



## 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.

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## 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 PAPADOPOULOS<sup>46</sup>, MIKKO PELTONIEMI<sup>47</sup>, ANY MARY PETRITAN<sup>13, 48</sup>, BRIGITTE ROHNER<sup>1, 13</sup>, GABRIEL SANGÜESA-BARREDA<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>

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- 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

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

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

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

#### **Keywords:**

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

#### **Article type:**

Primary research article

#### **Introduction**

Accelerating rates of tree mortality and forest die-off events have been reported worldwide (e.g., van Mantgem et al. 2009; Allen et al. 2010). These trends have been attributed to direct and indirect impacts of drought stress and higher temperatures (e.g., higher competition intensity as a result of growth enhancement in environments limited by low temperature; Luo and Chen 2015), and are expected to continue as a result of further global warming and drying in many regions (Cook et al. 2014; Allen et al. 2015). Tree mortality has large impacts on both short-term forest functioning (e.g., forest productivity, water and carbon cycles; Anderegg et al. 2016b) and long-term ecosystem dynamics (Franklin 1987; Millar et al. 2015), yet our physiological understanding of the mechanisms leading to mortality and our ability to predict mortality and its impacts over space and time is still limited (McDowell et al. 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-

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

## Materials and Methods

### *Tree-ring width database*

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

The sampling approach varied widely across studies. Tree-ring data were derived from cores or cross-sections taken at different sampling heights, from the base to eight meters of height. At 30 sites (15.8% of the sites), tree-ring data were only available for the outermost rings (i.e., partial data). Estimates of cambial age and measures of tree diameter at breast height (DBH) at the time of coring were missing for 58 (30.5%) and 21 (11.1%) sites, respectively, which renders these data inappropriate for our analyses. Trees can die during the growing season before ring formation is complete, which induces an incomplete outermost ring. As the precise (intra-annual) timing of tree death was not available, we did not consider the last ring of the dead trees. The year of death was defined as the year of formation of 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

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

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

### Early growth rate

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

### Designation of the main factors that triggered mortality

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

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

### *Statistical analyses*

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

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

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

## Results

### *Change in growth rates before mortality*

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

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

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

#### *Early growth rates*

Dying trees tended to have lower averaged early growth rate than conspecific surviving ones, especially when a short time period is used to calculate mean juvenile growth rate (Fig. 3b). Considering the first 50 years of a tree's lifetime as representative of its juvenile phase, this trend was observed in 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 not significantly different from one ( $p > 0.1$ ).

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

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

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

Finally, long and strong growth reductions before death were found when mortality was caused by neither drought nor biotic agents, or when the cause was not specified. This group especially included trees that died because of high competition intensity, confirming that shading can suppress trees for a long period before they actually die (Abrams and Orwig 1996). However, the effects of shading (and competition in general) and other stress factors frequently interact (Das et al. 2016; Myers and Kitajima 2007) and are difficult to disentangle in field settings.

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

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

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

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

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

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

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

As reported by Bigler (2016), methodological aspects related to the experimental design and the sampling strategy may explain differences in the relationship between early growth rates and longevity among sites, species or studies. In our database, most of the samples did not cover large gradients of early growth and lifespan (e.g. very old trees or very rapidly/slowly growing trees are missing), mainly because of the relatively low number of dead trees at each site and for each species (Appendix S5). Thus, the lack of consistent trade-off between early growth rates and longevity, and the lack of strong differences among species and mortality sources observed in our synthesis likely reflects high variability in sampling design among sites, and highlights the need for further research on this important topic. Our results show that radial growth reductions before tree mortality are nearly universal. However, their magnitude and the corresponding growth-mortality relationships varied among sources of mortality, between gymnosperms and angiosperms, and among species. These differences largely support our initial hypothesis: angiosperms, trees attacked by bark beetles or stress-sensitive species (e.g., with narrow hydraulic safety margins) typically show a short-term growth decline prior to mortality, while long-lasting growth reductions tend to occur in gymnosperms, stress-tolerant species and may indicate a long-term (chronic) deterioration of the carbon and water economies. Our analyses show that the temporal changes in growth level before death may provide useful insights into the mechanisms underlying tree mortality, and its complex, multi-scale processes. In addition, our results have strong implications for the use of growth data as early warning signal of mortality and for the simulation of tree mortality in dynamic vegetation models. Species- or functional type- specific growth-based mortality algorithms may be powerful for predicting mortality induced by multi-annual stress factors and forecasting gymnosperm death. However, for angiosperms and in case of intense drought or bark-beetle outbreaks, growth-based algorithms are unlikely to be predictive, and must be complemented by physiological and/or anatomical information.

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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.

		Drought	Drought + Biotic	Biotic agents	Others
species	angiosperms	6	3	2	3
	gymnosperms	12	6	9	8
sites	angiosperms	10	9	4	4
	gymnosperms	65	28	43	27
mortality events	angiosperms	31	93	25	103
	gymnosperms	301	252	318	373
dying trees	angiosperms	151	160	86	191
	gymnosperms	564	455	570	793
surviving trees	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.

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

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

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

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

	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



**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 significance of the relationship) for angiosperms (A.) and gymnosperms (G.). For each species-specific variable, linear models were fitted to the 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-transformed. Models were not fitted (NA) when data were available for fewer than 4 species (*nb. species*). Significant relationships are in bold. (\*) $P < 0.1$ ; \* $P < 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$ . (-): negative relationship; (+): positive relationship. The hydraulic safety margin was measured at water potential corresponding to 50% loss of xylem conductivity. Drought and shade tolerance parameters (DrTol and ShTol) were available from Niinemets and Valladares (2006; NV06) and from the ForClim forest model (Bugmann 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**

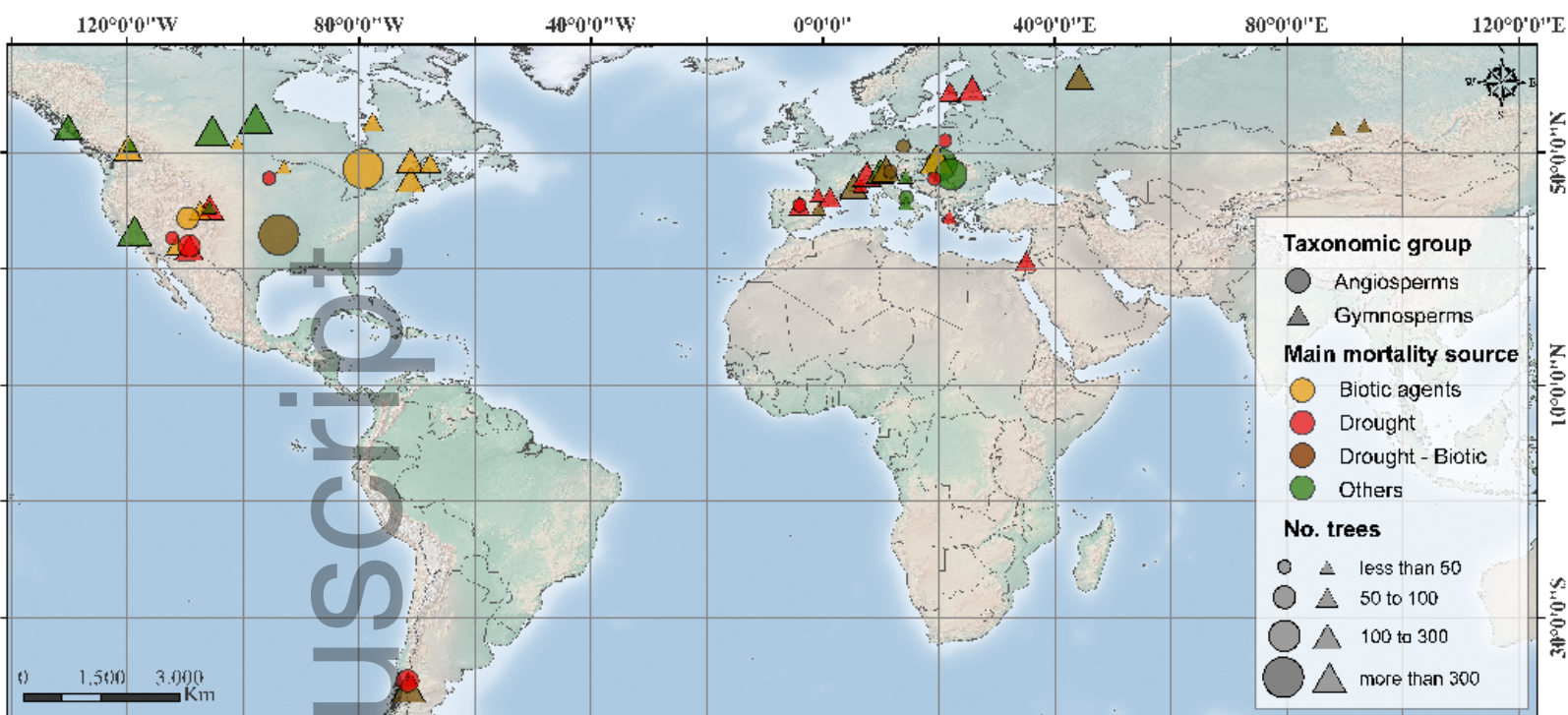
941 **Figure 1.** Geographical distribution of the sites included in the tree-ring database. Sites with similar  
942 species and mortality source in close geographic proximity (difference in latitude and longitude lower  
943 than  $1^\circ$ ) were pooled to improve the clarity of the map; thus the 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.

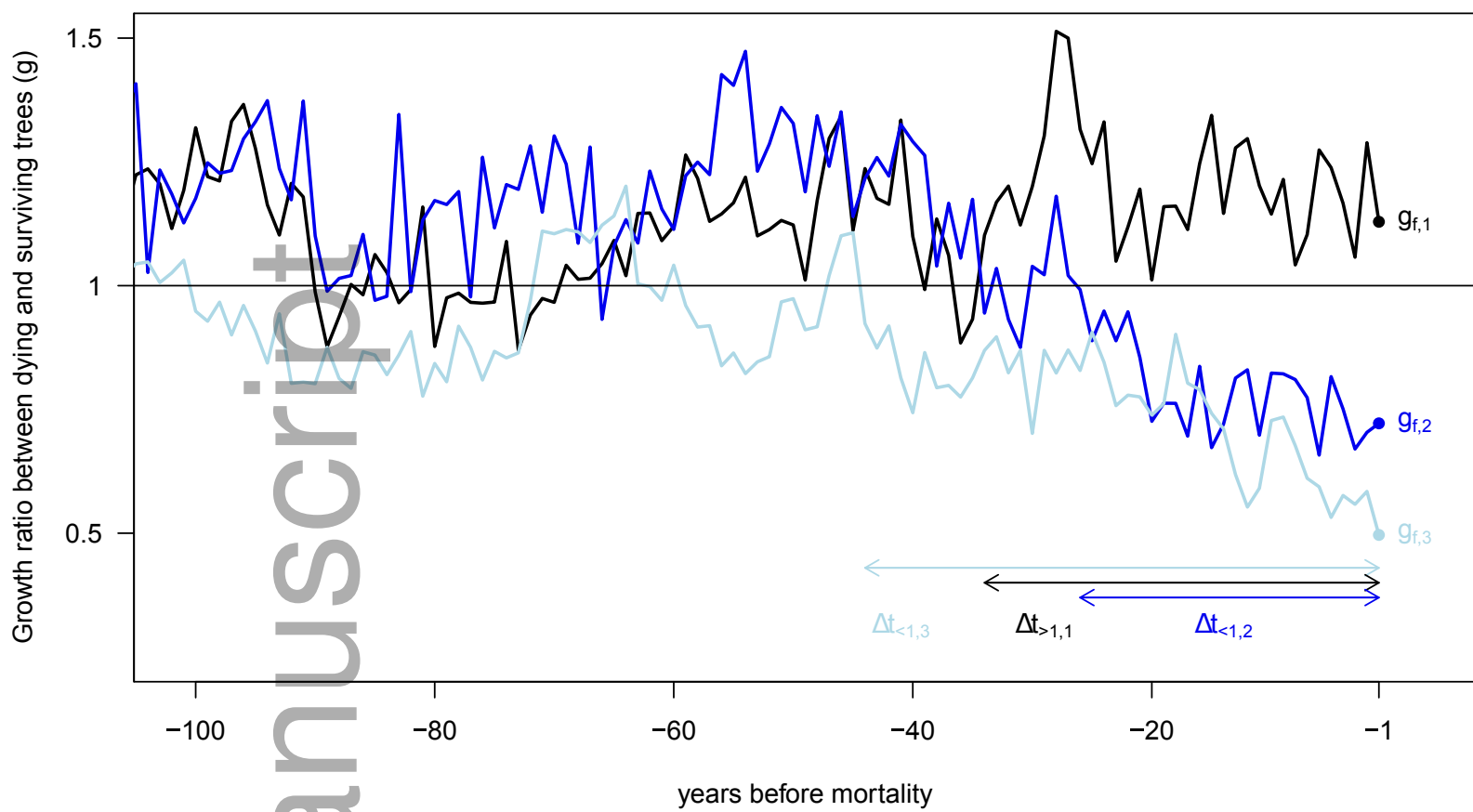
950 **Figure 3:** (a) Temporal change in growth ratio between dying and surviving trees before mortality, and  
951 (b) ontogenetic change in growth ratio calculated using ring-width data (RW) and considering all mor-  
952 tality events. Shaded areas represent the 95% confidence intervals of the medians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

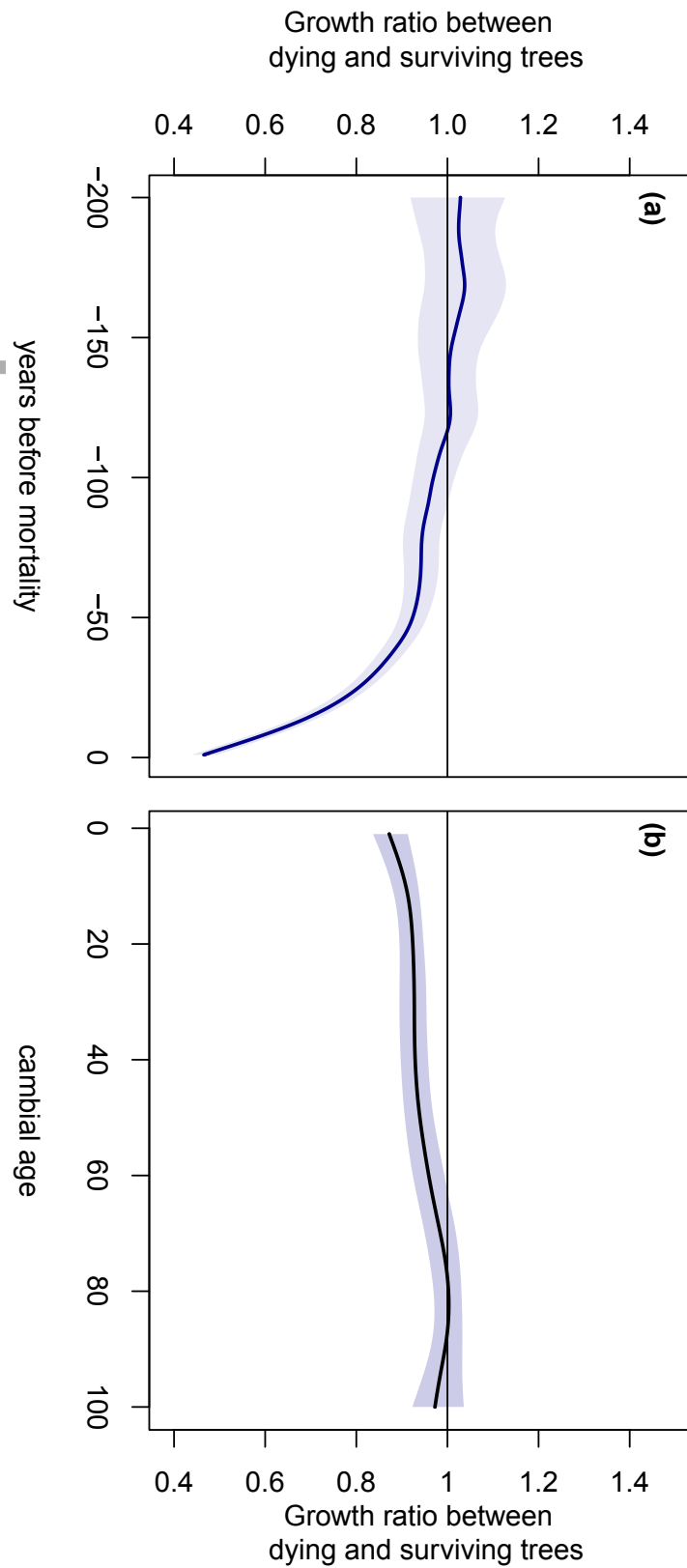
959 **Figure 5:** Differences in the distribution of the growth ratio the year before death ( $g_t$ ) and the duration  
960 of the period with reduced or increased growth ( $\Delta t$ ) predicted by the generalized and linear mixed mod-  
961 els among groups of mortality sources (Fig. 5a) and between 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.



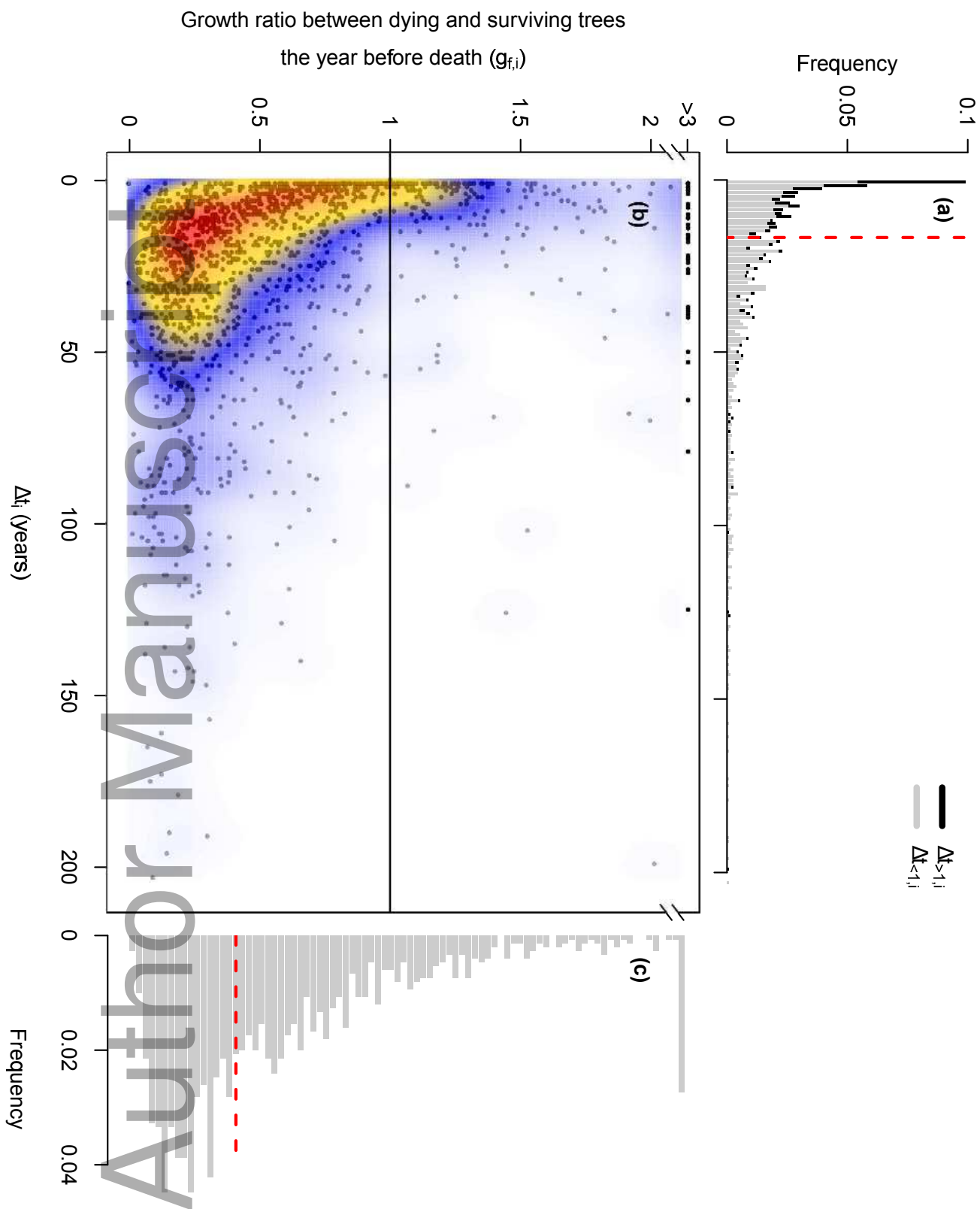
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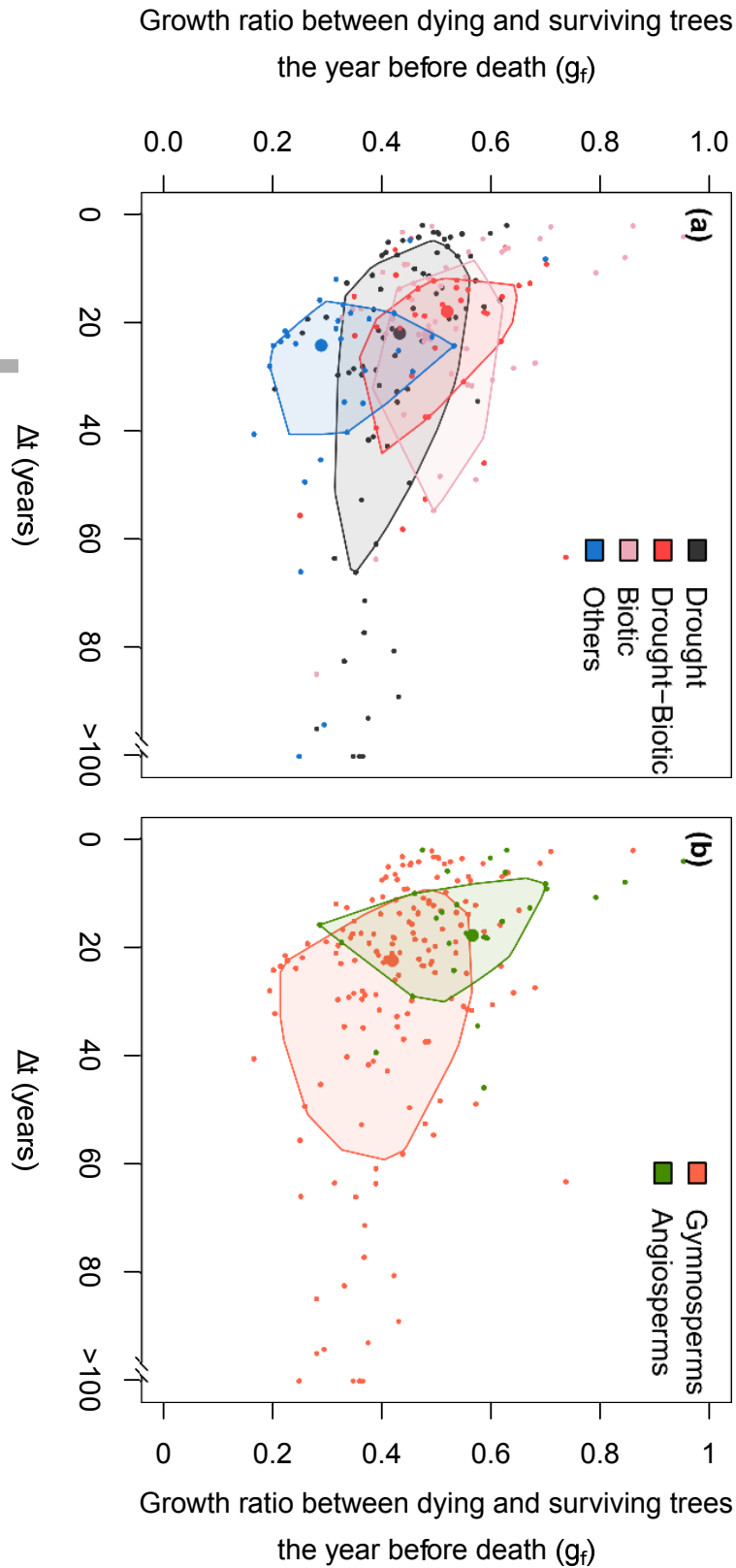
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gcb\_13535\_f4.eps



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