responses of trees to fire.

## Authors' contributions

R.H.N., C.C.R., and S.M.H. contributed equally to the conceptualization. R.N.H. led the writing; C.C.R. led the development of the conceptual diagram. All authors contributed to editing and revising the manuscript.

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## Conflict of interest

None declared.

## Data availability

No new data or materials were created or analyzed in this study. Data sharing is not applicable to this article.

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