



## The contribution of competition to tree mortality in old-growth coniferous forests

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### ABSTRACT

Competition is a well-documented contributor to tree mortality in temperate forests, with numerous studies documenting a relationship between tree death and the competitive environment. Models frequently rely on competition as the only non-random mechanism affecting tree mortality. However, for mature forests, competition may cease to be the primary driver of mortality.

We use a large, long-term dataset to study the importance of competition in determining tree mortality in old-growth forests on the western slope of the Sierra Nevada of California, U.S.A. We make use of the comparative spatial configuration of dead and live trees, changes in tree spatial pattern through time, and field assessments of contributors to an individual tree's death to quantify competitive effects.

Competition was apparently a significant contributor to tree mortality in these forests. Trees that died tended to be in more competitive environments than trees that survived, and suppression frequently appeared as a factor contributing to mortality. On the other hand, based on spatial pattern analyses, only three of 14 plots demonstrated compelling evidence that competition was dominating mortality. Most of the rest of the plots fell within the expectation for random mortality, and three fit neither the random nor the competition model. These results suggest that while competition is often playing a significant role in tree mortality processes in these forests it only infrequently governs those processes. In addition, the field assessments indicated a substantial presence of biotic mortality agents in trees that died.

While competition is almost certainly important, demographics in these forests cannot accurately be characterized without a better grasp of other mortality processes. In particular, we likely need a better understanding of biotic agents and their interactions with one another and with competition.

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### 1. Introduction

The death of a tree often involves many interacting factors, including competition, pathogen and insect attack, mechanical failure, climate-induced environmental stress, and localized edaphic constraints (Franklin et al., 1987; Waring, 1987). Identifying the multiple contributors to a given tree's death and determining their relative importance can be exceedingly difficult (Franklin et al., 1987; Das et al., 2008). Yet improving our understanding of tree mortality is essential in this era of global change. In many respects, the death of the extant tree canopy is the prelude to any compositional, structural, or functional shifts in forest ecosystems.

Competition is a well-documented contributor to tree mortality in temperate forests (Eid and Tuhus, 2001; Yang et al., 2003; Monserud et al., 2004; Temesgen and Mitchell, 2005; Bravo-Oviedo et al., 2006) and is often considered to be the primary agent of mortality. For example, many forest simulation models rely on the simplification – via growth-dependent mortality relationships – that competition for resources is the only non-climatic, non-catastrophic, non-random mechanism that affects likelihood of mortality (e.g., Pacala et al., 1996; Bugmann, 2001; Moravie and Robert, 2003). While the assumption appears valid for dense, young stands undergoing self-thinning (Yoda et al., 1963; Oliver and Larson, 1990), competition may cease to be the primary driver of mortality in older forests. For example, pathogen and insect attacks may play a greater role in tree mortality as forests age (Franklin and van Pelt, 2004). We might therefore expect competition to be less important in an old-growth forest. However, while biotic enemies might be operating within a forest, if they are only killing those trees that are in highly competitive environments, then competition might still ultimately dominate tree mortality. Quantifying the contribution of competition to tree mortality in mature forests

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should therefore lead to insights about underlying mortality processes.

Given the stature, complexity, and longevity of trees (not to mention aesthetic and economic value), research on tree mortality often relies on observation rather than experimentation. However, the fact that the spatial pattern of forest trees is fundamentally linked to demographic processes provides a powerful inferential framework (He and Duncan, 2000). Changes in pattern are driven by births and deaths. Demography in turn is affected by existing spatial pattern, as spacing plays a large part in defining the risks of mortality and opportunities for recruitment. This link provides the rationale for inferring biological process from spatial pattern. In effect, the pattern becomes the ‘footprint’ of the process (Picard et al., 2009).

The pattern–process relationship is often used to study competitive mortality (e.g., Kenkel, 1988; Getzin et al., 2006). Mortality due to competition is presumed to increase as local density increases. Thus the expectation is that competitive mortality should shift the spacing of surviving trees to a more uniform arrangement (Leps and Kindlmann, 1987; Kenkel, 1988; e.g., Stanislav and Leps, 1996; although see Murrell, 2009). Under this framework, the existence of uniformity in tree spacing or an increase in the uniformity in tree spacing with tree age or size is taken as evidence of competitive mortality (e.g., Phillips and Macmahon, 1981; Prentice and Werger, 1985; Jose et al., 1991; Skarpe, 1991; Moeur, 1997; Druckenbrod et al., 2005).

In this paper, we quantify the contribution of competition to tree mortality using an extensive network of spatially explicit monitoring plots, which encompass several forest types, incorporate up to 23 years of annual resolution mortality data, and include comprehensive field evaluations of factors contributing to mortality. We combine three lines of evidence from these data to test the importance of competition as a driver of mortality in these old-growth forests: local competitive environment for each tree; the change in spatial pattern within each plot; and field assessments of contributors to individual mortalities. Specifically, we ask whether competition appears to be strongly contributing to tree mortality processes in these forests and, further, whether those competitive effects are dominating these mortality processes.

## 2. Materials and methods

### 2.1. Research sites

The study sites are located in the coniferous forests of Sequoia National Park and Yosemite National Park on the western slope of the Sierra Nevada in California, U.S.A. Soils are generally coarse loams derived from granitic parent material. At 2000 m, annual precipitation averages 1200 mm/yr with the majority of that falling between December and March (35–65% as snow). The sites were never logged and have not experienced any stand-replacing disturbances in the last several centuries (Swetnam, 1993). Before Euroamerican settlement, low to moderate intensity surface fires were common, with the mean fire return intervals along an elevational (1575–2180 m) transect at Sequoia varying between 4 and 30 years (Caprio and Swetnam, 1993). The forests are dominated by *Abies concolor*, *Abies magnifica*, *Calocedrus decurrens*, and *Pinus lambertiana*. In addition, some areas contain significant numbers of *Sequoiadendron giganteum*, *Pinus ponderosa*, and *Quercus kelloggii*.

Plot locations were chosen to be both accessible and representative of uneven-aged Sierra Nevada forests. All living trees taller than 1.37 m were mapped, tagged, and had their diameters measured at breast height (1.37 m). The 14 plots encompassed a variety of stand characteristics (Tables 1 and 2), minimizing the chance of drawing unreliable inferences from a small sample of plots (Moravie and

Robert, 2003). They ranged in elevation from 1500 m to 2600 m, were established between 1982 and 1994, and cover four different forest types: Ponderosa pine-mixed conifer, white fir-mixed conifer, red fir, and Jeffrey pine forest (sensu Fites-Kaufman et al., 2007). Inventory data indicate that although the tree composition has changed in individual plots over time, there have been no strong trends in density or basal area among these sites during the last two decades (van Mantgem and Stephenson, 2007). Taken as a whole, recruitment and mortality rates were roughly balanced, though mortality rates have increased in recent decades (Stephenson and van Mantgem, 2005; van Mantgem et al., 2009).

Trees were checked annually for mortality, and every five years tree diameters were re-measured and newly established trees (recruitment) mapped and measured. Trees that died within the last year were evaluated for factors potentially contributing to mortality, including an inspection of physical damage and signs of tree-killing or -weakening pathogens and insects. As part of that evaluation, field crews made a visual assessment of a given tree’s competitive environment and listed ‘suppression’ as a potential mortality factor when crowding among trees appeared to be important.

### 2.2. Analytical approach

Our core data consist of the comparative spatial configuration of dead and live trees, changes in tree spatial pattern through time, and field assessments of contributors to an individual tree’s death. Note that our primary interest for this study was to examine changes in pattern for the assemblage of trees as a whole rather than for any individual species. As Picard et al. (2009) noted, species that have different dynamics can collectively contribute to a common emergent pattern. Therefore, for this work, we have not subdivided our analyses by species.

Tree spacing was evaluated in the context of a local neighborhood, as processes that affect tree population dynamics often act on local scales (Gratzer et al., 2004). For example, competition (Antonovics and Levin, 1980), root rots (Slaughter and Parmeter, 1995; Garbelotto et al., 1997), and bark beetles (Wood, 1982) all act locally in non-outbreak conditions. Therefore we defined a neighborhood size at which these processes are likely to occur (McIntire and Fajardo, 2009). We wanted our scale of analysis to encompass an area big enough to allow at least two of the largest trees to interact. We used crown-diameter equations developed by Gersonde et al. (2004) and bole diameters from plot data to determine that a 9 m radius was the maximum distance at which at least two of the largest canopy trees for the dominant species could presumably be included in the same neighborhood (i.e., the distance beyond which the largest trees would not have overlapping crowns). Note that a neighborhood of this size should capture local processes affecting trees both large and small. Since demographic processes do not act at discrete distances but rather operate across a range, we evaluated the aggregate spatial pattern for the entire 9 m radius neighborhood. For reference, the median number of neighbors for trees in all plots combined was 25 in a 9-m neighborhood, with median values varying by plot between 11 and 58.

### 2.3. Comparative spatial configuration

We calculated a competition index for each tree at the time of plot establishment. We used the Hegyi index, which estimates competition by weighting the contribution of each competitor by size and distance (Hegyi, 1974; Biging and Dobbertin, 1992). The Hegyi index in this case was calculated based on all trees within a 9 m radius of the subject tree. The competitive environments of dead and surviving trees in each plot were compared using Wilcoxon rank-sum tests. We used a Bonferroni correction to determine a

**Table 1**

Descriptions of the 14 monitoring plots. Characteristics reported are for the populations at the time of plot establishment. Location of plot (in parentheses next to plot name): SEKI = Sequoia Kings-Canyon National Park; YOSE = Yosemite National Park.

| Plot            | Elevation (m) | Year Established | Size (ha) | Density (trees/ha) | Basal area (m <sup>2</sup> /ha) | % Species composition by density  |
|-----------------|---------------|------------------|-----------|--------------------|---------------------------------|---|
| YOHPIPO (YOSE)  | 1500          | 1991             | 1.000     | 2980               | 67                              | <i>A. concolor</i> 39<br><i>C. decurrens</i> 32<br><i>P. lambertiana</i> 23<br><i>P. ponderosae</i> 5<br><i>P. menziesii</i> 1<br><i>Q. kelloggii</i> 1 |
| BBBPIPO (SEKI)  | 1609          | 1992             | 1.000     | 1273               | 69                              | <i>A. concolor</i> 12<br><i>C. decurrens</i> 50<br><i>P. lambertiana</i> 7<br><i>P. ponderosae</i> 4<br><i>Q. kelloggii</i> 23                          |
| CCRPIPO (SEKI)  | 1622          | 1991             | 1.125     | 1868               | 63                              | <i>A. concolor</i> 37<br><i>C. decurrens</i> 34<br><i>P. lambertiana</i> 7<br><i>P. ponderosae</i> 6<br><i>Q. kelloggii</i> 15                          |
| CRCRPIPO (YOSE) | 1637          | 1993             | 1.000     | 1753               | 71                              | <i>A. concolor</i> 46<br><i>C. decurrens</i> 28<br><i>P. lambertiana</i> 18<br><i>P. ponderosae</i> 6<br><i>Q. kelloggii</i> 2                          |
| SUW (SEKI)      | 2035          | 1982–1983        | 3.375     | 732                | 68                              | <i>A. concolor</i> 60<br><i>A. magnifica</i> 3<br><i>C. decurrens</i> 19<br><i>P. lambertiana</i> 17  |
| LMCC (SEKI)     | 2128          | 1982             | 1.75      | 249                | 160 <sup>a</sup>                | <i>A. concolor</i> 68<br><i>A. magnifica</i> 24<br><i>P. lambertiana</i> 2<br><i>S. giganteum</i> 5   |
| LOGSEGI (SEKI)  | 2170          | 1983             | 2.500     | 422                | 145 <sup>a</sup>                | <i>A. concolor</i> 76<br><i>A. magnifica</i> 17<br><i>P. lambertiana</i> 4<br><i>S. giganteum</i> 2   |
| LOLOG (SEKI)    | 2207          | 1987             | 1.125     | 407                | 62                              | <i>A. concolor</i> 71<br><i>A. magnifica</i> 27<br><i>P. lambertiana</i> 1<br><i>S. giganteum</i> 1   |
| UPLOG (SEKI)    | 2210          | 1987             | 0.937     | 378                | 54                              | <i>A. concolor</i> 88<br><i>C. decurrens</i> 3<br><i>P. jeffreyi</i> 1<br><i>P. lambertiana</i> 6<br><i>Q. kelloggii</i> 1                              |
| LOGPIJE (SEKI)  | 2405          | 1985             | 1.000     | 121                | 18                              | <i>A. concolor</i> 60<br><i>A. magnifica</i> 2<br><i>P. jeffreyi</i> 33   |
| SFTRABMA (YOSE) | 2484          | 1992             | 1.000     | 1631               | 101                             | <i>A. magnifica</i> 100   |
| WTABMA (SEKI)   | 2521          | 1993             | 1.000     | 459                | 57                              | <i>A. magnifica</i> 99<br><i>P. monticola</i> 1   |
| POFLABMA (YOSE) | 2542          | 1994             | 1.000     | 589                | 105                             | <i>A. magnifica</i> 95<br><i>P. contorta</i> 5  |
| PGABMA (SEKI)   | 2576          | 1992             | 1.000     | 765                | 97                              | <i>A. magnifica</i> 100   |

<sup>a</sup> The basal area of these plots is inflated by *S. giganteum*.

*p*-value for rejecting or accepting a hypothesis, considering each of our 14 plots a separate test. This yields a *p*-value of 0.004. We also wished to consider how tree size might affect our results. We expect small trees to be more strongly affected by competitive mortality than large trees, which based on their size and canopy status are expected to be less susceptible to competitive effects. Therefore, we also performed these analyses for trees with a diameter at breast height (DBH) less than 20 cm and for trees with a DBH  $\geq$  40 cm. Trees with a DBH < 20 cm include mainly understory trees in these forests, while trees with a DBH  $\geq$  40 cm represent the canopy dominant trees (van Mantgem and Stephenson, 2005).

#### 2.4. Spatial pattern analysis

Changes in spatial patterns were analyzed using 'marked point pattern' techniques (Illian et al., 2008). By 'marked' we mean that

each tree is considered to be marked (labeled) as live or dead. The empirical marking (i.e., the actual patterns of live and dead trees) is then compared to that of simulations, where trees are labeled as live or dead based on a hypothetical process. Most commonly, a hypothesis of complete randomness is used, with trees labeled at random. An advantage of a marked approach is that the simulations are conditioned upon the underlying pattern, allowing tests of hypotheses even in cases where the underlying pattern might exhibit first order heterogeneity (see Appendix A).

We studied the effect of mortality on pattern by comparing empirical results to 1000 simulations of two modeled processes: random mortality and mortality dominated by competition. For the random mortality simulation, we randomly labeled a number of trees as dead equal to the number of trees that had actually died within a given plot during the measurement period. For dominant competitive mortality, we simulated the expected out-

**Table 2**  
Number of trees for the spatial analyses in 14 monitoring plots in Sequoia Kings-Canyon and Yosemite National Parks. Establishment column gives the number of live trees at plot establishment. Refer to Table 1 for establishment dates.

| Plot     | All trees     |             | Small trees (DBH < 20 cm) |             | Large trees (DBH ≥ 40 cm) |                |
|----------|---------------|-------------|---------------------------|-------------|---------------------------|----------------|
|          | Establishment | Mortalities | Establishment             | Mortalities | Establishment             | Mortalities    |
| YOHPIPO  | 2980          | 849         | 2692                      | 828         | 109                       | 3 <sup>a</sup> |
| BBBPIPO  | 1273          | 231         | 736                       | 172         | 75                        | 14             |
| CCRPIPO  | 1868          | 543         | 1715                      | 510         | 107                       | 9 <sup>a</sup> |
| CRCRPIPO | 1753          | 312         | 1403                      | 288         | 117                       | 3 <sup>a</sup> |
| SUW      | 732           | 718         | 1682                      | 511         | 391                       | 110            |
| LMCC     | 249           | 103         | 291                       | 68          | 149                       | 23             |
| LOGSEGI  | 422           | 233         | 610                       | 179         | 228                       | 26             |
| LOLOG    | 407           | 98          | 267                       | 76          | 104                       | 13             |
| UPLOG    | 378           | 60          | 239                       | 39          | 80                        | 12             |
| LOGPIJE  | 121           | 25          | 78                        | 18          | 32                        | 5 <sup>a</sup> |
| SFTRABMA | 1631          | 326         | 1407                      | 313         | 131                       | 5 <sup>a</sup> |
| WTABMA   | 459           | 55          | 293                       | 32          | 105                       | 13             |
| POFLABMA | 589           | 51          | 316                       | 35          | 176                       | 11             |
| PGABMA   | 765           | 139         | 451                       | 91          | 179                       | 25             |

<sup>a</sup> Indicates plots that were not included in the large tree analysis due to insufficient trees dying (less than 5% of the starting population or less than 10 trees dying).

comes of mortality processes in which competition dominated. We assumed that an equal number of trees had died each year (i.e., yearly increment equaled the total number of trees that had died in a given plot during the measurement period divided by the number of years of measure). We calculated a competition index for each tree, as described above, and then labeled a number of trees as dead equal to our yearly increment, with the chance of a tree being labeled weighted by its competition index (i.e., trees with higher competition indices were more likely to die). We then recalculated the competition index without those trees in the sample and repeated the process until the number of trees labeled equaled the number that had actually died in the plot.

We also performed the analyses described above for trees with DBH < 20 cm and for trees with a DBH ≥ 40 cm (understory and canopy dominant trees respectively). Analyses and simulations for each were performed as described above. We did not treat the trees within the individual size classes in isolation for our simulations because it is unrealistic to assume that small trees exist in isolation from large trees or vice versa in assessing their competitive environment. Competitive indices included all trees, meaning that trees were killed during simulation besides those within the size class. The only difference from the all-tree simulations was that simulations halted only after enough trees had been killed within the given size class to match the number of deaths seen within that size class during the measurement period.

Finally, we examined changes in pattern in an extended neighborhood to evaluate whether results observed in the local neighborhood scaled to larger distances. The extended neighborhood was determined by doubling the distance for the immediate neighborhood (10–18 m). Distances beyond 18 m (i.e., larger neighborhoods) were not considered due to plot size restrictions. For the extended neighborhood, the median number of neighbors was 87, with plot values ranging from 39 to 210. For the analysis of spacing in the extended neighborhood, we used a pair correlation function (Appendix B) in addition to the *K* statistic (described below). Since the *K* statistic is a cumulative function, there is the potential that patterns observed in local neighborhood would influence results in the extended neighborhood. The pair correlation function avoids such bias.

## 2.5. Quantifying the patterns

We quantified the patterns using the *K*-function (Ripley, 1976; Bailey and Gatrell, 1995). The *K*-function is a statistic that can be

used with fully mapped point patterns to examine spatial structure at various scales or distances. Larger values of *K* indicate increased clumping of the point pattern. The bivariate form of the *K*-function is estimated by:

$$\hat{K}_{1,2}(h) = \frac{A}{n_1 n_2} \sum_{i=1}^{n_1} \sum_{j=1}^{n_2} I_h(d_{ij})$$

where *h* is the distance at which  $\hat{K}_{1,2}$  is being calculated; *n*<sub>1</sub> is the number of trees in pattern 1 (dead trees); *n*<sub>2</sub> is the number of trees in pattern 2 (surviving trees); *A* is the area of the plot; *d*<sub>*ij*</sub> is the distance between the *i*th and *j*th trees; and *I*<sub>*h*</sub> is an indicator function which is 1 if *d*<sub>*ij*</sub> < *h* and 0 otherwise. All statistics were calculated in S-plus using code derived from the Splanacs library for S-plus (Rowlingson and Diggle, 1993). Details of the function and edge correction used can be found in Rowlingson and Diggle (1991, 1993).

To test for departures from random mortality and subsequently from competitive mortality, we measured the change in pattern over time due to tree mortality. Frequently, processes are inferred from static patterns without reference to changes over time (Wiegand et al., 2000; Druckenbrod et al., 2005). However, as Wiegand et al. (2000) demonstrated, static patterns that are attributed to density-dependent mortality can be reproduced by alternative mechanisms. In addition He and Duncan (2000) showed that unexamined environmental factors can also lead to patterns that mimic density-dependent effects. The effect of process on pattern can only safely be inferred by examining changes through time (Leps, 1990; Wiegand et al., 2000). We followed this recommendation and relied on changes in pattern. With this approach, we can directly assess the effects of mortality on spatial pattern while controlling for environmental heterogeneity.

Specifically, we compared the pattern of survivors in 2005 to the pattern of living trees at the first mapping. In marked point-process terms this bivariate *K* statistic is expressed as:  $\hat{K}_{2,2} - \hat{K}_{1+2,1+2}$ , where the subscript 2 refers to the trees that survived the interval and the subscript 1 refers to trees that died in the interval. The expectation under random mortality is 0, with values greater than 0 indicating increased clumping after mortality and values less than 0 indicating increased uniformity.

To test the consistency of empirical results with our hypothesized processes, we used Loosmore and Ford's (2006) *u* statistic to test goodness of fit of  $\hat{K}_{2,2} - \hat{K}_{1+2,1+2}$  from the observed pattern to

the simulated patterns.

$$u_i = \sum_{t_k=t_{\min}}^{t_{\max}} [\hat{H}(t_k) - \bar{H}(t_k)]^2 \delta t_k$$

where  $t_k$  is distance,  $t_{\min}$  and  $t_{\max}$  are lower and upper limits of the neighborhood,  $\hat{H}(t_k)$  is the empirical result for pattern  $i$  for the given test statistic, and  $\bar{H}(t_k)$  is the mean result for all the patterns except  $i$ , and  $\delta t_k$  is the width of the distance interval.  $\bar{H}(t_k)$  is given by

$$\bar{H}(t) = \frac{1}{s-1} \left[ \sum_{j=1, j \neq i}^s \hat{H}_j(t) \right]$$

where  $s-1$  is the number of simulations. The  $p$ -value for the  $u$  statistic is given by

$$\hat{p} = 1 - \frac{\sum_j I[u_1 > u_j]}{s}$$

where  $j=2, \dots, s$  and  $I$  is an indicator function that is 1 if  $u_1$  is greater than a given  $u_j$  and 0 otherwise.

The  $u$  test avoids the underestimation of Type I error rates inherent in approaches that rely on simulation envelopes (Loosmore and Ford, 2006). Moreover, the  $u$  statistic is an aggregate measure that matches our neighborhood approach, since it evaluates the goodness of fit for a range of distances simultaneously.

The first step in the statistical inference of the spatial pattern analysis was to check for significant departures from random processes (Diggle, 2003). Again, we used the Bonferroni correction to account for the 14 independent tests (i.e., 14 plots) of departures from random mortality. Thus the  $p$ -value cut-off was 0.004. We tested plots that rejected random mortality against the competitive mortality hypothesis with the cut-off for significant departures at 0.05. No adjustments were made for multiple testing since the comparisons were made only after an initial rejection (i.e., a protected test).

## 2.6. Field assessments

Field assessments of dead trees occurred within one year of tree death and contained valuable first-hand information on the potential causes of mortality. Such assessments provided a qualitative supplement to the quantitative spatial analyses. Field crews listed all signs and symptoms of possible contributors to mortality. We organized these observations into three categories: biotic – evidence of tree-killing pathogens or insects; suppression – trees in which suppression was listed as a factor associated with mortality; and mechanical – evidence of crushing, snapping, or uprooting. The suppression category presumably encompasses mortality for which competition (density-dependent mortality) was a factor, though this assignment rests on a visual assessment of the competitive environment by field crews. Since multiple factors can contribute to an individual tree's death, our summaries of the field assessments were not mutually exclusive. The death of some trees was attributed to more than one category. In addition, a subset of trees were assigned a mortality factor of 'unknown' when field inspection did not yield evidence of other causal factors or when the noted mortality agents did not appear sufficient to have caused mortality. In general, assessing agents of tree mortality from non-destructive field inspection of signs and symptoms is limited to what can be seen from the ground. Insects in the crown and pathogens in the roots can be difficult to detect. Moreover, rarely do trees in forests of the Sierra Nevada die from a single specific cause (Franklin et al., 1987; Das et al., 2008). Detailed analyses of the contribution of particular agents are likely to be confounded by

unseen or overlapping factors. Therefore, we used the field assessments to inform our quantitative analyses but, given the limitations of this information, we did not attempt to directly analyze spatial patterns of specific mortality agents in this study.

## 3. Results

### 3.1. Competitive environment of dead trees versus survivors

Dead trees experienced a more competitive local environment than surviving trees. On average, the median competition index for dead trees was 70% greater than surviving trees in the same plot (Fig. 1). In nine of the 14 plots, dead trees had significantly higher competition indices than surviving trees (Fig. 1). In two cases, the differences were extreme. The competition indices for dead trees in CCRPIPO and SFTRABMA were nearly three times greater than surviving trees (Fig. 1). The differences in competitive environment were most consistent in the ponderosa pine-mixed conifer plots. The Hegyi indices for dead trees were significantly higher in all four of the plots in this forest type (Table 3 and Fig. 1). For trees with a DBH < 20 cm, seven of the 14 plots had significantly higher competition indices for dead trees, with both LOGSEGI and LOLOG no longer showing a significant difference (results not shown). For trees with DBH ≥ 40 cm, none of the plots showed a significant difference between live and dead trees (results not shown).

### 3.2. Spatial analyses

#### 3.2.1. Primary analysis: all sizes, local neighborhood

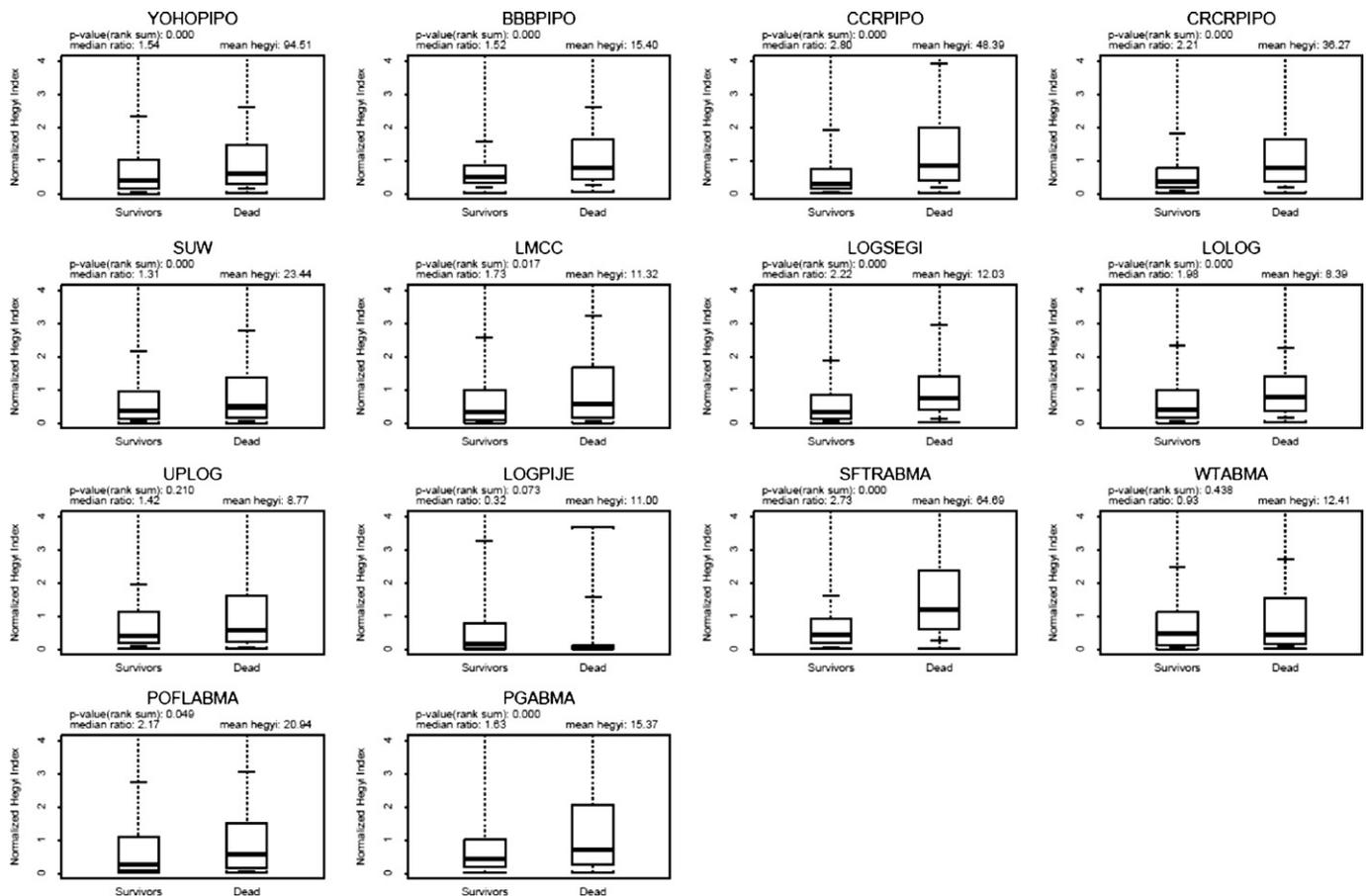
The two mortality simulations (random, competition) generated distinctly different realizations of the potential changes in spatial pattern (Fig. 2). As expected, the random simulations bracketed the zero line (no change in pattern) and the competition simulations resulted in more uniform spacing of trees (less than 0). The variation in the simulations, especially the random mortality model, reflects the density of trees. For plots with fewer trees, the simulations produced a wider range of values, which in turn gave less power in our ability to distinguish between hypotheses (LOGPIJE for example).

Eight of the 14 plots could not be distinguished from random mortality based on changes in spatial pattern (Table 3 and Fig. 2). Of the remaining six, three showed changes in pattern consistent with dominant competitive mortality (YOHPIPO, CCRPIPO, and LOGSEGI). In these three cases, the observed pattern fell within the bounds of the competition simulations. In the remaining three cases with non-random patterns (CRCPIPO, SUW, SFTRABMA), the observed pattern fell between the random and the competition simulations (Fig. 2 and Table 3).

In the three plots consistent with dominant competitive mortality, initial tree density was more than double that of other plots ( $1757 \pm 740$  trees/ha, mean  $\pm$  standard error) and suppression was noted in more than half the dead trees (53%). By contrast, initial tree density in the random plots was  $359 \pm 127$  trees/ha and in the three non-random plots was  $1372 \pm 322$  trees/ha. Suppression was also less commonly observed in the random plots (21%) than in the non-random plots (46%). Two of the plots (YOHPIPO, CCRPIPO) that were consistent with dominant competitive mortality had an average initial Hegyi index much higher than most of the other plots (Fig. 1). Interestingly, the third plot (LOGSEGI) did not, and SFTRABMA, which was not consistent with dominant competitive mortality, had a very high average Hegyi index.

#### 3.2.2. Size class analysis

For small trees (DBH < 20 cm), 12 of the 14 plots could not be distinguished from random mortality based on changes in spatial pattern (Table 4 and Fig. 3). Of the remaining two



**Fig. 1.** Box diagrams of the normalized Hegyi index at plot establishment for trees that survived and for trees that died over the measurement period. The Hegyi index has been normalized by dividing by the mean Hegyi index for all trees for the given plot. The box encompasses the center half of the data with the line inside the box representing the median. The whiskers are drawn to the full range of the data. Narrower horizontal black lines represent the 10th and 90th quantiles of the data. The *p*-values are results from a Wilcoxon rank-sum test between the competition indices for the dead and surviving trees. The median ratio is the ratio of the median competition index for dead trees and that of surviving trees. The mean Hegyi is the mean Hegyi index at plot establishment for all trees.

**Table 3**  
P-values of *u* statistic for  $K_{2,2} - K_{1+2,1+2}$  for all trees.

| Plot     | Random mortality hypothesis | Competition mortality hypothesis | Mean Hegyi index |
|----------|-----------------------------|----------------------------------|------------------|
| YOHAPIPO | <b>0.002</b>                | 0.203                            | 94.51            |
| BBBPIPO  | 0.957                       | –                                | 15.40            |
| CCRPIPO  | <b>0.001</b>                | 0.134                            | 48.39            |
| CRCRPIPO | <b>0.001</b>                | <b>0.018</b>                     | 36.27            |
| SUW      | <b>0.003</b>                | <b>0.001</b>                     | 23.44            |
| LMCC     | 0.412                       | –                                | 11.32            |
| LOGSEGI  | <b>0.001</b>                | 0.153                            | 12.03            |
| LOLOG    | 0.034                       | –                                | 8.39             |
| UPLOG    | 0.885                       | –                                | 8.77             |
| LOGPIJE  | 0.070                       | –                                | 11.00            |
| SFTRABMA | <b>0.001</b>                | <b>0.005</b>                     | 64.69            |
| WTABMA   | 0.164                       | –                                | 12.41            |
| POFLABMA | 0.419                       | –                                | 20.94            |
| PGABMA   | 0.663                       | –                                | 15.37            |

*Note:* Bolded numbers indicate that a given hypothesis is rejected. For the random mortality hypothesis, a Bonferroni corrected *p*-value of 0.004 was used. For the competition hypothesis, a *p*-value of 0.05 was used since only plots where random mortality was rejected were considered (protected test).

plots, only one (CRCRPIPO) was consistent with dominant competitive mortality. Contrary to expectation, there was little evidence that competition was affecting small tree mortality in a disproportionate fashion relative to the population as a whole.

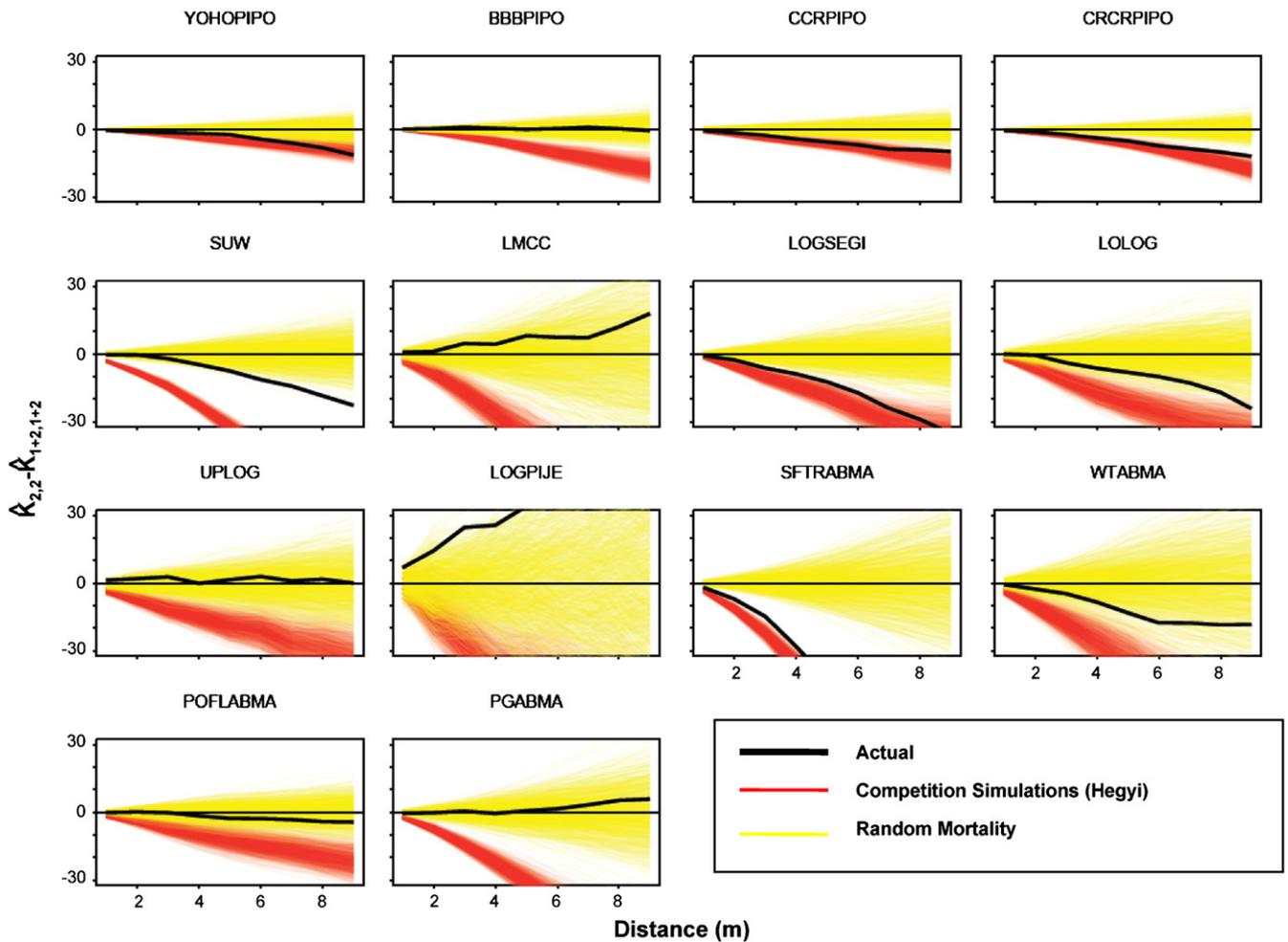
For large trees (DBH  $\geq 40$  cm), only plots where more than 5% of the trees had died and greater than 10 individuals had died were

**Table 4**  
P-values of *u* statistic for  $K_{2,2} - K_{1+2,1+2}$  for trees with DBH < 20.

| Plot     | Random mortality hypothesis | Competition mortality hypothesis |
|----------|-----------------------------|----------------------------------|
| YOHAPIPO | 0.026                       | –                                |
| BBBPIPO  | 0.304                       | –                                |
| CCRPIPO  | 0.329                       | –                                |
| CRCRPIPO | <b>0.001</b>                | 0.269                            |
| SUW      | 0.164                       | –                                |
| LMCC     | 0.140                       | –                                |
| LOGSEGI  | 0.443                       | –                                |
| LOLOG    | 0.349                       | –                                |
| UPLOG    | 0.686                       | –                                |
| LOGPIJE  | 0.081                       | –                                |
| SFTRABMA | <b>0.001</b>                | <b>0.025</b>                     |
| WTABMA   | 0.330                       | –                                |
| POFLABMA | 0.417                       | –                                |
| PGABMA   | 0.323                       | –                                |

*Note:* Bolded numbers indicate that a given hypothesis is rejected. For the random mortality hypothesis, a Bonferroni corrected *p*-value of 0.004 was used. For the hypothesis, a *p*-value of 0.05 was used since only plots where random mortality was rejected were considered (protected test).

considered to avoid excessively small sample sizes. Of the nine remaining plots, none could be distinguished from random mortality, in keeping with expectations that large trees would be relatively less affected by competition (Table 5 and Fig. 4). However, even with the restrictions, sample size is still quite small for the large trees in most plots (Table 2), so the results should be interpreted with caution.



**Fig. 2.** Change in overall structure for each plot, as characterized by  $\hat{K}_{2,2} - \hat{K}_{1+2,1+2}$ , the difference in pattern between survivors and the full set of trees prior to mortality. The solid black line indicates the actual change in pattern in the plot, with values above zero indicating increased clumping and values below zero indicating increased uniformity. The red lines are the results of 1000 simulations of a hypothesis in which all mortality is due to competition. The yellow lines are the results of 1000 simulations of random mortality.

**Table 5**  
*p*-values of *u* statistic for  $K_{2,2} - K_{1+2,1+2}$  for trees with DBH  $\geq 40$ .

| Plot     | Random mortality hypothesis | Competition mortality hypothesis |
|----------|-----------------------------|----------------------------------|
| YOHPIPO  | NA                          | NA                               |
| BBBPIPO  | 0.563                       | -                                |
| CCRPIPO  | NA                          | NA                               |
| CRCRPIPO | NA                          | NA                               |
| SUW      | 0.157                       | -                                |
| LMCC     | 0.545                       | -                                |
| LOGSEGI  | 0.092                       | -                                |
| LOLOG    | 0.098                       | -                                |
| UPLOG    | 0.129                       | -                                |
| LOGPIJE  | NA                          | NA                               |
| SFTRABMA | NA                          | NA                               |
| WTABMA   | 0.566                       | -                                |
| POFLABMA | 0.510                       | -                                |
| PGABMA   | 0.745                       | -                                |

Note: No hypotheses were rejected by goodness of fit tests. For the random mortality hypothesis, a Bonferroni corrected *p*-value of 0.004 was used. For the competition hypothesis, a *p*-value of 0.05 was used since only plots where random mortality was rejected were considered (protected test). 'NA' indicates plots that were not included in the large tree analysis due to insufficient trees dying (less than 5% of the starting population or less than 10 trees dying).

### 3.2.3. Extended neighborhood analysis

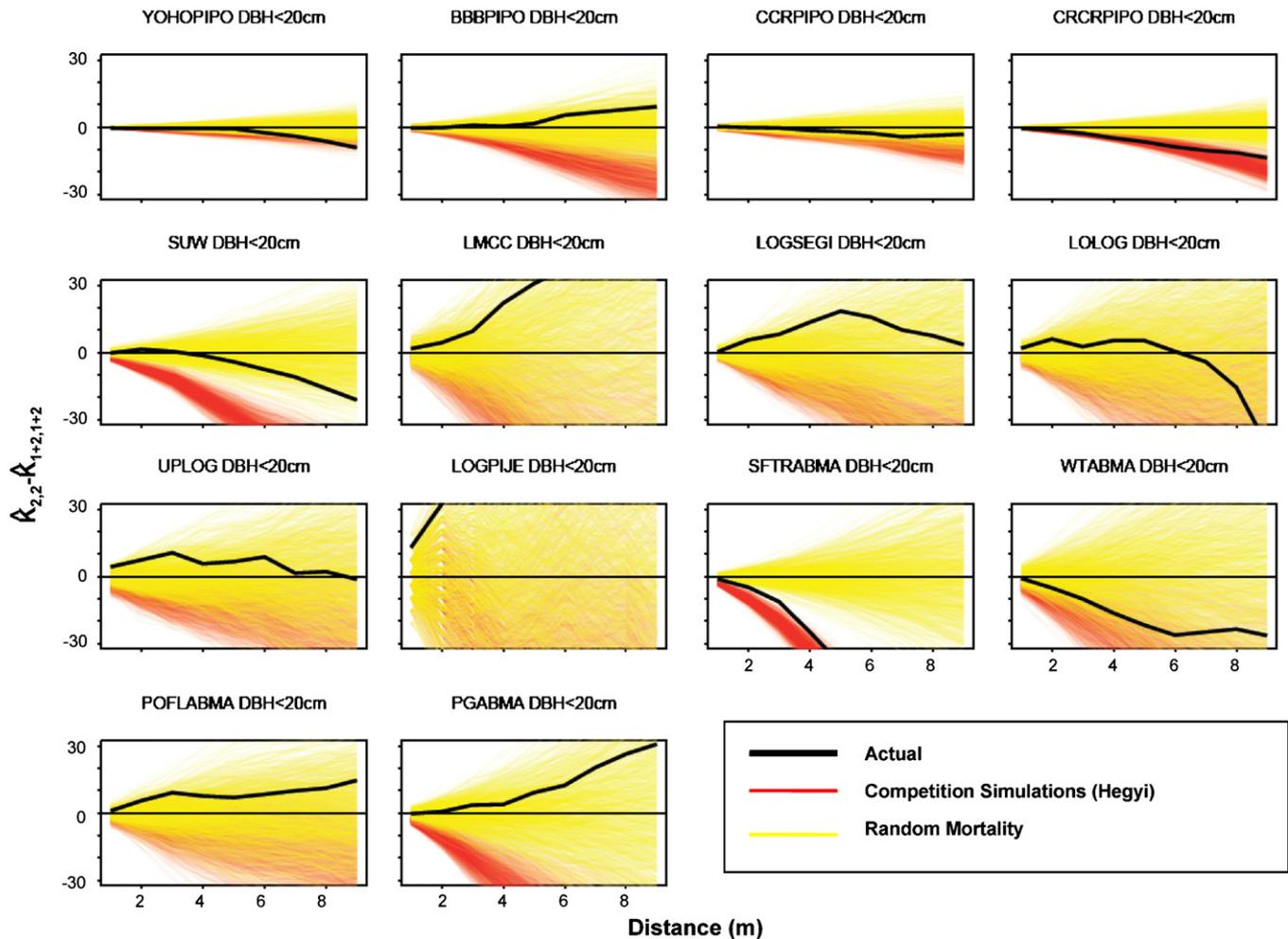
One additional plot (CCRPIPO) was not distinguishable from random in the extended neighborhood, and none of the plots

**Table 6**  
*p*-values of *u* statistic for  $K_{2,2} - K_{1+2,1+2}$  for all trees in the extended neighborhood.

| Plot     | Random mortality hypothesis | Competition mortality hypothesis |
|----------|-----------------------------|----------------------------------|
| YOHPIPO  | <b>0.001</b>                | <b>0.004</b>                     |
| BBBPIPO  | 0.491                       | -                                |
| CCRPIPO  | 0.008                       | -                                |
| CRCRPIPO | <b>0.003</b>                | <b>0.015</b>                     |
| SUW      | <b>0.001</b>                | <b>0.001</b>                     |
| LMCC     | 0.203                       | -                                |
| LOGSEGI  | <b>0.001</b>                | <b>0.001</b>                     |
| LOLOG    | 0.015                       | -                                |
| UPLOG    | 0.985                       | -                                |
| LOGPIJE  | 0.082                       | -                                |
| SFTRABMA | <b>0.001</b>                | <b>0.001</b>                     |
| WTABMA   | 0.458                       | -                                |
| POFLABMA | 0.381                       | -                                |
| PGABMA   | 0.421                       | -                                |

Note: Bolded numbers indicate that a given hypothesis is rejected. For the random mortality hypothesis, a Bonferroni corrected *p*-value of 0.004 was used. For the competition hypothesis, a *p*-value of 0.05 was used since only plots where random mortality was rejected were considered (protected test).

were consistent with a hypothesis of mortality dominated by competition (Table 6). The paired correlation function analysis was essentially identical, except that one more plot was not distinguishable from random (CCRPIPO, Appendix B).



**Fig. 3.** Change in overall structure for each plot for trees with DBH < 20 cm, as characterized by  $\hat{K}_{2,2} - \hat{K}_{1+2,1+2}$  the difference in pattern between survivors and the full set of trees prior to mortality. The solid black line indicates the actual change in pattern in the plot, with values above zero indicating increased clumping and values below zero indicating increased uniformity. The red lines are the results of 1000 simulations of a hypothesis in which all mortality is due to competition. The yellow lines are the results of 1000 simulations of random mortality.

### 3.3. Field assessments

Suppression was the most common mortality factor noted in the field assessments. More than half (52%) of the 3743 tree mortalities over our 23 year study period had suppression listed as a contributor. Biotic factors were also frequently observed (54%) while mechanical factors were the least common (26%). However, at the plot level, suppression was most common in only two plots while biotic factors were the most common in eight plots and mechanical in four (Table 7). Not surprisingly, plot level tree density was significantly correlated with suppression ( $r=0.85$ ,  $p<0.001$ ) as was the mean Hegyi index at plot establishment ( $r=0.93$ ,  $p<0.001$ ). Also, the contribution to mortality by mechanical factors increased with elevation ( $r=0.71$ ,  $p=0.004$ ). There were no apparent patterns in the distribution of mortality agents by forest type (Table 7) except those related to forest type differences in tree density and elevation.

## 4. Discussion

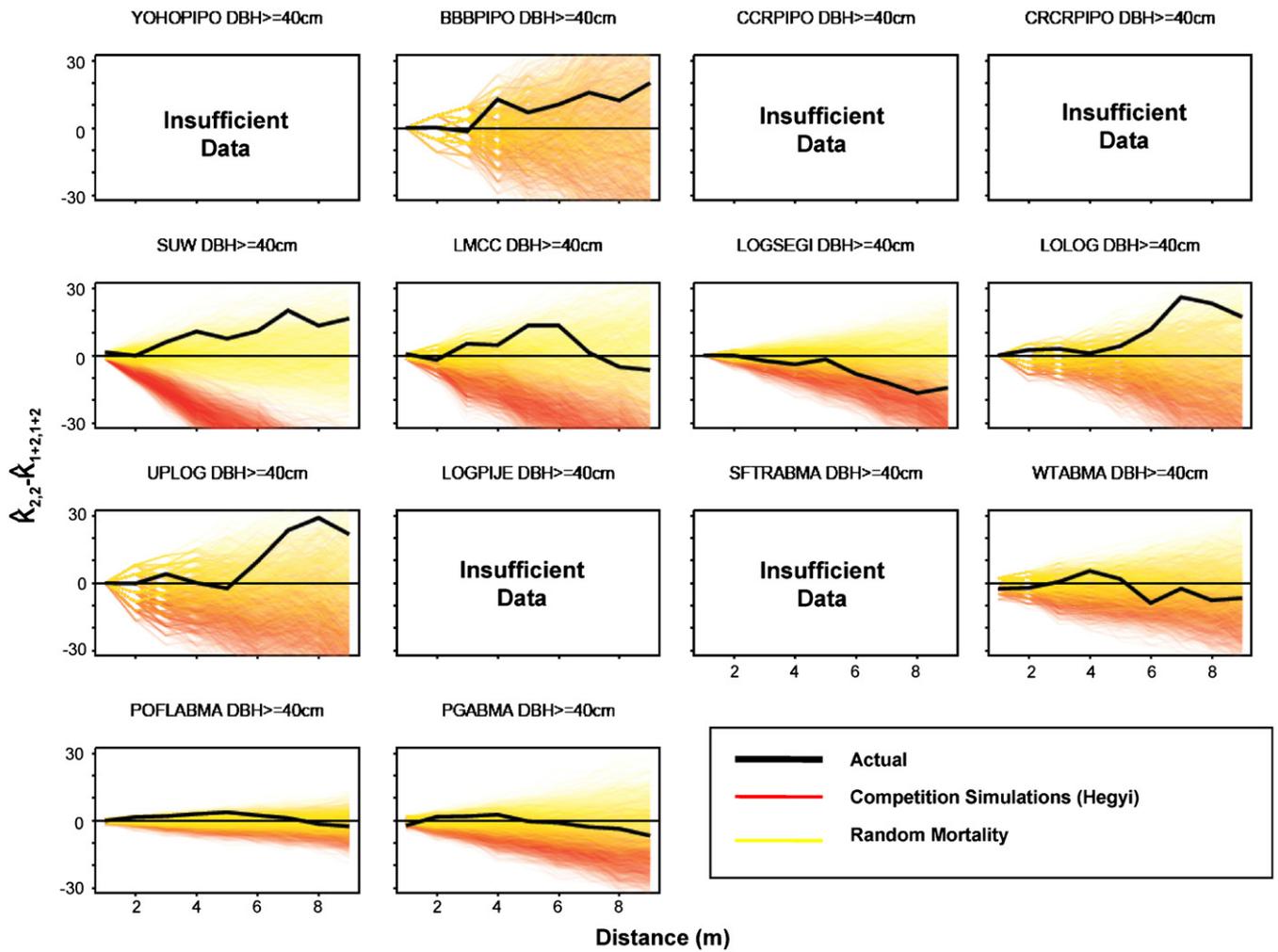
### 4.1. Competition and mortality

Competition was apparently a significant contributor to tree mortality in these forests. Trees that died tended to be in more competitive environments than trees that survived, and suppression frequently appeared as a factor contributing to mortality. On

the other hand, based on spatial pattern analyses, only three of 14 plots demonstrated compelling evidence that competition was *dominating* mortality. Most of the rest of the plots fell within the expectation for random mortality, and three fit neither the random nor the competition model. These results suggest that while competition is often playing a substantial role in tree mortality processes in these forests it only infrequently governs those processes.

The finding that competition is likely a significant factor contributing to mortality in our study is in keeping with the extensive literature demonstrating such relationships in other forests (Eid and Tuhus, 2001; Yang et al., 2003; Monserud et al., 2004; Temesgen and Mitchell, 2005; Bravo-Oviedo et al., 2006) and in a previous study at this site (Das et al., 2008). The further finding that it often does not dominate mortality processes agrees with the contention of Franklin and van Pelt (2004) that other agents become increasingly important as a stand ages. In fact, their observation that old growth forests are a fine-scale mosaic “in which all stand development processes are simultaneously present within the stand” seems well in keeping with our results, which show the strength of competition varying from one locale to another.

Not surprisingly, given other studies (e.g., Yang et al., 2003; Gonzalez et al., 2004; Temesgen and Mitchell, 2005), overall plot density appears to be related to the strength of competitive effects. Two of the three plots with mortality apparently dominated by competition had high overall stand densities, and two other dense



**Fig. 4.** Change in overall structure for each plot for trees with DBH  $\geq 40$  cm, as characterized by  $K_{2,2} - K_{1+2,1+2}$  the difference in pattern between survivors and the full set of trees prior to mortality. The solid black line indicates the actual change in pattern in the plot, with values above zero indicating increased clumping and values below zero indicating increased uniformity. The red lines are the results of 1000 simulations of a hypothesis in which all mortality is due to competition. The yellow lines are the results of 1000 simulations of random mortality.

**Table 7**  
Field assessments of factors associated with tree death.

| Plot     | Biotic | Suppression | Mechanical | Forest type                    |
|----------|--------|-------------|------------|--------------------------------|
| YOHOPIPO | 65     | 59          | 12         | Ponderosa pine – mixed conifer |
| BBBPIPO  | 37     | 42          | 23         | Ponderosa pine – mixed conifer |
| CCRPIPO  | 33     | 62          | 15         | Ponderosa pine – mixed conifer |
| CRCRPIPO | 56     | 44          | 29         | Ponderosa pine – mixed conifer |
| SUW      | 57     | 31          | 20         | White fir – mixed conifer      |
| LMCC     | 41     | 24          | 50         | White fir – mixed conifer      |
| LOGSEGI  | 52     | 39          | 27         | White fir – mixed conifer      |
| LOLOG    | 26     | 15          | 35         | White fir – mixed conifer      |
| UPLOG    | 37     | 12          | 40         | White fir – mixed conifer      |
| LOGPIJE  | 40     | 8           | 24         | Jeffrey pine                   |
| SFTRABMA | 76     | 64          | 25         | Red fir                        |
| WTABMA   | 69     | 20          | 60         | Red fir                        |
| POFLABMA | 73     | 16          | 65         | Red fir                        |
| PGABMA   | 48     | 27          | 65         | Red fir                        |

*Note:* Results indicate percentages of dead trees in the plot with biotic agents, suppression, or mechanical damage listed. Note that categories are not mutually exclusive, allowing the total to sum to more than 100. Also note that some trees have ‘unknown’ as the only mortality factor, allowing the sum to be less than 100.

plots (SFTRABMA and CRCRPIPO) had changes in pattern strongly tending toward the competition simulations.

However, plot density was not a consistent measure of the strength of competitive interactions. The third plot in which competition processes explained the spatial pattern (LOGSEGI) had a relatively low density and mean competition index. Furthermore,

several plots with low densities (e.g., LOLOG, LMCC, WTABMA) showed significant differences between the competition indices of live and dead trees. Some of these inconsistencies are likely due to the inability of stand level measures of competition to capture individual variation in the competitive environment in spatially heterogeneous stands.

Contrary to expectation, our analysis did not find evidence of increased competitive effects among the smallest trees. In fact, fewer plots rejected the random mortality hypothesis (Fig. 3 and Table 4), and fewer plots showed a significant difference between live and dead trees with regard to their competitive environment. In general, the results for the smaller trees track the results for the population as a whole – trees in competitive neighborhoods have a higher probability of dying but this tendency does not generate a strong spatial signature of competition.

For large trees, our results followed the expectation that canopy dominants would be less affected by competition. Competition also did not show persistent and strong effects in the extended neighborhood, suggesting that its action may be most prevalent at finer scales and that other agents may be influencing patterns more strongly at coarser scales.

The Hegyi index is a straightforward measure of competition that we have successfully used in the past to develop relationships between mortality and competition (Das et al., 2008). However it does not take species identity into account. We did find in our previous study (Das et al. 2008) that a more complex index including species identity did not improve the models. In addition, a significant relationship exists between the Hegyi index and growth rate for our trees (not shown). Nevertheless, future work would certainly benefit from more refined measures of competition, both incorporating species identity and empirically fitting neighborhood sizes. Recent work has shown that the competitive contribution of trees can vary by species (Canham et al., 2004; Uriarte et al., 2004a) and that competitive effects can be asymmetric between species (Canham et al., 2006). Even when species identity does not matter in terms of competitive strength, the response to competition can vary among species (Uriarte et al., 2004b).

In addition to refining the competition index, we might also benefit from fitting more general relationships between competition and mortality risk. Rather than a simple weighting, we could fit an absolute survival probability for each tree using empirical models that relate competition to mortality risk. Developing such relationships would require teasing out the various factors that affect a given tree's risk of mortality, but such models are not necessarily intractable.

#### 4.2. Biotic factors and moving forward

The spatial pattern analyses suggest that factors in addition to competition are playing a substantial role in mortality processes. While trees that die are on average in more crowded environments, changes in spatial pattern indicate that competitive interactions *alone* can only infrequently explain changes in tree pattern. In keeping with that result, over 50% of the mortalities across all the plots had a biotic factor listed as a contributor, and field evaluations indicated the presence of at least two different root rots and several bark beetles. Our results are also consistent with the finding that non-competitive spatial elements of mortality, such as proximity to disease vectors, may play an important role in determining individual tree mortality risk (Das et al., 2008). Certainly, in the Sierra Nevada, many insects and pathogens are considered threats to tree health (Wood et al., 2003), and several other studies have examined the effect of non-competitive agents on forests (Hansen and Goheen, 2000; Dobbertin et al., 2001; Rademacher et al., 2004; Davis et al., 2005; Rigg, 2005).

Given the circumstantial evidence, future work in these forests should incorporate direct tests for the action of biotic agents. For example, since many root rots and beetles kill trees in localized centers (e.g., Slaughter and Parmeter, 1995), one could simulate mortality patterns that matched the expected action of these agents and then compare the results against the expectations for competitive and random mortality. More broadly, a multi-factor analysis

would likely provide a more comprehensive picture of mortality processes.

In general, one might expect factors other than competition to be important not just for old growth forests but potentially for any mature forest (sensu Franklin and van Pelt, 2004). Therefore, reliance on the simplification that competition is the primary driver of mortality may result in inaccurate forecasts of forest change – a particularly relevant problem in an age of unprecedented anthropogenic global changes.

Currently, most forest gap models make the simplification that competition is the only non-random driver of mortality (Keane et al., 2001). Since those models do incorporate a random component and most of the plots in this study might be described by some combination of the random and competitive mortality, one could theoretically just adjust the proportions of mortality killed by each mechanism to try to match empirical reality. For example, early versions of the SORTIE model (Pacala et al., 1996) made such an adjustment by making canopy trees only susceptible to random mortality and not to competitive mortality.

However, as Keane et al. (2001) note, the random mortality in such models is a 'catch all' submodel without an underlying mechanism and would be unlikely to perform well under changing conditions. For example, biotic agents would not be expected to operate completely at random, given such factors as host preferences, aggregation pheromones, and a tendency in some cases to seek out more vulnerable trees. Moreover, spatial processes are rarely truly random in biological systems (Loosmore and Ford, 2006). This suggests a need for more refined tests to detect departures from randomness in our plots (e.g., Raventos et al., 2010) – tests that also include more precise hypotheses for biological agents as noted above.

Advancing our understanding of tree mortality will require holistic approaches that embrace more biological complexity. We have here – by incorporating multiple lines of evidence and testing a specific biological hypothesis – attempted to take a first step in that direction.

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#### Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.foreco.2010.12.035.

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