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

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149

150 **Abstract**

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

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

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

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

172

173

174 **Keywords:**

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

176

177 **Article type:**

178 Primary research article

179 **Introduction**

180

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

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

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

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

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

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

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

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

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

263

264 **Materials and Methods**

265

266 *Tree-ring width database*

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

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

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

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

298 *Growth patterns before mortality*

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

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

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

325 *Early growth rate*

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

339 *Designation of the main factors that triggered mortality*

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

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

353 *Statistical analyses*

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

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

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

390

391 **Results**

392

393 *Change in growth rates before mortality*

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

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

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

409 The variation in $g_{f,m}$ and Δt_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 Δt_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 Δt_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, Δt_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 Δt_m and higher $g_{f,m}$, respectively), when trees
425 were killed by biotic agents alone ($p < 0.1$; Table 2) and, particularly, when trees were attacked by bark
426 beetles ($p < 0.05$; Appendix S12). Trees that died because of other factors (including inter-individual
427 competition) showed the longest and strongest period of reduced growth before death (predicted medi-
428 an in $\Delta t_m = 24$ years and in $g_{f,m} = 0.29$; Fig. 5a; Table 2).

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

435 *Species characteristics associated with growth patterns before mortality*

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

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

449 *Early growth rates*

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

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

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

467

468 **Discussion**

469

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

476 *General growth patterns before mortality*

477 Our synthesis supports that dying trees commonly show lower growth rates prior to death than surviv-
478 ing ones ($g_{f,m} < 1$). Considering all mortality events, the decrease in growth the year before death aver-
479 aged ~60% (median in $g_{f,m} \sim 0.4$). This substantial growth reduction may have been overestimated be-
480 cause of the reduction in the competitive ability of dying trees, which may have benefited the growth of
481 surviving individuals (Cavin et al. 2013). However, this effect was compensated, at least partially, by
482 the fact that the group of ‘surviving’ trees at a given mortality event may include trees with reduced
483 growth that died shortly after the event. Although growth reductions before mortality are nearly
484 universal, our results show that they can be abrupt or gradual, and the duration of the period with
485 reduced growth (Δt_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 (Δt_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 Δt_m and $g_{f,m}$ was not explained by the categorical variables considered here (low
507 variance explained by the generalized and linear mixed models), the high dimensionality of the tree-
508 ring database in terms of sample size, diversity of species, and mortality causes allowed us to detect
509 differences among these groups. Considering that the outputs of the generalized and linear mixed mod-
510 els were coherent no matter what methodology was used to calculate growth ratios (Appendices S6, S9
511 and S11), and what calibration dataset was used to fit them (Table 2; Appendix S10), we are confident
512 about the reliability of our results.

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

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

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

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

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

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

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

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

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

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

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

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

617

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685

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

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

- 757 Delpierre N, Berveiller D, Granda E, Dufrêne E (2015) Wood phenology, not carbon input, controls the
758 interannual variability of wood growth in a temperate oak forest. *New Phytologist*, **210**, 459-470.
- 759 Dickman LT, McDowell NG, Sevanto S, Pangle RE, Pockman WT (2015) Carbohydrate dynamics and mortality
760 in a piñon-juniper woodland under three future precipitation scenarios. *Plant, Cell and Environment*, **38**,
761 729-739.
- 762 Dietze MC, Sala A, Carbone MS, Czimczik CI, Mantooth JA, Richardson AD, Vargas R (2014) Nonstructural
763 carbon in woody plants. *Annual Review of Plant Biology*, **65**, 667-687.
- 764 Dobbertin M (2005) Tree growth a indicator of tree vitality and of tree reaction to environmental stress: a re-
765 view. *European Journal of Forest Research*, **124**, 319-333.
- 766 Ferrenberg S, Kane JM, Mitton JB (2014). Resin duct characteristics associated with tree resistance to bark bee-
767 tles across lodgepole and limber pines. *Oecologia*, **174**, 1283-1292.
- 768 Franklin JF, Shugart, HH, Harmon ME (1987) Tree death as an ecological process. *BioScience*, **37**, 550-556.
- 769 Friend AD, Lucht W, Rademacher TT *et al.* (2014) Carbon residence time dominates uncertainty in terrestrial
770 vegetation responses to future climate and atmospheric CO₂. *Proceedings of the National Academy of*
771 *Sciences of the United States of America*, **111**, 3280-3285.
- 772 Hartmann H (2015) Carbon starvation during drought-induced tree mortality – are we chasing a myth? *Journal*
773 *of Plant Hydraulics*, **2**, e005.
- 774 Hartmann H, Adams HD, Anderegg WRL, Jansen S, Zeppel MJ (2015) Research frontiers in drought-induced
775 tree mortality: crossing scales and disciplines. *New Phytologist*, **205**, 965-969.
- 776 Hartmann H, Messier C (2008) The role of forest tent caterpillar defoliations and partial harvest in the decline
777 and death of sugar maple. *Annals of Botany*, **102**, 377-387.
- 778 Hereş AM, Martínez-Vilalta J, López BC (2012) Growth patterns in relation to drought-induced mortality at two
779 Scots pine (*Pinus sylvestris* L.) sites in NE Iberian Peninsula. *Trees*, **26**, 621-630.
- 780 Herguido E, Granda E, Benavides R, García-Cervigón AI, Camarero JJ, Valladares F (2016) Contrasting growth
781 and mortality responses to climate warming of two pine species in a continental Mediterranean ecosystem.
782 *Forest Ecology and Management*, **363**, 149-158.
- 783 Herms DA, Mattson, WJ (1992) The dilemma of plants: to grow or defend. *Quarterly Review of Biology*, **67**,
784 283-335.
- 785 Hubbard RM, Rhoades CC, Elder K, Negrón J (2013) Changes in transpiration and foliage growth in lodgepole
786 pine trees following mountain pine beetle attack and mechanical girdling. *Forest Ecology and Management*,
787 **289**, 312-317.
- 788 Hultine KR, Dudley TL, Leavitt SW (2013) Herbivory-induced mortality increases with radial growth in an in-
789 vasive riparian phreatophyte. *Annals of Botany*, **111**, 1197-1206.
- 790 Ireland KB, Moore MM, Fulé PZ, Ziegler TJ, Keane RE (2014) Slow lifelong growth predisposes *Populus*
791 *tremuloides* trees to mortality. *Oecologia*, **175**, 847-859.

- 792 Kane JM, Kolb TE (2010) Importance of resin ducts in reducing ponderosa pine mortality from bark beetle at-
793 tack. *Oecologia*, **164**, 601-609.
- 794 Körner C (2015) Paradigm shift in plant growth control. *Current Opinion in Plant Biology*, **25**, 107-114.
- 795 Lin YS, Medlyn BE, Duursma RA *et al.* (2015) Optimal stomatal behaviour around the world. *Nature Climate*
796 *Change*, **5**, 459-464.
- 797 Luo Y, Chen HY (2015) Climate change-associated tree mortality increases without decreasing water availabil-
798 ity. *Ecology Letters*, **18**, 1207-1215.
- 799 Macalady AK, Bugmann H (2014) Growth-mortality relationships in pinon pine (*Pinus edulis*) during severe
800 droughts of the past century: shifting processes in space and time. *Plos One*, **9**, e92770.
- 801 Manion PD (1991) *Tree Disease Concepts*. Prentice-Hall, Englewood Cliffs.
- 802 van Mantgem PJ, Stephenson NL, Byrne JC *et al.* (2009) Widespread increase of tree mortality rates in the west-
803 ern United States. *Science*, **323**, 521–524.
- 804 Mayr S, Schmid P, Laur J, Rosner S, Charra-Vaskou K, Dämon B, Hacke UG (2014) Uptake of water via
805 branches helps timberline conifers refill embolized xylem in late winter. *Plant Physiology*, **164**, 1731-1740.
- 806 McDowell NG, Beerling, DJ, Breshears DD, Fisher RA, Raffa KF, Stitt M (2011) The interdependence of mech-
807 anisms underlying climate-driven vegetation mortality. *Trends in Ecology and Evolution*, **26**, 523-532.
- 808 McDowell NG, Fisher RA, Xu C *et al.* (2013). Evaluating theories of drought-induced vegetation mortality us-
809 ing a multimodel–experiment framework. *New Phytologist*, **200**, 304-321.
- 810 Meddens AJ, Hicke JA, Ferguson CA (2012) Spatiotemporal patterns of observed bark beetle-caused tree mor-
811 tality in British Columbia and the western United States. *Ecological Applications*, **22**, 1876-1891.
- 812 Mencuccini M, Onate M, Penuelas J, Rico L, Munné-Bosch S (2014) No signs of meristem senescence in old
813 Scots pine. *Journal of Ecology*, **102**, 555-565.
- 814 Millar CI, Stephenson NL (2015) Temperate forest health in an era of emerging megadisturbance. *Science*, **349**,
815 823-826.
- 816 Morris H, Plavcová L, Cvecko P *et al.* (2016) A global analysis of parenchyma tissue fractions in secondary
817 xylem of seed plants. *New Phytologist*, **209**, 1553-1565
- 818 Myers JA, Kitajima K (2007) Carbohydrate storage enhances seedling shade and stress tolerance in a
819 Neotropical forest. *Journal of Ecology*, **95**, 383-395
- 820 Nakagawa S, Schielzeth H (2013) A general and simple method for obtaining R² from generalized linear
821 mixed-effects models. *Methods in Ecology and Evolution*, **4**, 133-142.
- 822 Nerhass-Ahles C, Babst F, Klesse S, Nötzli M, Bouriaud O, Neukom R, Dobbertin M, Frank D (2014) The
823 influence of sampling design on tree-ring-based quantification of forest growth. *Global Change Biology*, **20**,
824 2867-2885.
- 825 Nesmith JCB, Das AJ, O’Hara KL, van Mantgem PJ (2015) The influence of prefire tree growth and crown con-
826 dition on postfire mortality of sugar pine following prescribed fire in Sequoia National Park. *Canadian Jour-
827 nal of Forest Research*, **45**, 910-919

- 828 Niinemets Ü, Valladares F (2006) Tolerance to shade, drought, and waterlogging of temperate Northern Hemi-
829 sphere trees and shrubs. *Ecological Monographs*, **76**, 521–547.
- 830 Ogle K, Whitham TG, Cobb NS (2000) Tree-ring variation in pinyon predicts likelihood of death following se-
831 vere drought. *Ecology*, **81**, 3237-3243.
- 832 Oliva J, Stenlid J, Martínez-Vilalta J (2014) The effect of fungal pathogens on the water and carbon economy of
833 trees: implications for drought-induced mortality. *New Phytologist*, **203**, 1028-1035.
- 834 Pedersen BS (1998) The role of stress in the mortality of midwestern oaks as indicated by growth prior to death.
835 *Ecology*, **79**, 79-93.
- 836 Pellizzari E, Camarero JJ, Gazol A, Sangüesa-Barreda G, Carrer M (2016) Wood anatomy and carbon-isotope
837 discrimination support long-term hydraulic deterioration as a major cause of drought-induced dieback. *Global*
838 *Change Biology*, **22**, 2125-2137
- 839 Piper FI, Gundale MJ, Fajardo A (2015) Extreme defoliation reduces tree growth but not C and N storage in a
840 winter-deciduous species. *Annals of Botany*, **115**, 1093-1103.
- 841 Plavcová L, Hoch G, Morris H, Ghiasi S, Jansen S (2016) The amount of parenchyma and living fibres affects
842 storage of non-structural carbohydrates in young stems and roots of temperate trees. *American Journal of*
843 *Botany*, **103**, 603-612
- 844 R Core Team (2015) R: A language and environment for statistical computing. R Foundation for Statistical
845 Computing, Vienna, Austria. <http://www.R-project.org/>.
- 846 Rodríguez-Calcerrada J, López, R., Salomón, R., Gordaliza, GG, Valbuena-Carabaña M, Oleksyn J, Gil L
847 (2015) Stem CO₂ efflux in six co-occurring tree species: underlying factors and ecological implications.
848 *Plant, Cell and Environment*, **38**, 1104-1115.
- 849 Rose KE, Atkinson RL, Turnbull LA, Rees M (2009) The costs and benefits of fast living. *Ecology Letters*, **12**,
850 1379-1384.
- 851 Rowland L, da Costa ACL, Galbraith DR *et al.* (2015) Death from drought in tropical forests is triggered by
852 hydraulics not carbon starvation. *Nature*, **528**, 119-122.
- 853 Sangüesa-Barreda G, Linares JC, Camarero JJ (2013) Drought and mistletoe reduce growth and water-use effi-
854 ciency of Scots pine. *Forest Ecology and Management*, **296**, 64-73.
- 855 Sangüesa-Barreda G, Linares JC, Camarero JJ (2015) Reduced growth sensitivity to climate in bark-beetle in-
856 fested Aleppo pines: Connecting climatic and biotic drivers of forest dieback. *Forest Ecology and Manage-*
857 *ment*, **357**, 126-137.
- 858 Schwarze FWMR, Fink,S, Deflorio G (2003) Resistance of parenchyma cells in wood to degradation by brown
859 rot fungi. *Mycological Progress*, **2**, 267-274.
- 860 Seidl R, Fernandes PM, Fonseca TF *et al.* (2011) Modelling natural disturbances in forest ecosystems: a re-
861 view. *Ecological Modelling*, **222**, 903-924.

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

871

872 **Supporting information caption**

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

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

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

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

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

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

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

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

885 **Appendix S9:** Summary of the fitted mixed effect models for Δt_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 Δt_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 Δt_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 Δt_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 (Δt_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 Δt_m while linear models were fitted to log-transformed $g_{f,m}$ and $g_{50,m}$ values.

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

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

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

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

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

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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)	0.13 (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 ² marginal	0.03	0.06	0.03
R ² conditional	0.26	0.18	0.22

930

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

(a) All mortality sources	nb. species		Duration of the period with reduced/increased growth (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 *

(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

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°) 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.

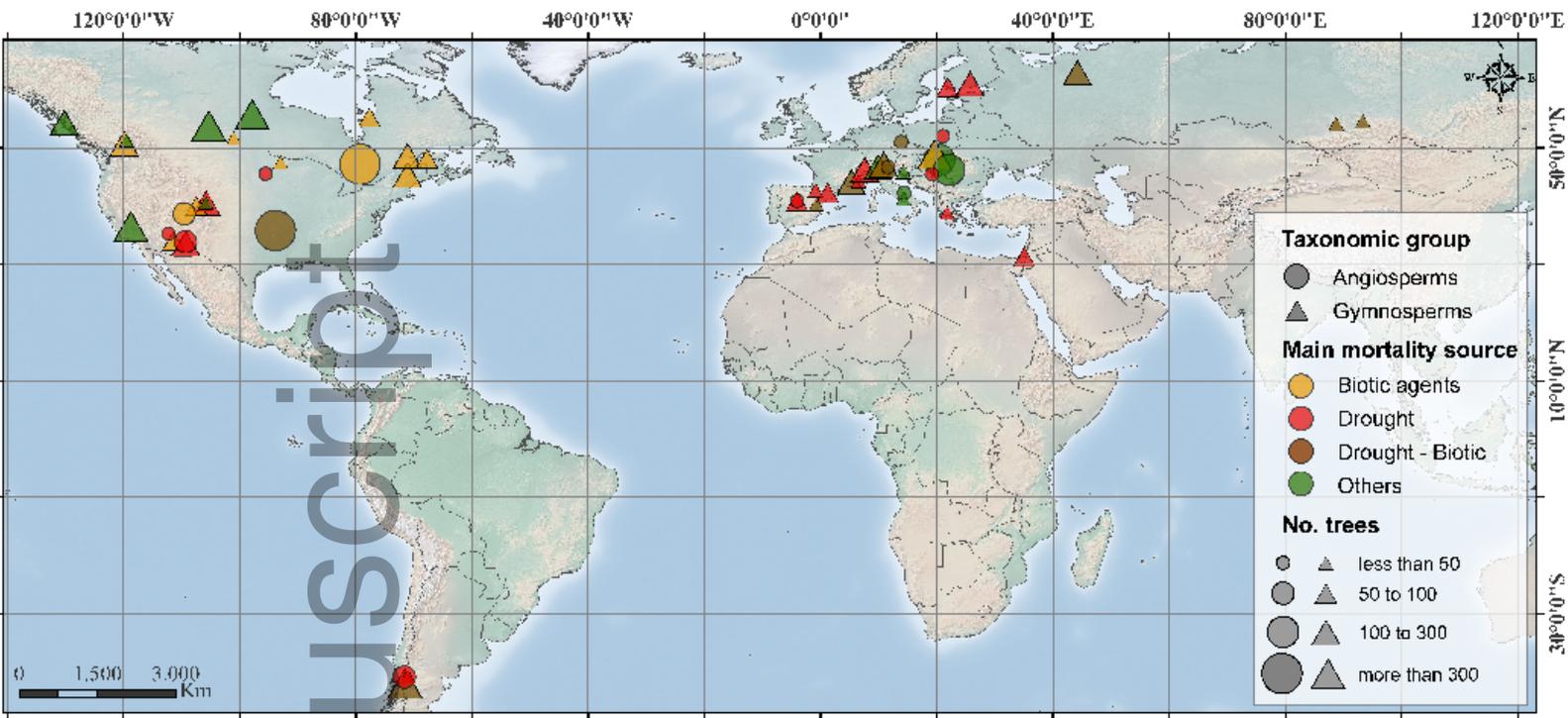
945 **Figure 2.** Example of time-series in growth ratio before mortality (dying / surviving trees) calculated
946 for *Quercus petraea* trees growing at the site 'Runcu' (Romania; Petritan et al. unpublished dataset) for
947 three different mortality events (1: 2009; 2: 2000; 3: 2010). The duration 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).

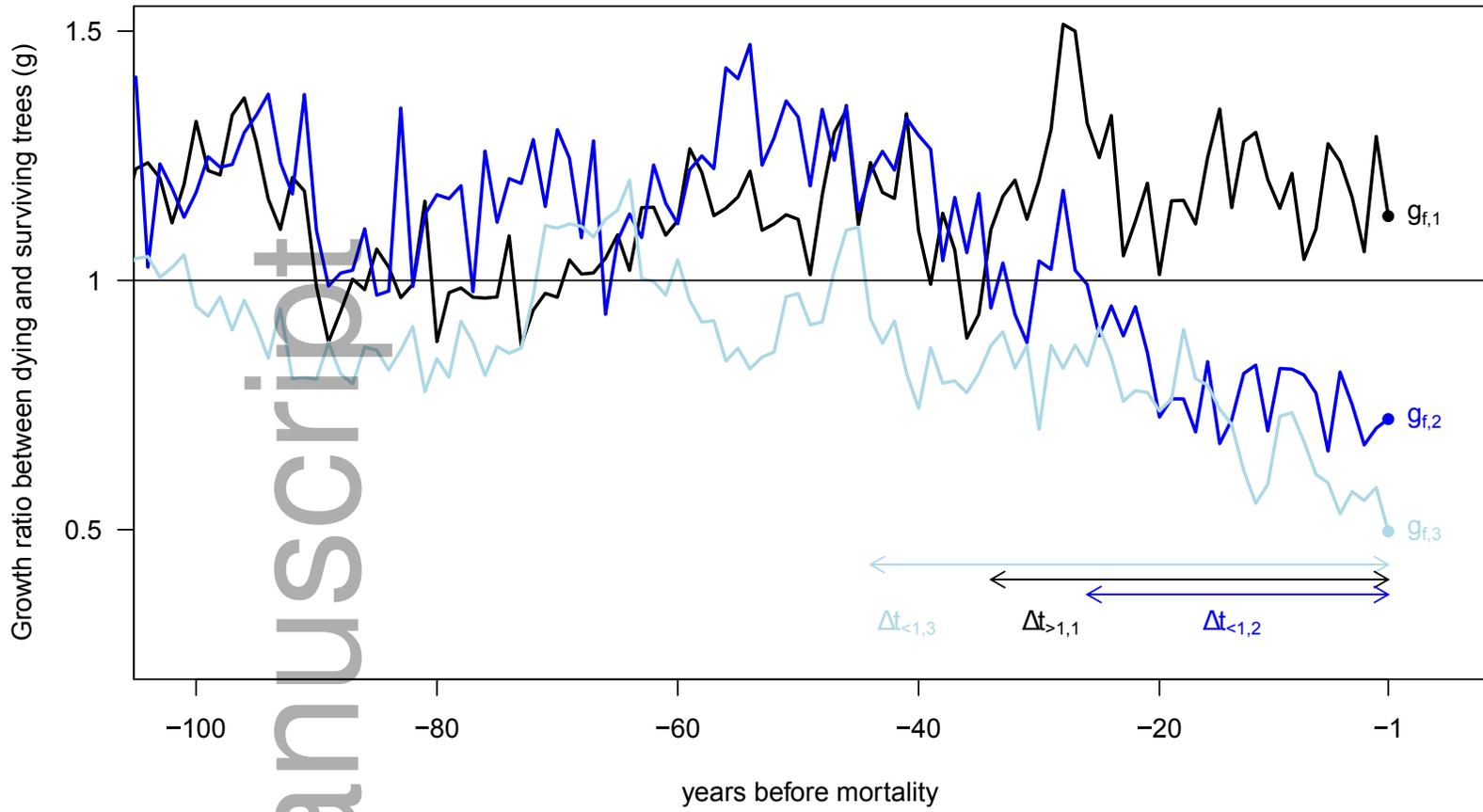
954 **Figure 4:** Distribution of the duration of the period with reduced or increased growth before death (a;
955 $\Delta t_{g<1,m}$ and $\Delta t_{g>1,m}$, respectively), and the growth ratio the year before death (c; $g_{f,m}$) and both variables
956 (b) calculated using ring-width data. Moving from 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 (Δt) 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.

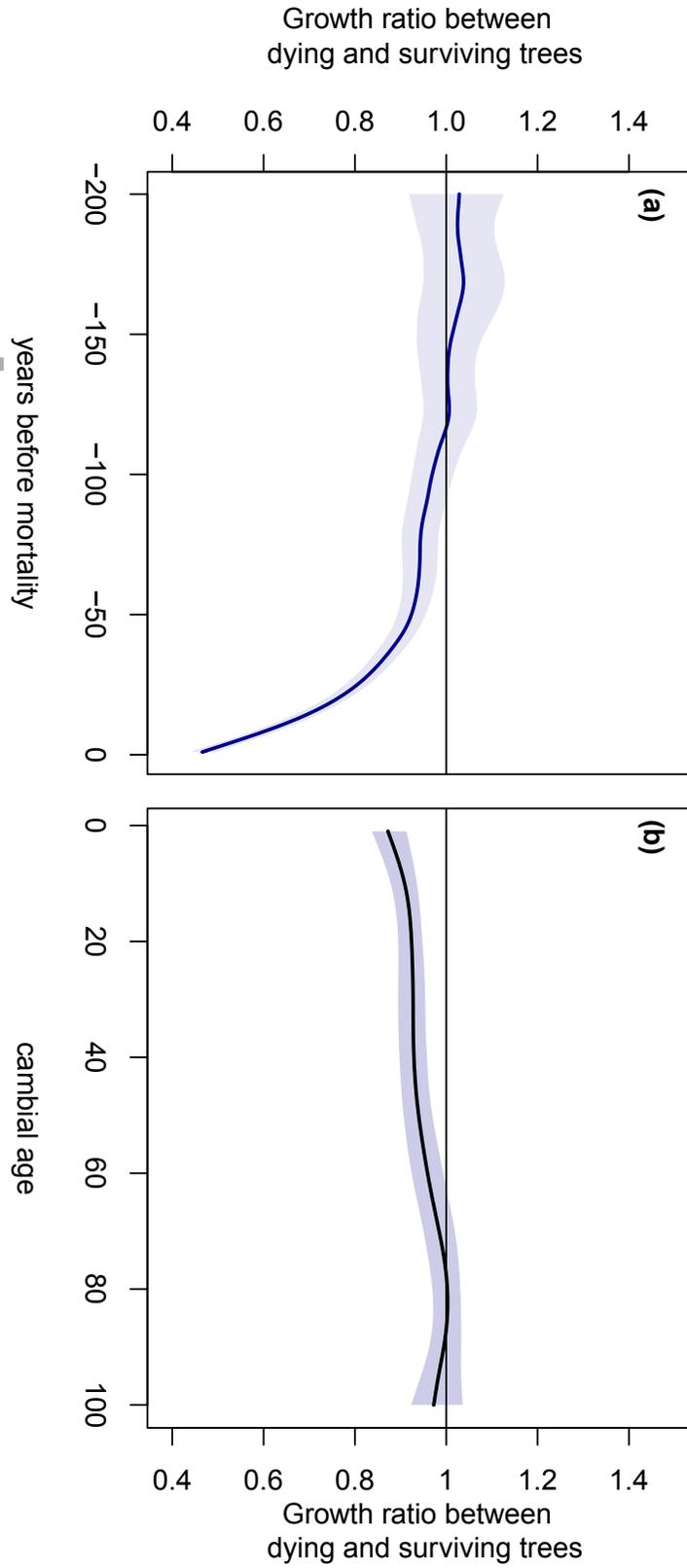
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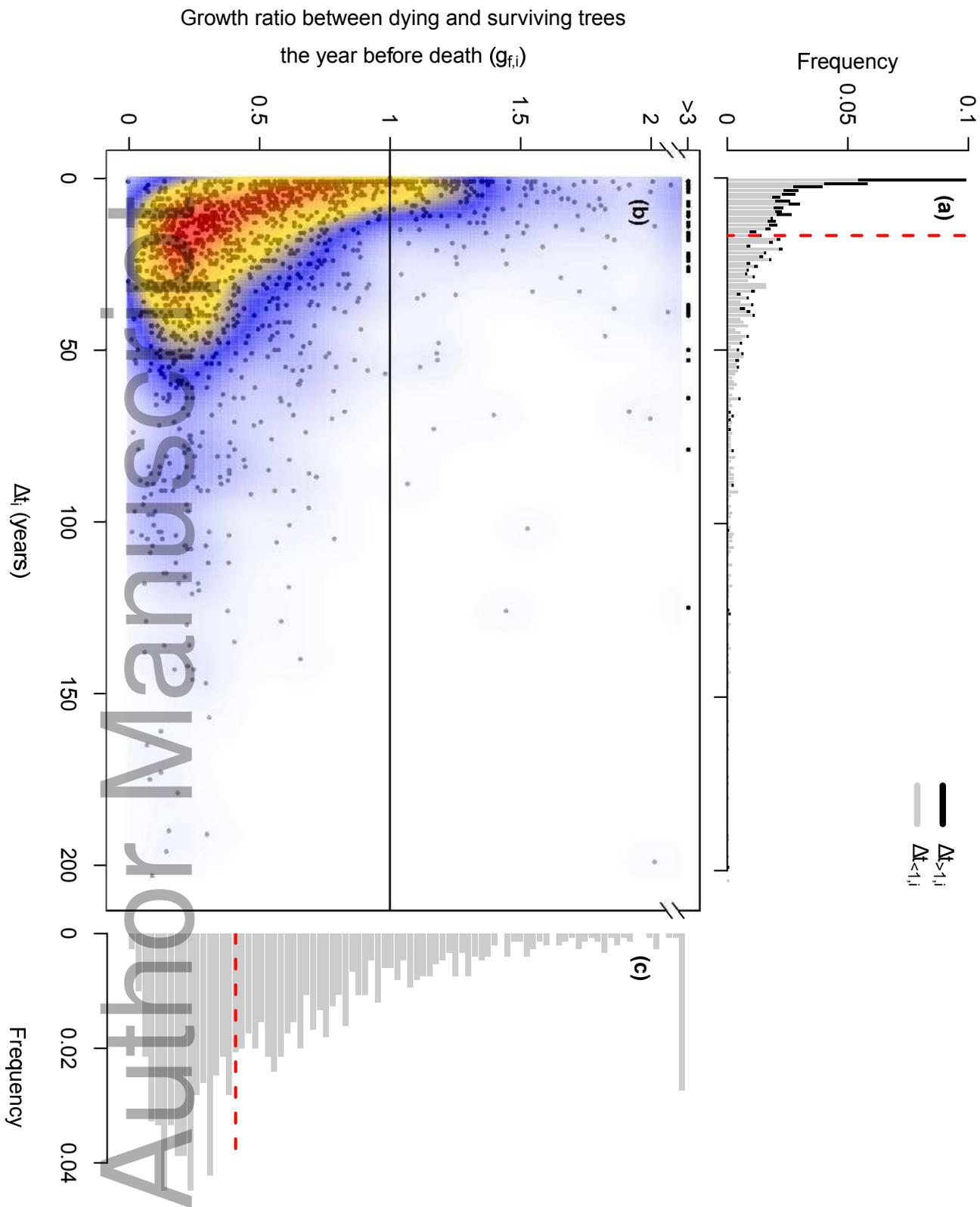
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