

1 A synthesis of radial growth patterns preceding tree mortality

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

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This is the accepted version of the following article: Cailleret, M. et al. "A synthesis of radial growth patterns preceding tree mortality" in Global change biology (Ed. Wiley), vol. 23 issue 4 (April 2017), p. 1675-1690, which has been published in final form at DOI 10.1111/gcb.13535. **This article may be used for non-commercial purposes in accordance with Wiley Terms and Conditions for Self-Archiving.**

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143 **Abstract**

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

149 We observed a decrease in radial growth before death in ca. 84% of the mortality events. The extent
150 and duration of these reductions were highly variable (1-100 years in 96% of events) due to the com-
151 plex interactions among study species and the source(s) of mortality. Strong and long-lasting declines
152 were found for gymnosperms, shade- and drought-tolerant species, and trees that died from competi-
153 tion. Angiosperms and trees that died due to biotic attacks (especially bark-beetles) typically showed
154 relatively small and short-term growth reductions. Our analysis did not highlight any universal trade-
155 off between early growth and tree longevity within a species, although this result may also reflect high
156 variability in sampling design among sites.

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

165

166

167 **Keywords:**

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

169

170 **Article type:**

171 Primary research article

172 **Introduction**

173

174 Accelerating rates of tree mortality and forest die-off events have been reported worldwide (e.g., van
175 Mantgem et al. 2009; Allen et al. 2010). These trends have been attributed to direct and indirect im-
176 pacts of drought stress and higher temperatures (e.g., higher competition intensity as a result of growth
177 enhancement in environments limited by low temperature; Luo and Chen 2015), and are expected to
178 continue as a result of further global warming and drying in many regions (Cook et al. 2014; Allen et
179 al. 2015). Tree mortality has large impacts on both short-term forest functioning (e.g., forest produc-
180 tivity, water and carbon cycles; Anderegg et al. 2016b) and long-term ecosystem dynamics (Franklin
181 1987; Millar et al. 2015), yet our physiological understanding of the mechanisms leading to mortality
182 and our ability to predict mortality and its impacts over space and time is still limited (McDowell et al.
183 2013; Hartmann et al. 2015). As a result, most dynamic vegetation models that aim to project future
184 forest development are still based on simple mortality algorithms despite their high sensitivity to mor-
185 tality assumptions (Friend et al. 2014; Bircher et al. 2015). In addition, reliable indicators that can be
186 used to predict individual mortality in the field from local to regional scales are lacking (McDowell et
187 al. 2013).

188 In contrast to most mortality events caused by short-term external disturbances, such as windthrow, fire
189 or flooding, stress-induced mortality is usually preceded by changes in tree function (e.g., hydraulic
190 conductivity, carbon assimilation) and structure (e.g., individual leaf area) (McDowell et al. 2011; Seidl
191 et al. 2011; but see Nesmith et al. 2015 for potential influence of pre-fire growth on post-fire mortality).
192 In this context, focusing on the temporal variations in radial stem growth rates is pertinent as they re-
193 flect changes in individual vitality, productivity, and carbon availability (Babst et al. 2014; Aguadé et
194 al. 2015; Dobbertin 2005). Although the inter-annual variability in wood growth is primarily driven by
195 cambial phenology and activity (Delpierre et al. 2015; Körner 2015) – thus by water availability, air

196 temperature and photoperiod – several studies have shown the utility of radial growth data for predict-
197 ing tree mortality probability (e.g., Pedersen 1998; Bigler and Bugmann 2004; Wunder et al. 2008;
198 Cailleret et al. 2016). Most studies used ring-width data as they allow for a long-term (i.e., >20 years)
199 retrospective quantification of annual growth for numerous individuals, sites, and species (e.g., An-
200 deregg et al. 2015a). Such data offer the further advantage of combining a large sample size (in contrast
201 to, for example, dendrometers) with a annual temporal resolution that is helpful to estimate the year of
202 tree death and to detect immediate reactions to intense stress such as drought or insect defoliation
203 (Dobbertin 2005), unlike forest inventories with multi-year re-measurement periods. Moreover, ring-
204 width data are usually available for almost the entire lifespan of a tree, which is valuable for exploring
205 long-term and delayed effects of stress on mortality (see Bigler et al. 2007) that would not be detected
206 using methods such as carbon flux measurements or remote sensing.

207 In most studies, dying trees showed lower radial growth rates prior to death than surviving ones (e.g.,
208 Pedersen 1998; Bigler and Bugmann 2004; Cailleret et al. 2016). Despite this common pattern, a large
209 variety of growth patterns before mortality have been described in the literature from abrupt or gradual
210 growth reductions to increases in growth before death. This variability is likely associated with differ-
211 ences in species' strategies to face environmental stress, and in their carbon allocation patterns related
212 to growth, defense, and storage (Dietze et al. 2014); for example, stress-tolerant species may survive
213 for many years with low growth rates under continuously stressful conditions (e.g., old *Pinus longaeva*),
214 while stress-sensitive species cannot (e.g., *Populus tremuloides*; Ireland et al. 2014). There is also sub-
215 stantial variability at the intra-specific level: drought-induced mortality events of *Pinus sylvestris* may
216 be preceded by fast declines (Herguido et al. 2016), or by slow and long-lasting growth reductions
217 (Bigler et al. 2006; Hereş et al. 2012).

218 Growth patterns before death are also influenced by the type, duration, frequency, and intensity of
219 stress factors that predisposed and triggered mortality. For *Picea engelmannii*, dying trees had lower

220 growth rates than surviving trees when mortality was caused by drought (Bigler et al. 2007), while no
221 differences were observed in two pine species when trees died because of bark beetles (Kane and Kolb
222 2010; Ferrenberg et al. 2014; Sangüesa-Barreda et al. 2015). In case of lethal episodic defoliation, tree
223 death can even be preceded by growth increases (e.g., on *Tamarix spp.* in Hultine et al. 2013). Similar-
224 ly, intra-specific trade-offs between early growth rates (defined as the first 50 years of a tree's life) and
225 longevity were commonly - but not consistently - observed (Bigler 2016; but see Ireland et al. 2014),
226 highlighting the potential disadvantage of investment in growth instead of defenses (Herms and
227 Mattson 1992; Rose et al. 2009).

228 Considering the multifactorial character of the mortality process (McDowell et al. 2011; Aguadé et al.
229 2015; Allen et al. 2015; Anderegg et al. 2015b), and the limited number of species and sites analyzed in
230 most earlier studies, we lack a global, comprehensive appraisal of the changes in growth rates before
231 mortality. This is especially relevant to the detection of variations among sources of mortality (e.g.,
232 drought, insect outbreak), environmental conditions, and species, and to the simulation of tree mortality
233 using growth-based models (Bircher et al. 2015). Moreover, the available studies applied different
234 methodologies to derive growth-mortality relationships (see Cailleret et al. 2016), which reduces the
235 strength of meta-analyses. Thus, we compiled a new pan-continental tree-ring width database from
236 published and unpublished datasets that include both dead and living trees growing at the same sites.
237 We compare the growth rates between trees that died and those that survived stress events. In particu-
238 lar, we address the following questions: (i) Are there characteristic changes in recent radial growth pri-
239 or to mortality? (ii) Did dead trees have higher growth rates when they were young than surviving
240 trees? (iii) To what extent are these growth patterns affected by structure-function differences between
241 gymnosperms and angiosperms, and by the shade and/or drought tolerance of a particular species?, and
242 (iv) are these patterns different depending on the main cause of mortality?

243 We hypothesize on the one hand that short-term (i.e., <5 years) or no decline in growth before death
244 will occur in case of severe biotic attack (especially bark beetles), or in case of drought-induced embo-
245 lism of xylem conduits that impedes water transport to the canopy and leads to tissue desiccation ('hy-
246 draulic failure' hypothesis; McDowell et al. 2011; Rowland et al. 2015). On the other hand, long-term
247 growth reductions (i.e., >20 years) before mortality will be more likely in response to repeated and
248 gradually increasing environmental stress such as shading or parasitism (e.g., mistletoe), where a slow
249 deterioration of the water and carbon economy may lead to tree death because of a lack of non-
250 structural carbohydrates (NSC) to sustain metabolic processes like respiration or to build defense com-
251 pounds ('carbon starvation' hypothesis; McDowell et al. 2011; Hartmann 2015). Accordingly, we ex-
252 pect longer-term growth reductions in shade- and drought-tolerant species than in stress-sensitive ones,
253 and in gymnosperms than in angiosperms, especially due to the wider hydraulic safety margins of coni-
254 fers (Choat et al. 2012). We also hypothesize that trees that died during a specific mortality event will
255 show higher juvenile growth rates than surviving trees (Bigler 2016).

256

257 **Materials and Methods**

258

259 *Tree-ring width database*

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

266 190 sites mostly in North America and Europe in the boreal, temperate and Mediterranean biomes (Fig.
267 1; Table 1; see details in Appendix S1).

268 The sampling approach varied widely across studies. Tree-ring data were derived from cores or cross-
269 sections taken at different sampling heights, from the base to eight meters of height. At 30 sites (15.8%
270 of the sites), tree-ring data were only available for the outermost rings (i.e., partial data). Estimates of
271 cambial age and measures of tree diameter at breast height (DBH) at the time of coring were missing
272 for 58 (30.5%) and 21 (11.1%) sites, respectively, which renders these data inappropriate for our anal-
273 yses. Trees can die during the growing season before ring formation is complete, which induces an in-
274 complete outermost ring. As the precise (intra-annual) timing of tree death was not available, we did
275 not consider the last ring of the dead trees. The year of death was defined as the year of formation of
276 the outermost ring, and considered as a proxy (cf. Bigler & Rigling 2013). At the site scale, tree mortal-
277 ity could be synchronous (all events occurring in one year), or spread in time over many years (the
278 maximum range being > 100 years; Appendix S1).

279 A total of 36 species were included in the database, which covered several gymnosperm and angio-
280 sperm families, although our dataset mainly included gymnosperms (64% of the species and 86% of the
281 sites), with Pinaceae being the most represented family in terms of the number of species and sites
282 sampled, followed by Fagaceae. Species life history strategies were characterized using two sets of
283 shade and drought tolerance indices derived from Niinemets and Valladares (2006) and from the For-
284 Clim dynamic vegetation model (Bugmann 1996; details in Appendix S2). In addition, species struc-
285 tural traits such as wood density (Chave et al. 2009), total and axial parenchyma (Rodríguez-Calcerrada
286 et al. 2015; Morris et al. 2016), Huber value (ratio of conducting xylem area per supported leaf area;
287 Xylem Functional Traits Database; Choat et al. 2012) as well as species' hydraulic safety margin
288 (difference between minimum seasonal water potential measured in the field and the water potential

289 causing 50% loss of xylem conductivity in the stem; Choat et al. 2012) were used to characterize
290 species responses to drought (see Appendix S2).

291 *Growth patterns before mortality*

292 We assumed that all deaths observed for each species within a given site and a given mortality year
293 were consequences of the same mortality process, while deaths that differed in time could be the result
294 of separate processes. Consequently, growth patterns were analyzed for each combination species, site,
295 and mortality year, hereafter referred to as a “mortality event”. Because of the variable methodologies
296 used across sites (see above), we standardized the data among studies to better detect consistent growth
297 patterns. First, for each mortality event (m) we calculated annual growth ratios (g_m) between trees that
298 died (*dying tree*) and conspecific trees that survived that specific mortality event (*surviving tree*) for
299 their entire lifespan up to the mortality year (Berdanier and Clark 2016; Fig. 2). A $g_m < 1$ for a given
300 year indicated that dying trees had lower growth rates than surviving ones. Analyzing this variable was
301 useful to quantify relative changes in growth rate over time, which are better linked with mortality
302 probability than absolute growth rates (Das et al. 2015), but also to remove potential biases due to dif-
303 ferences in sampling schemes among studies (Cailleret et al. 2016). Second, to maximize sample size,
304 g_m were calculated using RW data (1,496 mortality events). RW data capture geometric and size effects
305 (Bowman et al. 2013) that must be removed by adequate data standardization. Thus, we only consid-
306 ered surviving trees with a DBH similar to the dying tree measured at a given mortality year (± 2.5
307 cm). In cases where none of the surviving trees fulfilled this condition, the corresponding mortality
308 event was discarded (123 events were not considered). When not measured in the original study, DBH
309 was estimated as twice the sum of all previous ring-width measurements. Direct age effects were not
310 considered here assuming that senescence only marginally affects tree function (Mencuccini et al.
311 2014). Finally, to assess the dependency of the results to the growth data used, g_m values were also cal-

312 culated using basal area increment (BAI; mm^2) for trees whose DBH was measured (1,000 mortality
313 events).

314 For each of the g_m time-series, we calculated (1) the growth ratio for the year before death ($g_{f,m}$; f for
315 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
316 $g_{f,m} < 1$) or the duration of the continuous period with a $g_m > 1$ before tree death ($\Delta t_{g \geq 1, m}$; in case of $g_{f,m}$
317 ≥ 1 (cf. Fig. 2).

318 *Early growth rate*

319 At each site for which tree cambial age was available, and instead of focusing on growth patterns *per*
320 *se*, we analyzed the ratio in mean RW calculated for the first 50 years of each tree's life between trees
321 that died and trees that survived a given mortality event ($g_{50,m}$). A 50 years period has been used in pre-
322 vious studies linking longevity with growth rates during this period (see Bigler 2016 and Ireland et al.
323 2014). To standardize the data and remove age effects, only surviving trees with an age comparable to
324 the dying one were sampled (± 2 years). When no surviving tree fulfilled this criterion, the correspond-
325 ing mortality event was not considered. This approach has the advantage of using the growth infor-
326 mation from surviving trees. However, as species-specific relationships between early growth rates and
327 mortality risk can be affected by methodological choices (Bigler 2016), we also assessed them (i) by
328 varying the number of years used to calculate early mean RW (Appendix S3), (ii) using different age
329 windows to sample surviving trees corresponding to each dead one (Appendix S4), and (iii) with a
330 method that is more commonly used, i.e., by comparing the growth rate and longevity of dead trees
331 only (Appendix S5).

332 *Designation of the main factors that triggered mortality*

333 The two major sources of mortality were determined for each site based on the expert assessment of the
334 authors of each study, normally combining climatic analyses, growth and mortality data, and the pres-

335 ence/absence of biotic agents. For the present study, we grouped mortality sources into four groups:
336 ‘drought’, ‘biotic’, ‘drought and biotic’, and ‘others’. The first group corresponds to drought-induced
337 mortality caused by a single or several drought events without obvious impact of biotic agents. The
338 group ‘biotic’ includes sites in which mortality was induced primarily by biotic factors, including bark
339 beetle outbreaks, intense leaf or bud herbivory by insects, and/or fungal infection. In the third group,
340 the impact of biotic agents (including mistletoes and wood-borers) was associated with drought. Final-
341 ly, the group ‘others’ included snowbreak, frost events, high competition intensity, and cases in which
342 mortality was induced by a combination of causes without a clear preponderating factor or, simply,
343 where mortality causes were not specified. The proportion of mortality events was uniformly distribut-
344 ed among these four classes ranging from 31.4% to 22.2% for the groups ‘others’ and ‘drought’, re-
345 spectively (Table 1).

346 *Statistical analyses*

347 As the frequency distributions of $g_{f,m}$ and $g_{50,m}$ were right-skewed and long-tailed, i.e., most of the val-
348 ues ranged between 0 and 2 but values exceeding 100 were possible when RW values of living trees \sim
349 0.01mm, and as the distribution in Δt_m was not normal, we analyzed median rather than mean values
350 for interpreting “average” growth patterns. To explore how growth variables differed among species
351 groups (gymnosperms vs. angiosperms) and mortality sources (drought, drought and biotic, biotic, oth-
352 ers), we fitted a generalized linear mixed model for Δt_m , and two linear mixed models for $g_{f,m}$ and $g_{50,m}$,
353 considering these categorical components as fixed effects. The variables $g_{f,m}$ and $g_{50,m}$ were log-
354 transformed to better satisfy normality of the residuals, and we used a Poisson model with a log-link
355 function for Δt_m as this response variable represents count data (see Bolker et al. 2008). As these varia-
356 bles may change among species and sites irrespective of the fixed effects, random effects were estimat-
357 ed for the intercept with site as grouping factor.

358 The variation among sites was not examined itself as we lack specific information on their environment
359 (e.g., climate, soil, forest type). However, aggregating the conditional means of the generalized and
360 linear mixed models by species allowed for estimating the variation in growth variables within and
361 among species (e.g., with species drought tolerance) irrespective of their group and of the mortality
362 source. As data on life history and structural traits were not available for every species, these variables
363 were not included as fixed effects in the models to avoid loss of statistical power. Interactions among
364 species groups and mortality sources were not considered in the final models as model fit was reduced
365 in their presence (higher AIC, Akaike Information Criterion). Type-III chi-squares and type-II sum of
366 squares variance analyses were used to estimate the respective impact of species group and source of
367 mortality on Δt_m as well as on $g_{f,m}$ and $g_{50,m}$, respectively. Coefficients of determination were used to
368 assess the percent contribution of fixed effects alone (R^2 marginal) and both fixed and random effects
369 (R^2 conditional) for explaining the variability in growth patterns (Nakagawa & Schielzeth, 2013).

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

383

384 **Results**

385

386 *Change in growth rates before mortality*

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

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

402 The variation in $g_{f,m}$ and Δt_m was high within species groups and mortality groups, with the same order
403 of magnitude as the variation within species and sites (quantile coefficients of dispersion; Appendix
404 S8). As a consequence, the fixed effects considered in the generalized and linear mixed models ex-
405 plained only a small part of the variance in $g_{f,m}$ and Δt_m (R^2 marginal = 0.06 and 0.03, respectively);

406 however significant differences among species groups and mortality sources could be detected (Table
407 2). Inter-site variability explained a larger part of the variance (R^2 conditional = 0.18 and 0.26) that
408 could be related with inter-specific differences in shade and drought tolerance (within species group).
409 Results of the generalized and linear mixed models were consistent regardless of the data source (RW
410 or BAI data; Appendix S9), regardless of the properties of the calibration dataset in terms of the distri-
411 bution of mortality events per site and species (Table 2 and Appendix S10), and regardless of whether
412 dying trees were grouped per mortality year or not (Appendix S11).

413 In case of drought-induced mortality, the median in RW $g_{f,m}$ and Δt_m predicted by the mixed effect
414 models was 0.42 and 19 years, respectively (Fig. 5a), identical to the values obtained when considering
415 all sources of mortality. Relative to cases in which drought was the main source of mortality, Δt_m and
416 $g_{f,m}$ did not significantly differ when drought was associated with biotic agents. Growth reductions,
417 however, tended to be shorter and more intense (lower Δt_m and higher $g_{f,m}$, respectively), when trees
418 were killed by biotic agents alone ($p < 0.1$; Table 2) and, particularly, when trees were attacked by bark
419 beetles ($p < 0.05$; Appendix S12). Trees that died because of other factors (including inter-individual
420 competition) showed the longest and strongest period of reduced growth before death (predicted medi-
421 an in $\Delta t_m = 24$ years and in $g_{f,m} = 0.29$; Fig. 5a; Table 2).

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

428 *Species characteristics associated with growth patterns before mortality*

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

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

442 *Early growth rates*

443 Dying trees tended to have lower averaged early growth rate than conspecific surviving ones, especial-
444 ly when a short time period is used to calculate mean juvenile growth rate (Fig. 3b). Considering the
445 first 50 years of a tree's lifetime as representative of its juvenile phase, this trend was observed in
446 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
447 significantly different from one ($p > 0.1$).

448 Significant differences among mortality groups were highlighted by the generalized linear mixed mod-
449 els. Early growth ratio was highest when mortality was caused by drought alone, and lowest when it
450 was induced by drought combined with biotic agents and by other factors. These differences were sig-
451 nificant using $g_{50,m}$ (Table 2), and also by averaging early growth rate over different time-windows

452 (number of years fixed across species or as a function of species lifespan; Appendix S3). There was a
453 tendency towards higher early growth ratio for gymnosperms than angiosperms, but this result was not
454 consistent when comparing different approaches to define the early growth ratio (Appendix S3).

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

460

461 **Discussion**

462

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

469 *General growth patterns before mortality*

470 Our synthesis supports that dying trees commonly show lower growth rates prior to death than surviv-
471 ing ones ($g_{f,m} < 1$). Considering all mortality events, the decrease in growth the year before death aver-
472 aged ~60% (median in $g_{f,m} \sim 0.4$). This substantial growth reduction may have been overestimated be-
473 cause of the reduction in the competitive ability of dying trees, which may have benefited the growth of
474 surviving individuals (Cavin et al. 2013). However, this effect was compensated, at least partially, by

475 the fact that the group of ‘surviving’ trees at a given mortality event may include trees with reduced
476 growth that died shortly after the event. Although growth reductions before mortality are nearly
477 universal, our results show that they can be abrupt or gradual, and the duration of the period with
478 reduced growth (Δt_m) was highly variable, ranging from 1 to 100 years in 96% of the cases. Overall,
479 62% of the mortality events showed reduced growth 5–50 years preceding tree death, consistent with
480 previous studies (e.g., ~5 years in Bond-Lamberty et al. 2014; 6–12 years in Wyckoff and Clark 2002;
481 10–15 years in Ogle et al. 2000; ~15 years in Camarero et al. 2015; ~30 years in Macalady and Bug-
482 mann 2014). These results confirm that trees can survive a long time with low growth, and emphasize
483 the role of accumulated stress or slow-acting processes (e.g., competition) in tree mortality (Das et al.
484 2008). However, it is noticeable that in 18% of the mortality events, trees died after a fast (≤ 5 years)
485 growth decline in comparison to trees that survived, highlighting quick tree responses to intense stress.
486 In 19% of the mortality events, trees died after experiencing only a slight decrease or even a short-term
487 increase in growth ($g_{f,m} > 0.9$). Similar observations are rather rare in the literature (but see Ferrenberg
488 et al. 2014; Rowland et al. 2015; Berdanier and Clark 2016; Herguido et al. 2016), and indicate either
489 that radial growth can be prioritized until the point of death irrespective of environmental stress, or that
490 stress can be strong enough to kill trees without any impact on the carbon budget and its allocation to
491 growth.

492 In addition to this general pattern, a wide range of growth patterns (Δt_m and $g_{f,m}$) within mortality
493 sources, within species, and within sites was observed. This variability likely reflects: (i) the classifica-
494 tion of mortality into four broad groups, disregarding the multifactorial character of mortality in many
495 cases and the inherent complexity of mortality processes (Allen et al. 2015; Anderegg et al. 2015b), (ii)
496 the difficult and somewhat arbitrary identification of the sources of mortality and quantification of their
497 respective role under field conditions, and (iii) the high spatio-temporal heterogeneity in micro-climate,
498 soil and stand density conditions and pressure from biotic agents within some sites. Even though most

499 of the variability in Δt_m and $g_{f,m}$ was not explained by the categorical variables considered here (low
500 variance explained by the generalized and linear mixed models), the high dimensionality of the tree-
501 ring database in terms of sample size, diversity of species, and mortality causes allowed us to detect
502 differences among these groups. Considering that the outputs of the generalized and linear mixed mod-
503 els were coherent no matter what methodology was used to calculate growth ratios (Appendices S6, S9
504 and S11), and what calibration dataset was used to fit them (Table 2; Appendix S10), we are confident
505 about the reliability of our results.

506 *Growth patterns before mortality vary among sources of mortality*

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

520 The slower growth signal associated with mortality induced by bark-beetle outbreaks may reflect a
521 negative effect of carbon allocation to growth rather than defense on tree survival (growth-
522 differentiation balance hypothesis; Herms and Mattson 1992) and could be explained by several

523 hypotheses. First, the disruption of carbohydrate transport due to phloem feeding by bark beetles and
524 xylem occlusion by the fungi they introduce (Hubbard et al. 2013) usually have major consequences for
525 tree functioning, leading to leaf shedding and tree death within a few years (Meddens et al. 2012;
526 Wiley et al. 2016). Second, in the endemic phase, bark beetles may not preferentially attack trees with
527 slow growth (Sangüesa-Barreda et al. 2015; but see Macalady and Bugmann 2014), but rather trees
528 with specific size and/or bark thickness, and with lower defense capacities (less resin duct production;
529 Kane and Kolb 2010; Ferrenberg et al. 2014). Third, considering that tree growth is frequently sink-
530 driven (Körner 2015), and that defoliation does not increase water stress (but may actually decrease it
531 due to lower whole-tree transpiration), a single biotic defoliation event may not strongly affect tree
532 growth (but see Piper et al. 2015).

533 Finally, long and strong growth reductions before death were found when mortality was caused by nei-
534 ther drought nor biotic agents, or when the cause was not specified. This group especially included
535 trees that died because of high competition intensity, confirming that shading can suppress trees for a
536 long period before they actually die (Abrams and Orwig 1996). However, the effects of shading (and
537 competition in general) and other stress factors frequently interact (Das et al. 2016; Myers and Kitajima
538 2007) and are difficult to disentangle in field settings.

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

540 As hypothesized, angiosperm species, and especially *Quercus* species, did not commonly show long-
541 lasting reduced growth periods before death but rather died after a fast decline, or even after a short-
542 term increase in growth before death. In contrast, gymnosperm species commonly showed long-term
543 and slow growth reductions before death. Angiosperms tend to recover quickly from extreme events,
544 whereas gymnosperms feature substantial legacy effects (e.g., after drought; Anderegg et al. 2015a),
545 which may reveal the slow but chronic deterioration of their carbon balance and hydraulic performance
546 under gradual or repeated environmental stress (Dickman et al. 2015; Pellizzari et al. 2016). This inter-

547 pretation is consistent with recent findings showing that reduced NSC concentrations are frequently
548 associated with drought-induced mortality in gymnosperms but not in angiosperms (Anderegg et al.,
549 2016a). Higher growth fluctuations in angiosperms than gymnosperms are likely associated to a num-
550 ber of attributes, including: (i) high growth efficiency (Brodribb et al. 2012) and productivity in fertile
551 conditions (Augusto et al. 2014), associated with less conservative water use and higher stomatal con-
552 ductance (Lin et al. 2015); (ii) higher amount of wood parenchyma that may serve to increase storage
553 capacity of NSC and symplastic water (Morris et al. 2016; Plavcová et al. 2016), (iii) high capacity to
554 resprout unlike most species in the Pinaceae family (Zeppel et al. 2015); (iv) narrower hydraulic safety
555 margins (Choat et al. 2012); and, possibly, (v) potential capacity to refill embolized xylem conduits
556 (Choat et al. 2012, 2015; but see Mayr et al. 2014 for passive hydraulic recovery in conifers). However,
557 because of the rather small number of angiosperm tree species studied, we acknowledge that more re-
558 search using a larger number of species, including tropical angiosperms, is needed to validate our hy-
559 pothesis.

560 Similarly, growth patterns before death differed among species according to their stress tolerance and
561 resistance and the related structural and functional traits. Because of the relatively low number of the
562 species studied and the limited availability of functional trait data, the correlation among traits was not
563 captured by the univariate analysis we used. Therefore, sufficient care should be taken while interpret-
564 ing these results. Nevertheless, our findings provide some physiological explanations for the differ-
565 ences between angiosperms and gymnosperms mentioned above. Long-term, strong reductions in
566 growth before death were more frequently observed for drought-tolerant species – according to For-
567 Clim’s parameters – with wide hydraulic safety margins, a low amount of wood parenchyma, and low
568 Huber values (for gymnosperms). Shade-tolerant species showed longer and stronger reductions in
569 growth before death than intolerant ones, as evident from comparing species-specific tolerance indices
570 derived from ForClim and Niinemets and Valladares (2006), confirming their ability to survive under

571 shading for a long period (Wyckoff and Clark 2002; Wunder et al. 2008). Despite the probable link
572 between wood density and mortality risk of angiosperms (Anderegg et al. 2016a), this trait was not
573 associated with particular growth patterns before death.

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

575 Intra-specific trade-offs between growth rates during the juvenile phase and tree longevity have been
576 observed frequently for angiosperm and gymnosperm species, while positive relationships have been
577 rarely found (Black et al. 2008; Ireland et al. 2014; Bigler 2016). In our synthesis we did not find evi-
578 dence of a consistent trade-off in gymnosperms and in angiosperms (Appendix S5). In 58.6% of the
579 mortality events, dying trees had lower early growth rates than surviving ones ($g_{50,m} < 1$), especially
580 when mortality was caused by other agents or by drought and biotic attack than drought alone. Early
581 investment in rapid growth may provide a strong advantage under light-limited conditions (e.g., in
582 dense stands). However, as highlighted by the high $g_{50,m}$ values in case of drought-induced mortality
583 and for species with low wood density, it may constitute a disadvantage under dry conditions, where
584 investment into mechanisms to increase water uptake capacity and hydraulic function may be favored.
585 Similarly, promoting early growth instead of whole-tree defenses may be a disadvantage in case of bio-
586 tic attack or insect defoliation (Rose et al. 2009), but our analysis did not fully support this hypothesis.
587 As reported by Bigler (2016), methodological aspects related to the experimental design and the sam-
588 pling strategy may explain differences in the relationship between early growth rates and longevity
589 among sites, species or studies. In our database, most of the samples did not cover large gradients of
590 early growth and lifespan (e.g. very old trees or very rapidly/slowly growing trees are missing), mainly
591 because of the relatively low number of dead trees at each site and for each species (Appendix S5).
592 Thus, the lack of consistent trade-off between early growth rates and longevity, and the lack of strong
593 differences among species and mortality sources observed in our synthesis likely reflects high variabil-
594 ity in sampling design among sites, and highlights the need for further research on this important topic.

595 *Conclusions*

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

611

612 **Acknowledgments**

613

614 This study generated from the COST Action STReSS (FP1106) financially supported by the EU
615 Framework Programme for Research and Innovation HORIZON 2020. We are particularly grateful to
616 Professor Dr. Ute Sass-Klaassen from Wageningen University (The Netherlands), chair of the action,
617 for making this meta-study possible. We also thank members of the Laboratory of Plant Ecology from

618 the University of Ghent (Belgium) for their help while compiling the database; Louise Filion for shar-
619 ing her dataset; Dario Martin-Benito for providing some ForClim parameters; the ARC-NZ Vegetation
620 Function Network for supporting the compilation of the Xylem Functional Traits dataset; Edurne Mar-
621 tinez del Castillo for the creation of Figure 1; and two anonymous reviewers and Phillip van Mantgem
622 (USGS) for their suggestions to improve the quality of the paper.

623 MC was funded by the Swiss National Science Foundation (project number 140968), SJ by the German
624 Research Foundation (JA 2174/3-1), EMRR by the Research Foundation - Flanders (FWO, Belgium)
625 and by the EU HORIZON 2020 Programme through a Marie Skłodowska-Curie IF fellowship (No
626 659191), LDS by a postdoctoral fellowship from the Portuguese *Fundação para a Ciência e a Tecno-*
627 *logia* (FCT) (SFRH/ BPD/70632/2010), TA by the Academy of Finland (proj. no. 252629 and 276255),
628 JAA by the British Columbia Forest Science Program and the Forest Renewal BC (Canada), BB and
629 WO by the Austrian Science Fund (FWF, Hertha Firnberg Programme project T667-B16 and FWF
630 P25643-B16), VC, PJ, MS and VT by the Czech Ministry of Education (MŠMT, project COST CZ no.
631 LD13064 and LD14074), JJC, JCLC and GSB by the Spanish Ministry of Economy (projects
632 CGL2015-69186-C2-1-R, CGL2013-48843-C2-2-R, and CGL2012-32965) and the EU (project FED-
633 ER 0087 TRANSHABITAT), MRC by the Natural Sciences and Engineering Research Council of
634 Canada (NSERC) and by the *Service de la protection contre les insectes et les maladies du ministère*
635 *des forêts du Québec* (Canada), KC by the Slovenian Research Agency (ARRS) - program P4-0015,
636 AD by the United States Geological Survey (USGS), HD by the French National Research Agency
637 (ANR, DRYADE project ANR-06-VULN-004) and the metaprogramme Adaptation of Agriculture and
638 Forests to Climate Change (AAFCC) of the French National Institute for Agricultural Research
639 (INRA), MD by the Israeli Ministry of Agriculture and Rural Development as a chief scientist and by
640 the Jewish National Fund (Israel), GGI by the Spanish Ministry of Economy and Competitiveness (Pro-
641 ject AGL2014-61175-JIN), SG by the *Bundesministerium für Bildung und Forschung* (BMBF) through
642 the project REGKLAM (Grant number: 01 LR 0802) (Germany), LJH by the Arkansas Agricultural

643 Experiment Station (United States of America) and the United States Department of Agriculture - For-
644 est Service, HH by the Natural Sciences and Engineering Research Council of Canada, AMH by the
645 Spanish Ministry of Science and Innovation (projects CGL2007-60120 and CSD2008-0040) and by the
646 Spanish Ministry of Education via a FPU scholarship, VIK by the Russian Science Foundation (grant
647 #14-24-00112), TKi and RV by the *Consejo Nacional de Investigaciones Científicas y Técnicas* (CO-
648 NICET Grant PIP 112-201101-00058 and PIP 112-2011010-0809) (Argentina), TKI by the Weizmann
649 Institute of Science (Israel) under supervision of Professor Dan Yakir, by the Keren Kayemeth LeIsrael
650 (KKL) – Jewish National Fund (JNF) (Alberta-Israel program 90-9-608-08), by the Sussman Center
651 (Israel), by the Cathy Wills and Robert Lewis Program in Environmental Science (United Kingdom),
652 by the France-Israel High Council for Research Scientific and Technological Cooperation (project 3-
653 6735) and by the Minerva Foundation (Germany), KK by the project "Resilience of Forests" of the
654 Ministry of Economic Affairs (The Netherlands - WUR Investment theme KB19), TL by the program
655 and research group P4-0107 Forest Ecology, Biology and Technology (Slovenia), RLV by a postdoc-
656 toral fellowship from the *Portuguese Fundação para a Ciência e a Tecnologia* (FCT;
657 SFRH/BPD/86938/2012), RLR by the EU FP7 Programme through a Marie Skłodowska-Curie IOF
658 fellowship (No 624473), HM by the Academy of Finland (Grant Nos. 257641 and 265504), SM by
659 Sparkling Science of the Federal Ministry of Science, Research and Economy (BMWFW) of Austria,
660 IM by the Hungarian Scientific Research Fund (No. K101552), JMM by the Circumpolar-Boreal Al-
661 berta grants program from the Natural Science and Engineering Research Council of Canada, MP by
662 the EU project LIFE12 ENV/FI/000409, AMP by a Swiss Research Fellowship (Sciex-NMSch, Project
663 13.272 – OAKAGE), JMS by the American National Science Foundation (grant 0743498), ABS by the
664 British Columbia Ministry of Forests, Lands and Natural Resource Operations (Canada), DS by the
665 Public Enterprise “Vojvodinasume” (project Improvement of Lowland Forest Management), MLS by
666 the *Consejo Nacional de Investigaciones Científicas y Técnicas* (CONICET Grant PIP
667 11420110100080) and by *El Fondo para la Investigación Científica y Tecnológica* (FONCyT Grant

668 PICT 2012-2009), RT by the Italian Ministry of Education (University and Research 2008, *Ciclo del*
669 *Carbonio ed altri gas serra in ecosistemi forestali, naturali ed artificiali dell’America Latina: analisi*
670 *preliminare, studio di fattibilità e comparazione con ecosistemi italiani*) and by the EU LIFE+ project
671 MANFOR C.BD. (Environment Policy and Governance 2009, Managing forests for multiple purposes:
672 carbon, biodiversity and socio-economic wellbeing), ARW by the Natural Sciences and Engineering
673 Council (NSERC) (Canada) through the University of Winnipeg and by Manitoba Conservation (Cana-
674 da) and JMV by the Spanish Ministry of Economy and Competitiveness (grant CGL2013-46808-R).
675 Any use of trade names is for descriptive purposes only and does not imply endorsement by the U.S.
676 Government.

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866 **Supporting information caption**

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

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

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

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

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

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

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

878 **Appendix S8:** Quartile coefficients of dispersion of Δt_m , $g_{f,m}$, and $g_{50,m}$ values

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

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

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

885 **Appendix S12:** Summary of the fitted mixed effect models for Δt_m , $g_{f,m}$ and $g_{50,m}$ for which the class
886 ‘biotic agents’ was divided into two groups: ‘Contributing and inciting’ and ‘Predisposing’ biotic
887 agents.

888 **Appendix S13:** Summary of the fitted mixed effect models for Δt_m , $g_{f,m}$ and $g_{50,m}$ for which the class
889 ‘angisperms’ was divided into two groups: ‘Quercus’ and ‘non-Quercus’ species.

890

891 **Tables**

892 **Table 1:** Main characteristics of the tree-ring database (ring-width data) compiled from 58 published
 893 papers and unpublished data (Appendix S1), showing details about the number of species and sites
 894 studied, the number of mortality events and the number of dying and surviving trees by group of mor-
 895 tality source. Note that we also considered ‘surviving’ information from dying trees (when they were
 896 still alive); thus the number of ‘surviving’ sets of information is larger than the number of surviving
 897 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

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

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

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

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

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

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

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	Duration of the period with reduced/increased growth (Δt_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) [-0.21 – 0.47]	0.13 (ns) [-0.14 – 0.29]	-0.21 ** [-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 ² marginal	0.03	0.06	0.03
R ² conditional	0.26	0.18	0.22

925 **Table 3** Summary of the relationships between Δt_m , $g_{f,m}$, and $g_{50,m}$, and species characteristics (sign in brackets; adjusted R^2 ; and significance
926 of the relationship) for angiosperms (A.) and gymnosperms (G.). For each species-specific variable, linear models were fitted to the condi-
927 tional 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-
928 transformed. Models were not fitted (NA) when data were available for fewer than 4 species (*nb. species*).

929 Significant relationships are in bold. (*) $P < 0.1$; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$. (-): negative relationship; (+): positive relationship.

930 The hydraulic safety margin was measured at water potential corresponding to 50% loss of xylem conductivity. Drought and shade tolerance
931 parameters (DrTol and ShTol) were available from Niinemets and Valladares (2006; NV06) and from the ForClim forest model (Bugmann
932 1996; FC)

(a) All mortality sources	nb. species		Duration of the period with reduced/increased growth (Δt_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	(+) 0.03 *	2	7	NA	0.01
Hydraulic safety margin	7	12	0.01	-0.01	(-) 0.19 *	(-) 0.03 *	5	8	-0.08	(-) 0.06 *
Wood density	12	20	-0.05	(-) 0.07 ***	-0.05	-0.01	6	14	0.01	(-) 0.03 (*)
Total parenchyma	7	12	0.02	(-) 0.04 *	(+) 0.42 **	(+) 0.05 **	4	8	0.13	-0.02
Axial parenchyma	7	3	(-) 0.17 (*)	NA	(+) 0.48 **	NA	4	8	-0.06	-0.01
DrTol_NV06	10	20	-0.04	-0.01	(+) 0.30 **	-0.01	4	13	0.07	-0.01
DrTol_FC	12	15	-0.03	(+) 0.01 (*)	-0.05	-0.01	6	11	-0.08	0.00
ShTol_NV06	10	20	(+) 0.20 *	(+) 0.01 (*)	(-) 0.32 **	-0.00	4	13	0.01	-0.01
ShTol_FC	12	15	-0.01	(+) 0.02 (*)	(-) 0.28 **	-0.00	6	10	-0.21 (*)	(-) 0.06 *

933

(b) Drought-related mortality	nb. species		Duration of the period with reduced/increased growth (Δt_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	(-) 0.25 ***	NA	(+) 0.08 *	2	4	NA	-0.02
Hydraulic safety margin	5	9	-0.06	-0.00	-0.07	(-) 0.03 (*)	4	7	(+) 0.36 *	(-) 0.11 *
Wood density	9	12	-0.06	(-) 0.12 ***	0.05	0.00	4	9	(-) 0.40 *	(-) 0.26 ***
Total parenchyma	5	6	-0.06	(-) 0.29 ***	0.00	(+) 0.21 ***	3	4	NA	-0.00
Axial parenchyma	5	3	(-) 0.32 *	NA	(+) 0.74 ***	NA	3	4	NA	(-) 0.18 *
DrTol_NV06	7	11	-0.07	-0.01	(+) 0.27 *	-0.01	3	8	NA	(-) 0.05 (*)
DrTol_FC	9	8	0.04	(+) 0.15 ***	0.02	(-) 0.11 **	4	6	0.05	-0.02

934 **Figures caption**

935 **Figure 1.** Geographical distribution of the sites included in the tree-ring database. Sites with similar
936 species and mortality source in close geographic proximity (difference in latitude and longitude lower
937 than 1°) were pooled to improve the clarity of the map; thus the number of symbols does not equal to
938 the number of sites considered here.

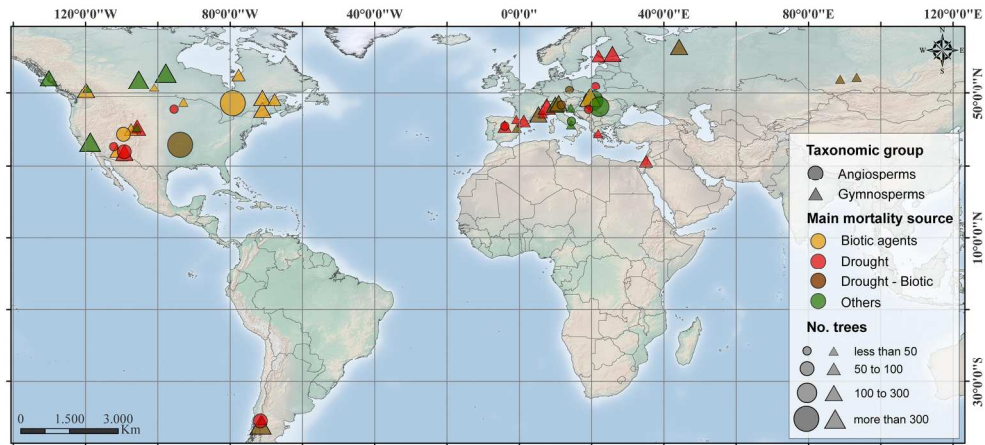
939 **Figure 2.** Example of time-series in growth ratio before mortality (dying / surviving trees) calculated
940 for *Quercus petraea* trees growing at the site 'Runcu' (Romania; Petritan et al. unpublished dataset) for
941 three different mortality events (1: 2009; 2: 2000; 3: 2010). The duration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

944 **Figure 3:** (a) Temporal change in growth ratio between dying and surviving trees before mortality, and
945 (b) ontogenetic change in growth ratio calculated using ring-width data (RW) and considering all mor-
946 tality events. Shaded areas represent the 95% confidence intervals of the medians from bootstrapping
947 (1000 re-samplings).

948 **Figure 4:** Distribution of the duration of the period with reduced or increased growth before death (a;
949 $\Delta t_{g<1,m}$ and $\Delta t_{g>1,m}$, respectively), and the growth ratio the year before death (c; $g_{f,m}$) and both variables
950 (b) calculated using ring-width data. Moving from blue to yellow to red indicates increasing density of
951 mortality events. Red dotted lines plotted on histograms represent median values ($\Delta t = 17$ years; $g_f =$
952 0.42).

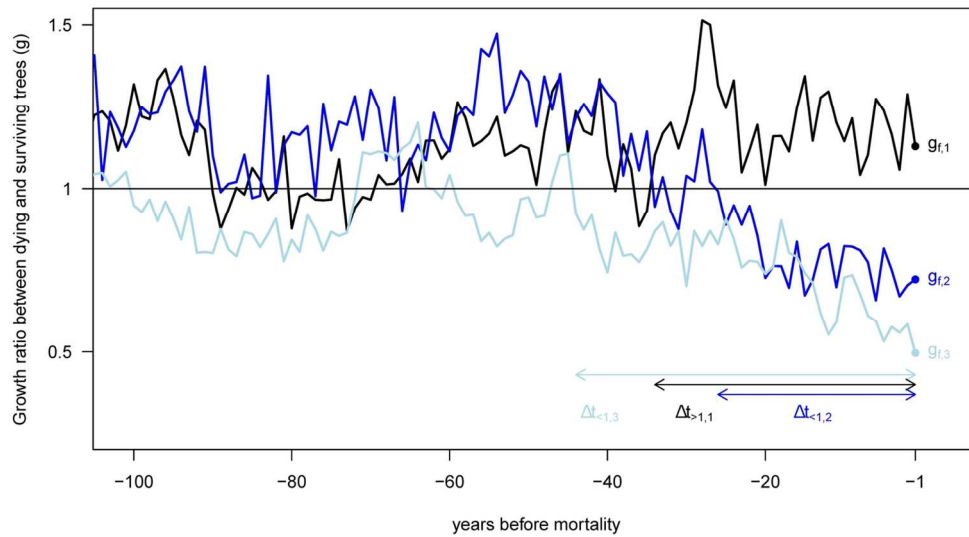
953 **Figure 5:** Differences in the distribution of the growth ratio the year before death (g_f) and the duration
954 of the period with reduced or increased growth (Δt) predicted by the generalized and linear mixed mod-
955 els among groups of mortality sources (Fig. 5a) and between angiosperms and gymnosperms (Fig. 5b).

956 50% of the values are included in the convex polygons (bags) whose center (median) is represented by
957 the large dots.



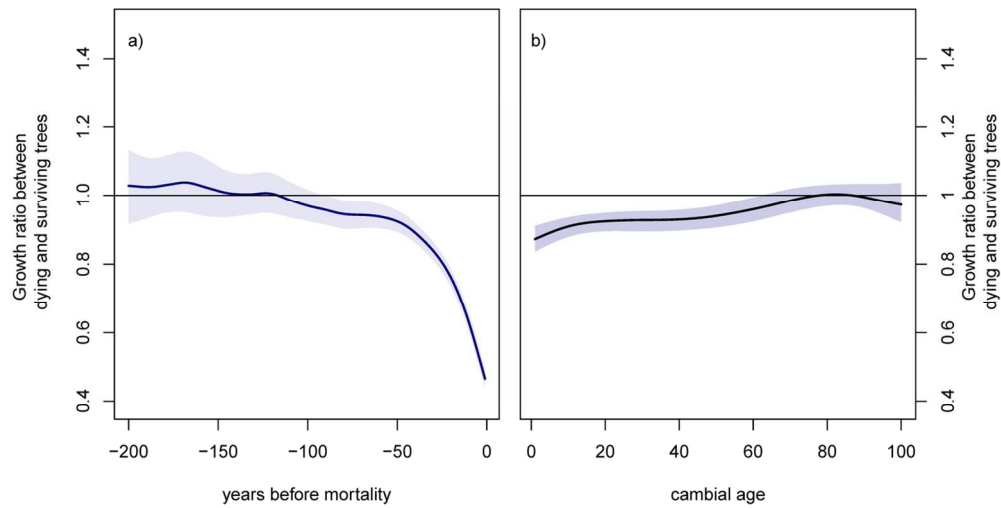
Geographical distribution of the sites included in the tree-ring database. Sites with similar species and mortality source in close geographic proximity (difference in latitude and longitude lower than 1°) were pooled to improve the clarity of the map; thus the number of symbols does not equal to the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

190x85mm (300 x 300 DPI)



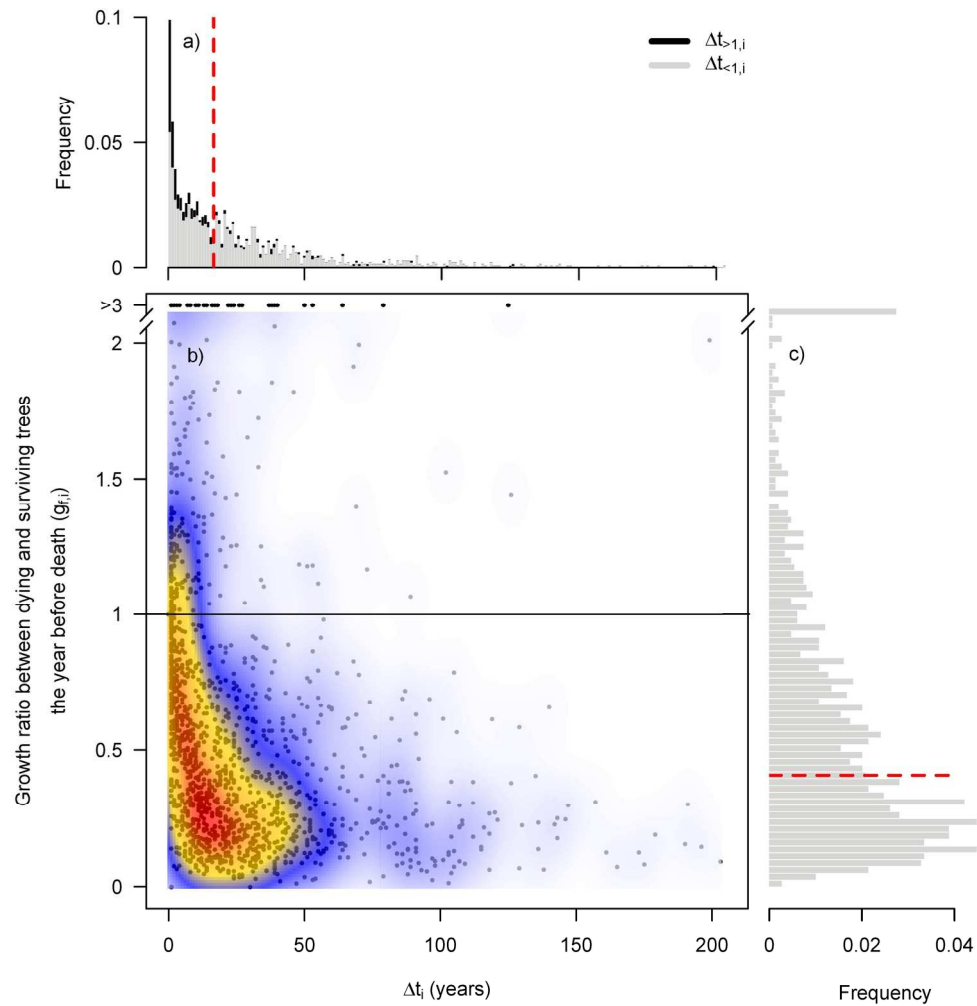
Example of time-series in growth ratio before mortality (dying / surviving trees) calculated for *Quercus petraea* trees growing at the site 'Runcu' (Romania; Petritan et al. unpublished dataset) for three different mortality events (1: 2009; 2: 2000; 3: 2010). The duration of the period with reduced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

129x69mm (300 x 300 DPI)



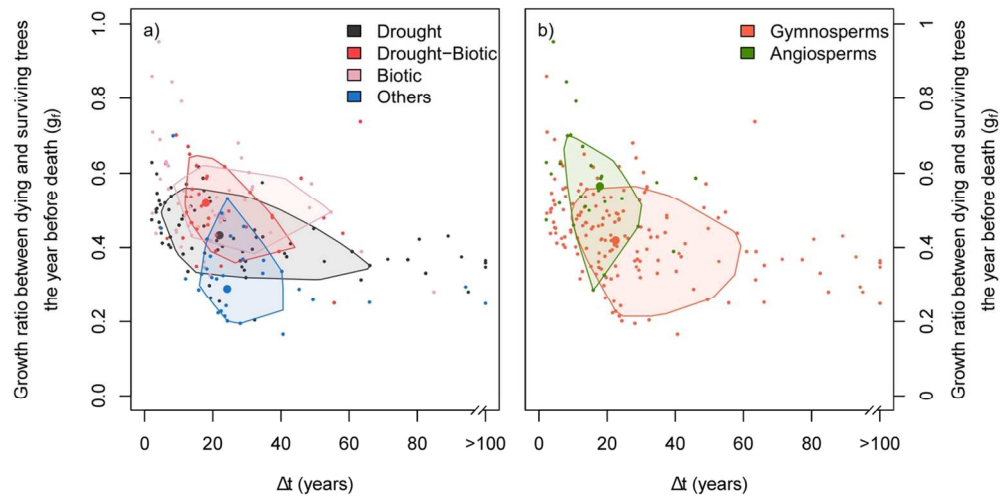
(a) Temporal change in growth ratio between dying and surviving trees before mortality, and (b) ontogenetic change in growth ratio calculated using ring-width data (RW) and considering all mortality events. Shaded areas represent the 95% confidence intervals of the medians from bootstrapping (1000 resamplings).

121x61mm (300 x 300 DPI)



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

177x177mm (300 x 300 DPI)



Differences in the distribution of the growth ratio the year before death (g_t) and the duration of the period with reduced or increased growth (Δt) predicted by the generalized and linear mixed models among groups of mortality sources (Fig. 5a) and between angiosperms and gymnosperms (Fig. 5b). 50% of the values are included in the convex polygons (bags) whose center (median) is represented by the large dots.

116x58mm (300 x 300 DPI)