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A multi-species synthesis of physiological mechanisms in drought-induced tree mortality

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1 **A multi-species synthesis of physiological mechanisms in drought-induced tree mortality**

2

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91

92 **Widespread tree mortality associated with drought has been observed on all**
93 **forested continents, and global change is expected to exacerbate vegetation vulnerability.**
94 **Forest mortality has implications for future biosphere-atmosphere interactions of carbon,**
95 **water, and energy balance, and is poorly represented in dynamic vegetation models.**
96 **Reducing uncertainty requires improved mortality projections founded on robust**
97 **physiological processes. However, the proposed mechanisms of drought-induced mortality,**
98 **including hydraulic failure and carbon starvation, are unresolved. A growing number of**
99 **empirical studies have investigated these mechanisms, but data have not been consistently**
100 **analyzed across species and biomes using a standardized physiological framework. Here**
101 **we show that xylem hydraulic failure was ubiquitous across multiple tree taxa at drought-**
102 **induced mortality. All species assessed had 60% or higher loss of xylem hydraulic**
103 **conductivity, consistent with proposed theoretical and modelled survival thresholds. We**
104 **found diverse responses in non-structural carbohydrate reserves at mortality, indicating**
105 **that evidence supporting carbon starvation was not universal. Reduced non-structural**
106 **carbohydrates were more common for gymnosperms than angiosperms, associated with**
107 **xylem hydraulic vulnerability, and may have a role in reducing hydraulic function. Our**
108 **finding that hydraulic failure at drought-induced mortality was persistent across species**
109 **indicates that substantial improvement in vegetation modelling can be achieved using**
110 **thresholds in hydraulic function.**

111 Increasing forest mortality from global change has been observed in all forested biomes^{1,2}
112 and will have profound implications for future energy and element fluxes³⁻⁵. Predictions of
113 vegetation responses to future climate are uncertain due to the lack of realistic mortality
114 mechanisms in vegetation models^{3,6-9}. Recent research supports at least two tightly inter-related

115 physiological mechanisms associated with tree mortality by drought: (a) hydraulic failure
116 through partial or complete loss of xylem function from embolism that inhibits water transport
117 through the vasculature, leading to tissue desiccation; and (b) carbon starvation via imbalance
118 between carbohydrate demand and supply that may lead to an inability to meet osmotic,
119 metabolic, and defensive carbon requirements^{3,6,7,10-15}. Hydraulic failure is most typically
120 assessed via percent loss of xylem conductivity (PLC), and carbon starvation via changes in
121 tissue non-structural carbohydrate (NSC) concentrations¹²⁻¹⁶. There has been significant debate
122 over these co-occurring mechanisms of mortality, particularly regarding the prevalence of carbon
123 starvation and whether reduced carbohydrate reserves can be lethal during drought^{11,17-22}.

124 Though a number of studies on the mechanism of drought-induced mortality in trees have
125 been conducted for a variety of tree species over the last decade, the prevalence of these
126 mechanisms at a global scale remains uncertain. Differences in approach, variables measured,
127 and species and life stage studied have limited global assessment of drought-induced tree
128 mortality mechanism. Here, we provide the first cross-species synthesis of tree drought
129 mortality mechanisms. We used a standardized physiological framework to analyze drought-
130 induced tree mortality across species and assessed hydraulic function as PLC, and carbohydrate
131 status as NSC normalized relative to controls. We examined data from 19 recent experimental
132 and observational studies on 26 species from around the globe. Most tree species were assessed
133 in only one study, but for several species, data were available from more than one study,
134 resulting in 34 cases (species-study combinations). However, data were not available for all
135 analyses from all cases: more cases had NSC data (31 cases from 24 species) than PLC data (14
136 cases from 9 species) which could be used to compare NSC and PLC at mortality with that of
137 surviving control trees (see Methods below, Supplementary Table 1). In order to make our

138 synthesis comprehensive, we worked with all of the data that were available, including data from
139 studies on a range of tree sizes and ontogenetic life stages (i.e. seedlings, saplings, and large
140 trees), conducted in a variety of settings, including potted plants in greenhouses or growth
141 chambers, and trees grown in the field (Supplementary Tables 1, 2). Given the diversity of
142 studies synthesized, these data were not ideal for a statistical meta-analysis; therefore, we limited
143 our analyses to a standard comparison within each case between plants that died and plants that
144 remained healthy (Supplementary Methods). We also compared differences in degree of
145 embolism and carbohydrate concentrations between plants at mortality and control plants to
146 differences in functional traits^{3,24,25}. For each species, we obtained available data for traits that
147 are easily measured, widely available, and likely relevant for drought tolerance, including wood
148 density and specific leaf area²⁶. We also obtained data for hydraulic traits that are directly related
149 to drought tolerance, but harder to measure, including xylem water potential at 50% loss of
150 hydraulic conductivity (Ψ_{50}), point of embolism entry (Ψ_e), and corresponding hydraulic safety
151 margins^{24,27} (Supplementary Methods). We used this dataset to address the following
152 hypotheses: 1) given the potential role of NSC in the maintenance of water transport during
153 drought^{6, 28}, both high PLC and reduced NSC reserves are common at tree death from drought,
154 and 2) among species, species-level functional traits that have been positively related to drought
155 tolerance (e.g. low xylem vulnerability to embolism, low SLA, high wood density) are associated
156 with high NSC at tree death. According to this hypothesis we expect that for species with greater
157 xylem vulnerability (quantified by Ψ_{50} , Ψ_e , and hydraulic safety margin), NSC at death will be
158 relatively lower. This hypothesis is based on prior proposals that drought-sensitive trees which
159 close their stomata earlier during drought would be more likely to show a reduction in NSC
160 associated with carbon starvation^{3,25,29,30}.

161

162 **Results.**

163 For the cases where PLC data at mortality were available (Supplementary Methods), PLC
164 was 60% or higher (Figure 1A), demonstrating that a high degree of xylem embolism at drought-
165 induced death was a universal aspect of mortality physiology in these species. Mean PLC was
166 84.3% at mortality, and PLC was significantly higher at mortality than for control trees in every
167 case ($p < 0.05$, Student's t-test). For NSC, we focused our analysis on differences in NSC
168 concentration between trees that died from drought and controls which did not die, measured at
169 the same point in time for both groups, although we also considered differences over time for
170 trees that died (Supplementary Discussion). Reductions in NSC at mortality were common
171 among species, but not universal, and no common NSC threshold for mortality was identified.
172 For 48% of cases and 38% of species with available data, NSCs were significantly lower at
173 mortality in dying trees compared to surviving or control trees (for observational and
174 experimental studies, respectively) in at least one tissue ($p < 0.05$, ANOVA; Figure 1B-D).
175 Among all species, mean NSCs at mortality for leaves, above-ground woody tissues (bole,
176 branch, stem, or twig), and roots were 13, 17, and 35% lower in dying trees than control
177 measurements.

178 For boreal and temperate angiosperms, lower NSCs at mortality relative to control trees
179 were observed in 56% of cases and 63% of the species for at least one tissue, and NSC
180 reductions exceeded 50% in approximately 33% of these cases and 38% of these species (Figure
181 1B). Higher NSCs at mortality relative to controls were common for tropical angiosperm
182 seedlings³¹, more than 100% higher in some cases, and reduced NSCs were not observed in this
183 group, suggesting different physiological responses to severe drought in non-tropical and tropical

184 tree species (Figure 1B, C). In a similar seedling study with the same tropical species, however,
185 lower pre-drought NSCs were consistently correlated with a shorter time to mortality though
186 NSCs did not decline during drought¹⁴. Lower NSCs at mortality relative to controls were most
187 common in root tissues³², and typically resulted from lower starch concentrations, consistent
188 with a starch to sugar conversion to meet metabolic and osmoregulatory demands during drought
189 stress⁶ (Supplementary Figure 1). Notably, only a few cases exhibited the hypothesized time-
190 series trend in NSCs of initial small increase and then a more pronounced decrease in NSCs
191 over time²⁸ (Supplementary Figures 2, 3, 4).

192 Reductions in NSCs at mortality were more prevalent for gymnosperms than
193 angiosperms (Figure 1, Supplementary Figures 2, 3, 4). Among gymnosperms, 83% of cases and
194 67% of species had lower NSC at mortality relative to controls for at least one tissue (Figure
195 1D). This occurred in at least one tissue for all four species of the Pinaceae, but not for the two
196 species in the Cupressaceae, which is consistent with divergent evolutionary pathways for
197 stomatal control between these families³³. Relative reductions in NSCs were also generally
198 greater in gymnosperms than angiosperms, e.g. *Pinus sylvestris* had NSC reductions of >80% in
199 some tissues prior to mortality (Figure 1D).

200 Functional traits related to xylem embolism resistance and stomatal control have been
201 suggested as useful predictors of the physiological causes of drought-induced mortality^{3,25,30,34}.
202 For all species, the deviation of NSCs in trees at mortality from their controls was not
203 significantly associated with wood density or specific leaf area ($p > 0.05$, linear regression),
204 regardless of whether the relationships were assessed for angiosperms, gymnosperms, or all
205 species together. For gymnosperms, reduced NSCs at mortality in aboveground woody tissues
206 (bole, branch, stem, or twig) were associated with lower resistance to xylem embolism (i.e.

207 higher Ψ_{50} and Ψ_e ; $r^2 = 0.88$ and 0.91 , respectively, $p < 0.001$, linear regression; Figure 2),
208 indicating that hydraulic features in gymnosperms associated with drought resistance were
209 related to NSC dynamics during lethal drought. Normalized NSCs in other tissues were
210 positively correlated with embolism resistance at mortality (leaf NSC with Ψ_{50} , root NSC with
211 Ψ_e ; $p < 0.05$, linear regression), and normalized NSCs in aboveground woody tissue and roots at
212 mortality were also positively correlated with the Ψ_{50} hydraulic safety margin for gymnosperms
213 ($p < 0.001$, linear regression; Supplementary Figure 5), but these relationships were strongly
214 influenced by one species, *Callitris rhomboidea* (Supplementary Methods). Variation in PLC at
215 mortality was not related to any functional traits assessed ($p > 0.05$, linear regression).

216

217 **Discussion.**

218 We found that tree mortality from drought was always associated with substantial loss of
219 hydraulic function, and that lower NSCs at mortality were common but not universal (Figure 1).
220 Our findings for PLC at mortality (Figure 1A) are close to modeling and theoretical predictions
221 of a stem PLC mortality threshold near or above 60%^{7,10,35-37}. In all cases, we found that PLC at
222 mortality was at least 60%, but values were much higher in a number of cases. The studies in
223 our synthesis were not designed to quantify lethal PLC thresholds, which deserve future
224 investigation to determine the duration and intensity of drought required to trigger mortality and
225 the mechanisms underlying such a threshold. The physiological effects of a particular level of
226 PLC likely vary among species, mediated by traits such as the capacity to refill embolism and
227 replace conducting area via new growth^{6,38}. Nonetheless, a sustained stem PLC at or above 60%
228 provides a generally supported starting point for modeling vegetation response across spatial
229 scales, a point beyond which the probability of mortality increases^{7,10,36,37}.

230 For the cases where both NSC and PLC data were available at mortality, all trees died
231 with high PLC (100% of cases), but only 62% of cases also had low NSCs at mortality relative to
232 controls (Figure 3). This suggests that trees died from either hydraulic failure alone, or hydraulic
233 failure in combination with reduced NSCs. This finding should help lay to rest the
234 misconception of a dichotomy between hydraulic failure and carbon starvation, which are often
235 mistakenly thought to represent mutually exclusive mechanisms¹⁵. Clearly, our results
236 underscore the importance of maintaining a functional plant hydraulic system for survival, while
237 suggesting a relationship between hydraulic failure and carbon starvation mechanisms in this
238 process. The majority of studies included in our analysis were not designed to distinguish the
239 drivers of mortality from the non-causative symptoms of dying. Thus, it is not possible with our
240 data to conclusively determine if changes in either NSC or PLC facilitated death or were the
241 result of the mortality process (Supplementary Discussion). Results from studies in which light
242 and CO₂ concentration were manipulated to regulate carbon fixation do suggest a role for NSC as
243 a survival mechanism against mortality via hydraulic failure during drought, even when NSC
244 does not decline during drought or is not reduced below control values¹²⁻¹⁶ (Supplementary
245 Discussion).

246 Given the diversity of NSC responses found at mortality, there is an obvious need to
247 develop frameworks for the sensitivity of plant metabolism to changes in NSC levels, including
248 the potential for lethal thresholds^{22,39}. Specific NSC thresholds for survival or mortality during
249 drought are not well-resolved in our data, nor yet in the literature. Such survival thresholds
250 likely vary with factors including tree species, ontogeny, tree tissue, canopy position,
251 seasonality, environmental conditions, and interactions with other organisms, but empirical
252 investigation of these thresholds is needed^{22,40,41}. Determination of these thresholds is hampered

253 by an incomplete understanding of the role of NSC storage in plant function, and its regulatory
254 mechanism^{22,39}. However, significantly lower NSCs at mortality were relatively common for a
255 variety of species in our analysis, such that reduced NSCs can no longer be considered a rare or
256 atypical response during tree death.

257 Our finding that reduced NSCs at mortality were more common for gymnosperms, than
258 for angiosperms (Figure 1, Supplementary Figures 2, 3, 4), is consistent with the wider hydraulic
259 safety margins of gymnosperms relative to angiosperms^{24,42}. For gymnosperms, our functional
260 trait analysis revealed that species with greater xylem embolism resistance had higher NSC at
261 mortality in boles, branches, stems, or twigs than surviving controls, indicating that species'
262 hydraulic traits can affect C balance during lethal drought (Figure 2). As embolism resistance is
263 often associated with an ability to keep stomata open at lower water potentials^{30,43}, our results
264 suggest that tree species which can maintain stomatal conductance and photosynthesis at higher
265 xylem tension during drought are less likely to have reduced NSC at mortality²⁹. These resistant
266 tree species would be more likely to die from hydraulic failure alone without reduced NSC —
267 consistent with hypotheses that stomatal regulation and hydraulic transport strategies influence
268 the contribution of carbon starvation and hydraulic failure to mortality mechanism among
269 species^{3,25,30}. Caution, however, should be used in assuming stomatal regulation is highly
270 coupled with water potential regulation and hydraulic strategy⁴⁴. Importantly, we did not find a
271 relationship between NSC reduction and embolism resistance for angiosperms, nor did any other
272 trait predict mortality physiology in these species.

273 Our synthesis of data from multiple studies on the physiology of drought-induced tree
274 mortality exposes several key knowledge gaps in the field. Our dataset of only 26 species under-
275 represents the enormous diversity of tree species found in forests globally, particularly so for

276 tropical forests, where drought-induced mortality can have substantial implications for the global
277 carbon cycle⁹. *Pinus* was relatively over-represented in this synthesis (nine cases from three
278 species), although it is widely distributed and has been widely affected by forest die-off on
279 multiple continents¹. Also, our dataset is dominated by data from seedlings and saplings, often
280 from studies conducted with potted plants, which may be predisposed to die quickly from
281 hydraulic failure due to limited rooting volume and lack of access to deeper soil water pools
282 (Supplementary Tables 1, 2). Data at mortality for more than one life stage were available for
283 only three species (Figure 1), and the consistency of NSC and PLC responses at mortality across
284 a gradient of size and ontogeny varied in these species. Clearly, more research on the physiology
285 of mortality in large trees in the field and the effect of size and ontogeny on the mortality process
286 is needed. Nonetheless, our overall observation that hydraulic failure was universal, and NSC
287 reduction was not, does not change if we only consider data for each life stage separately. In all
288 cases for which PLC data were available, mean PLC was 60% or greater at mortality,
289 irrespective of life stage (Figure 1A). Our finding that normalized NSC at mortality varied
290 among cases and species also holds when seedlings, saplings, and trees are considered separately
291 (Figure 1B-D, Supplementary Table 1).

292 Determining whether forests will continue to act as a global carbon sink or transition to a
293 carbon source is a critical uncertainty for the carbon cycle with large ramifications for society
294 and climate policy^{8,9,23}. Such a shift largely depends on tree mortality responses which could be
295 anticipated by resolving the relative roles of hydraulic and carbohydrate mechanisms in causing
296 tree death^{7,10,45}. We found that hydraulic failure was ubiquitous among the studies we compared,
297 that PLC at mortality in all cases with such data was at least 60%. These results affirm that
298 simulating hydraulic function should be a first priority for development of mechanistic tree

299 mortality algorithms in climate-vegetation models to improve projections of the future terrestrial
300 carbon budget. Hydraulic models that capture drought damage at tree and landscape scales are
301 rapidly developing^{7,10,36,37,45-47} and substantial improvement in vegetation model projections may
302 be possible with simulation of hydraulic-driven mortality, whether tree carbohydrate status is
303 represented or not. Reduced NSC in tree species dying from drought was common in
304 gymnosperms, but not angiosperms, suggesting an influence of NSC on hydraulic deterioration
305 in some trees that requires further investigation. Yet, the diversity of NSC responses among only
306 26 species and the design limitations of past studies in determining causality demonstrate that we
307 need to further assess the influence of carbon metabolism and storage on mortality³⁹. Ultimately,
308 an improved representation of the physiology of drought-induced tree mortality that includes
309 both water and carbon relations will be crucial for forecasting the fate of forests in a changing
310 climate.

311

312 **Methods**

313 **Data Synthesis.** We used literature search and extensive discussion with colleagues to identify
314 data from 19 experimental and observational studies on 26 species, for a total of 34 cases (study
315 and species combinations). Literature search terms included “non-structural carbohydrates”,
316 “water potential”, “tree mortality”, and “drought”. Our synthesis was not limited to an objective
317 literature search, as we sought to include all published data that fit our criteria for inclusion.
318 Criteria for inclusion were that studies included data on: 1) tree mortality from drought; 2) NSC
319 concentrations of at least one tissue, and/or PLC of aboveground woody tissue, either measured
320 directly, or estimated from plant water potential (Ψ_p) measured at mortality, or modeled from
321 hydraulic conductance^{48,49} (Supplementary Methods); and 3) that data were either: a)

322 concurrently collected for trees that died (either at or near mortality) and from trees that either
323 survived the drought or were in a paired control treatment, and/or: b) available prior to drought
324 or pre-treatment from the same trees that later died. We obtained data from each study directly
325 from contributors. Details on the specific studies synthesized can be found in Supplementary
326 Table 1. Determination of the point of mortality in dying trees was defined in each original
327 study, as detailed in Supplementary Table 3, and we relied on data contributors to provide the
328 appropriate data for at- (or near-) mortality assessments.

329 NSC measurements are methodologically challenging and comparisons of absolute
330 concentrations can be problematic across studies due to issues of standards, NSC technique, and
331 lab protocol disagreement^{50,51}. However, relative differences (treatment vs. control and changes
332 over time assessed with the same technique in the same laboratory) provide robust estimates of
333 NSC dynamics within studies^{50,51}. We limited all statistical analyses of absolute NSC data to
334 within each case (detailed below) and we only present relative differences in NSC in figures.
335 For studies where data were concurrently available for trees that died and control or surviving
336 trees, we calculated a normalized NSC deviation from the difference between values at or near
337 mortality and those for control or surviving trees divided by the control or surviving tree value.
338 For studies where data were available prior to the drought for the same trees that later died (or
339 seedlings in the same treatment harvested at measurement), normalized values were also
340 calculated as the difference between values at or near mortality and initial pre-treatment or pre-
341 drought values divided by the initial or pre-drought values. In both cases, normalized values
342 were expressed as a percent. For comparison of time series trends in NSC, we also calculated
343 normalized, proportional NSCs in trees that died by scaling values relative to the maximum
344 value in each time series, which was defined as a normalized value of 1. When possible,

345 normalizations were calculated for individual trees, and specifically for each tissue sampled. For
346 studies 3 and 9 (Supplementary Table 1), only means and standard errors for species and tissues
347 were available, so normalized values were calculated from these metrics.

348 Note that all types of data were not available for all cases in our synthesis. Among the 34
349 cases in our dataset, PLC measured at mortality was available for nine cases (eight species), PLC
350 was estimated in five cases (two species), NSC deviation from control/surviving trees at
351 mortality was available for 31 cases (24 species), and percent change in NSC was available for
352 28 cases (22 species). Sample sizes for PLC and NSC data are available in Supplementary
353 Tables 4 and 5. Because PLC values are already normalized to the maximum conductivity per
354 sample, no further normalization was conducted with these data. We also acknowledge that
355 direct measurements of PLC and generation of hydraulic vulnerability curves can be challenging,
356 and that method artifacts can effect results^{52,53}. Although the majority of hydraulic data we
357 report were collected following recommended practices (Supplementary Methods,
358 Supplementary Table 5), we cannot rule out the possibility of such artifacts influencing our data.

359 To compare physiological mortality indicators to tree species traits, we obtained trait data
360 for the species in this synthesis from a variety of sources. We investigated the relationships
361 between physiology at mortality and traits related to drought tolerance that are easily measured
362 and widely available, such as wood density and specific leaf area (SLA). We also included
363 hydraulic traits more directly related to drought tolerance that were measured with more-
364 challenging hydraulic vulnerability curve methods. Wood density data for most species were
365 obtained from the Global Wood Density database^{54,55} available through the DRYAD digital
366 repository (www.datadryad.org). We obtained SLA data from the TRY database (www.try-
367 db.org)⁵⁶⁻⁵⁸, for nearly all non-tropical species. We calculated species means for SLA from all

368 data available for each species of interest for our analysis. Data for *Acer pseudoplatanus* were
369 available from the mortality study population¹⁶. For *Callitris rhomboidea* and *Eucalyptus*
370 *smithii*, SLA data were not available. Additional sources of wood density data are detailed in
371 Supplementary Methods. Hydraulic trait data for the stem water potential at 50 PLC (Ψ_{50}) and
372 hydraulic safety margin ($\Psi_{50} - \text{minimum } \Psi$)^{24,27}, were obtained from multiple sources
373 (Supplementary Table 5, Supplementary Methods). Data for the embolism entry point (Ψ_e) were
374 not available in the literature, so we calculated Ψ_e from relevant hydraulic vulnerability curve for
375 each case by applying a Weibull fit to the data, and determining the x-intercept of the line
376 tangent to Ψ_{50} (Supplementary Table 5)^{27,59}. Hydraulic trait data were unavailable for
377 *Eucalyptus radiata*, *Eucalyptus smithii*, and *Nothofagus nitida*. No trait data were available for
378 the tropical angiosperm species from study 7 (Supplementary Table 1) for any of the traits we
379 assessed³¹.

380 The majority of datasets generated and analyzed during the current study are available
381 from the corresponding author on reasonable request. Trait data obtained for the current study
382 from the TRY Database were used under license and as restrictions apply to the availability of
383 these data, these are not available from the corresponding author, but can be requested from the
384 TRY Database (www.try-db.org).

385

386 **Statistical Analyses.** We used MATLAB R2012a (The Mathworks, Inc., Natick, MA, USA) for
387 all statistical analyses, with $\alpha = 0.05$. All NSC and PLC comparisons were performed using
388 ANOVA or Student's t-test individually for each case, between dead (or dying) and
389 control/surviving trees or between post-drought dead and corresponding pre-drought values, with
390 tissue as a factor for analysis of NSC. Since our NSC normalization could affect tissue

391 comparisons within the same case, these analyses were performed on non-normalized NSC data
392 to maintain the correct ratio among tissues, a conservative approach. Our within-individual case
393 analysis on relative differences in non-normalized NSC does not bear the risk of error introduced
394 by different NSC techniques or labs, or uncertainty in standards for determining absolute NSC,
395 and furthermore the inferences are based on large effect sizes compared to possible measurement
396 error^{50,51}. In experimental cases that included temperature or CO₂ concentration treatments in
397 addition to drought, we included these factors in ANOVA tests to determine if PLC and NSC
398 should be pooled or split among levels of these factors (Supplementary Table 1). For NSC, these
399 analyses also included tissue as a factor. Functional trait relationships with normalized NSC data
400 at mortality were analyzed with linear regression. Cook's distance was calculated for all points
401 in significant linear regressions, and a value greater than three times the mean of the Cook's
402 distance was used to identify outliers for exclusion.

403

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581 HDA, HH, JAP, JDL, JMK, JML, JSS, LDLA, LTD, MJBZ, MJG, MM, NGM, PJH, RCC, RV,
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583 DDB, DJB, DML, DTT, EAP, EAY, FIP, GBG, HD, HDA, HH, JAP, JDL, JMV, JQ, JSS, KR,
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589 SML, SS, TEF, TEH, TEK, UH, WRLA, and WTP contributed to the discussion of results.
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594

595 **Competing financial interests.** The authors declare they have no competing financial interests.

596

597 **Figure Legends**

598

599 **Figure 1.** Physiological responses at, or prior to, mortality from drought for multiple tree
600 species. Percent loss of hydraulic conductivity (PLC) for ambient moisture, control, or surviving
601 trees and concurrently at mortality from drought is shown for both angiosperm and gymnosperm
602 species (A). PLC was either measured directly (red) for control (open symbols) and dying
603 (closed symbols) trees or estimated from either water potential with a hydraulic vulnerability
604 curve (green) for control (open) and dying (closed) trees, or modeled from hydraulic
605 conductance (orange) for control (open), and dying (closed) trees. An “NA” indicates that
606 control PLC data were not available. In all panels for cases where individual data were
607 available, boxes indicate the 25% and 75% quartiles, whiskers indicate the extent of data, and
608 black bars indicate the mean. For cases where only means and a measure of variability were

609 available, means are indicated with squares and error bars are one standard error. For each case
610 in A where control and dying tree data were available, PLC was significantly higher at mortality
611 than for controls concurrently ($p < 0.05$, Student's t-test). A potential threshold for hydraulic
612 failure is indicated by a line at 60%. Non-structural carbohydrate concentration (NSC) at
613 mortality, normalized as the percent deviation from concurrent measurements of ambient,
614 control, or unaffected trees in each study for each plant tissue, is shown for deciduous and
615 evergreen non-tropical angiosperm (B), evergreen tropical angiosperm (C), and evergreen
616 gymnosperm (D) species. Significant differences for each tree tissue between drought trees at
617 mortality (black bar or square) and ambient, control, or surviving trees (0% line) are indicated
618 with an asterisk ($p < 0.05$, ANOVA). Note that the absolute values in NSC concentration used in
619 statistical analysis varied for each tissue in each case, such that distances between the mean and
620 zero in B-D are not a consistent indicator of statistical significance among cases or for tissues
621 within a case. An "M" indicates data from a study on mature trees; all other data are from studies
622 of seedlings, saplings, and small trees (Supplementary Tables 1, 2). Numbers after species
623 names in all panels designate original studies (Supplementary Table 1). Sample size for all data
624 analyzed for Figure 1 are shown in Supplementary Table 4.

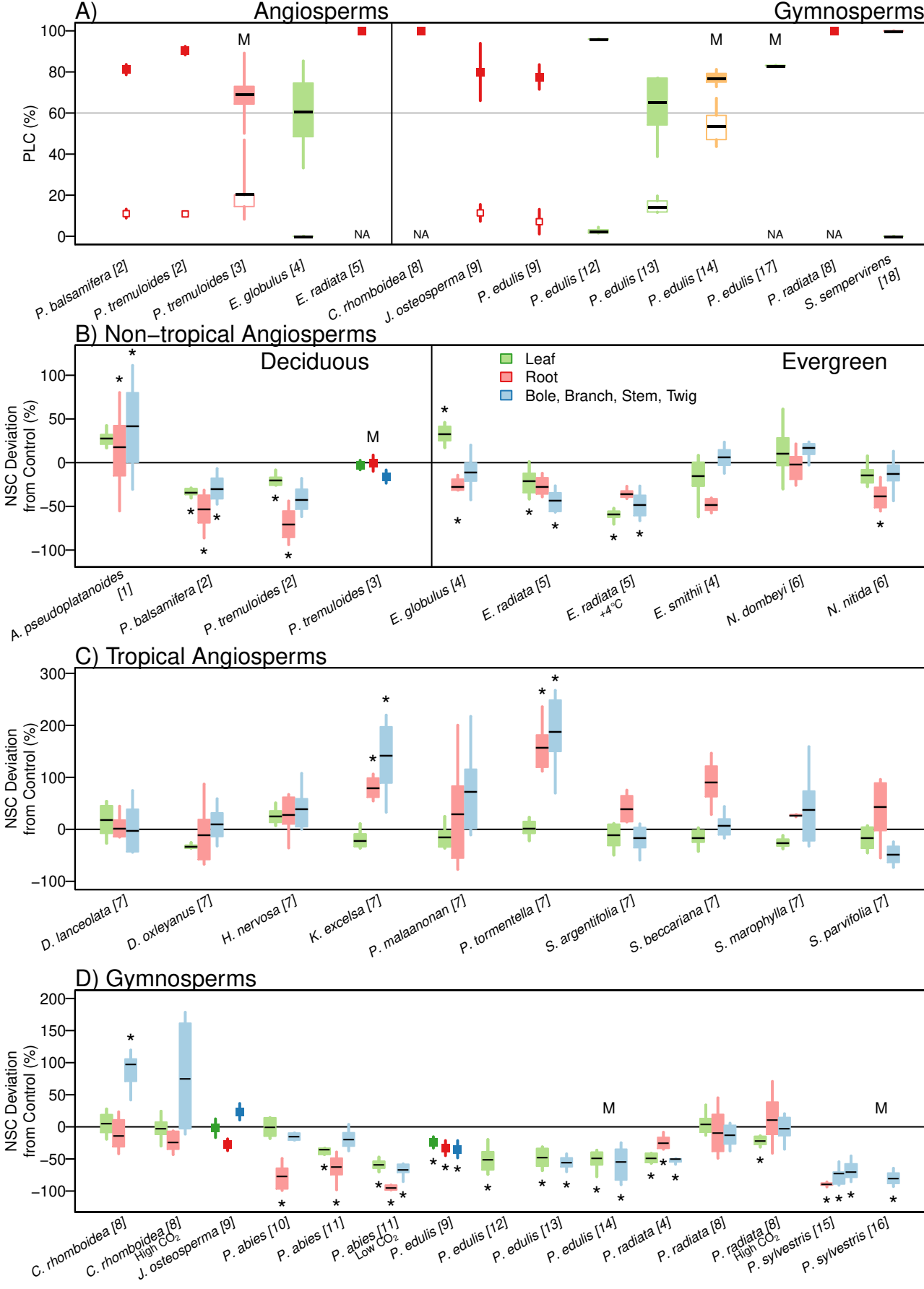
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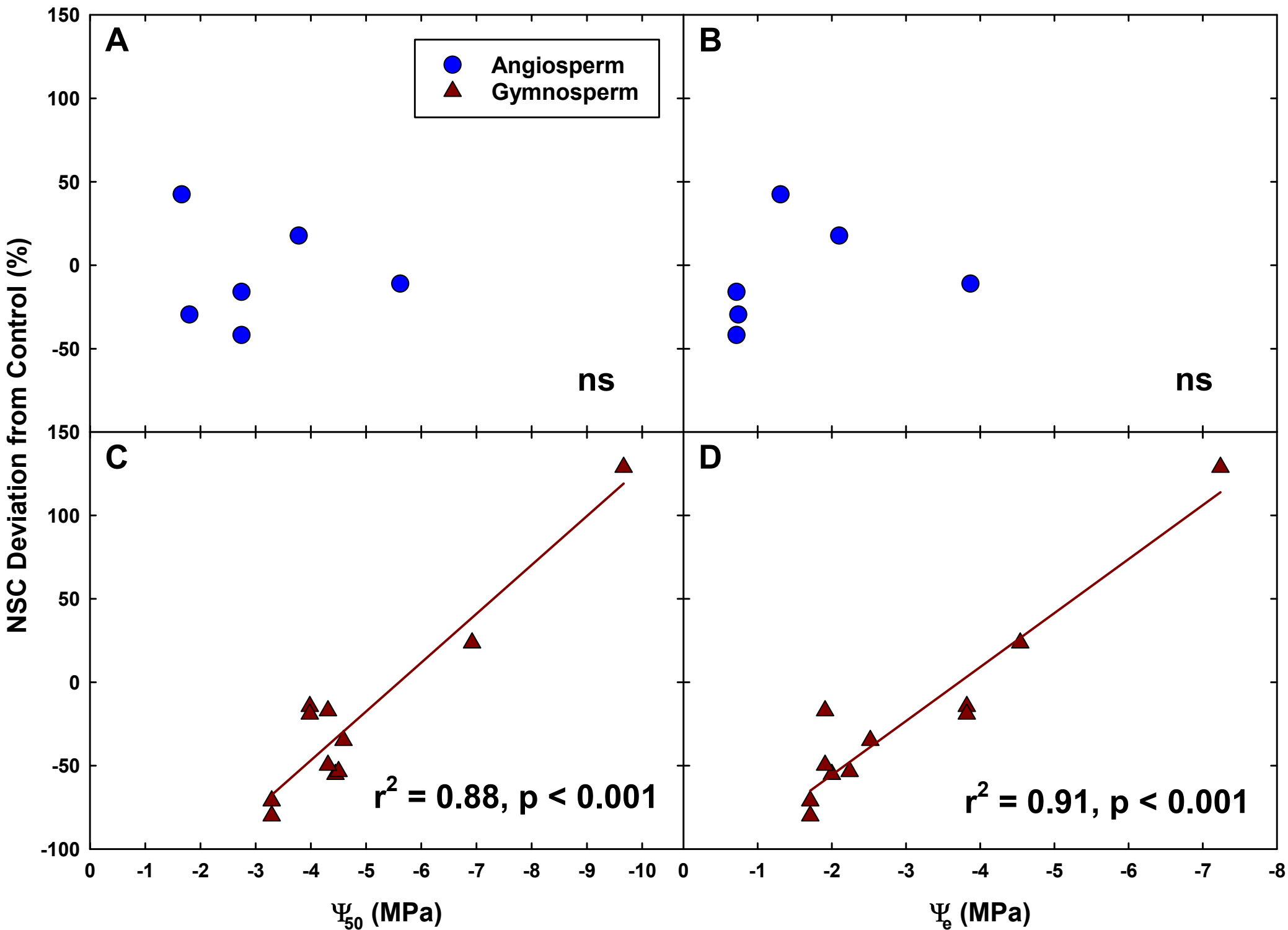
626 **Figure 2.** The relationship between the tree hydraulic traits related to xylem embolism resistance
627 and normalized non-structural carbohydrates (NSC) in aboveground woody tissue at, or prior to,
628 mortality from drought, expressed as a deviation from concurrent measurements of surviving
629 control trees, for angiosperm (blue circles; A, B) and gymnosperm (red triangles; C, D) species.
630 Tree hydraulic traits related to embolism resistance are the water potential at 50% loss of
631 hydraulic conductivity (Ψ_{50} ; A, C) and point of xylem embolism entry (Ψ_e ; B, D). Xylem

632 embolism resistance increases to the right. NSC data shown are means for aboveground woody
633 tissue (bole, branch, stem, or twig), normalized as a percent of ambient moisture, control, or
634 surviving trees in each case. Significant linear regressions were found for gymnosperms (C, D)
635 but not angiosperms (A, B). Values for *Callitris rhomboidea* (upper right in C, D) were
636 identified as potential outliers, but both relationships remain statistically significant ($p < 0.01$,
637 linear regression) for the remaining data with the removal of these points (Supplementary
638 Methods).

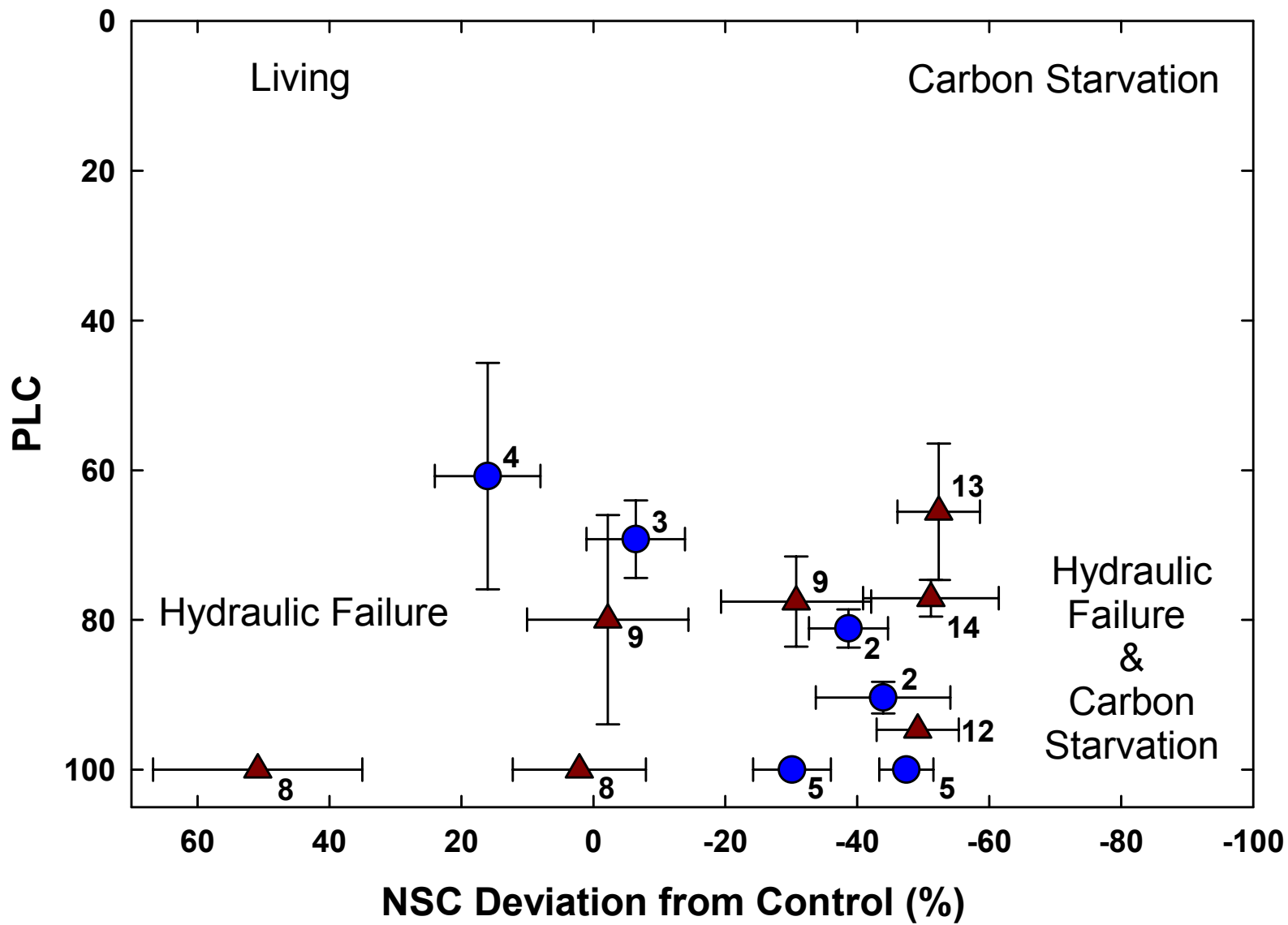
639

640 **Figure 3.** Physiological responses associated with hydraulic failure and carbon starvation, as
641 defined by PLC and NSC deviation from control in 13 cases (study \times species combinations) for
642 which both data were available. Among these cases, trees either died with high PLC and low
643 NSCs (8/13 cases), or with only high PLC (5/13 cases). NSC data are means for all sampled
644 tissues available for each case and normalized as a percent of difference from concurrent
645 measurements of control trees. PLC data are those shown in Figure 1A. NSC and PLC at
646 mortality for angiosperm (blue circles) and gymnosperm (red triangles) species are shown
647 relative to hypothesized drought mortality mechanisms. Numbers near points designate original
648 studies (Supplementary Table 1). Error bars are one standard error.





Increasing Hydraulic Failure



Increasing Carbon Starvation