



**HAL**  
open science

## A synthesis of radial growth patterns preceding tree mortality

Maxime Cailleret, Steven Jansen, Elisabeth M. R. Robert, Lucia Desoto, Tuomas Aakala, Joseph A. Antos, Barbara Beikircher, Christof Bigler, Harald Bugmann, Marco Caccianiga, et al.

► **To cite this version:**

Maxime Cailleret, Steven Jansen, Elisabeth M. R. Robert, Lucia Desoto, Tuomas Aakala, et al.. A synthesis of radial growth patterns preceding tree mortality. *Global Change Biology*, 2016, 23 (4), 16 p. 10.1111/gcb.13535 . hal-01506661

**HAL Id: hal-01506661**

**<https://hal.science/hal-01506661>**

Submitted on 1 Jan 2024

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

Received Date : 08-Jun-2016

Revised Date : 12-Sep-2016

Accepted Date : 11-Oct-2016

Article type : Primary Research Articles

## A synthesis of radial growth patterns preceding tree mortality

**Running head:** Growth patterns preceding tree mortality

MAXIME CAILLERET<sup>1\*</sup>, STEVEN JANSEN<sup>2</sup>, ELISABETH M. R. ROBERT<sup>3, 4, 5</sup>, LUCÍA DESOTO<sup>6</sup>, TUOMAS AAKALA<sup>7</sup>, JOSEPH A. ANTOS<sup>8</sup>, BARBARA BEIKIRCHER<sup>9</sup>, CHRISTOF BIGLER<sup>1</sup>, HARALD BUGMANN<sup>1</sup>, MARCO CACCIANIGA<sup>10</sup>, VOJTĚCH ČADA<sup>11</sup>, J. JULIO CAMARERO<sup>12</sup>, PAOLO CHERUBINI<sup>13</sup>, HERVÉ COCHARD<sup>14</sup>, MARIE R. COYEA<sup>15</sup>, KATARINA ČUFAR<sup>16</sup>, ADRIAN J. DAS<sup>17</sup>, HENDRIK DAVI<sup>18</sup>, SYLVAIN DELZON<sup>19</sup>, MICHAEL DORMAN<sup>20</sup>, GUILLERMO GEA-IZQUIERDO<sup>21</sup>, STEN GILLNER<sup>22, 23</sup>, LAUREL J. HAAVIK<sup>24, 25</sup>, HENRIK HARTMANN<sup>26</sup>, ANA-MARIA HERES<sup>3, 27</sup>, KEVIN R. HULTINE<sup>28</sup>, PAVEL JANDA<sup>11</sup>, JEFFREY M. KANE<sup>29</sup>, VYACHESLAV I. KHARUK<sup>30</sup>, THOMAS KITZBERGER<sup>31, 32</sup>, TAMIR KLEIN<sup>33</sup>, KOEN KRAMER<sup>34</sup>, FREDERIC LENS<sup>35</sup>, TOM LEVANIC<sup>36</sup>, JUAN CARLOS LINARES CALDERON<sup>37</sup>, FRANCISCO LLORET<sup>3, 38</sup>, RAQUEL LOBO-DO-VALE<sup>39</sup>, FABIO LOMBARDI<sup>40</sup>, ROSANA LÓPEZ RODRÍGUEZ<sup>41, 42</sup>, HARRI MÄKINEN<sup>43</sup>, STEFAN MAYR<sup>9</sup>, ILONA MÉSZÁROS<sup>44</sup>, JUHA M. METSARANTA<sup>45</sup>, FRANCESCO MINUNNO<sup>7</sup>, WALTER OBERHUBER<sup>9</sup>, ANDREAS PAPAPOULOS<sup>46</sup>, MIKKO PELTONIEMI<sup>47</sup>, ANY MARY PETRITAN<sup>13, 48</sup>, BRIGITTE ROHNER<sup>1, 13</sup>, GABRIEL SANGÜESA-BARRERA<sup>12</sup>, DIMITRIOS SARRIS<sup>49, 50, 51</sup>, JEREMY M. SMITH<sup>52</sup>, AMANDA B. STAN<sup>53</sup>, FRANK STERCK<sup>54</sup>, DEJAN B. STOJANOVIĆ<sup>55</sup>, MARIA LAURA SUAREZ<sup>32</sup>, MIROSLAV SVOBODA<sup>11</sup>, ROBERTO TOGNETTI<sup>56, 57</sup>, JOSÉ M. TORRES-RUIZ<sup>19</sup>, VOLODYMYR TROTSIUK<sup>11</sup>, RICARDO VILLALBA<sup>58</sup>, FLOOR VODDE<sup>59</sup>, ALANA R. WESTWOOD<sup>60</sup>, PETER H. WYCKOFF<sup>61</sup>, NIKOLAY ZAFIROV<sup>62</sup> and JORDI MARTÍNEZ-VILALTA<sup>3, 38</sup>

This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the [Version of Record](#). Please cite this article as [doi: 10.1111/gcb.13535](https://doi.org/10.1111/gcb.13535)

This article is protected by copyright. All rights reserved

30 <sup>1</sup>Forest Ecology, Institute of Terrestrial Ecosystems, Department of Environmental Systems Science,  
31 ETH Zürich, Universitätstrasse 22, 8092 Zürich, Switzerland

32 <sup>2</sup>Institute of Systematic Botany and Ecology, Ulm University, Albert-Einstein-Allee 11, 89081 Ulm,  
33 Germany

34 <sup>3</sup>CREAF, Campus UAB, 08193 Cerdanyola del Vallès, Spain

35 <sup>4</sup>Laboratory of Plant Biology and Nature Management (APNA), Vrije Universiteit Brussel, Pleinlaan 2,  
36 1050 Brussels, Belgium

37 <sup>5</sup>Laboratory of Wood Biology and Xylarium, Royal Museum for Central Africa (RMCA),  
38 Leuvensesteenweg 13, 3080 Tervuren, Belgium

39 <sup>6</sup>Centre for Functional Ecology, Department of Life Sciences, University of Coimbra, Calçada Martim  
40 de Freitas, 3000-456 Coimbra, Portugal

41 <sup>7</sup>Department of Forest Sciences, University of Helsinki, P.O. Box 27 (Latokartanonkaari 7), 00014  
42 Helsinki, Finland

43 <sup>8</sup>Department of Biology, University of Victoria, PO Box 3020, STN CSC, Victoria, B.C. V8W 3N5,  
44 Canada

45 <sup>9</sup>Institute of Botany, University of Innsbruck, Sternwartestrasse 15, 6020 Innsbruck, Austria

46 <sup>10</sup>Dipartimento di Bioscienze, Università degli Studi di Milano, Via Giovanni Celoria 26, 20133  
47 Milano, Italy

48 <sup>11</sup>Faculty of Forestry and Wood Sciences, Czech University of Life Sciences, Kamýcká 961/129, 165  
49 21 Praha 6-Suchbát, Czech Republic

50 <sup>12</sup>Instituto Pirenaico de Ecología (IPE-CSIC), Avenida Montañana 1005, 50192 Zaragoza, Spain

51 <sup>13</sup>Swiss Federal Institute for Forest, Snow and Landscape Research - WSL, Zürcherstrasse 111, 8903  
52 Birmensdorf, Switzerland

53 <sup>14</sup>Unité Mixte de Recherche (UMR) 547 PIAF, Institut National de la Recherche Agronomique (IN-  
54 RA), Université Clermont Auvergne, 63100 Clermont-Ferrand, France

55 <sup>15</sup>Centre for Forest Research, Département des sciences du bois et de la forêt, Faculté de foresterie, de  
56 géographie et de géomatique, Université Laval, 2405 rue de la Terrasse, Québec, Québec G1V 0A6,  
57 Canada

58 <sup>16</sup>Biotechnical Faculty, University of Ljubljana, Jamnikarjeva 101, 1000 Ljubljana, Slovenia

59 <sup>17</sup> U.S. Geological Survey, Western Ecological Research Center, 47050 Generals Highway, Three Riv-  
60 ers, California 93271 USA

- 61 <sup>18</sup>Ecologie des Forest Méditerranéennes (URFM), Institut National de la Recherche Agronomique (IN-  
62 RA), Domaine Saint Paul, Site Agroparc, 84914 Avignon Cedex 9, France
- 63 <sup>19</sup>Unité Mixte de Recherche (UMR) 1202 BIOGECO, Institut National de la Recherche Agronomique  
64 (INRA), Université de Bordeaux, 33615 Pessac, France
- 65 <sup>20</sup>Department of Geography and Environmental Development, Ben-Gurion University of the Negev,  
66 84105 Beer-Sheva, Israel
- 67 <sup>21</sup>Centro de Investigación Forestal (CIFOR), Instituto Nacional de Investigación y Tecnología Agraria  
68 y Alimentaria (INIA), Carretera La Coruña km 7.5, 28040 Madrid, Spain
- 69 <sup>22</sup>Institute of Forest Botany and Forest Zoology, TU Dresden, 01062 Dresden, Germany
- 70 <sup>23</sup>Fachgebiet Vegetationstechnik und Pflanzenverwendung, Institut für Landschaftsarchitektur und  
71 Umweltplanung, TU Berlin, 10623 Berlin, Germany
- 72 <sup>24</sup>Department of Entomology, University of Arkansas, Fayetteville, Arkansas 72701, United States of  
73 America
- 74 <sup>25</sup>Department of Ecology and Evolutionary Biology, University of Kansas, 1450 Jayhawk Boulevard,  
75 Lawrence, Kansas 66045, United States of America
- 76 <sup>26</sup>Max-Planck Institute for Biogeochemistry, Hans Knöll Strasse 10, 07745 Jena, Germany
- 77 <sup>27</sup>Department of Biogeography and Global Change, National Museum of Natural History (MNCN),  
78 Consejo Superior de Investigaciones Científicas (CSIC), C/Serrano 115bis, 28006 Madrid, Spain
- 79 <sup>28</sup>Department of Research, Conservation and Collections, Desert Botanical Garden, 1201 N Galvin  
80 Parkway, Phoenix, Arizona, United States of America
- 81 <sup>29</sup>Department of Forestry and Wildland Resources, Humboldt State University, 1 Harpst Street, Arcata,  
82 California 95521, United States of America
- 83 <sup>30</sup>Sukachev Institute of Forest, Siberian Division of the Russian Academy of Sciences (RAS), Krasno-  
84 yarsk 660036, Russia
- 85 <sup>31</sup>Department of Ecology, Universidad Nacional del Comahue, Quintral S/N, Barrio Jardín Botánico,  
86 8400 San Carlos de Bariloche, Río Negro, Argentina
- 87 <sup>32</sup>Instituto de Investigaciones de Biodiversidad y Medio Ambiente (INIBOMA), Consejo Nacional de  
88 Investigaciones Científicas y Técnicas (CONICET), Quintral 1250, 8400 San Carlos de Bariloche, Río  
89 Negro, Argentina
- 90 <sup>33</sup>Institute of Soil, Water, and Environmental Sciences, Agricultural Research Organization (ARO),  
91 Volcani center, PO Box 6, 50250 Beit Dagan, Israel
- 92 <sup>34</sup>Alterra - Green World Research, Wageningen University, Droevendaalse steeg 1, 6700AA  
93 Wageningen, the Netherlands

- 94 <sup>35</sup>Naturalis Biodiversity Center, Leiden University, PO Box 9517, 2300RA Leiden, The Netherlands
- 95 <sup>36</sup>Department of Yield and Silviculture, Slovenian Forestry Institute, Večna pot 2, 1000 Ljubljana, Slo-
- 96 venia
- 97 <sup>37</sup>Department of Physical, Chemical and Natural Systems, Pablo de Olavide University, Carretera de
- 98 Utrera km 1, 41013 Seville, Spain
- 99 <sup>38</sup>Universitat Autònoma de Barcelona, 08193 Cerdanyola del Vallès, Spain
- 100 <sup>39</sup>Forest Research Centre, School of Agriculture, University of Lisbon, Tapada da Ajuda, 1349-017
- 101 Lisboa, Portugal
- 102 <sup>40</sup>Department of Agricultural Science, Mediterranean University of Reggio Calabria, loc. Feo di Vito,
- 103 89060 Reggio Calabria, Italy
- 104 <sup>41</sup>Forest Genetics and Physiology Research Group, Technical University of Madrid, Calle Ramiro de
- 105 Maeztu 7, 28040 Madrid, Spain
- 106 <sup>42</sup>Hawkesbury Institute for the Environment, University of Western Sydney, Science Road, Richmond,
- 107 New South Wales 2753, Australia
- 108 <sup>43</sup>Natural Resources Institute Finland (Luke), Viikinkaari 4, 00790 Helsinki, Finland
- 109 <sup>44</sup>Department of Botany, Faculty of Science and Technology, University of Debrecen, Egyetem tér 1,
- 110 4032 Debrecen, Hungary
- 111 <sup>45</sup>Northern Forestry Centre, Canadian Forest Service, Natural Resources Canada, 5320-122nd Street,
- 112 Edmonton, Alberta, Canada T6H 3S5
- 113 <sup>46</sup>Department of Forestry and Natural Environment Management, Technological Educational Institute
- 114 (TEI) of Stereas Elladas, Ag Georgiou 1, 36100 Karpenissi, Greece
- 115 <sup>47</sup>Natural Resources Institute Finland (Luke), PO Box 18 (Jokiniemenkuja 1), 01301 Vantaa, Finland
- 116 <sup>48</sup>National Institute for Research-Development in Forestry ‘‘Marin Dracea’’, Eroilor 128, 077190
- 117 Voluntari, Romania
- 118 <sup>49</sup>Faculty of Pure and Applied Sciences, Open University of Cyprus, Latsia, 2252 Nicosia, Cyprus
- 119 <sup>50</sup>Department of Biological Sciences, University of Cyprus, PO Box 20537, 1678 Nicosia, Cyprus
- 120 <sup>51</sup>Division of Plant Biology, Department of Biology, University of Patras, 26500 Patras, Greece
- 121 <sup>52</sup>Department of Geography, University of Colorado, Boulder, Colorado 80309-0260, United States of
- 122 America
- 123 <sup>53</sup>Department of Geography, Planning and Recreation, Northern Arizona University, PO Box 15016,
- 124 Flagstaff, Arizona 86011, United States of America
- 125 <sup>54</sup>Forest Ecology and Forest Management Group, Wageningen University, Droevendaalsesteeg 3a,
- 126 6708 PB Wageningen, The Netherlands

127 <sup>55</sup>Institute of Lowland Forestry and Environment, University of Novi Sad, Antona Cehova 13, PO Box  
128 117, 21000 Novi Sad, Serbia

129 <sup>56</sup>Dipartimenti di Bioscienze e Territorio, Università del Molise, C. da Fonte Lappone, 86090 Pesche,  
130 Italy

131 <sup>57</sup>European Forest Institute (EFI) Project Centre on Mountain Forests (MOUNTFOR), Via E. Mach 1,  
132 38010 San Michele all'Adige, Italy

133 <sup>58</sup>Laboratorio de Dendrocronología e Historia Ambiental, Instituto Argentino de Nivología, Glaciología  
134 y Ciencias Ambientales (IANIGLA), CCT CONICET Mendoza, Av. Ruiz Leal s/n, Parque General  
135 San Martín, Mendoza, Argentina CP 5500

136 <sup>59</sup>Institute of Forestry and Rural Engineering, Estonian University of Life Sciences, Kreutzwaldi 5,  
137 51014 Tartu, Estonia

138 <sup>60</sup>Boreal Avian Modelling Project, Department of Renewable Resources, University of Alberta, 751  
139 General Services Building, Edmonton, Alberta, Canada T6G 2H1

140 <sup>61</sup>University of Minnesota, 600 East 4<sup>th</sup> Street, Morris, Minnesota 56267, United States of America

141 <sup>62</sup>University of Forestry, Kliment Ohridski Street 10, 1756 Sofia, Bulgaria.

142

143 \*Corresponding author:

144 Maxime Cailleret

145 Forest Ecology, Institute of Terrestrial Ecosystems, Department of Environmental Systems Science,  
146 ETH Zürich, CHN G77, Universitätstrasse 16, 8092 Zürich, Switzerland.

147 Tel: +41 44 632 52 08; Fax: +41 44 632 13 58

148 E-Mail: cailleret.maxime@gmail.com

149

## 150 **Abstract**

151 Tree mortality is a key factor influencing forest functions and dynamics, but our understanding of the  
152 mechanisms leading to mortality and the associated changes in tree growth rates are still limited. We  
153 compiled a new pan-continental tree-ring width database from sites where both dead and living trees  
154 were sampled (2,970 dead and 4,224 living trees from 190 sites, including 36 species), and compared  
155 early and recent growth rates between trees that died and those that survived a given mortality event.

156 We observed a decrease in radial growth before death in ca. 84% of the mortality events. The extent  
157 and duration of these reductions were highly variable (1-100 years in 96% of events) due to the com-  
158 plex interactions among study species and the source(s) of mortality. Strong and long-lasting declines

159 were found for gymnosperms, shade- and drought-tolerant species, and trees that died from competi-  
160 tion. Angiosperms and trees that died due to biotic attacks (especially bark-beetles) typically showed  
161 relatively small and short-term growth reductions. Our analysis did not highlight any universal trade-  
162 off between early growth and tree longevity within a species, although this result may also reflect high  
163 variability in sampling design among sites.

164 The inter-site and inter-specific variability in growth patterns before mortality provides valuable infor-  
165 mation on the nature of the mortality process, which is consistent with our understanding of the physio-  
166 logical mechanisms leading to mortality. Abrupt changes in growth immediately before death can be  
167 associated with generalized hydraulic failure and/or bark beetle attack, while long-term decrease in  
168 growth may be associated with a gradual decline in hydraulic performance coupled with depletion in  
169 carbon reserves. Our results imply that growth-based mortality algorithms may be a powerful tool for  
170 predicting gymnosperm mortality induced by chronic stress, but not necessarily so for angiosperms and  
171 in case of intense drought or bark-beetle outbreaks.

172

173

174 **Keywords:**

175 Tree mortality, growth, ring-width, drought, pathogens, angiosperms, gymnosperms, death

176

177 **Article type:**

178 Primary research article

179 **Introduction**

180

181 Accelerating rates of tree mortality and forest die-off events have been reported worldwide (e.g., van  
182 Mantgem et al. 2009; Allen et al. 2010). These trends have been attributed to direct and indirect im-  
183 pacts of drought stress and higher temperatures (e.g., higher competition intensity as a result of growth  
184 enhancement in environments limited by low temperature; Luo and Chen 2015), and are expected to  
185 continue as a result of further global warming and drying in many regions (Cook et al. 2014; Allen et  
186 al. 2015). Tree mortality has large impacts on both short-term forest functioning (e.g., forest produc-  
187 tivity, water and carbon cycles; Anderegg et al. 2016b) and long-term ecosystem dynamics (Franklin  
188 1987; Millar et al. 2015), yet our physiological understanding of the mechanisms leading to mortality  
189 and our ability to predict mortality and its impacts over space and time is still limited (McDowell et al.  
190 2013; Hartmann et al. 2015). As a result, most dynamic vegetation models that aim to project future

191 forest development are still based on simple mortality algorithms despite their high sensitivity to mor-  
192 tality assumptions (Friend et al. 2014; Bircher et al. 2015). In addition, reliable indicators that can be  
193 used to predict individual mortality in the field from local to regional scales are lacking (McDowell et  
194 al. 2013).

195 In contrast to most mortality events caused by short-term external disturbances, such as windthrow, fire  
196 or flooding, stress-induced mortality is usually preceded by changes in tree function (e.g., hydraulic  
197 conductivity, carbon assimilation) and structure (e.g., individual leaf area) (McDowell et al. 2011; Seidl  
198 et al. 2011; but see Nesmith et al. 2015 for potential influence of pre-fire growth on post-fire mortality).  
199 In this context, focusing on the temporal variations in radial stem growth rates is pertinent as they re-  
200 flect changes in individual vitality, productivity, and carbon availability (Babst et al. 2014; Aguadé et  
201 al. 2015; Dobbertin 2005). Although the inter-annual variability in wood growth is primarily driven by  
202 cambial phenology and activity (Delpierre et al. 2015; Körner 2015) – thus by water availability, air  
203 temperature and photoperiod – several studies have shown the utility of radial growth data for predict-  
204 ing tree mortality probability (e.g., Pedersen 1998; Bigler and Bugmann 2004; Wunder et al. 2008;  
205 Cailleret et al. 2016). Most studies used ring-width data as they allow for a long-term (i.e., >20 years)  
206 retrospective quantification of annual growth for numerous individuals, sites, and species (e.g.,  
207 Anderegg et al. 2015a). Such data offer the further advantage of combining a large sample size (in con-  
208 trast to, for example, dendrometers) with a annual temporal resolution that is helpful to estimate the  
209 year of tree death and to detect immediate reactions to intense stress such as drought or insect defolia-  
210 tion (Dobbertin 2005), unlike forest inventories with multi-year re-measurement periods. Moreover,  
211 ring-width data are usually available for almost the entire lifespan of a tree, which is valuable for ex-  
212 ploring long-term and delayed effects of stress on mortality (see Bigler et al. 2007) that would not be  
213 detected using methods such as carbon flux measurements or remote sensing.

214 In most studies, dying trees showed lower radial growth rates prior to death than surviving ones (e.g.,  
215 Pedersen 1998; Bigler and Bugmann 2004; Cailleret et al. 2016). Despite this common pattern, a large  
216 variety of growth patterns before mortality have been described in the literature from abrupt or gradual  
217 growth reductions to increases in growth before death. This variability is likely associated with differ-  
218 ences in species' strategies to face environmental stress, and in their carbon allocation patterns related  
219 to growth, defense, and storage (Dietze et al. 2014); for example, stress-tolerant species may survive  
220 for many years with low growth rates under continuously stressful conditions (e.g., old *Pinus longaeva*),  
221 while stress-sensitive species cannot (e.g., *Populus tremuloides*; Ireland et al. 2014). There is also sub-



222 stantial variability at the intra-specific level: drought-induced mortality events of *Pinus sylvestris* may  
223 be preceded by fast declines (Herguido et al. 2016), or by slow and long-lasting growth reductions  
224 (Bigler et al. 2006; Hereş et al. 2012).

225 Growth patterns before death are also influenced by the type, duration, frequency, and intensity of  
226 stress factors that predisposed and triggered mortality. For *Picea engelmannii*, dying trees had lower  
227 growth rates than surviving trees when mortality was caused by drought (Bigler et al. 2007), while no  
228 differences were observed in two pine species when trees died because of bark beetles (Kane and Kolb  
229 2010; Ferrenberg et al. 2014; Sangüesa-Barreda et al. 2015). In case of lethal episodic defoliation, tree  
230 death can even be preceded by growth increases (e.g., on *Tamarix spp.* in Hultine et al. 2013). Similar-  
231 ly, intra-specific trade-offs between early growth rates (defined as the first 50 years of a tree's life) and  
232 longevity were commonly - but not consistently - observed (Bigler 2016; but see Ireland et al. 2014),  
233 highlighting the potential disadvantage of investment in growth instead of defenses (Herms and  
234 Mattson 1992; Rose et al. 2009).

235 Considering the multifactorial character of the mortality process (McDowell et al. 2011; Aguadé et al.  
236 2015; Allen et al. 2015; Anderegg et al. 2015b), and the limited number of species and sites analyzed in  
237 most earlier studies, we lack a global, comprehensive appraisal of the changes in growth rates before  
238 mortality. This is especially relevant to the detection of variations among sources of mortality (e.g.,  
239 drought, insect outbreak), environmental conditions, and species, and to the simulation of tree mortality  
240 using growth-based models (Bircher et al. 2015). Moreover, the available studies applied different  
241 methodologies to derive growth-mortality relationships (see Cailleret et al. 2016), which reduces the  
242 strength of meta-analyses. Thus, we compiled a new pan-continental tree-ring width database from  
243 published and unpublished datasets that include both dead and living trees growing at the same sites.  
244 We compare the growth rates between trees that died and those that survived stress events. In particu-  
245 lar, we address the following questions: (i) Are there characteristic changes in recent radial growth pri-  
246 or to mortality? (ii) Did dead trees have higher growth rates when they were young than surviving  
247 trees? (iii) To what extent are these growth patterns affected by structure-function differences between  
248 gymnosperms and angiosperms, and by the shade and/or drought tolerance of a particular species?, and  
249 (iv) are these patterns different depending on the main cause of mortality?

250 We hypothesize on the one hand that short-term (i.e., <5 years) or no decline in growth before death  
251 will occur in case of severe biotic attack (especially bark beetles), or in case of drought-induced embo-  
252 lism of xylem conduits that impedes water transport to the canopy and leads to tissue desiccation ('hy-

253 draumatic failure' hypothesis; McDowell et al. 2011; Rowland et al. 2015). On the other hand, long-term  
254 growth reductions (i.e., >20 years) before mortality will be more likely in response to repeated and  
255 gradually increasing environmental stress such as shading or parasitism (e.g., mistletoe), where a slow  
256 deterioration of the water and carbon economy may lead to tree death because of a lack of non-  
257 structural carbohydrates (NSC) to sustain metabolic processes like respiration or to build defense com-  
258 pounds ('carbon starvation' hypothesis; McDowell et al. 2011; Hartmann 2015). Accordingly, we ex-  
259 pect longer-term growth reductions in shade- and drought-tolerant species than in stress-sensitive ones,  
260 and in gymnosperms than in angiosperms, especially due to the wider hydraulic safety margins of conif-  
261 ers (Choat et al. 2012). We also hypothesize that trees that died during a specific mortality event will  
262 show higher juvenile growth rates than surviving trees (Bigler 2016).

263

## 264 **Materials and Methods**

265

### 266 *Tree-ring width database*

267 We compiled tree-ring width data (RW; mm) from 58 published and unpublished studies dealing with  
268 tree growth and mortality and that satisfied the following constraints: (1) mortality was mainly induced  
269 by stress, and not by abrupt abiotic disturbances such as windthrow, fire or flooding that may kill trees  
270 irrespective of their vitality and growth (but see Nesmith et al. 2015); (2) both dying and surviving  
271 trees were growing together at the same site; and (3) all individual chronologies had been successfully  
272 cross-dated. Overall, the dataset analyzed here included 2,970 dead and 4,224 living trees growing at  
273 190 sites mostly in North America and Europe in the boreal, temperate and Mediterranean biomes (Fig.  
274 1; Table 1; see details in Appendix S1).

275 The sampling approach varied widely across studies. Tree-ring data were derived from cores or cross-  
276 sections taken at different sampling heights, from the base to eight meters of height. At 30 sites (15.8%  
277 of the sites), tree-ring data were only available for the outermost rings (i.e., partial data). Estimates of  
278 cambial age and measures of tree diameter at breast height (DBH) at the time of coring were missing  
279 for 58 (30.5%) and 21 (11.1%) sites, respectively, which renders these data inappropriate for our anal-  
280 yses. Trees can die during the growing season before ring formation is complete, which induces an in-  
281 complete outermost ring. As the precise (intra-annual) timing of tree death was not available, we did  
282 not consider the last ring of the dead trees. The year of death was defined as the year of formation of  
283 the outermost ring, and considered as a proxy (cf. Bigler & Rigling 2013). At the site scale, tree mortal-

284 ity could be synchronous (all events occurring in one year), or spread in time over many years (the  
285 maximum range being > 100 years; Appendix S1).

286 A total of 36 species were included in the database, which covered several gymnosperm and angio-  
287 sperm families, although our dataset mainly included gymnosperms (64% of the species and 86% of the  
288 sites), with Pinaceae being the most represented family in terms of the number of species and sites  
289 sampled, followed by Fagaceae. Species life history strategies were characterized using two sets of  
290 shade and drought tolerance indices derived from Niinemets and Valladares (2006) and from the  
291 ForClim dynamic vegetation model (Bugmann 1996; details in Appendix S2). In addition, species  
292 structural traits such as wood density (Chave et al. 2009), total and axial parenchyma (Rodríguez-  
293 Calcerrada et al. 2015; Morris et al. 2016), Huber value (ratio of conducting xylem area per supported  
294 leaf area; Xylem Functional Traits Database; Choat et al. 2012) as well as species' hydraulic safety  
295 margin (difference between minimum seasonal water potential measured in the field and the water  
296 potential causing 50% loss of xylem conductivity in the stem; Choat et al. 2012) were used to  
297 characterize species responses to drought (see Appendix S2).

#### 298 *Growth patterns before mortality*

299 We assumed that all deaths observed for each species within a given site and a given mortality year  
300 were consequences of the same mortality process, while deaths that differed in time could be the result  
301 of separate processes. Consequently, growth patterns were analyzed for each combination species, site,  
302 and mortality year, hereafter referred to as a “mortality event”. Because of the variable methodologies  
303 used across sites (see above), we standardized the data among studies to better detect consistent growth  
304 patterns. First, for each mortality event ( $m$ ) we calculated annual growth ratios ( $g_m$ ) between trees that  
305 died (*dying tree*) and conspecific trees that survived that specific mortality event (*surviving tree*) for  
306 their entire lifespan up to the mortality year (Berdanier and Clark 2016; Fig. 2). A  $g_m < 1$  for a given  
307 year indicated that dying trees had lower growth rates than surviving ones. Analyzing this variable was  
308 useful to quantify relative changes in growth rate over time, which are better linked with mortality  
309 probability than absolute growth rates (Das et al. 2015), but also to remove potential biases due to dif-  
310 ferences in sampling schemes among studies (Cailleret et al. 2016). Second, to maximize sample size,  
311  $g_m$  were calculated using RW data (1,496 mortality events). RW data capture geometric and size effects  
312 (Bowman et al. 2013) that must be removed by adequate data standardization. Thus, we only consid-  
313 ered surviving trees with a DBH similar to the dying tree measured at a given mortality year ( $\pm 2.5$   
314 cm). In cases where none of the surviving trees fulfilled this condition, the corresponding mortality

315 event was discarded (123 events were not considered). When not measured in the original study, DBH  
316 was estimated as twice the sum of all previous ring-width measurements. Direct age effects were not  
317 considered here assuming that senescence only marginally affects tree function (Mencuccini et al.  
318 2014). Finally, to assess the dependency of the results to the growth data used,  $g_m$  values were also  
319 calculated using basal area increment (BAI;  $\text{mm}^2$ ) for trees whose DBH was measured (1,000 mortality  
320 events).

321 For each of the  $g_m$  time-series, we calculated (1) the growth ratio for the year before death ( $g_{f,m}$ ;  $f$  for  
322 final), and (2) the duration of the continuous period with a  $g_m < 1$  before tree death ( $\Delta t_{g < 1, m}$ ; in case of  
323  $g_{f,m} < 1$ ) or the duration of the continuous period with a  $g_m > 1$  before tree death ( $\Delta t_{g \geq 1, m}$ ; in case of  
324  $g_{f,m} \geq 1$  (cf. Fig. 2).

### 325 *Early growth rate*

326 At each site for which tree cambial age was available, and instead of focusing on growth patterns *per*  
327 *se*, we analyzed the ratio in mean RW calculated for the first 50 years of each tree's life between trees  
328 that died and trees that survived a given mortality event ( $g_{50,m}$ ). A 50 years period has been used in  
329 previous studies linking longevity with growth rates during this period (see Bigler 2016 and Ireland et  
330 al. 2014). To standardize the data and remove age effects, only surviving trees with an age comparable  
331 to the dying one were sampled ( $\pm 2$  years). When no surviving tree fulfilled this criterion, the corre-  
332 sponding mortality event was not considered. This approach has the advantage of using the growth in-  
333 formation from surviving trees. However, as species-specific relationships between early growth rates  
334 and mortality risk can be affected by methodological choices (Bigler 2016), we also assessed them (i)  
335 by varying the number of years used to calculate early mean RW (Appendix S3), (ii) using different  
336 age windows to sample surviving trees corresponding to each dead one (Appendix S4), and (iii) with a  
337 method that is more commonly used, i.e., by comparing the growth rate and longevity of dead trees  
338 only (Appendix S5).

### 339 *Designation of the main factors that triggered mortality*

340 The two major sources of mortality were determined for each site based on the expert assessment of the  
341 authors of each study, normally combining climatic analyses, growth and mortality data, and the pres-  
342 ence/absence of biotic agents. For the present study, we grouped mortality sources into four groups:  
343 'drought', 'biotic', 'drought and biotic', and 'others'. The first group corresponds to drought-induced  
344 mortality caused by a single or several drought events without obvious impact of biotic agents. The

345 group ‘biotic’ includes sites in which mortality was induced primarily by biotic factors, including bark  
346 beetle outbreaks, intense leaf or bud herbivory by insects, and/or fungal infection. In the third group,  
347 the impact of biotic agents (including mistletoes and wood-borers) was associated with drought. Final-  
348 ly, the group ‘others’ included snowbreak, frost events, high competition intensity, and cases in which  
349 mortality was induced by a combination of causes without a clear preponderating factor or, simply,  
350 where mortality causes were not specified. The proportion of mortality events was uniformly distribut-  
351 ed among these four classes ranging from 31.4% to 22.2% for the groups ‘others’ and ‘drought’, re-  
352 spectively (Table 1).

### 353 *Statistical analyses*

354 As the frequency distributions of  $g_{f,m}$  and  $g_{50,m}$  were right-skewed and long-tailed, i.e., most of the val-  
355 ues ranged between 0 and 2 but values exceeding 100 were possible when RW values of living trees  $\sim$   
356 0.01mm, and as the distribution in  $\Delta t_m$  was not normal, we analyzed median rather than mean values  
357 for interpreting “average” growth patterns. To explore how growth variables differed among species  
358 groups (gymnosperms vs. angiosperms) and mortality sources (drought, drought and biotic, biotic, oth-  
359 ers), we fitted a generalized linear mixed model for  $\Delta t_m$ , and two linear mixed models for  $g_{f,m}$  and  $g_{50,m}$ ,  
360 considering these categorical components as fixed effects. The variables  $g_{f,m}$  and  $g_{50,m}$  were log-  
361 transformed to better satisfy normality of the residuals, and we used a Poisson model with a log-link  
362 function for  $\Delta t_m$  as this response variable represents count data (see Bolker et al. 2008). As these varia-  
363 bles may change among species and sites irrespective of the fixed effects, random effects were estimat-  
364 ed for the intercept with site as grouping factor.

365 The variation among sites was not examined itself as we lack specific information on their environment  
366 (e.g., climate, soil, forest type). However, aggregating the conditional means of the generalized and  
367 linear mixed models by species allowed for estimating the variation in growth variables within and  
368 among species (e.g., with species drought tolerance) irrespective of their group and of the mortality  
369 source. As data on life history and structural traits were not available for every species, these variables  
370 were not included as fixed effects in the models to avoid loss of statistical power. Interactions among  
371 species groups and mortality sources were not considered in the final models as model fit was reduced  
372 in their presence (higher AIC, Akaike Information Criterion). Type-III chi-squares and type-II sum of  
373 squares variance analyses were used to estimate the respective impact of species group and source of  
374 mortality on  $\Delta t_m$  as well as on  $g_{f,m}$  and  $g_{50,m}$ , respectively. Coefficients of determination were used to

375 assess the percent contribution of fixed effects alone ( $R^2$  marginal) and both fixed and random effects  
376 ( $R^2$  conditional) for explaining the variability in growth patterns (Nakagawa & Schielzeth, 2013).  
377 Finally, re-sampling procedures were used to assess the dependency of mixed models estimates to the  
378 properties of the calibration dataset and to account for the heterogeneity in the number of mortality  
379 events per site and per species. For each species, we randomly sampled 21 or 17 mortality events (me-  
380 dians in the database for recent and early growth rates, respectively) with replacement. Depending on  
381 the species, the information from a given mortality event could be either replicated (when sample size  
382 was low e.g., for *Nothofagus dombeyi*), or excluded (e.g., for *Quercus rubra*). This sampling procedure  
383 was repeated 500 times and mixed-effects models were fitted to each of these 500 datasets. With this  
384 approach, each species has the same weight in the calibration dataset and contributes to the same extent  
385 to the model estimates. We also generated 500 different datasets with a bootstrap re-sampling ap-  
386 proach. In that case, the number of mortality events was identical to the original dataset but they were  
387 randomly selected with replacement, irrespective of the site or species. Mixed models fitting and selec-  
388 tion, and variance analyses were performed using the packages *lme4*, *lmerTest*, *MuMIn*, and *car* of the  
389 open-source software R (R Development Core Team 2015).

390

## 391 **Results**

392

### 393 *Change in growth rates before mortality*

394 In 83.9% of the mortality events, dying trees showed reduced growth rates prior to death compared to  
395 surviving trees ( $g_{f,m} < 1$ ). This reduction was frequently substantial and lasted for many years (Fig. 3a).  
396 On average, growth of dying trees in the year before mortality ( $g_{f,m}$ ) was ca. 40% of the growth of sur-  
397 viving trees with a similar DBH (median in RW  $g_{f,m} = 0.42$ ), but  $g_{f,m}$  was highly variable among mor-  
398 tality events (Fig. 4). The distribution of  $g_{f,m}$  was right-skewed with highest frequencies between 0.1  
399 and 0.3 (Fig. 4) and did not significantly change with the approach used to sample surviving trees (Ap-  
400 pendix S6). The duration of the period with reduced growth of dying trees ( $\Delta t_{g<1,m}$ ) was highly variable  
401 from 1 to 100 years in 96% of the mortality events, and followed an exponential-like probability  
402 density function with a median of 19 years. Around 17% of the mortality events showed a  $\Delta t_{g<1} \leq 5$   
403 years, and 15% showed a decline period  $> 50$  years. Similar results were obtained using BAI data  
404 (Appendix S7), but median values of  $g_{f,m}$  (0.39) and  $\Delta t_{g<1,m}$  (18 years) were slightly lower than with  
405 RW data. Finally, in 241 mortality events (16.1%), dying trees had higher RW than surviving ones the

406 year before death ( $g_{f,m} \geq 1$ ). For these mortality events, the increase in growth was much more recent,  
407 as the median of  $\Delta t_{g \geq 1, m}$  was 4 years (Fig. 4).

#### 408 *Differences in growth patterns before mortality across species groups and mortality sources*

409 The variation in  $g_{f,m}$  and  $\Delta t_m$  was high within species groups and mortality groups, with the same order  
410 of magnitude as the variation within species and sites (quantile coefficients of dispersion; Appendix  
411 S8). As a consequence, the fixed effects considered in the generalized and linear mixed models ex-  
412 plained only a small part of the variance in  $g_{f,m}$  and  $\Delta t_m$  ( $R^2$  marginal = 0.06 and 0.03, respectively);  
413 however significant differences among species groups and mortality sources could be detected (Table  
414 2). Inter-site variability explained a larger part of the variance ( $R^2$  conditional = 0.18 and 0.26) that  
415 could be related with inter-specific differences in shade and drought tolerance (within species group).  
416 Results of the generalized and linear mixed models were consistent regardless of the data source (RW  
417 or BAI data; Appendix S9), regardless of the properties of the calibration dataset in terms of the distri-  
418 bution of mortality events per site and species (Table 2 and Appendix S10), and regardless of whether  
419 dying trees were grouped per mortality year or not (Appendix S11).

420 In case of drought-induced mortality, the median in RW  $g_{f,m}$  and  $\Delta t_m$  predicted by the mixed effect  
421 models was 0.42 and 19 years, respectively (Fig. 5a), identical to the values obtained when considering  
422 all sources of mortality. Relative to cases in which drought was the main source of mortality,  $\Delta t_m$  and  
423  $g_{f,m}$  did not significantly differ when drought was associated with biotic agents. Growth reductions,  
424 however, tended to be shorter and more intense (lower  $\Delta t_m$  and higher  $g_{f,m}$ , respectively), when trees  
425 were killed by biotic agents alone ( $p < 0.1$ ; Table 2) and, particularly, when trees were attacked by bark  
426 beetles ( $p < 0.05$ ; Appendix S12). Trees that died because of other factors (including inter-individual  
427 competition) showed the longest and strongest period of reduced growth before death (predicted medi-  
428 an in  $\Delta t_m = 24$  years and in  $g_{f,m} = 0.29$ ; Fig. 5a; Table 2).

429 Considering all sources of mortality, the period with reduced growth was longer and the associated  
430 reduction in growth was stronger for gymnosperms than for angiosperms (predicted medians  $\Delta t_m = 22$   
431 and 16 years, and  $g_{f,m} = 0.41$  and 0.53, respectively; Table 2; Fig. 5b), and, to a lower extent, for 'non-  
432 *Quercus*' angiosperms relative to *Quercus* species (Appendix S13). Interestingly, this trend occurred  
433 whatever the mortality source, as there was no significant interaction between the effects of species  
434 group and mortality source (higher AIC of the mixed models when interactions were included).

#### 435 *Species characteristics associated with growth patterns before mortality*

436 At the species level, long-term reductions in growth (high  $\Delta t_m$ ) were mainly observed for shade-  
437 tolerant angiosperms, shade- and drought- tolerant gymnosperms, gymnosperms with low wood  
438 density, and species with a low amount of wood parenchyma (especially axial parenchyma for  
439 angiosperms; ray parenchyma for gymnosperms) (Table 3a). Results were similar when only drought-  
440 induced mortality was considered. In this case, gymnosperms with a low Huber value were also  
441 characterized by long-term growth reductions before mortality (Table 3b).

442 Strong reductions in growth before death (low  $g_{f,m}$ ) were detected for species with a low amount of  
443 wood parenchyma, for shade-tolerant angiosperms, and for species with high hydraulic safety margin  
444 (Table 3a). In case of drought-induced mortality, gymnosperms with low Huber values had also  
445 stronger growth reductions (Table 3b). The relationship between  $g_{f,m}$  and species drought tolerance was  
446 inconsistent, as opposite trends were found for gymnosperms and angiosperms and results differed de-  
447 pending on whether the tolerance indices used were derived from Niinemets and Valladares (2006) or  
448 from ForClim (Table 3b).

#### 449 *Early growth rates*

450 Dying trees tended to have lower averaged early growth rate than conspecific surviving ones, especial-  
451 ly when a short time period is used to calculate mean juvenile growth rate (Fig. 3b). Considering the  
452 first 50 years of a tree's lifetime as representative of its juvenile phase, this trend was observed in  
453 58.6% of the mortality events ( $g_{50,m} < 1$ ; 361/617), but the median in  $g_{50,m}$  was around 0.93 and was  
454 not significantly different from one ( $p > 0.1$ ).

455 Significant differences among mortality groups were highlighted by the generalized linear mixed mod-  
456 els. Early growth ratio was highest when mortality was caused by drought alone, and lowest when it  
457 was induced by drought combined with biotic agents and by other factors. These differences were sig-  
458 nificant using  $g_{50,m}$  (Table 2), and also by averaging early growth rate over different time-windows  
459 (number of years fixed across species or as a function of species lifespan; Appendix S3). There was a  
460 tendency towards higher early growth ratio for gymnosperms than angiosperms, but this result was not  
461 consistent when comparing different approaches to define the early growth ratio (Appendix S3).

462 Considering all sources of mortality,  $g_{50,m}$  showed a negative relationship with species shade tolerance  
463 (both species groups; according to ForClim's parameters), and with wood density and the hydraulic  
464 safety margin in gymnosperms (Table 3a). The same trends were observed in case of drought-induced  
465 mortality, while for angiosperms  $g_{50,m}$  was positively related with their hydraulic safety margin, and  
466 negatively linked with their wood density (Table 3b).



467

## 468 **Discussion**

469

470 Based on a new tree-ring width database from temperate, boreal and Mediterranean forests, our analy-  
471 sis shows that tree mortality is preceded by a growth reduction in ~84% of the mortality events, and  
472 supports our initial hypothesis, i.e., the decrease in growth before death is most likely stronger and  
473 longer for various stress-tolerant gymnosperms than some angiosperms, and also longer when trees are  
474 affected by repeated, mild, but gradually increasing environmental stress such as shading rather than by  
475 a severe attack of biotic agents.

### 476 *General growth patterns before mortality*

477 Our synthesis supports that dying trees commonly show lower growth rates prior to death than surviv-  
478 ing ones ( $g_{f,m} < 1$ ). Considering all mortality events, the decrease in growth the year before death aver-  
479 aged ~60% (median in  $g_{f,m} \sim 0.4$ ). This substantial growth reduction may have been overestimated be-  
480 cause of the reduction in the competitive ability of dying trees, which may have benefited the growth of  
481 surviving individuals (Cavin et al. 2013). However, this effect was compensated, at least partially, by  
482 the fact that the group of ‘surviving’ trees at a given mortality event may include trees with reduced  
483 growth that died shortly after the event. Although growth reductions before mortality are nearly  
484 universal, our results show that they can be abrupt or gradual, and the duration of the period with  
485 reduced growth ( $\Delta t_m$ ) was highly variable, ranging from 1 to 100 years in 96% of the cases. Overall,  
486 62% of the mortality events showed reduced growth 5–50 years preceding tree death, consistent with  
487 previous studies (e.g., ~5 years in Bond-Lamberty et al. 2014; 6–12 years in Wyckoff and Clark 2002;  
488 10–15 years in Ogle et al. 2000; ~15 years in Camarero et al. 2015; ~30 years in Macalady and  
489 Bugmann 2014). These results confirm that trees can survive a long time with low growth, and  
490 emphasize the role of accumulated stress or slow-acting processes (e.g., competition) in tree mortality  
491 (Das et al. 2008). However, it is noticeable that in 18% of the mortality events, trees died after a fast ( $\leq$   
492 5 years) growth decline in comparison to trees that survived, highlighting quick tree responses to  
493 intense stress. In 19% of the mortality events, trees died after experiencing only a slight decrease or  
494 even a short-term increase in growth ( $g_{f,m} > 0.9$ ). Similar observations are rather rare in the literature  
495 (but see Ferrenberg et al. 2014; Rowland et al. 2015; Berdanier and Clark 2016; Herguido et al. 2016),  
496 and indicate either that radial growth can be prioritized until the point of death irrespective of environ-

497 mental stress, or that stress can be strong enough to kill trees without any impact on the carbon budget  
498 and its allocation to growth.

499 In addition to this general pattern, a wide range of growth patterns ( $\Delta t_m$  and  $g_{f,m}$ ) within mortality  
500 sources, within species, and within sites was observed. This variability likely reflects: (i) the classifica-  
501 tion of mortality into four broad groups, disregarding the multifactorial character of mortality in many  
502 cases and the inherent complexity of mortality processes (Allen et al. 2015; Anderegg et al. 2015b), (ii)  
503 the difficult and somewhat arbitrary identification of the sources of mortality and quantification of their  
504 respective role under field conditions, and (iii) the high spatio-temporal heterogeneity in micro-climate,  
505 soil and stand density conditions and pressure from biotic agents within some sites. Even though most  
506 of the variability in  $\Delta t_m$  and  $g_{f,m}$  was not explained by the categorical variables considered here (low  
507 variance explained by the generalized and linear mixed models), the high dimensionality of the tree-  
508 ring database in terms of sample size, diversity of species, and mortality causes allowed us to detect  
509 differences among these groups. Considering that the outputs of the generalized and linear mixed mod-  
510 els were coherent no matter what methodology was used to calculate growth ratios (Appendices S6, S9  
511 and S11), and what calibration dataset was used to fit them (Table 2; Appendix S10), we are confident  
512 about the reliability of our results.

### 513 *Growth patterns before mortality vary among sources of mortality*

514 Although a stronger and longer decrease in growth prior to death could be expected when drought was  
515 associated with biotic agents, growth patterns under these conditions were similar to those from trees  
516 undergoing drought only. This may be the result of two opposite influences of pathogens on the  
517 growth-mortality relationships, depending on their role within the mortality spiral (predisposing vs.  
518 contributing factor; Manion 1991). On the one hand, a recurrence of moderate biotic attacks (e.g.,  
519 insect defoliators) and pathogen infection or parasite infestation (e.g., mistletoes or root fungi) reduce  
520 carbon, water and nutrient availability of individual trees, and thus may reduce their growth over both  
521 short- and long-term periods and predispose them to subsequent stress factors, and finally to mortality  
522 (Schwarze et al. 2003; Hartmann and Messier 2008; Sangüesa-Barreda et al. 2013; Macalady and  
523 Bugmann 2014; Oliva et al. 2014). On the other hand, massive insect outbreaks may lead to faster tree  
524 death that is largely decoupled from growth. Consistent with that interpretation, the decrease in growth  
525 before death was shorter and smaller when mortality was related to biotic agents than by drought, and  
526 was especially low in case of bark-beetle attacks (contributing factor; Appendix S12).

527 The slower growth signal associated with mortality induced by bark-beetle outbreaks may reflect a  
528 negative effect of carbon allocation to growth rather than defense on tree survival (growth-  
529 differentiation balance hypothesis; Herms and Mattson 1992) and could be explained by several  
530 hypotheses. First, the disruption of carbohydrate transport due to phloem feeding by bark beetles and  
531 xylem occlusion by the fungi they introduce (Hubbard et al. 2013) usually have major consequences for  
532 tree functioning, leading to leaf shedding and tree death within a few years (Meddens et al. 2012;  
533 Wiley et al. 2016). Second, in the endemic phase, bark beetles may not preferentially attack trees with  
534 slow growth (Sangüesa-Barreda et al. 2015; but see Macalady and Bugmann 2014), but rather trees  
535 with specific size and/or bark thickness, and with lower defense capacities (less resin duct production;  
536 Kane and Kolb 2010; Ferrenberg et al. 2014). Third, considering that tree growth is frequently sink-  
537 driven (Körner 2015), and that defoliation does not increase water stress (but may actually decrease it  
538 due to lower whole-tree transpiration), a single biotic defoliation event may not strongly affect tree  
539 growth (but see Piper et al. 2015).

540 Finally, long and strong growth reductions before death were found when mortality was caused by nei-  
541 ther drought nor biotic agents, or when the cause was not specified. This group especially included  
542 trees that died because of high competition intensity, confirming that shading can suppress trees for a  
543 long period before they actually die (Abrams and Orwig 1996). However, the effects of shading (and  
544 competition in general) and other stress factors frequently interact (Das et al. 2016; Myers and Kitajima  
545 2007) and are difficult to disentangle in field settings.

546 *Low, short-term growth reductions before death are more common in angiosperms*

547 As hypothesized, angiosperm species, and especially *Quercus* species, did not commonly show long-  
548 lasting reduced growth periods before death but rather died after a fast decline, or even after a short-  
549 term increase in growth before death. In contrast, gymnosperm species commonly showed long-term  
550 and slow growth reductions before death. Angiosperms tend to recover quickly from extreme events,  
551 whereas gymnosperms feature substantial legacy effects (e.g., after drought; Anderegg et al. 2015a),  
552 which may reveal the slow but chronic deterioration of their carbon balance and hydraulic performance  
553 under gradual or repeated environmental stress (Dickman et al. 2015; Pellizzari et al. 2016). This inter-  
554 pretation is consistent with recent findings showing that reduced NSC concentrations are frequently  
555 associated with drought-induced mortality in gymnosperms but not in angiosperms (Anderegg et al.,  
556 2016a). Higher growth fluctuations in angiosperms than gymnosperms are likely associated to a num-  
557 ber of attributes, including: (i) high growth efficiency (Brodribb et al. 2012) and productivity in fertile

558 conditions (Augusto et al. 2014), associated with less conservative water use and higher stomatal con-  
559 ductance (Lin et al. 2015); (ii) higher amount of wood parenchyma that may serve to increase storage  
560 capacity of NSC and symplastic water (Morris et al. 2016; Plavcová et al. 2016), (iii) high capacity to  
561 resprout unlike most species in the Pinaceae family (Zeppel et al. 2015); (iv) narrower hydraulic safety  
562 margins (Choat et al. 2012); and, possibly, (v) potential capacity to refill embolized xylem conduits  
563 (Choat et al. 2012, 2015; but see Mayr et al. 2014 for passive hydraulic recovery in conifers). However,  
564 because of the rather small number of angiosperm tree species studied, we acknowledge that more re-  
565 search using a larger number of species, including tropical angiosperms, is needed to validate our hy-  
566 pothesis.

567 Similarly, growth patterns before death differed among species according to their stress tolerance and  
568 resistance and the related structural and functional traits. Because of the relatively low number of the  
569 species studied and the limited availability of functional trait data, the correlation among traits was not  
570 captured by the univariate analysis we used. Therefore, sufficient care should be taken while interpret-  
571 ing these results. Nevertheless, our findings provide some physiological explanations for the differ-  
572 ences between angiosperms and gymnosperms mentioned above. Long-term, strong reductions in  
573 growth before death were more frequently observed for drought-tolerant species – according to  
574 ForClim's parameters – with wide hydraulic safety margins, a low amount of wood parenchyma, and  
575 low Huber values (for gymnosperms). Shade-tolerant species showed longer and stronger reductions in  
576 growth before death than intolerant ones, as evident from comparing species-specific tolerance indices  
577 derived from ForClim and Niinemets and Valladares (2006), confirming their ability to survive under  
578 shading for a long period (Wyckoff and Clark 2002; Wunder et al. 2008). Despite the probable link  
579 between wood density and mortality risk of angiosperms (Anderegg et al. 2016a), this trait was not  
580 associated with particular growth patterns before death.

#### 581 *No clear intra-specific trade-off between early growth rates and longevity*

582 Intra-specific trade-offs between growth rates during the juvenile phase and tree longevity have been  
583 observed frequently for angiosperm and gymnosperm species, while positive relationships have been  
584 rarely found (Black et al. 2008; Ireland et al. 2014; Bigler 2016). In our synthesis we did not find evi-  
585 dence of a consistent trade-off in gymnosperms and in angiosperms (Appendix S5). In 58.6% of the  
586 mortality events, dying trees had lower early growth rates than surviving ones ( $g_{50,m} < 1$ ), especially  
587 when mortality was caused by other agents or by drought and biotic attack than drought alone. Early  
588 investment in rapid growth may provide a strong advantage under light-limited conditions (e.g., in

589 dense stands). However, as highlighted by the high  $g_{50,m}$  values in case of drought-induced mortality  
590 and for species with low wood density, it may constitute a disadvantage under dry conditions, where  
591 investment into mechanisms to increase water uptake capacity and hydraulic function may be favored.  
592 Similarly, promoting early growth instead of whole-tree defenses may be a disadvantage in case of bio-  
593 tic attack or insect defoliation (Rose et al. 2009), but our analysis did not fully support this hypothesis.

594 As reported by Bigler (2016), methodological aspects related to the experimental design and the sam-  
595 pling strategy may explain differences in the relationship between early growth rates and longevity  
596 among sites, species or studies. In our database, most of the samples did not cover large gradients of  
597 early growth and lifespan (e.g. very old trees or very rapidly/slowly growing trees are missing), mainly  
598 because of the relatively low number of dead trees at each site and for each species (Appendix S5).  
599 Thus, the lack of consistent trade-off between early growth rates and longevity, and the lack of strong  
600 differences among species and mortality sources observed in our synthesis likely reflects high variabil-  
601 ity in sampling design among sites, and highlights the need for further research on this important topic.

602 Our results show that radial growth reductions before tree mortality are nearly universal. However,  
603 their magnitude and the corresponding growth-mortality relationships varied among sources of mortali-  
604 ty, between gymnosperms and angiosperms, and among species. These differences largely support our  
605 initial hypothesis: angiosperms, trees attacked by bark beetles or stress-sensitive species (e.g., with  
606 narrow hydraulic safety margins) typically show a short-term growth decline prior to mortality, while  
607 long-lasting growth reductions tend to occur in gymnosperms, stress-tolerant species and may indicate  
608 a long-term (chronic) deterioration of the carbon and water economies. Our analyses show that the  
609 temporal changes in growth level before death may provide useful insights into the mechanisms under-  
610 lying tree mortality, and its complex, multi-scale processes. In addition, our results have strong impli-  
611 cations for the use of growth data as early warning signal of mortality and for the simulation of tree  
612 mortality in dynamic vegetation models. Species- or functional type- specific growth-based mortality  
613 algorithms may be powerful for predicting mortality induced by multi-annual stress factors and fore-  
614 casting gymnosperm death. However, for angiosperms and in case of intense drought or bark-beetle  
615 outbreaks, growth-based algorithms are unlikely to be predictive, and must be complemented by physi-  
616 ological and/or anatomical information.

617

## 618 **Acknowledgments**

619

620 This study generated from the COST Action STReSS (FP1106) financially supported by the EU  
621 Framework Programme for Research and Innovation HORIZON 2020. We are particularly grateful to  
622 Professor Dr. Ute Sass-Klaassen from Wageningen University (The Netherlands), chair of the action,  
623 for making this meta-study possible. We also thank members of the Laboratory of Plant Ecology from  
624 the University of Ghent (Belgium) for their help while compiling the database; Louise Filion for shar-  
625 ing her dataset; Dario Martin-Benito for providing some ForClim parameters; the ARC-NZ Vegetation  
626 Function Network for supporting the compilation of the Xylem Functional Traits dataset; Edurne Mar-  
627 tinez del Castillo for the creation of Figure 1; and two anonymous reviewers and Phillip van Mantgem  
628 (USGS) for their suggestions to improve the quality of the paper.

629 MC was funded by the Swiss National Science Foundation (project number 140968), SJ by the German  
630 Research Foundation (JA 2174/3-1), EMRR by the Research Foundation - Flanders (FWO, Belgium)  
631 and by the EU HORIZON 2020 Programme through a Marie Skłodowska-Curie IF fellowship (No  
632 659191), LDS by a postdoctoral fellowship from the Portuguese *Fundação para a Ciência e a*  
633 *Tecnologia* (FCT) (SFRH/ BPD/70632/2010), TA by the Academy of Finland (proj. no. 252629 and  
634 276255), JAA by the British Columbia Forest Science Program and the Forest Renewal BC (Canada),  
635 BB and WO by the Austrian Science Fund (FWF, Hertha Firnberg Programme project T667-B16 and  
636 FWF P25643-B16), VC, PJ, MS and VT by the Czech Ministry of Education (MŠMT, project COST  
637 CZ no. LD13064 and LD14074), JJC, JCLC and GSB by the Spanish Ministry of Economy (projects  
638 CGL2015-69186-C2-1-R, CGL2013-48843-C2-2-R, and CGL2012-32965) and the EU (project  
639 FEDER 0087 TRANSHABITAT), MRC by the Natural Sciences and Engineering Research Council of  
640 Canada (NSERC) and by the *Service de la protection contre les insectes et les maladies du ministère*  
641 *des forêts du Québec* (Canada), KC by the Slovenian Research Agency (ARRS) - program P4-0015,  
642 AD by the United States Geological Survey (USGS), HD by the French National Research Agency  
643 (ANR, DRYADE project ANR-06-VULN-004) and the metaprogramme Adaptation of Agriculture and  
644 Forests to Climate Change (AAFCC) of the French National Institute for Agricultural Research  
645 (INRA), MD by the Israeli Ministry of Agriculture and Rural Development as a chief scientist and by  
646 the Jewish National Fund (Israel), GGI by the Spanish Ministry of Economy and Competitiveness (Pro-  
647 ject AGL2014-61175-JIN), SG by the *Bundesministerium für Bildung und Forschung* (BMBF) through  
648 the project REGKLAM (Grant number: 01 LR 0802) (Germany), LJH by the Arkansas Agricultural  
649 Experiment Station (United States of America) and the United States Department of Agriculture - For-  
650 est Service, HH by the Natural Sciences and Engineering Research Council of Canada, AMH by the  
651 Spanish Ministry of Science and Innovation (projects CGL2007-60120 and CSD2008-0040) and by the  
652 Spanish Ministry of Education via a FPU scholarship, VIK by the Russian Science Foundation (grant

653 #14-24-00112), TKi and RV by the *Consejo Nacional de Investigaciones Científicas y Técnicas*  
654 (CONICET Grant PIP 112-201101-00058 and PIP 112-2011010-0809) (Argentina), TKI by the Weiz-  
655 mann Institute of Science (Israel) under supervision of Professor Dan Yakir, by the Keren Kayemeth  
656 LeIsrael (KKL) – Jewish National Fund (JNF) (Alberta-Israel program 90-9-608-08), by the Sussman  
657 Center (Israel), by the Cathy Wills and Robert Lewis Program in Environmental Science (United King-  
658 dom), by the France-Israel High Council for Research Scientific and Technological Cooperation (pro-  
659 ject 3-6735) and by the Minerva Foundation (Germany), KK by the project "Resilience of Forests" of  
660 the Ministry of Economic Affairs (The Netherlands - WUR Investment theme KB19), TL by the pro-  
661 gram and research group P4-0107 Forest Ecology, Biology and Technology (Slovenia), RLV by a  
662 postdoctoral fellowship from the *Portuguese Fundação para a Ciência e a Tecnologia* (FCT;  
663 SFRH/BPD/86938/2012), RLR by the EU FP7 Programme through a Marie Skłodowska-Curie IOF  
664 fellowship (No 624473), HM by the Academy of Finland (Grant Nos. 257641 and 265504), SM by  
665 Sparkling Science of the Federal Ministry of Science, Research and Economy (BMFWF) of Austria,  
666 IM by the Hungarian Scientific Research Fund (No. K101552), JMM by the Circumpolar-Boreal Al-  
667 berta grants program from the Natural Science and Engineering Research Council of Canada, MP by  
668 the EU project LIFE12 ENV/FI/000409, AMP by a Swiss Research Fellowship (Sciex-NMSch, Project  
669 13.272 – OAKAGE), JMS by the American National Science Foundation (grant 0743498), ABS by the  
670 British Columbia Ministry of Forests, Lands and Natural Resource Operations (Canada), DS by the  
671 Public Enterprise “Vojvodinasume” (project Improvement of Lowland Forest Management), MLS by  
672 the *Consejo Nacional de Investigaciones Científicas y Técnicas* (CONICET Grant PIP  
673 11420110100080) and by *El Fondo para la Investigación Científica y Tecnológica* (FONCyT Grant  
674 PICT 2012-2009), RT by the Italian Ministry of Education (University and Research 2008, *Ciclo del*  
675 *Carbonio ed altri gas serra in ecosistemi forestali, naturali ed artificiali dell’America Latina: analisi*  
676 *preliminare, studio di fattibilità e comparazione con ecosistemi italiani*) and by the EU LIFE+ project  
677 MANFOR C.BD. (Environment Policy and Governance 2009, Managing forests for multiple purposes:  
678 carbon, biodiversity and socio-economic wellbeing), ARW by the Natural Sciences and Engineering  
679 Council (NSERC) (Canada) through the University of Winnipeg and by Manitoba Conservation (Cana-  
680 da) and JMV by the Spanish Ministry of Economy and Competitiveness (grant CGL2013-46808-R).  
681 Any use of trade names is for descriptive purposes only and does not imply endorsement by the U.S.  
682 Government.

683

## 684 **References**

This article is protected by copyright. All rights reserved

685

- 686 Abrams MD, Orwig DA (1996) A 300-year history of disturbance and canopy recruitment of co-occurring white  
687 pine and hemlock on the Allegheny Plateau, USA. *Journal of Ecology*, **84**, 353-363
- 688 Aguadé D, Poyatos R, Gómez M, Oliva J, Martínez-Vilalta J (2015) The role of defoliation and root rot patho-  
689 gen infection in driving the mode of drought-related physiological decline in Scots pine (*Pinus sylvestris* L.).  
690 *Tree physiology*, **35**, 229-242.
- 691 Allen CD, Macalady AK, Chenchouni H *et al.* (2010) A global overview of drought and heat-induced tree mor-  
692 tality reveals emerging climate change risks for forests. *Forest Ecology and Management*, **259**, 660–684.
- 693 Allen CD, Breshears DD, McDowell NG (2015) On underestimation of global vulnerability to tree mortality and  
694 forest die-off from hotter drought in the Anthropocene. *Ecosphere*, **6**, art129.
- 695 Anderegg WRL, Kane JM, Anderegg LD (2013) Consequences of widespread tree mortality triggered by  
696 drought and temperature stress. *Nature Climate Change*, **3**, 30-36.
- 697 Anderegg WRL, Schwalm C, Biondi F *et al.* (2015a) Pervasive drought legacies in forest ecosystems and their  
698 implications for carbon cycle models. *Science*, **349**, 528-532.
- 699 Anderegg WRL, Hicke JA, Fisher RA *et al.* (2015b) Tree mortality from drought, insects, and their interactions  
700 in a changing climate. *New Phytologist*, **208**, 674-683.
- 701 Anderegg WRL, Klein T, Bartlett M, Sack L, Pellegrini AFA, Choat B, Jansen S (2016a) Meta-analysis reveals  
702 that hydraulic traits explain cross-species patterns of drought-induced tree mortality across the globe. *Pro-  
703 ceedings of the National Academy of Sciences of the United States of America*, **113**, 5024-5029.
- 704 Anderegg WR, Martinez-Vilalta J, Cailleret M *et al.* (2016b) When a tree dies in the forest: scaling climate-  
705 driven tree mortality to ecosystem water and carbon fluxes. *Ecosystems*, **19**, 1133-1147.
- 706 Augusto L, Davies TJ, Delzon S, Schrijver A (2014) The enigma of the rise of angiosperms: can we untie the  
707 knot? *Ecology Letters*, **17**, 1326-1338.
- 708 Babst F, Bouriaud O, Papale D *et al.* (2014) Above-ground woody carbon sequestration measured from tree  
709 rings is coherent with net ecosystem productivity at five eddy-covariance sites. *New Phytologist*, **201**, 1289-  
710 1303.
- 711 Berdanier AB, Clark JS (2016) Multi-year drought-induced morbidity preceding tree death in Southeastern US  
712 forests. *Ecological Applications*, **26**, 17-23.
- 713 Bigler C (2016) Trade-offs between growth rate, tree size and lifespan of mountain pine (*Pinus montana*) in the  
714 Swiss National Park. *Plos One*, **11**, e0150402. doi:10.1371/journal.pone.0150402
- 715 Bigler C, Bugmann H (2004) Predicting the time of tree death using dendrochronological data. *Ecological Ap-  
716 plications*, **14**, 902-914.
- 717 Bigler C, Bräker OU, Bugmann H, Dobbertin M, Rigling A (2006) Drought as an inciting mortality factor in  
718 Scots pine stands of the Valais, Switzerland. *Ecosystems*, **9**, 330-343.
- 719 Bigler C, Gavin DG, Gunning C, Veblen TT (2007) Drought induces lagged tree mortality in a subalpine forest  
720 in the Rocky Mountains. *Oikos*, **116**, 1983-1994.



- 721 Bigler C, Rigling A, (2013) Precision and accuracy of tree-ring-based death dates of mountain pines in the Swiss  
722 National Park. *Trees*, **27**, 1703-1712.
- 723 Bircher N, Cailleret M, Bugmann H (2015) The agony of choice: different empirical models lead to sharply dif-  
724 ferent future forest dynamics. *Ecological Applications*, **25**, 1303–1318.
- 725 Black BA, Colbert JJ, Pederson N (2008) Relationships between radial growth rates and lifespan within North  
726 American tree species. *Ecoscience*. **15**, 349–357.
- 727 Bolker BM, Brooks ME, Clark CJ, Geange SW, Poulsen JR, Stevens MHH, White JSS (2009) Generalized line-  
728 ar mixed models: a practical guide for ecology and evolution. *Trends in Ecology and Evolution*, **24**, 127-135.
- 729 Bowman DMJS, Brienens RJW, Gloor E, Phillips OL, Prior LD (2013) Detecting trends in tree growth: not so  
730 simple. *Trends in Plant Science*, **18**, 11–17.
- 731 Brodribb TJ, Bowman, DJ, Nichols S, Delzon S, Burrett R (2010) Xylem function and growth rate interact to  
732 determine recovery rates after exposure to extreme water deficit. *New Phytologist*, **188**, 533-542.
- 733 Brodribb TJ, Pittermann J, Coomes DA (2012) Elegance versus speed: examining the competition between coni-  
734 fer and angiosperm trees. *International Journal of Plant Sciences*, **173**, 673-694.
- 735 Bugmann HK (1996) A simplified forest model to study species composition along climate gradients. *Ecology*,  
736 **77**, 2055-2074.
- 737 Cailleret M, Bigler C, Bugmann H *et al.* (2016) Towards a common methodology for developing logistic tree  
738 mortality models based on ring-width data. *Ecological Applications*, **26**, 1827-1841.
- 739 Camarero JJ, Gazol A, Sangüesa-Barreda G, Oliva J, Vicente-Serrano SM (2015) To die or not to die: early  
740 warnings of tree dieback in response to a severe drought. *Journal of Ecology*, **103**, 44-57.
- 741 Cavin L, Mountford EP, Peterken GF, Jump AS (2013) Extreme drought alters competitive dominance within  
742 and between tree species in a mixed forest stand. *Functional Ecology*, **27**, 1424-1435.
- 743 Chave J, Coomes D, Jansen S, Lewis SL, Swenson NG, Zanne AE (2009) Towards a worldwide wood econom-  
744 ics spectrum. *Ecology Letters*, **12**, 351-366.
- 745 Choat B, Jansen S, Brodribb TJ *et al.* (2012) Global convergence in the vulnerability of forests to drought. *Nature*,  
746 **491**, 752-755.
- 747 Choat B, Brodersen CR, McElrone AJ (2015) Synchrotron X-ray microtomography of xylem embolism in *Se-*  
748 *quoia sempervirens* saplings during cycles of drought and recovery. *New Phytologist*, **205**, 1095-1105.
- 749 Cook BI, Smerdon JE, Seager R, Coats S (2014) Global warming and 21st century drying. *Climate Dynamics*,  
750 **43**, 2607-2627.
- 751 Das A, Battles J, van Mantgem PJ, Stephenson NL (2008) Spatial elements of mortality risk in old-growth for-  
752 ests. *Ecology*, **89**, 1744-1756.
- 753 Das AJ, Stephenson NL (2015) Improving estimates of tree mortality probability using potential growth rate.  
754 *Canadian Journal of Forest Research*, **45**, 920-928.
- 755 Das AJ, Stephenson NL, Davis KP (2016) Why do trees die? Characterizing the drivers of background tree mor-  
756 tality. *Ecology*, **97**, 2616-2627.

- 757 Delpierre N, Berveiller D, Granda E, Dufrêne E (2015) Wood phenology, not carbon input, controls the  
758 interannual variability of wood growth in a temperate oak forest. *New Phytologist*, **210**, 459-470.
- 759 Dickman LT, McDowell NG, Sevanto S, Pangle RE, Pockman WT (2015) Carbohydrate dynamics and mortality  
760 in a piñon-juniper woodland under three future precipitation scenarios. *Plant, Cell and Environment*, **38**,  
761 729-739.
- 762 Dietze MC, Sala A, Carbone MS, Czimczik CI, Mantooh JA, Richardson AD, Vargas R (2014) Nonstructural  
763 carbon in woody plants. *Annual Review of Plant Biology*, **65**, 667-687.
- 764 Dobbertin M (2005) Tree growth a indicator of tree vitality and of tree reaction to environmental stress: a re-  
765 view. *European Journal of Forest Research*, **124**, 319-333.
- 766 Ferrenberg S, Kane JM, Mitton JB (2014). Resin duct characteristics associated with tree resistance to bark bee-  
767 tles across lodgepole and limber pines. *Oecologia*, **174**, 1283-1292.
- 768 Franklin JF, Shugart, HH, Harmon ME (1987) Tree death as an ecological process. *BioScience*, **37**, 550-556.
- 769 Friend AD, Lucht W, Rademacher TT *et al.* (2014) Carbon residence time dominates uncertainty in terrestrial  
770 vegetation responses to future climate and atmospheric CO<sub>2</sub>. *Proceedings of the National Academy of*  
771 *Sciences of the United States of America*, **111**, 3280-3285.
- 772 Hartmann H (2015) Carbon starvation during drought-induced tree mortality – are we chasing a myth? *Journal*  
773 *of Plant Hydraulics*, **2**, e005.
- 774 Hartmann H, Adams HD, Anderegg WRL, Jansen S, Zeppel MJ (2015) Research frontiers in drought-induced  
775 tree mortality: crossing scales and disciplines. *New Phytologist*, **205**, 965-969.
- 776 Hartmann H, Messier C (2008) The role of forest tent caterpillar defoliations and partial harvest in the decline  
777 and death of sugar maple. *Annals of Botany*, **102**, 377-387.
- 778 Hereş AM, Martínez-Vilalta J, López BC (2012) Growth patterns in relation to drought-induced mortality at two  
779 Scots pine (*Pinus sylvestris* L.) sites in NE Iberian Peninsula. *Trees*, **26**, 621-630.
- 780 Herguido E, Granda E, Benavides R, García-Cervigón AI, Camarero JJ, Valladares F (2016) Contrasting growth  
781 and mortality responses to climate warming of two pine species in a continental Mediterranean ecosystem.  
782 *Forest Ecology and Management*, **363**, 149-158.
- 783 Herms DA, Mattson, WJ (1992) The dilemma of plants: to grow or defend. *Quarterly Review of Biology*, **67**,  
784 283-335.
- 785 Hubbard RM, Rhoades CC, Elder K, Negrón J (2013) Changes in transpiration and foliage growth in lodgepole  
786 pine trees following mountain pine beetle attack and mechanical girdling. *Forest Ecology and Management*,  
787 **289**, 312-317.
- 788 Hultine KR, Dudley TL, Leavitt SW (2013) Herbivory-induced mortality increases with radial growth in an in-  
789 vasive riparian phreatophyte. *Annals of Botany*, **111**, 1197-1206.
- 790 Ireland KB, Moore MM, Fulé PZ, Ziegler TJ, Keane RE (2014) Slow lifelong growth predisposes *Populus*  
791 *tremuloides* trees to mortality. *Oecologia*, **175**, 847-859.

- 792 Kane JM, Kolb TE (2010) Importance of resin ducts in reducing ponderosa pine mortality from bark beetle at-  
793 tack. *Oecologia*, **164**, 601-609.
- 794 Körner C (2015) Paradigm shift in plant growth control. *Current Opinion in Plant Biology*, **25**, 107-114.
- 795 Lin YS, Medlyn BE, Duursma RA *et al.* (2015) Optimal stomatal behaviour around the world. *Nature Climate*  
796 *Change*, **5**, 459-464.
- 797 Luo Y, Chen HY (2015) Climate change-associated tree mortality increases without decreasing water availabil-  
798 ity. *Ecology Letters*, **18**, 1207-1215.
- 799 Macalady AK, Bugmann H (2014) Growth-mortality relationships in pinon pine (*Pinus edulis*) during severe  
800 droughts of the past century: shifting processes in space and time. *Plos One*, **9**, e92770.
- 801 Manion PD (1991) *Tree Disease Concepts*. Prentice-Hall, Englewood Cliffs.
- 802 van Mantgem PJ, Stephenson NL, Byrne JC *et al.* (2009) Widespread increase of tree mortality rates in the west-  
803 ern United States. *Science*, **323**, 521–524.
- 804 Mayr S, Schmid P, Laur J, Rosner S, Charra-Vaskou K, Dämon B, Hacke UG (2014) Uptake of water via  
805 branches helps timberline conifers refill embolized xylem in late winter. *Plant Physiology*, **164**, 1731-1740.
- 806 McDowell NG, Beerling, DJ, Breshears DD, Fisher RA, Raffa KF, Stitt M (2011) The interdependence of mech-  
807 anisms underlying climate-driven vegetation mortality. *Trends in Ecology and Evolution*, **26**, 523-532.
- 808 McDowell NG, Fisher RA, Xu C *et al.* (2013). Evaluating theories of drought-induced vegetation mortality us-  
809 ing a multimodel–experiment framework. *New Phytologist*, **200**, 304-321.
- 810 Meddens AJ, Hicke JA, Ferguson CA (2012) Spatiotemporal patterns of observed bark beetle-caused tree mor-  
811 tality in British Columbia and the western United States. *Ecological Applications*, **22**, 1876-1891.
- 812 Mencuccini M, Onate M, Penuelas J, Rico L, Munné-Bosch S (2014) No signs of meristem senescence in old  
813 Scots pine. *Journal of Ecology*, **102**, 555-565.
- 814 Millar CI, Stephenson NL (2015) Temperate forest health in an era of emerging megadisturbance. *Science*, **349**,  
815 823-826.
- 816 Morris H, Plavcová L, Cvecko P *et al.* (2016) A global analysis of parenchyma tissue fractions in secondary  
817 xylem of seed plants. *New Phytologist*, **209**, 1553-1565
- 818 Myers JA, Kitajima K (2007) Carbohydrate storage enhances seedling shade and stress tolerance in a  
819 Neotropical forest. *Journal of Ecology*, **95**, 383-395
- 820 Nakagawa S, Schielzeth H (2013) A general and simple method for obtaining R<sup>2</sup> from generalized linear  
821 mixed-effects models. *Methods in Ecology and Evolution*, **4**, 133-142.
- 822 Nerhass-Ahles C, Babst F, Klesse S, Nötzli M, Bouriaud O, Neukom R, Dobbertin M, Frank D (2014) The  
823 influence of sampling design on tree-ring-based quantification of forest growth. *Global Change Biology*, **20**,  
824 2867-2885.
- 825 Nesmith JCB, Das AJ, O’Hara KL, van Mantgem PJ (2015) The influence of prefire tree growth and crown con-  
826 dition on postfire mortality of sugar pine following prescribed fire in Sequoia National Park. *Canadian Jour-  
827 nal of Forest Research*, **45**, 910-919

- 828 Niinemets Ü, Valladares F (2006) Tolerance to shade, drought, and waterlogging of temperate Northern Hemi-  
829 sphere trees and shrubs. *Ecological Monographs*, **76**, 521–547.
- 830 Ogle K, Whitham TG, Cobb NS (2000) Tree-ring variation in pinyon predicts likelihood of death following se-  
831 vere drought. *Ecology*, **81**, 3237-3243.
- 832 Oliva J, Stenlid J, Martínez-Vilalta J (2014) The effect of fungal pathogens on the water and carbon economy of  
833 trees: implications for drought-induced mortality. *New Phytologist*, **203**, 1028-1035.
- 834 Pedersen BS (1998) The role of stress in the mortality of midwestern oaks as indicated by growth prior to death.  
835 *Ecology*, **79**, 79-93.
- 836 Pellizzari E, Camarero JJ, Gazol A, Sangüesa-Barreda G, Carrer M (2016) Wood anatomy and carbon-isotope  
837 discrimination support long-term hydraulic deterioration as a major cause of drought-induced dieback. *Global*  
838 *Change Biology*, **22**, 2125-2137
- 839 Piper FI, Gundale MJ, Fajardo A (2015) Extreme defoliation reduces tree growth but not C and N storage in a  
840 winter-deciduous species. *Annals of Botany*, **115**, 1093-1103.
- 841 Plavcová L, Hoch G, Morris H, Ghiasi S, Jansen S (2016) The amount of parenchyma and living fibres affects  
842 storage of non-structural carbohydrates in young stems and roots of temperate trees. *American Journal of*  
843 *Botany*, **103**, 603-612
- 844 R Core Team (2015) R: A language and environment for statistical computing. R Foundation for Statistical  
845 Computing, Vienna, Austria. <http://www.R-project.org/>.
- 846 Rodríguez-Calcerrada J, López, R., Salomón, R., Gordaliza, GG, Valbuena-Carabaña M, Oleksyn J, Gil L  
847 (2015) Stem CO<sub>2</sub> efflux in six co-occurring tree species: underlying factors and ecological implications.  
848 *Plant, Cell and Environment*, **38**, 1104-1115.
- 849 Rose KE, Atkinson RL, Turnbull LA, Rees M (2009) The costs and benefits of fast living. *Ecology Letters*, **12**,  
850 1379-1384.
- 851 Rowland L, da Costa ACL, Galbraith DR *et al.* (2015) Death from drought in tropical forests is triggered by  
852 hydraulics not carbon starvation. *Nature*, **528**, 119-122.
- 853 Sangüesa-Barreda G, Linares JC, Camarero JJ (2013) Drought and mistletoe reduce growth and water-use effi-  
854 ciency of Scots pine. *Forest Ecology and Management*, **296**, 64-73.
- 855 Sangüesa-Barreda G, Linares JC, Camarero JJ (2015) Reduced growth sensitivity to climate in bark-beetle in-  
856 fested Aleppo pines: Connecting climatic and biotic drivers of forest dieback. *Forest Ecology and Manage-*  
857 *ment*, **357**, 126-137.
- 858 Schwarze FW, Fink, S, Deflorio G (2003) Resistance of parenchyma cells in wood to degradation by brown  
859 rot fungi. *Mycological Progress*, **2**, 267-274.
- 860 Seidl R, Fernandes PM, Fonseca TF *et al.* (2011) Modelling natural disturbances in forest ecosystems: a re-  
861 view. *Ecological Modelling*, **222**, 903-924.

862 Wyckoff PH, Clark JS (2002) The relationship between growth and mortality for seven co-occurring tree species  
863 in the southern Appalachian Mountains. *Journal of Ecology*, **90**, 604-615.  
864 Wunder J, Brzeziecki B, Żybura H, Reineking B, Bigler C, Bugmann H (2008) Growth–mortality relationships  
865 as indicators of life-history strategies: a comparison of nine tree species in unmanaged European forests.  
866 *Oikos*, **117**, 815-828.  
867 Wiley E, Rogers BJ, Hodgkinson R, Landhäusser SM (2016) Nonstructural carbohydrate dynamics of lodgepole  
868 pine dying from mountain pine beetle attack. *New Phytologist*, **209**, 550-562.  
869 Zeppel MJ, Harrison SP, Adams HD *et al.* (2015) Drought and resprouting plants. *New Phytologist*, **206**, 583-  
870 589.

871

## 872 **Supporting information caption**

873 **Appendix S1:** Database built-up.

874 **Appendix S2:** Species parameters and wood anatomical variables

875 **Appendix S3:** Effect of the number of years used to calculate early growth ratio on the estimates of the  
876 mixed-effect models fitted to early growth ratio values.

877 **Appendix S4:** Effect of the age-window used to generate the pair of sample of dead and living trees on  
878 the estimates of the mixed-effect models fitted to early growth ratio values.

879 **Appendix S5:** Relationship between the longevity of dead trees and their mean early growth rate

880 **Appendix S6:** Effect of the sampling scheme used to generate the pairs of dying and surviving trees on  
881 the  $g_{f,m}$  values

882 **Appendix S7:** Temporal change in growth ratio before mortality calculated using ring-width (RW) and  
883 basal area increment (BAI) data.

884 **Appendix S8:** Quartile coefficients of dispersion of  $\Delta t_m$ ,  $g_{f,m}$ , and  $g_{50,m}$  values

885 **Appendix S9:** Summary of the fitted mixed effect models for  $\Delta t_m$  and  $g_{f,m}$  calculated using both ring-  
886 width (RW) and basal area increment (BAI) data

887 **Appendix S10:** Summary of the mixed effect models fitted to datasets derived using a bootstrap ap-  
888 proach

889 **Appendix S11:** Summary of the fitted mixed effect models for  $\Delta t_m$ ,  $g_{f,m}$  and  $g_{50,m}$  calculated for each  
890 pair of dying tree / surviving trees with a similar DBH

891 **Appendix S12:** Summary of the fitted mixed effect models for  $\Delta t_m$ ,  $g_{f,m}$  and  $g_{50,m}$  for which the class  
892 ‘biotic agents’ was divided into two groups: ‘Contributing and inciting’ and ‘Predisposing’ biotic  
893 agents.

894 **Appendix S13:** Summary of the fitted mixed effect models for  $\Delta t_m$ ,  $g_{f,m}$  and  $g_{50,m}$  for which the class  
 895 ‘angiosperms’ was divided into two groups: ‘Quercus’ and ‘non-Quercus’ species.

896

897 **Tables**

898 **Table 1:** Main characteristics of the tree-ring database (ring-width data) compiled from 58 published  
 899 papers and unpublished data (Appendix S1), showing details about the number of species and sites  
 900 studied, the number of mortality events and the number of dying and surviving trees by group of mor-  
 901 tality source. Note that we also considered ‘surviving’ information from dying trees (when they were  
 902 still alive); thus the number of ‘surviving’ sets of information is larger than the number of surviving  
 903 trees.

		<b>Drought</b>	<b>Drought + Biotic</b>	<b>Biotic agents</b>	<b>Others</b>
<b>species</b>	angiosperms	6	3	2	3
	gymnosperms	12	6	9	8
<b>sites</b>	angiosperms	10	9	4	4
	gymnosperms	65	28	43	27
<b>mortality events</b>	angiosperms	31	93	25	103
	gymnosperms	301	252	318	373
<b>dying trees</b>	angiosperms	151	160	86	191
	gymnosperms	564	455	570	793
<b>surviving trees</b>	angiosperms	143	565	354	293
	gymnosperms	646	629	658	936

904

905 **Table 2** Summary of the fitted generalized and linear mixed effect models for the duration of the peri-  
 906 od with reduced/increased growth before death ( $\Delta t_m$ ), the growth rate of dying trees relative to surviv-  
 907 ing trees the year before death ( $g_{f,m}$ ), and the growth ratio calculated for the first 50 years of each tree’s  
 908 life ( $g_{50,m}$ ). All variables were calculated using ring-width data (RW). A Poisson model was used for  
 909  $\Delta t_m$  while linear models were fitted to log-transformed  $g_{f,m}$  and  $g_{50,m}$  values.

910 *Top:* For  $\Delta t_m$ , chi-square values and significance levels of the chi-square tests of the variable effects are  
 911 shown, which were derived from type-II variance analysis. Sum of squares and significance levels of  
 912 the variable effects on  $g_{f,m}$  and  $g_{50,m}$  were calculated using type-III variance analysis.

913 *Center:* Estimates of regression coefficients, significance levels (in brackets), and 95% confidence intervals  
 914 intervals of regression coefficients (in square brackets). The intercept corresponds to the reference species  
 915 group (angiosperms) and the reference mortality source (drought). Confidence intervals were calculated  
 916 based on mixed effect models fitted to 500 different datasets generated using a random sample  
 917 of 21 or 17 mortality events per species with replacement (medians in the database for recent and early  
 918 growth ratios, respectively).

919 *Bottom:*  $R^2$  marginal and  $R^2$  conditional indicate the variance explained by fixed effects and by both  
 920 fixed and random effects, respectively.

921 (ns)not significant; (\*) $P < 0.1$ ; \* $P < 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$

922 n: number of mortality events considered in each model. d.f.: degrees of freedom

923

924

925

926

927

928

929

	Duration of the period with reduced/increased growth ( $\Delta t_m$ ; Chi Sq.) RW, n=1496	Growth ratio the year before death ( $g_{f,m}$ ; Sum Sq.) log(RW), n=1496	Early growth ratio ( $g_{50,m}$ ; Sum Sq.) log(RW), n=617
Species group (d.f. = 1)	9.33 **	5.60 **	0.25 (ns)
Mortality group (d.f. = 3)	9.67 *	19.26 ***	1.58 *
Intercept	2.43 *** [2.09 – 2.52]	-0.62 *** [-0.70 – -0.38]	0.02 (ns) [-0.08 – 0.11]
Gymnosperms	0.57 ** [0.28 – 0.71]	-0.28 ** [-0.47 – -0.17]	0.09 (ns) [-0.01 – 0.18]
Drought - Biotic	0.08 (ns)	0.13 (ns)	-0.21 **

	[-0.21 – 0.47]	[-0.14 – 0.29]	[-0.29 – -0.07]
Biotic agents	-0.30 (*) [-0.51 – 0.10]	0.22 * [0.02 – 0.44]	-0.10 (ns) [-0.17 – 0.01]
Others	0.31 (*) [0.00 – 0.68]	-0.28 ** [-0.53 – -0.09]	-0.21 * [-0.36 – -0.06]
R <sup>2</sup> marginal	0.03	0.06	0.03
R <sup>2</sup> conditional	0.26	0.18	0.22

930

Author Manuscript



931 **Table 3** Summary of the relationships between  $\Delta t_m$ ,  $g_{f,m}$ , and  $g_{50,m}$ , and species characteristics (sign in brackets; adjusted  $R^2$ ; and signifi-  
932 cance of the relationship) for angiosperms (A.) and gymnosperms (G.). For each species-specific variable, linear models were fitted to the  
933 conditional means (random effect of the site aggregated by species) of the generalized and linear mixed models.  $g_{f,m}$ , and  $g_{50,m}$  were log-  
934 transformed. Models were not fitted (NA) when data were available for fewer than 4 species (*nb. species*).  
935 Significant relationships are in bold. (\*) $P < 0.1$ ; \* $P < 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$ . (-): negative relationship; (+): positive relationship.  
936 The hydraulic safety margin was measured at water potential corresponding to 50% loss of xylem conductivity. Drought and shade tolerance  
937 parameters (DrTol and ShTol) were available from Niinemets and Valladares (2006; NV06) and from the ForClim forest model (Bugmann  
938 1996; FC)

(a) All mortality sources	nb. species		Duration of the period with reduced/increased growth ( $\Delta t_m$ ; RW)		Growth ratio the year before death ( $g_{f,m}$ ; log RW)		nb. species		Early growth ratio ( $g_{50,m}$ ; log RW)	
	A.	G.	A.	G.	A.	G.	A.	G.	A.	G.
Huber Value	4	10	0.05	-0.01	0.04	(+) <b>0.03</b> *	2	7	NA	0.01
Hydraulic safety margin	7	12	0.01	-0.01	(-) <b>0.19</b> *	(-) <b>0.03</b> *	5	8	-0.08	(-) <b>0.06</b> *
Wood density	12	20	-0.05	(-) <b>0.07</b> ***	-0.05	-0.01	6	14	0.01	(-) <b>0.03</b> (*)
Total parenchyma	7	12	0.02	(-) <b>0.04</b> *	(+) <b>0.42</b> **	(+) <b>0.05</b> **	4	8	0.13	-0.02
Axial parenchyma	7	3	(-) <b>0.17</b> (*)	NA	(+) <b>0.48</b> **	NA	4	8	-0.06	-0.01
DrTol_NV06	10	20	-0.04	-0.01	(+) <b>0.30</b> **	-0.01	4	13	0.07	-0.01
DrTol_FC	12	15	-0.03	(+) <b>0.01</b> (*)	-0.05	-0.01	6	11	-0.08	0.00
ShTol_NV06	10	20	(+) <b>0.20</b> *	(+) <b>0.01</b> (*)	(-) <b>0.32</b> **	-0.00	4	13	0.01	-0.01
ShTol_FC	12	15	-0.01	(+) <b>0.02</b> (*)	(-) <b>0.28</b> **	-0.00	6	10	<b>-0.21</b> (*)	(-) <b>0.06</b> *

(b) Drought-related mortality	nb. species		Duration of the period with reduced/increased growth ( $\Delta t_m$ ; RW)		Growth ratio the year before death ( $g_{f,m}$ ; log RW)		nb. species		Early growth ratio ( $g_{50,m} - \log RW$ )	
	A.	G.	A.	G.	A.	G.	A.	G.	A.	G.
Huber Value	3	6	NA	(-) <b>0.25</b> ***	NA	(+) <b>0.08</b> *	2	4	NA	-0.02
Hydraulic safety margin	5	9	-0.06	-0.00	-0.07	(-) <b>0.03</b> (*)	4	7	(+) <b>0.36</b> *	(-) <b>0.11</b> *
Wood density	9	12	-0.06	(-) <b>0.12</b> ***	0.05	0.00	4	9	(-) <b>0.40</b> *	(-) <b>0.26</b> ***
Total parenchyma	5	6	-0.06	(-) <b>0.29</b> ***	0.00	(+) <b>0.21</b> ***	3	4	NA	-0.00
Axial parenchyma	5	3	(-) <b>0.32</b> *	NA	(+) <b>0.74</b> ***	NA	3	4	NA	(-) <b>0.18</b> *
DrTol_NV06	7	11	-0.07	-0.01	(+) <b>0.27</b> *	-0.01	3	8	NA	(-) <b>0.05</b> (*)
DrTol_FC	9	8	0.04	(+) <b>0.15</b> ***	0.02	(-) <b>0.11</b> **	4	6	0.05	-0.02

940 **Figures caption**

941 **Figure 1.** Geographical distribution of the sites included in the tree-ring database. Sites with similar  
942 species and mortality source in close geographic proximity (difference in latitude and longitude lower  
943 than  $1^\circ$ ) were pooled to improve the clarity of the map; thus the number of symbols does not equal to  
944 the number of sites considered here.

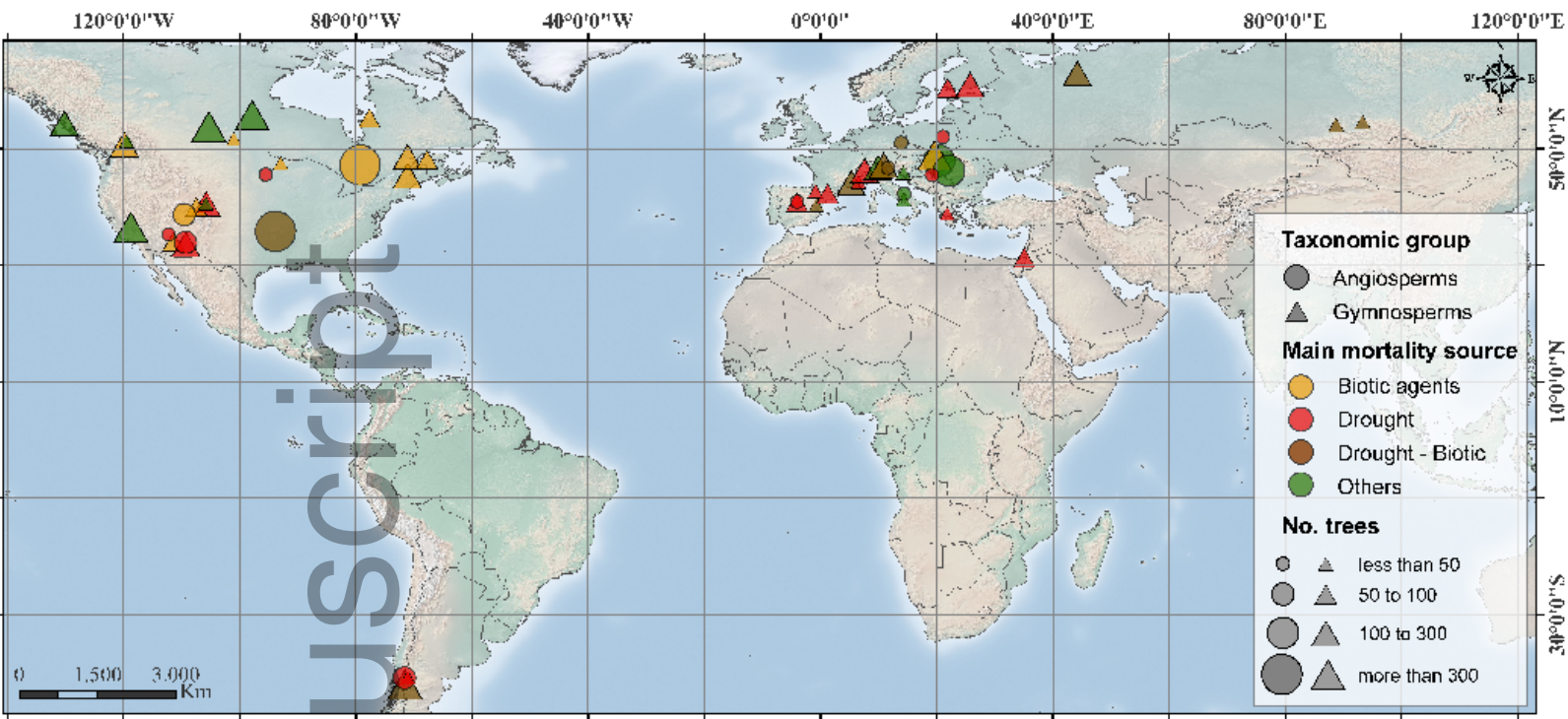
945 **Figure 2.** Example of time-series in growth ratio before mortality (dying / surviving trees) calculated  
946 for *Quercus petraea* trees growing at the site 'Runcu' (Romania; Petritan et al. unpublished dataset) for  
947 three different mortality events (1: 2009; 2: 2000; 3: 2010). The duration of the period with reduced or  
948 increased growth before death ( $\Delta t_{g<1,m}$  and  $\Delta t_{g>1,m}$  respectively, in arrows), and the growth ratio the  
949 year before death ( $g_{f,m}$ ) were used to quantify recent changes in growth rates.

950 **Figure 3:** (a) Temporal change in growth ratio between dying and surviving trees before mortality, and  
951 (b) ontogenetic change in growth ratio calculated using ring-width data (RW) and considering all mor-  
952 tality events. Shaded areas represent the 95% confidence intervals of the medians from bootstrapping  
953 (1000 re-samplings).

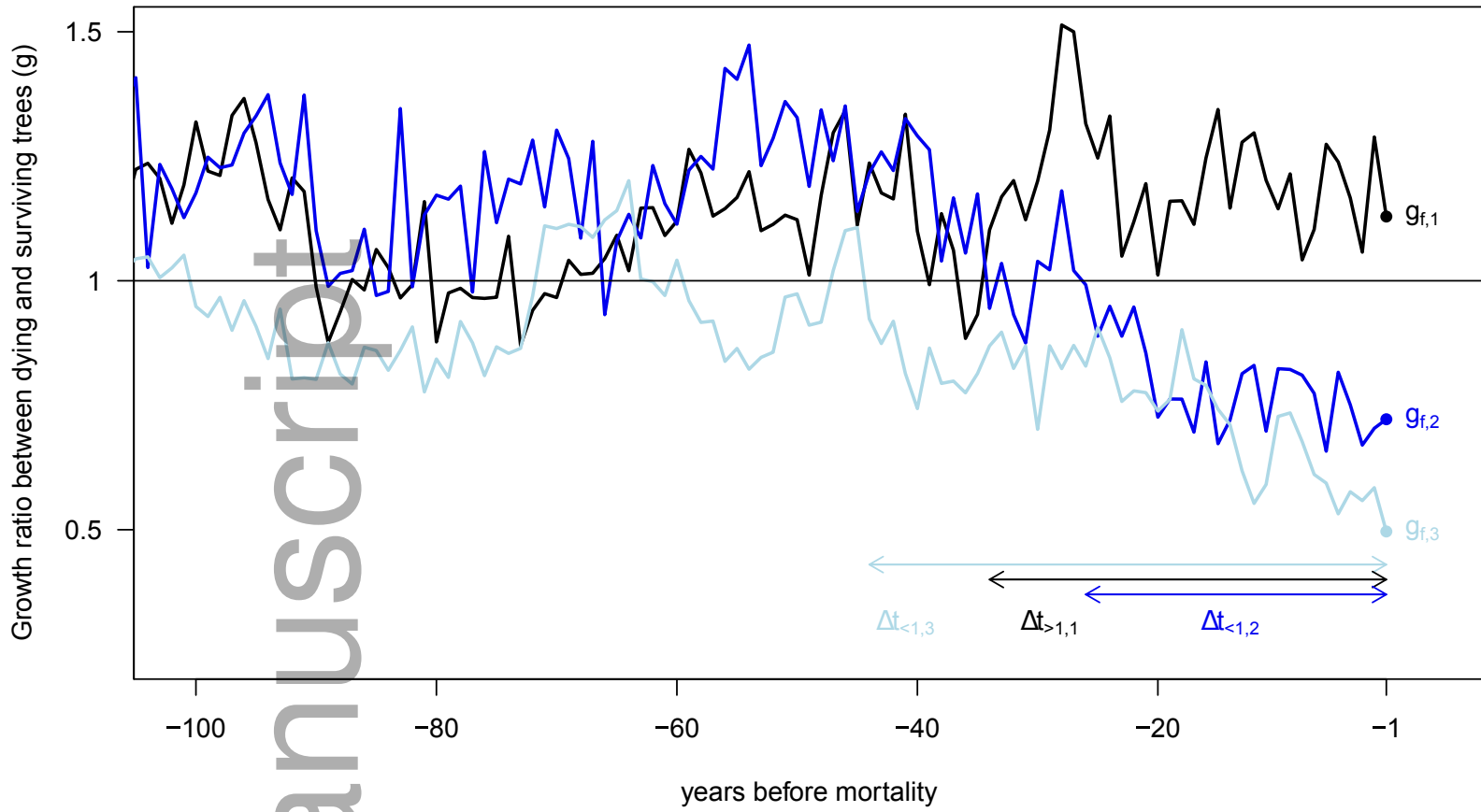
954 **Figure 4:** Distribution of the duration of the period with reduced or increased growth before death (a;  
955  $\Delta t_{g<1,m}$  and  $\Delta t_{g>1,m}$ , respectively), and the growth ratio the year before death (c;  $g_{f,m}$ ) and both variables  
956 (b) calculated using ring-width data. Moving from blue to yellow to red indicates increasing density of  
957 mortality events. Red dotted lines plotted on histograms represent median values ( $\Delta t = 17$  years;  $g_f =$   
958  $0.42$ ).

959 **Figure 5:** Differences in the distribution of the growth ratio the year before death ( $g_t$ ) and the duration  
960 of the period with reduced or increased growth ( $\Delta t$ ) predicted by the generalized and linear mixed mod-  
961 els among groups of mortality sources (Fig. 5a) and between angiosperms and gymnosperms (Fig. 5b).  
962 50% of the values are included in the convex polygons (bags) whose center (median) is represented by  
963 the large dots.

Author Manuscript

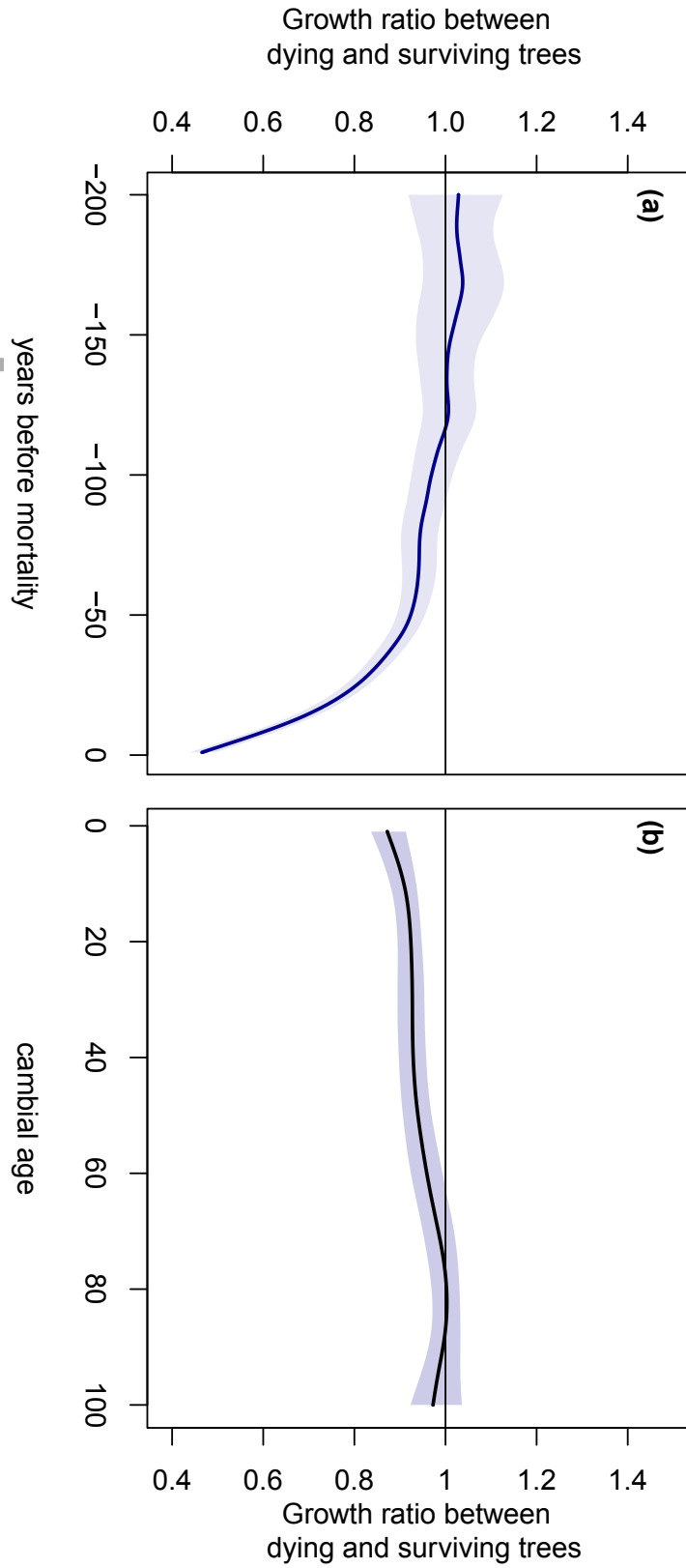


gcb\_13535\_f1.eps

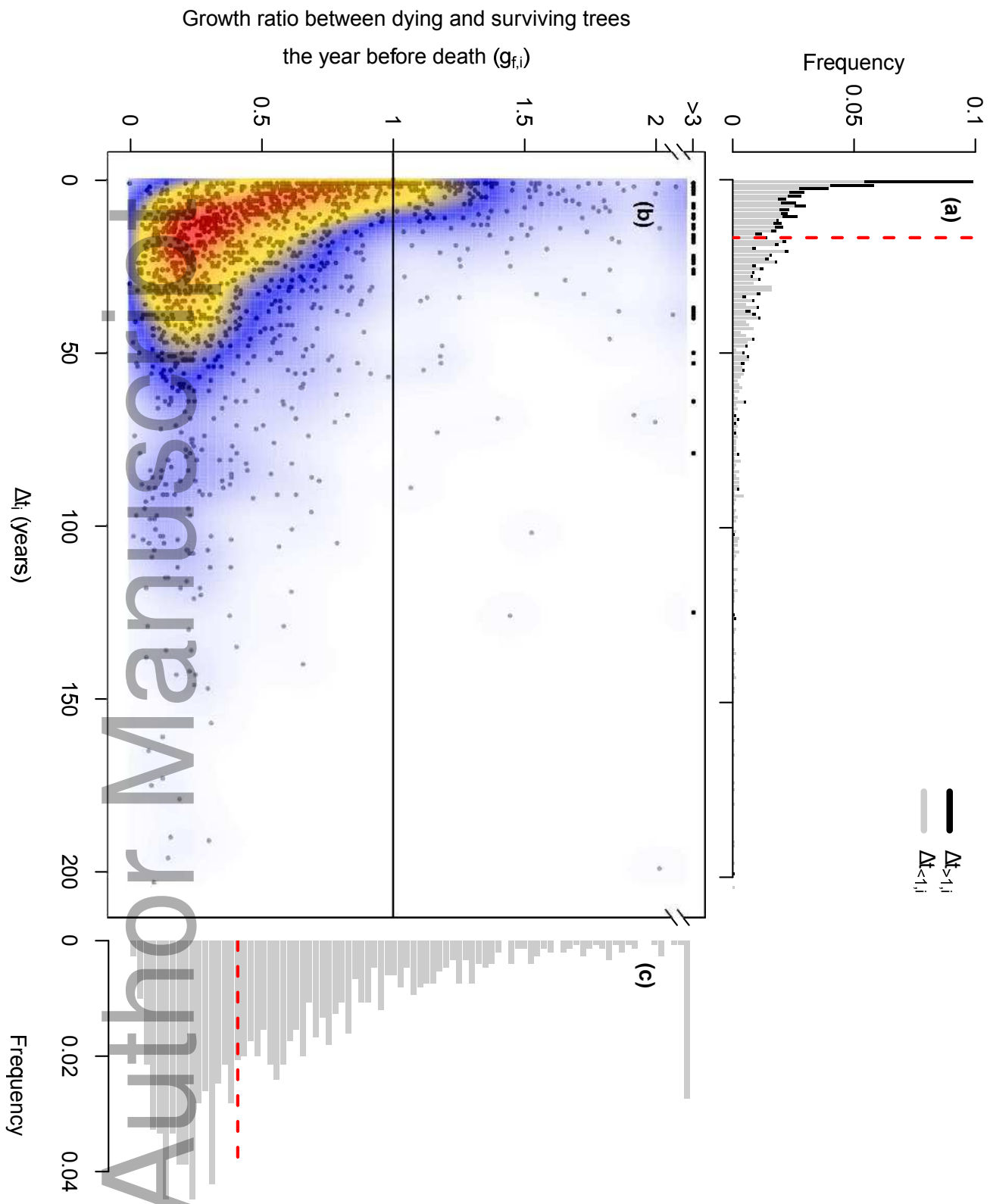


gcb\_13535\_f2.eps

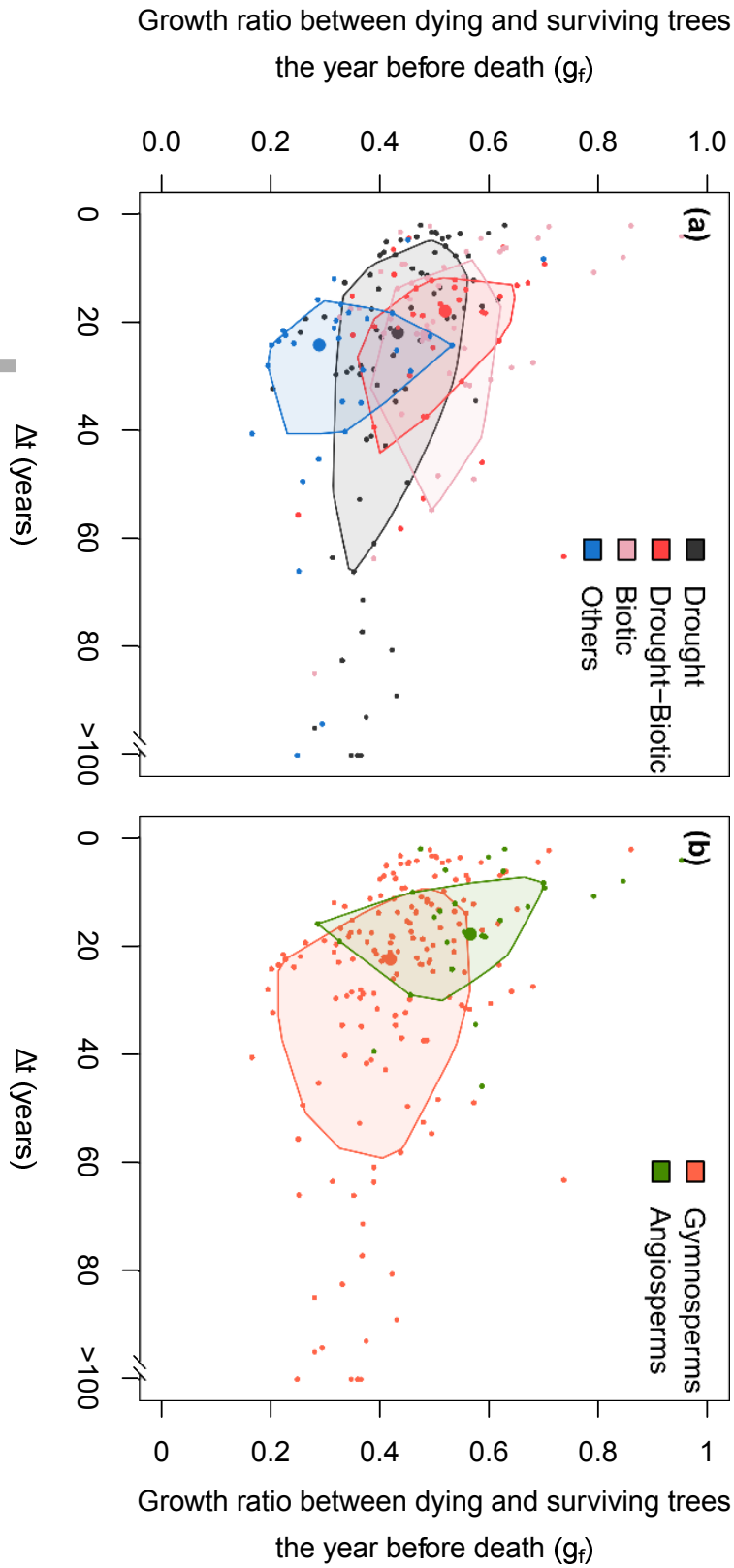
Author Manuscript



gcb\_13535\_f3.eps



gcb\_13535\_f4.eps



gcb\_13535\_f5.eps