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# Causes and Implications of the Correlation between Forest productivity and Tree Mortality Rates

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# Causes and implications of the correlation between forest productivity and tree mortality rates

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*Abstract.* At global and regional scales, tree mortality rates are positively correlated with forest net primary productivity (NPP). Yet causes of the correlation are unknown, in spite of potentially profound implications for our understanding of environmental controls of forest structure and dynamics and, more generally, our understanding of broad-scale environmental controls of population dynamics and ecosystem processes. Here we seek to shed light on the causes of geographic patterns in tree mortality rates, and we consider some implications of the positive correlation between mortality rates and NPP. To reach these ends, we present seven hypotheses potentially explaining the correlation, develop an approach to help distinguish among the hypotheses, and apply the approach in a case study comparing a tropical and temperate forest.

Based on our case study and literature synthesis, we conclude that no single mechanism controls geographic patterns of tree mortality rates. At least four different mechanisms may be at play, with the dominant mechanisms depending on whether the underlying productivity gradients are caused by climate or soil fertility. Two of the mechanisms are consequences of environmental selection for certain combinations of life-history traits, reflecting trade-offs between growth and defense (along edaphic productivity gradients) and between reproduction and persistence (as manifested in the adult tree stature continuum along climatic and edaphic gradients). The remaining two mechanisms are consequences of environmental influences on the nature and strength of ecological interactions: competition (along edaphic gradients) and pressure from plant enemies (along climatic gradients).

For only one of these four mechanisms, competition, can high mortality rates be considered to be a relatively direct consequence of high NPP. The remaining mechanisms force us to adopt a different view of causality, in which tree growth rates and probability of mortality can vary with at least a degree of independence along productivity gradients. In many cases, rather than being a direct cause of high mortality rates, NPP may remain high in spite of high mortality rates. The independent influence of plant enemies and other factors helps explain why forest biomass can show little correlation, or even negative correlation, with forest NPP.

*Key words: competition; consumer controls; forest dynamics; functional traits; life-history trade-offs; net primary productivity; permanent sample plots; plant enemies; temperate forest; tree growth; tree mortality; tropical forest.*

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## Introduction

Global patterns of insolation, temperature, precipitation, and nutrient supplies are the foundation of gradients of terrestrial net primary productivity (NPP). Although causation is still debated, broad-scale gradients of NPP have long been known to correlate with important characteristics of biotic communities, such as species diversity and vegetation structure (e.g., Whittaker 1975, Gillman and Wright 2006, Keeling and Phillips 2007, Moles et al. 2009*a*). Less well characterized, however, is the relationship between NPP and population dynamics, particularly demographic rates (see the references and summaries in Gaston et al. 2008 and Schemske et al. 2009). In one of the best characterized examples, background (non-catastrophic) mortality rates of forest trees follow global and regional patterns of forest productivity, with the most productive forest types having average mortality rates three to four times greater than those of the least productive forest types (Stephenson and van Mantgem 2005). This simple correlation hints at a possible causal relationship between population dynamics and ecosystem processes (cf. Clark 1990, Brown et al. 2004), with implications for our understanding of controls of forest structure, carbon storage, and NPP.

Causes of geographic variation in tree mortality rates have been explored in only a handful of studies (e.g., Chao et al. 2008, Lines et al. 2010; Dietze and Moorcroft, *in press*), without a systematic consideration of a range of hypotheses explaining the relationship between tree mortality rates and forest NPP. We therefore lack generalized answers, or even approaches for obtaining answers, to several key questions. For example, is high forest NPP a direct cause of high tree mortality rates, such as through enhanced competition (Clark 1990, Phillips et al. 2008, Enquist et al. 2009)? Or is mortality rate controlled by other factors, such as environmental effects on herbivore populations (Coley and Barone 1996), that may be largely independent of those directly mediated by NPP? Are mortality rates further influenced by environmental selection for species exhibiting certain life-history traits and trade-offs (Grime 2001)? Finally, what are the implications of these possibilities for ecological theory in general?

These questions are made especially compelling by recent observations that, at subcontinental to global scales, forest function, structure, and dynamics are changing. Over the last few decades average global forest NPP has been changing, most likely due to various combinations of changing temperature, precipitation, cloudless days, atmospheric $CO_2$, and nutrient deposition (Boisvenue and Running 2006, Zhao and Running 2010). Over roughly the same period, reports of drought- and temperature-induced episodes of elevated tree mortality have increased (Allen et al. 2010). In tropical Amazonia, apparent increases in forest NPP have been paralleled by increasing forest density, aboveground biomass, recruitment rates, and mortality rates (Laurance et al. 2004, 2009, Lewis et al. 2004, Phillips et al. 2008), and similar changes may be occurring in at least some other tropical regions (Chave et al. 2008, Lewis et al. 2009*a*, *b*). In contrast, in the temperate western United States, background tree mortality rates have increased while recruitment rates have remained unchanged, leading to a net decrease in forest density and basal area (van Mantgem et al. 2009). These observations, coupled with model results suggesting that small changes in tree mortality rates can, over time, profoundly affect the structure, composition, and dynamics of forests (e.g., Kobe 1996, Pacala et al. 1996, Bugmann 2001, Wyckoff and Clark 2002), point to a clear need for a better understanding of environmental controls of tree mortality.

This paper has two goals: to shed light on the causes of geographic patterns in background tree mortality rates, and to consider some implications of the positive correlation between mortality rate and NPP. To reach these ends, the paper is organized in four main sections. In the first, we provide background and theory, beginning with syntheses of hypothesized and observed relationships between tree traits and probability of mortality, both within and among tree life-history groups and within and among forest communities. This sets the stage for introducing two broad classes of proximate causes of differences in mortality rates between forests and seven hypothesized ultimate causes. The seven hypothesized ultimate causes are not all mutually exclusive, and include well-known conjectures about broad-scale gradients of selection for species exhibiting certain combinations of life-history traits and trade-offs, and direct environmental influences on ecological interactions.

In the second section, we develop an approach to help distinguish among the hypotheses. For each of the hypotheses, we identify the associated differences that would be expected between a high-mortality and a low-mortality forest in (1) the forests' relative proportions of trees belonging to groups defined by species- and tree-specific traits, and (2) mortality rates specific to each of these groups. Based on these expectations and the results of other published studies, we provide a framework for systematically determining which hypotheses to favor or reject.

In the third section, we apply our approach to a case study, both as a means of demonstrating the potential utility of our approach and as a basis for contributing, along with our literature synthesis, to the two primary goals of the paper. The case study compares two of the largest qualifying data sets from forest types that exhibit, at global scales, extreme high and low mortality rates (Stephenson and van Mantgem 2005): tropical angiosperm forest (data from Barro Colorado Island, Panama; mortality rate 2.22% per yr) and temperate gymnosperm forest (data from California, Oregon, and Washington, USA; mortality rate 1.10% per yr). We interpret the case study's results in light of the available

literature. Finally, in the *Discussion*, we use information and results from the preceding sections to frame our discussion of possible generalizations about mechanisms driving geographic patterns of tree mortality rates, and implications of the positive correlation between mortality rates and NPP.

## Background and Theory

### *Relationships between species' traits and probability of mortality*

Plants must allocate finite resources to three critical functions: growth, reproduction, and persistence. Relative allocations to these functions are at least partly determined by trade-offs subject to natural selection, and can affect mortality rates (Herms and Mattson 1992, Arendt 1997, Obeso 2002, Strauss et al. 2002, Reich et al. 2003, Stamp 2003, Westoby and Wright 2006). Not surprisingly, then, forest ecologists have recognized two continua of life-history trade-offs of particular importance in trees: growth vs. persistence (especially as manifested in the shade-tolerance continuum), and reproduction vs. persistence (as manifested in the continuum of adult tree stature) (Loehle 2000, Turner 2001, Poorter et al. 2003, 2006, Falster and Westoby 2005, Nascimento et al. 2005, Bohlman and O'Brien 2006, Wright et al. 2010).

Importantly, differences in life-history traits among species might manifest themselves locally, within forest communities, and regionally and globally, among forest communities along broad-scale environmental gradients. Our ability to understand causes of differences in mortality rates among forests requires that we make a clear distinction between the relative effects of alpha (within-community) and beta (among-community) variation in life-history traits and trade-offs (cf. Ackerly and Cornwell 2007). While we introduce this distinction in the following paragraphs, its importance will become especially evident in subsequent sections.

In the growth–persistence trade-off, resource-rich environments are thought to select for species exhibiting suites of traits that favor rapid growth, which confers a competitive advantage, at the expense of traits that can enhance long-term survival, especially defenses (Coley et al. 1985, Stamp 2003, Chave et al. 2009*a*, Endara and Coley 2011) but perhaps also failure-resistant hydraulic architecture (e.g., Markesteijn et al. 2011) and structural reinforcement (Zimmerman et al. 1994, King et al. 2006*b*, van Gelder et al. 2006, Chave et al. 2009*a*; but see Anten and Schieving 2010, Larjavaara and Muller-Landau 2010). Such species therefore experience both higher average growth rates and mortality rates than other species. Within a forest community, the trade-off is most familiarly expressed in the continuum between shade-tolerant and shade-intolerant species (Pacala et al. 1996, Turner 2001, Wright et al. 2003, Gilbert et al. 2006, Valladares and Niinemets 2008). Shade-intolerant species generally depend on the high-light environments of forest gaps, and usually have both high potential growth rates and high mortality rates.

Among different forest communities, the growth–persistence trade-off might be expressed in the continuum between "competitors" and "stress-tolerators" (Grime 2001). Broad-scale gradients of environmental potential for NPP could affect community-wide mortality rates by selecting for whole groups of species that, independent of their local shade tolerance, strike a particular balance in the trade-off between growth and persistence, particularly growth and defenses (Coley et al. 1985, Grime 2001, Stamp 2003, Endara and Coley 2011). For example, a trade-off between growth and defense is found in Amazonian tree species growing on soils of contrasting fertility (Fine et al. 2004, 2006). Another broad-scale growth–persistence trade-off might involve that between tree hydraulic efficiency and safety, in which more stressful environments select for species with hydraulic architectures that are more resistant to failure (embolism), at the expense of efficient water transport and thus potential for rapid growth and the competitive advantages it confers (e.g., Hacke and Sperry 2001, Sperry et al. 2008). (We call this the "growth–hydraulic-safety trade-off" to emphasize its membership in the broader class of growth–persistence trade-offs.) For such a trade-off to contribute to the positive correlation between forest NPP and tree mortality rates at global scales, the more efficient hydraulic architecture of trees in productive environments must also contribute to their higher mortality rates. Such a condition might arise if, for example, species in productive environments face especially strong selection for the competitive advantage conferred by rapid growth, even if the associated hydraulic architecture brings a greater risk of fatal failure relative to species in unproductive environments.

In contrast, in the reproduction–persistence trade-off, species that direct more resources toward reproduction may do so at the expense of growth and defenses, and thus may suffer higher mortality rates (Silvertown and Dodd 1999, Obeso 2002). Within forest communities, the trade-off may be expressed in the adult tree stature continuum. Compared to canopy species, subcanopy species (those that complete their entire life cycles without achieving canopy stature) direct more resources toward early and profuse reproduction at the expense of continued growth (Thomas 1996, Turner 2001, Kohyama et al. 2003, Kohyama and Takada 2009) and probably also defenses (Loehle 2000). Consequently, subcanopy species often have higher mortality rates than canopy species, even when trees of the same sizes and light environments are compared (Manokaran and Kochummen 1987, Korning and Balslev 1994, Nascimento et al. 2005, King et al. 2006*a*). (An exception is found in seedlings of subcanopy species, which have low mortality rates [King et al. 2006*c*], presumably because they have not yet begun to divert resources toward reproduction.) While the reproduction–persistence

trade-off is evident within forest communities, we are unaware of clear manifestations of the trade-off among forest communities (but see Moles et al. [2009*b*], who reported increasing proportions of NPP devoted to seed production with declining absolute latitude).

### *Relationships between individual tree characteristics and probability of mortality*

Mortality rate is also related to characteristics of individual trees that are independent of their species' life-history traits: in particular, tree growth rate and size relative to conspecifics. Within a given species, recent growth rate usually is negatively correlated with mortality rate (i.e., rapidly growing trees are the least likely to die, slowly growing trees the most likely to die; e.g., Buchman et al. 1983, Wyckoff and Clark 2002, Bigler and Bugmann 2004). This negative relationship exists because persistent slow growth relative to conspecifics usually reflects chronic stresses, including competition (Pedersen 1998, Canham et al. 2006), reduced defenses (Waring and Pitman 1985, Waring 1987), and (or) ongoing attack by herbivores or pathogens (Rosso and Hansen 1998, Noetzli et al. 2003). This negative relationship does not contradict the observed positive relationship between species' average (or maximum potential) growth rates and mortality rates, which is a result of a local growth–persistence trade-off (e.g., Condit et al. 1996*a*, Wright et al. 2003, Gilbert et al. 2006). The positive relationship between growth and mortality applies among species along the shade-tolerance continuum, whereas the negative relationship applies to individuals within those species.

Relationships between tree size and mortality rate are more difficult to generalize and interpret. Some studies have found no relationship between size and mortality, at least for trees $\geq$10 cm in diameter (e.g., Lieberman and Lieberman 1987). A more common observation is that small trees have higher mortality rates than large trees (e.g., Condit et al. 1999, Coomes et al. 2003, Muller-Landau et al. 2006*a*, Wunder et al. 2008), although in some cases this pattern may simply reflect that smaller trees more often are suppressed than larger trees, and therefore exhibit elevated mortality related to slow growth, not small size per se (Coomes et al. 2003, Uriarte et al. 2004). Another common relationship is that mortality rate is highest in the smallest and largest trees, and lowest in mid-sized trees (e.g., Buchman et al. 1983, Muller-Landau et al. 2006*a*, Lines et al. 2010). In Malaysia, Newbery et al. (1999) found that the relationship between size and mortality depended on adult tree stature: mortality rate increased with size within subcanopy species, but decreased with size within canopy species. These contrasting patterns may reflect costs incurred by subcanopy species, which, unlike canopy species, initiate reproduction in the shaded understory (expressing a local reproduction–persistence trade-off).

### *Proximate causes of differences in mortality rates between forest communities*

The preceding subsections identified several continua of variation in species' life-history traits and the characteristics of individual trees that are related to probability of mortality. For ease of presentation and to facilitate analyses, we divide these continua into discrete groups: LH groups, defined by species' local life-history traits (shade tolerance and adult tree stature relative to sympatric species), and GS groups, defined by tree growth rate and size. These divisions help us develop a quantitative tool (Eqs. 1 and 2) that both contributes conceptually to our hypotheses explaining geographic variation in tree mortality rates, and helps us distinguish among those hypotheses.

As suggested by the preceding subsection, two broad classes of proximate causes can explain differences in mortality rates between two forest communities. First, two forests might differ in *group proportions*: the high-mortality forest has greater proportions of trees belonging to groups with intrinsically high mortality rates, such as subcanopy species or slowly growing (suppressed) trees (Fig. 1). Second, the forests might differ in *group-specific mortality rates*: a given group in the high-mortality forest has a higher mortality rate than the same group in the low-mortality forest (Fig. 1). The two classes of proximate causes are not mutually exclusive, and their relative contributions to differences in mortality rates between forest communities can be calculated from data collected in permanent forest plots. Specifically, the relative proportion of the difference in community-wide mortality rates between two forests that results from differences in LH-group proportions is calculated as

$$\lambda = \frac{\sum_{i,j}(p_{ij}^{\text{high}} - p_{ij}^{\text{low}})(m_{ij}^{\text{high}} + m_{ij}^{\text{low}})}{2(M^{\text{high}} - M^{\text{low}})} \quad (1)$$

where $p_{ij}$ is the proportion of trees and $m_{ij}$ is the annual mortality rate in LH group $ij$ (shade-tolerance class $i$ and adult stature class $j$), and $M$ is overall (community-wide) mortality rate of a forest (see Appendix A for the derivation). Superscripts "high" and "low" indicate the high- and low-mortality forests, respectively. Similarly, the relative proportion of the difference in community-wide mortality rates between the two forests that results from differences in LH-group-specific mortality rates is calculated as

$$\mu = \frac{\sum_{i,j}(m_{ij}^{\text{high}} - m_{ij}^{\text{low}})(p_{ij}^{\text{high}} + p_{ij}^{\text{low}})}{2(M^{\text{high}} - M^{\text{low}})}. \quad (2)$$

Values of $\lambda$ and $\mu$ can be either positive or negative, but their sum must equal 1.

If calculations reveal that some of the difference in community-wide mortality rates between the forests can be attributed to differences in their LH-group-specific

mortality rates (i.e., if $\mu > 0$), we may wish to determine proximate causes of the differences in LH-group-specific mortality rates (see *Distinguishing among the hypotheses*). Eqs. 1 and 2 are then used to calculate the relative contributions of GS-group proportions and GS-group-specific mortality rates to the difference between forests in LH-group-specific mortality rates, where $p_{ij}$ now represents the proportion of trees, and $m_{ij}$ the annual mortality rate, in GS group $ij$ (growth-rate class $i$, diameter class $j$), and $M$ represents LH-group-specific mortality rate for a given LH group.

We highlight three limitations of Eqs. 1 and 2. First, species life-history traits, tree growth rates, and tree sizes fall along continua, not into discrete groups as assumed by the equations. To a degree, the continua can be represented by classifying species and trees into more (and more finely divided) groups, but this comes at the expense of reduced sample sizes within groups. The second (and related) limitation is that the quantitative results of the equations can be affected by the location of boundaries between groups; however, these effects should normally be small relative to the dominant patterns revealed by the equations. Finally, in their current forms the equations allow comparison of only two forests at once. In the future, some of these limitations might be overcome by adopting an approach conceptually similar to that used by Ackerly and Cornwell (2007) to partition species trait values into within- and among-community components. However, development of such an approach poses unique challenges that are beyond the scope of this paper.

### *Hypothesized ultimate causes*

Ultimate causes of differences in community-wide mortality rates between forests are those environmental and ecological factors that drive the observed proximate causes of the differences. For example, if a forest's higher mortality rate is at least partly attributed proximately to its greater proportion of trees belonging to subcanopy species, an ultimate cause explains the origin of that greater proportion.

We present seven possible ultimate causes of broad-scale differences in community-wide mortality rates between forests. The hypotheses are not all mutually exclusive, and reflect the premise that broad-scale patterns in background mortality rates must be a consequence of environmental selection for species exhibiting certain traits, environmental influences on the nature of ecological interactions, or both. (Environmental effects on mechanical stresses to trees are considered briefly in *Discussion*.) We emphasize aspects of the causes that would help explain the positive correlation, at regional and global scales, between tree mortality rates and forest NPP. For brevity, our use of the term "resource" can include temperature, in addition to light, water, and nutrients.

The first two hypothesized ultimate causes are related to differences between forests in LH-group proportions.

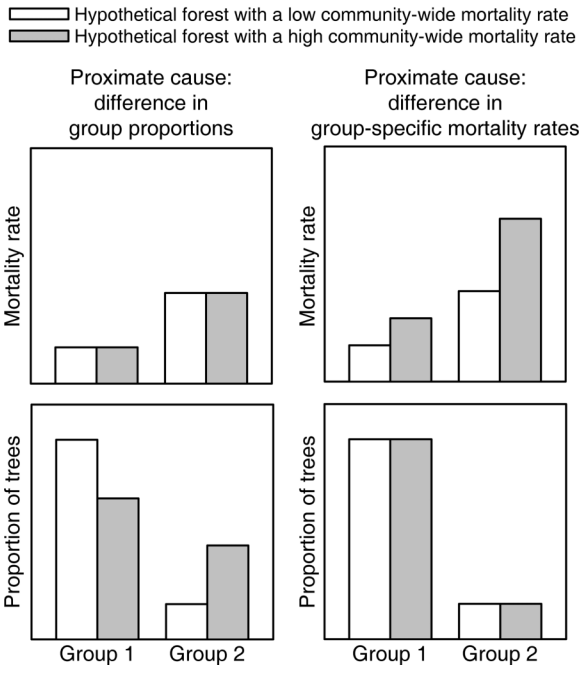


FIG. 1. The two classes of proximate causes of differences in community-wide mortality rates (white and gray bars represent hypothetical forests with low and high community-wide mortality rates, respectively). Difference in group proportions (left panels): a greater proportion of trees belong to groups with high mortality rates (in this case, Group 2), leading to the forest's higher community-wide mortality rate. Difference in group-specific mortality rates (right panels): mortality rates within some or all groups are higher, leading to the forest's higher community-wide mortality rate. Although each set of panels shows only one proximate cause acting at a time, the causes are not mutually exclusive, and they may work either in concert or in opposition. Group 2 can represent life-history groups with high mortality rates (such as shade-intolerant or subcanopy species), growth-rate and size classes with high mortality rates (such as slowly growing or small trees within a given life-history group), or combinations of these.

*The tolerant/intolerant proportions hypothesis*.—One forest could have a higher mortality rate than another because it has a greater proportion of trees belonging to shade-intolerant species (as measured relative to sympatric species), which have high mortality rates due to a local growth–persistence trade-off. The ultimate cause of this greater abundance would be any of the other six ultimate causes that result in higher mortality rates (especially in canopy trees), thereby creating more opportunities for successful recruitment of shade intolerants (cf. Chao et al. 2009). This hypothesis is unique in reflecting a secondary effect of one or more of the other ultimate causes; however, we find it conceptually useful to retain as a separate hypothesis.

*The canopy/subcanopy proportions hypothesis*.—One forest could have a higher mortality rate than another because it has a greater proportion of trees belonging to subcanopy species, which may have high mortality rates due to a local reproduction–persistence trade-off. The

proportion of forest trees belonging to subcanopy species roughly parallels global patterns of forest NPP; it increases strongly toward the equator (Niklas et al. 2003, King et al. 2006*c*), and within a latitudinal zone often increases with increasing precipitation and soil fertility (Gentry and Emmons 1987, Givnish 1999, Pitman et al. 2002, but see LaFrankie et al. 2006). The greater abundance of subcanopy species at low latitudes ultimately may result from the combined effects of year-round warmth and more vertical sun angles, which may allow the evolution of additional tree strata (Terborgh 1985, King et al. 2006*c*). Similarly, within a latitudinal zone increased moisture or soil fertility might reduce whole-plant light compensation points (thereby increasing shade tolerance) or have other effects that allow greater densities of plants (and species) to persist in the understory (Givnish 1999, Pitman et al. 2002, Coomes et al. 2009).

The remaining five hypotheses are related to differences between forest communities in LH-group-specific mortality rates. The first two concern broad-scale gradients of direct environmental influences on ecological interactions; the final three are related to broad-scale gradients of environmental selection for species exhibiting certain life-history traits.

*The competition hypothesis*.—Mortality rates within any given species or LH group might be influenced by the rate at which individuals die from the effects of competition, which in turn may be positively correlated with resource availability. In a self-thinning forest patch, additional resources usually increase the growth rate of the largest (dominant) trees. In turn, these large, rapidly growing trees suppress more trees more quickly than they would in a resource-poor environment, through asymmetric competition for "preemptable" resources such as light (Weiner 1990, Keddy et al. 1997, Schwinning and Weiner 1998, Bauer et al. 2004). (We will refer to this process as "enhanced" asymmetric competition relative to that in a resource-poor forest.) The net effect is a higher mortality rate (concentrated in the suppressed trees) and more rapid stand development, a phenomenon called the Sukatschew effect (Harper 1977:176; also see Weiner 1985, Clark 1990, Turnblom and Burk 2000). Since old forests near dynamic equilibrium comprise a mosaic of patches of all ages, many (or most) of which are at some stage of self-thinning (Coomes and Allen 2007), the forest as a whole will express a higher mortality rate.

*The enemies hypothesis*.—Mortality rates within a given species or LH group ultimately might be influenced by the rate and severity of attack by plant enemies, which in turn may be highest in climatic zones that favor high NPP. For example, the warm, moist, aseasonal environments that best favor rapid tree growth may also favor the herbivores, pathogens, and agents of decay that attack trees (Givnish 1999, Gilbert 2005, Frazier et al. 2006, Schemske et al. 2009). High attack rates could lead to higher mortality rates in many (or all) species or LH groups, either by killing trees directly or by making them more vulnerable to other causes of death, such as rot fungi making trees more vulnerable to windthrow (Franklin et al. 1987). (Higher attack rates could also select for increased tree defenses, a possibility considered under *Distinguishing among the hypotheses*.)

*The growth–defense hypothesis*.—At broad spatial scales, resource-rich environments might select for suites of species that, independent of their shade tolerance and adult stature relative to sympatrics (i.e., their local LH-group membership), sacrifice defenses in favor of the competitive advantage conferred by rapid growth (Coley et al. 1985, Grime 2001, Stamp 2003, King et al. 2006*b*, Coomes et al. 2009, Endara and Coley 2011). Thus, compared to forest communities with low resource availability, those with high availability may be more heavily dominated, within one or more LH groups, by species that both grow more rapidly and have higher mortality rates.

*The growth–hydraulic-safety hypothesis*.—Resource-rich environments might select for species that, independent of their local LH-group membership, sacrifice resistance to hydraulic failure in favor of the competitive advantage conferred by rapid growth (cf. Hacke and Sperry 2001, Maherali et al. 2004, Sperry et al. 2008). Thus, compared to forests with low resource availability, those with high availability may be more heavily dominated, within one or more LH groups, by species that both grow more rapidly and have higher mortality rates.

*The reproduction–persistence hypothesis*.—At broad spatial scales, certain environments (including productive environments; cf. Moles et al. 2009*b*) may select for suites of species that, independent of their local LH-group membership, sacrifice growth (hence competitive ability) or defenses in favor of reproduction (cf. Obeso 2002). Thus, compared to forests with low resource availability, those with high availability may be more heavily dominated, within one or more LH groups, by species with higher mortality rates.

We have not included a hypothesis related to the possibility that trees senesce more rapidly in warmer or more productive environments (cf. Brown et al. 2004, McCoy and Gillooly 2008). We are unaware of any convincing evidence that senescence, the endogenous degenerative processes that can lead to death (Noodén and Leopold 1988), is a common phenomenon in trees, and a growing body of evidence suggests it is not (e.g., Loehle 1988, Mencuccini et al. 2007, Munné-Bosch 2008, Peñuelas and Munné-Bosch 2010; but see Issartel and Coiffard 2011). Of course, trees do age, suffering cumulative exogenous damage that may make them more susceptible to death, but such damage is not caused by age-related changes in endogenous metabolic function (senescence). Even if trees did senesce, senescence would be a minor contributor to community-wide mortality rates in forests. Globally, most trees in

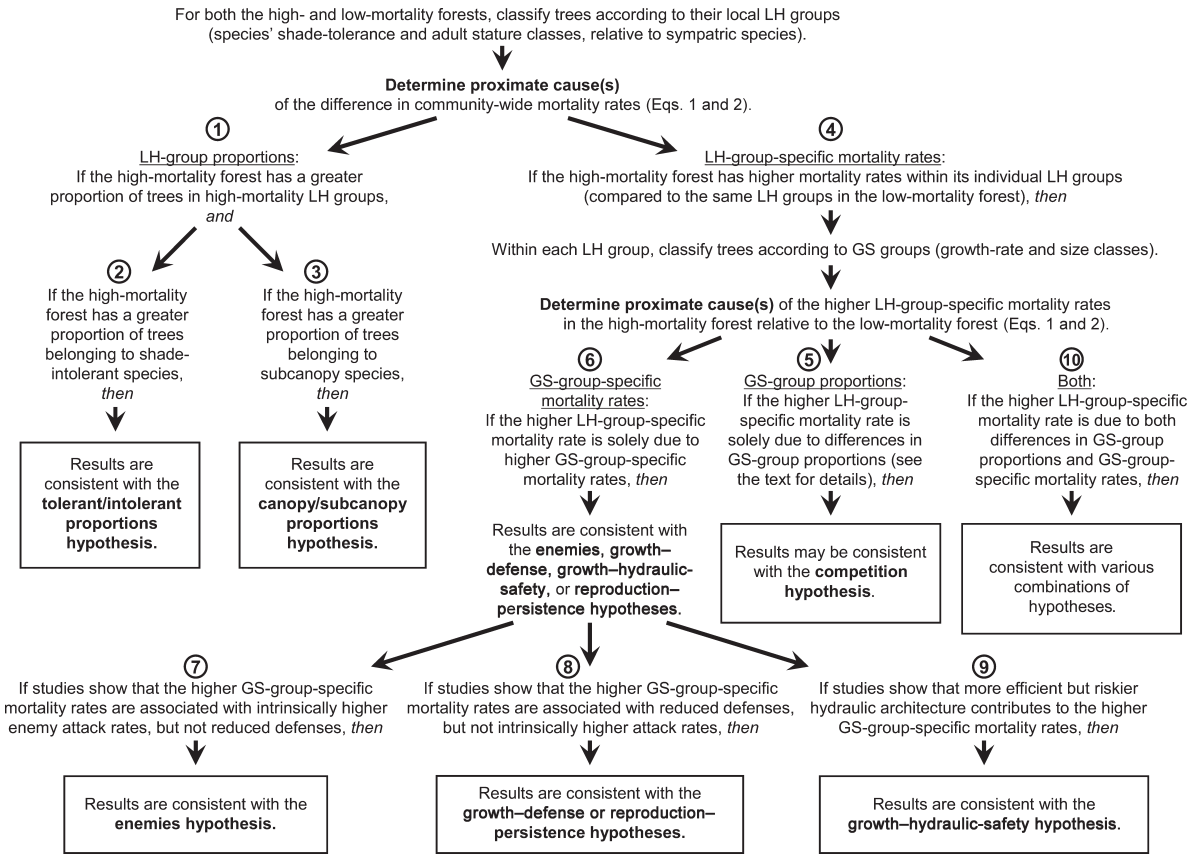


FIG. 2. Approach to distinguishing among the hypothesized ultimate causes. Arrows indicate paths of analysis and interpretation; boxes represent conclusions that are best supported by the indicated paths. Circled numerals are referenced in *Distinguishing among the hypotheses* and *Ultimate causes of BCI's higher mortality rate*. Key to abbreviations: LH, life history; GS, growth rate and size.

unmanaged forests are small and therefore young (e.g., Enquist and Niklas 2001), and nearly always have higher mortality rates than the relatively rare large, old trees (e.g., Condit et al. 1999, Coomes et al. 2003, Muller-Landau et al. 2006*a*). Young trees therefore strongly dominate in determining the overall mortality rates of forests (e.g., see the *Case study*).

### Distinguishing among the Hypotheses

Building from the preceding section, we developed an approach to help distinguish among the hypotheses (Fig. 2). The approach uses proximate causes of differences in mortality rates between forest communities, determined from data collected in long-term forest plots, to help identify probable ultimate causes. The following paragraphs outline the approach and its underlying logic and assumptions; our case study then offers a concrete example of its application.

We emphasize a point that is critical to understanding our approach: life-history (LH) groups are defined locally, relative to sympatric species. Thus, a species' classification as shade tolerant or intolerant and canopy or subcanopy is determined relative to other species within a particular forest community that is being compared to another forest community, but not on an absolute scale that includes species from different forest communities.

To begin, species within the two forests being compared (a high-mortality and a low-mortality forest) are assigned to LH groups defined by combinations of the two local life-history axes (shade tolerance and adult stature relative to sympatrics): i.e., shade-tolerant canopy species, shade-intolerant canopy species, shade-tolerant subcanopy species, and shade-intolerant subcanopy species. Eqs. 1 and 2 are then used to determine how much of the difference in overall mortality rates between the forest communities can be attributed to differences in LH-group proportions (associated with the first two hypothesized ultimate causes), and how much to differences in LH-group-specific mortality rates (associated with the last five hypothesized ultimate causes). If a portion of the high-mortality forest's higher mortality rate can be attributed to differences in LH-group proportions (location 1 in Fig. 2), the data are examined to determine whether this a consequence of higher proportions of shade-intolerant species (support-

ing the tolerant/intolerant proportions hypothesis), subcanopy species (supporting the canopy/subcanopy proportions hypothesis), or both (locations 2 and 3 in Fig. 2).

If part of the difference in overall mortality rates between the two forests can be attributed to differences between the forests in LH-group-specific mortality rates, further analyses are needed (location 4 in Fig. 2). For each LH group in each forest, trees are assigned to GS groups (e.g., small slowly growing trees, large slowly growing trees, and so on). Eqs. 1 and 2 are then used to determine how much of the difference in LH-group-specific mortality rates between the two forests can be attributed to differences in GS-group proportions, and how much to differences in GS-group-specific mortality rates.

If higher LH-group-specific mortality rates are entirely due to differences in GS-group proportions, results are inconsistent with the enemies, growth–defense, growth–hydraulic-safety, and reproduction–persistence hypotheses, but may be consistent with the competition hypothesis (location 5 in Fig. 2). The competition hypothesis is unique in that it neither proposes nor requires a mechanism by which GS-group-specific mortality rates would differ between the two forests. Instead, abundant resources allow a subset of trees to grow more rapidly than they would in a resource-poor environment, and thus to more quickly and effectively suppress other trees through enhanced asymmetric competition (see *Hypothesized ultimate causes*). During any given time period, a larger proportion of trees therefore suffers the elevated mortality associated with slow growth, leading to a higher mortality rate for the LH group as a whole. Thus, the specific expectation for the competition hypothesis (which we have confirmed with an individual-based forest model; N. L. Stephenson, P. J. van Mantgem, A. G. Bunn, H. Bruner, M. E. Harmon, K. B. O'Connell, D. L. Urban, and J. F. Franklin, *unpublished manuscript*) is a greater proportion of both slowly and rapidly growing trees in the resource-rich environment, both at the expense of trees with intermediate growth rates, with the more abundant slowly growing trees dominating in determining the higher community-wide mortality rate.

In contrast, if higher LH-group-specific mortality rates are entirely due to higher GS-group-specific mortality rates, results are inconsistent with the competition hypothesis, and consistent with the enemies, growth–defense, growth–hydraulic-safety, and reproduction–persistence hypotheses (location 6 in Fig. 2). In these last four hypotheses, trees of most growth rates and sizes are likely to suffer higher mortality rates in resource-rich than in resource-poor environments, either due to environmental favorability to plant enemies (the enemies hypothesis), reduced defenses or structural integrity (the growth–defense and reproduction–persistence hypotheses), or reduced hydraulic safety (the growth–hydraulic-safety hypothesis). The conditions hypothesized in this paragraph (i.e., that none of the higher LH-group-specific mortality rate can be attributed to differences in GS-group proportions) have an important additional implication: in the resource-rich environment, higher GS-group-specific mortality rates may be sufficient to counteract any potential for a resource-induced enhancement of asymmetric competition (see *Discussion*). That is, the high-mortality forest may have a smaller proportion of slowly growing trees, which would tend to reduce community-wide mortality rate, but this shift in proportions is not great enough to compensate for the increase in GS-group-specific mortality rates.

Evidence from other studies must be brought to bear to distinguish among the four hypotheses that are associated with higher GS-group-specific mortality rates. Studies of plant defenses and attack rates by plant enemies can help distinguish between the enemies hypothesis and the growth–defense and reproduction–persistence hypotheses (locations 7 and 8 in Fig. 2). If such studies show that higher mortality rates are associated with intrinsically high attack rates by plant enemies, but not reduced defenses, results are consistent with the enemies hypothesis. If studies show the opposite, results are consistent with the growth–defense and reproduction–persistence hypotheses. (Distinguishing between the last two hypotheses, in turn, would require additional field studies of growth rates and reproductive effort.) If studies show that higher GS-group-specific mortality rates are a consequence both of reduced defenses and intrinsically high attack rates (not shown in Fig. 2), results would be consistent with the enemies hypothesis acting in concert with the growth–defense or reproduction–persistence hypotheses.

The growth–hydraulic-safety hypothesis is not mutually exclusive of the other three hypotheses that are associated with higher GS-group-specific mortality rates. Evidence would be consistent with the hypothesis if studies demonstrated that (1) trees in the resource-rich environment have more efficient hydraulic architectures, and (2) under climatic conditions typical of the resource-rich environment, those more efficient hydraulic architectures contribute to higher mortality rates (location 9 in Fig. 2).

Finally, higher LH-group-specific mortality rates could be a consequence both of differences in GS-group proportions (e.g., a greater proportion of slowly growing, suppressed trees) and of higher GS-group-specific mortality rates (location 10 in Fig. 2). This outcome is consistent with the reproduction–persistence hypothesis (which postulates reduced allocation of resources to both growth and defenses), or the competition hypothesis working in concert with the enemies, growth–defense, growth–hydraulic-safety, or reproduction–persistence hypotheses. The last possibilities imply that the increases in GS-group-specific mortality rates, whatever their cause, are not sufficient

to counteract a resource-induced enhancement of asymmetric competition. Distinguishing among the possibilities would likely require a series of targeted studies.

If the resource-rich environment associated with high LH-group-specific mortality rates (location 4 in Fig. 2) is climatically more favorable to the growth and reproduction of plant enemies, added pressure from those enemies might select for increased tree defenses. If selection for increased defenses were great enough to counteract the increased mortality rates otherwise expected from the five hypotheses, then LH-group-specific mortality rates in the resource-rich environment would not be higher than those in the resource-poor environment, and differences in LH-group-specific mortality rates would not have been identified as a cause of differences in mortality rates between the forest communities in the first place. However, if increased defenses only partly counteracted the increase in mortality rates expected from the five hypotheses, the signatures of those hypotheses should still be evident as outlined in Fig. 2. Thus, our approach to distinguishing among the hypotheses should be robust in the face of selection favoring increased defenses in resource-rich environments.

## Case Study

### *Site selection and characteristics of the forests*

We required that sites for our case study met the following criteria. First, the forests being compared exhibited a large and persistent difference in mortality rates, and the difference appeared to be intrinsic rather than the result of unusual disturbance, introduced pathogens, etc. Second, to reduce possible confounding effects of stand development and succession, we compared only old forests. Third, to have a large enough sample to meaningfully compare various LH and GS groups, each sample included tens of thousands of trees. Fourth, to calculate growth rates and track subsequent mortality, each forest included at least three complete censuses. Fifth, we sought intervals between censuses of about five years: long enough to calculate growth and mortality with reasonable precision, but short enough to minimize problems such as bias in mortality rate calculation (e.g., Sheil 1995). Finally, the large majority of trees belonged to species already classified according to shade tolerance.

Data sets meeting all these criteria simultaneously—particularly with large and persistent differences in mortality rates, very large sample sizes, and accompanied by robust life-history information—are currently quite rare (a situation that is likely to change within the next decade; see *Summary and conclusions*). Only two well-qualified data sets were available to us (Appendix B). Tropical data came from the 50-ha moist forest plot on Barro Colorado Island, Panama (BCI; latitude 9° N), described in Leigh et al. (2004). Temperate data came from the pooled data of 65 plots totaling 58.1 ha in California, Oregon, and Washington, USA (latitudes 36°–48° N), hereafter referred to as "Pacific States" (Acker et al. 1998, Stephenson and van Mantgem 2005). While a comparison of data from one large tropical plot with pooled data from many small temperate plots is not ideal, all qualifying temperate plots were one to two orders of magnitude smaller than the BCI plot, requiring pooling to accumulate the tens of thousands of trees needed for analysis. (Conversely, otherwise qualifying tropical data sets comprising many small plots lack the rich depth of relevant background information and life-history data associated with the BCI plot.) In spite of the wide range of latitudes and elevations spanned by the Pacific States plots (Appendix B), individually those plots' mortality rates were low compared to that of BCI (cf. Stephenson and van Mantgem 2005, van Mantgem et al. 2009), and their combined data were dominated by only six species.

The BCI plot contains >300 free-standing woody species with dbh ≥1 cm (Condit et al. 1996*b*). BCI's climate, soils, forest structure, dynamics, species diversity, and floristics are not exceptional among tropical forests (Leigh 1999, Losos and Leigh 2004, Muller-Landau et al. 2006*a*, *b*). BCI has a dry season of about four months; normally about 10% of the canopy is deciduous at peak leaf loss. Over a period of a few decades, annual community-wide mortality ranged from 1.7% to 2.8% (depending in part on minimum dbh analyzed [Putz and Milton 1996, Condit et al. 1999]; e.g., E. Leigh, *personal communication*), averaging roughly 2.1%: somewhat higher than the global average of 1.7% for tropical forests, but well within the range of typical values (data from Stephenson and van Mantgem 2005). Sheil and Burslem (2003) argued that the BCI plot may still be recovering from centuries-old (or even more recent) disturbances; however, it probably has not experienced broad-scale, stand-replacing disturbance in >1000 years (Leigh et al. 2004). Muller-Landau et al. (2006*b*) found that the size structure of the BCI forest is close to dynamic equilibrium. Estimated aboveground NPP at BCI is 18 $Mg \cdot ha^{-1} \cdot yr^{-1}$ (Chave et al. 2003).

The Pacific States plots are dominated by evergreen conifers (Appendix B), with six species comprising 77% of trees (nomenclature follows Hickman 1993): *Tsuga heterophylla* (western hemlock; 22%), *Abies concolor* (white fir; 16%), *A. amabilis* (Pacific silver fir; 12%), *A. magnifica* (red fir; 10%), *Pseudotsuga menziesii* (Douglas fir; 9%), and *Calocedrus decurrens* (incense cedar; 8%). None of the remaining 27 species comprises >4% of trees, and the 11 angiosperm species collectively account for only 5%. The plots experience warm, dry summers and cold, wet winters typically dominated by snow. Climate, soils, and forest structure are described elsewhere (Franklin and Dyrness 1973, Barbour and Major 1977, Waring and Franklin 1979). The plots have not experienced stand-replacing disturbance in at least 200 years, and usually much longer (estimated by counting rings on increment cores or nearby stumps,

or by historical records and the sizes of the largest trees). Additionally, we excluded plots experiencing fire, avalanche, or major flood during their measurement periods. From 1972 through 2004, annual mortality rate averaged across the plots (excluding trees <5 cm in diameter) ranged from 0.4% to 1.5%, with a mean of 1.0% for the entire period: somewhat lower than the global average of 1.2% for all temperate forest types combined, and somewhat higher than the average of 0.7% for temperate gymnosperm forests (data from Stephenson and van Mantgem 2005). Although mortality rates in the Pacific States have increased through time (perhaps due to regional warming; van Mantgem et al. 2009), values have remained below those at BCI. Estimated aboveground NPP in old forests of the Pacific States (~5–13 $Mg \cdot ha^{-1} \cdot yr^{-1}$ [data from Harmon et al. 2004, Van Tuyl et al. 2005, Hudiburg et al. 2009]) is lower than that at BCI.

### *Data and analysis*

For all plots, we used the three most recent censuses available at the time of our analyses, referred to as censuses 1, 2, and 3 (Appendix B). Using standard approaches, data from censuses 1 and 2 were used to calculate tree growth rates, and data from censuses 2 and 3 to calculate subsequent mortality rates (Appendix C). We defined three diameter growth-rate classes (−2 to <2, 2 to <6, and 6 to 40 mm/yr) and three size classes (5 to <15, 15 to <50, and ≥50 cm dbh), for a total of nine GS groups (Appendix C).

To provide a simple definition of canopy and subcanopy species for both forest types, we defined subcanopy species as those that had no individual ≥50 cm dbh at census 2. This gave a classification of BCI species comparable to a separate classification based on maximum adult tree height (Welden et al. 1991), with only 5% of trees being classified differently by the two approaches.

For BCI, we used Condit et al.'s (1995; see Welden et al. 1991) "colonizing index" as a measure of shade tolerance relative to sympatrics; shade-intolerant species were defined as those with ≥30% of recruitment found in forest gaps. The index is strongly correlated with species' mean growth and mortality rates (Condit et al. 1996*a*), traits that commonly covary along the shade-tolerance continuum. We dropped from analysis the 9% of BCI trees belonging to rare species for which no shade-tolerance classification was available. Overall mortality rate and canopy/subcanopy proportions of these unclassified trees did not differ greatly from those of the remaining, classified trees (Appendix C), suggesting that unclassified trees did not represent a strongly demographically biased subset of BCI trees.

For the Pacific States, shade-intolerant species were those classified by Burns and Honkala (1990) as "intolerant" and "very intolerant" of shade; the remaining species were classified as shade tolerant. (For four minor species not classified by Burns and Honkala, we referred to Sudworth [1967].) Thus the Pacific States tolerance classifications ultimately were based on a number of experimental studies by different investigators, and consensus expert opinion. Although the approaches used to define shade tolerance relative to sympatrics differed between BCI and the Pacific States, the difference should not greatly affect our case study. As is typical in old forests globally, at both BCI and the Pacific States trees belonging to shade-intolerant species have low relative abundances (<10%) and thus relatively small effect on community-level mortality rates in either forest.

We used Eqs. 1 and 2 to determine relative contributions of differences in LH-group proportions and LH-group-specific mortality rates to the difference in mortality rates between the forests (Appendix A), and to determine the relative contributions of differences in GS-group proportions and GS-group-specific mortality rates to differences in LH-group-specific mortality rates. Significance of differences in proportions of trees belonging to different groups were calculated using Fisher's exact test for the contingency table of numbers of trees alive at census 2, by forest type (BCI or Pacific States). Significance of differences in mortality rates were calculated using Fisher's exact test for the contingency table of numbers of trees by survival status at census 3 (alive or dead) and forest type.

### *Proximate causes of BCI's higher mortality rate*

*Differences in LH-group proportions and LH-group-specific mortality rates.*—The two forests differed strongly in their LH-group proportions (Fig. 3). While the vast bulk of Pacific States trees belonged to shade-tolerant canopy species (94%), only one third of BCI trees did. Most of the remaining BCI trees belonged to shade-tolerant subcanopy species (57%), a group that was virtually absent from the Pacific States (1%). Trees belonging to shade-intolerant species were relatively minor components of both forests; however, by our classifications they were slightly more abundant at BCI than the Pacific States (9% and 6%, respectively).

Among LH groups at BCI, trees belonging to shade-tolerant canopy species had the lowest mortality rate, those belonging to shade-intolerant canopy and shade-tolerant subcanopy species were intermediate (and statistically indistinguishable by Fisher's exact test; $P = 0.13$), and those belonging to shade-intolerant subcanopy species had the highest mortality rate (Fig. 3). In contrast, in the Pacific States, mortality rates of trees belonging to shade-tolerant and shade-intolerant canopy species showed no detectable difference ($P = 0.29$), though both groups had significantly lower mortality rates than Pacific States trees belonging to shade-tolerant subcanopy species ($P < 0.001$). No Pacific States comparison of mortality rates that included shade-intolerant subcanopy species was significant, almost certainly due to the extremely small sample of trees in this LH group (Table 1).

Trees belonging to canopy species, both shade tolerant and intolerant, had higher mortality rates at BCI than the Pacific States ($P < 0.0001$; Fig. 3). In contrast, no significant difference was found in mortality rates of trees belonging to subcanopy species (tolerant or intolerant) between the forests ($P \geq 0.10$), probably due to small Pacific States samples (Table 1).

BCI's community-wide mortality rate was twice that of the Pacific States (2.22 and 1.10% per yr, respectively; $P < 0.0001$). BCI's higher mortality rate could be attributed both to differences in LH-group proportions (mostly due to BCI's much greater relative abundance of trees belonging to subcanopy species) and BCI's higher LH-group-specific mortality rates (at least in canopy species, which had large enough samples to meaningfully compare BCI and the Pacific States); each accounted for roughly one half of BCI's higher mortality rate (54% and 46%, respectively). However, in light of the broad confidence intervals on mortality rates for Pacific States subcanopy species, precise values of the relative contributions should be viewed with caution.

*Causes of differences in LH-group-specific mortality rates*.—Because LH-group-specific mortality rates of subcanopy species did not differ significantly between the two forests (almost certainly due to the small sample size for trees belonging to subcanopy species in the Pacific States), we determined proximate causes of the differences in LH-group-specific mortality rates only for canopy species. For both the between-forest comparisons of shade-tolerant canopy species and of shade-intolerant canopy species, the higher LH-group-specific mortality rates at BCI were entirely a consequence of higher GS-group-specific mortality rates (Table 2). Differences in GS-group proportions between the forests acted to diminish, not enhance, differences in LH-group-specific mortality rates between the forests (Table 2). Within each of the two canopy LH groups, BCI had significantly smaller proportions of slowly growing and greater proportions of rapidly growing trees than the Pacific States ($P \leq 0.0002$; Table 2).

For either shade-tolerant or shade-intolerant canopy species, mortality rate in any given GS group was always higher at BCI than in the Pacific States (Figs. 4 and 5). The probability that this is a chance occurrence (i.e., mortality rates in one forest type coincidentally exceeds those of the other in all 18 GS-group comparisons) is quite small: $P = (1/2)^{17} = 0.000008$. Individually, seven of the nine GS-group comparisons for shade-tolerants were significant by Fisher's exact test ($P < 0.05$); four were significant for shade-intolerants (Figs. 4 and 5).

### *Ultimate causes of BCI's higher mortality rate*

*Major contribution from canopy/subcanopy proportions*.—BCI's much greater relative abundance of trees belonging to subcanopy species (Fig. 3) may ultimately be a consequence of its more productive environment (see *Hypothesized ultimate causes*), and accounted for about one half of its 1.1% per yr higher community-wide mortality rate. BCI's subcanopy species have a higher overall mortality rate than its canopy species for two proximate reasons: they have proportionally more slowly growing individuals (which experience elevated mortality rates relative to faster-growing trees), and especially because they have higher mortality rates within all growth-rate and size classes of trees (data not shown). The former is consistent with a diversion of resources from growth toward reproduction (Silvertown

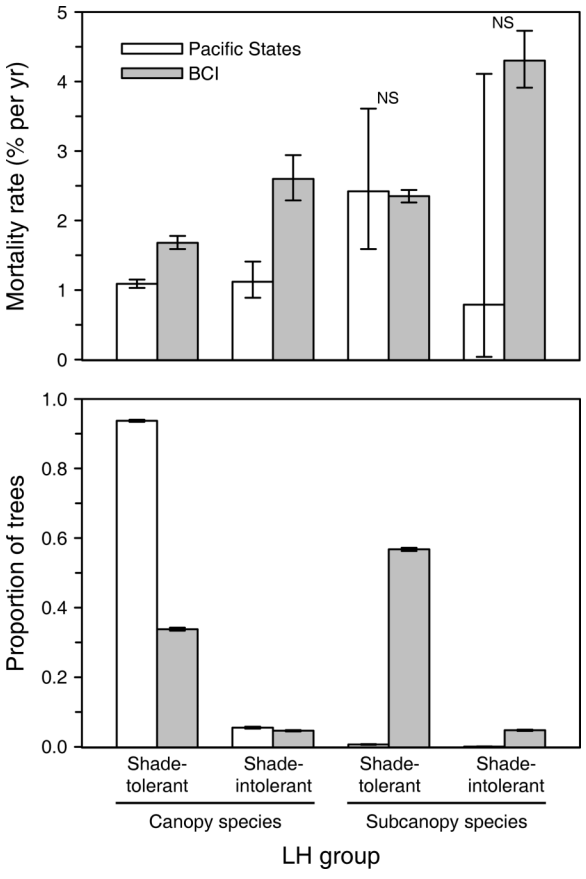


FIG. 3. Mortality rates and proportions of trees in each of the four combinations of life-history (LH) groups at the temperate Pacific States (white bars) and tropical BCI (gray bars). For mortality rates, the binomial 95% confidence intervals are based on number of dead trees at census 3 and number of living trees at census 2 for that particular LH group. Mortality rates differ significantly between California, Oregon, and Washington, USA (Pacific States) and Barro Colorado Island, Panama (BCI) for both shade-tolerant and shade-intolerant canopy species ($P < 0.0001$, Fisher's exact test), but not for shade-tolerant and shade-intolerant subcanopy species ($P > 0.05$, indicated by "NS"). The broad confidence intervals on mortality rates for Pacific States subcanopy species is a consequence of small sample sizes (Table 1). For proportions, binomial confidence intervals are based on number of living trees in the LH group and total number of living trees in the particular forest, both at census 2. Total numbers of living trees in the forests are so large that confidence intervals for proportions are vanishingly small, and proportions differ significantly for each pair of bars ($P < 0.0001$, Fisher's exact test).

TABLE 1. Numbers of living trees, dead trees, and species used in the case study, by life-history group and forest.

| Life-history group | Number of living trees at census 2 | | Number of dead trees at census 3 | | Number of species | |
|---|---|---|---|---|---|---|
| | BCI | PS | BCI | PS | BCI | PS |
| Shade-tolerant canopy | 14 915 | 23 859 | 1164 | 1392 | 17 | 18 |
| Shade-intolerant canopy | 2047 | 1405 | 243 | 72 | 26 | 7 |
| Shade-tolerant subcanopy | 25 037 | 175 | 2699 | 22 | 86 | 7 |
| Shade-intolerant subcanopy | 2107 | 22 | 401 | 1 | 20 | 1 |
| Total | 44 106 | 25 461 | 4507 | 1487 | 149 | 33 |

*Note:* Key to abbreviations: BCI, Barro Colorado Island, Panama; PS, Pacific States (California, Oregon, and Washington, USA).

and Dodd 1999, Turner 2001, Kohyama et al. 2003), thereby slowing growth relative to canopy species of the same size (which may delay reproduction until they reach canopy height). The latter may result from an additional diversion of resources, from defenses toward reproduction (Loehle 2000, Obeso 2002).

*Minor contribution from tolerant/intolerant proportions*.—Mortality rates of large trees are higher at BCI than in the Pacific States (Figs. 4 and 5). All else being equal, this should lead to a higher gap formation rate at BCI, providing more opportunities for recruitment of trees belonging to shade-intolerant species. However, the density of large trees ($\geq$50 cm dbh) at BCI is only one fourth that in the Pacific States (see *Discussion*), meaning fewer large trees are available to fall and create gaps. The nominal net effect of these opposing patterns was that BCI had a somewhat greater proportion of trees belonging to shade-intolerant species than the Pacific States (Fig. 3), though the difference could be an artifact of the different local shade-tolerance classifications used in the two forests. Regardless, because shade-intolerants comprise relatively small proportions of trees in either forest, the higher proportion at BCI contributes only slightly to the higher community-wide mortality rate there.

While BCI trees exhibited the expected pattern of higher mortality rates among shade-intolerant than shade-tolerant species, the difference was negligible (and statistically insignificant) in the Pacific States. This unexpected outcome might have resulted either from small sample sizes or from the origin of the Pacific States data in numerous widely distributed plots. Regarding the latter, several of the shade-intolerant species were of the genus *Pinus*, which, compared to most other Pacific States species, is more often found on low-productivity sites (e.g., Stephenson 1998). Trees on low-productivity sites generally have lower mortality rates than those on high-productivity sites (Stephenson and van Mantgem 2005), potentially obscuring any difference in mortality rates between shade intolerants and tolerants when data from all plots were combined.

*No apparent contribution from competition*.—Of the five hypotheses potentially explaining higher LH-group-specific mortality rates at BCI (location 4 in Fig. 2), we can with reasonable confidence eliminate the competition hypothesis. The competition hypothesis is unique in that higher LH-group-specific mortality rates are solely a consequence of increased proportions of slowly growing (suppressed) trees; they are not even partly the result of higher GS-group-specific mortality rates (see *Background and theory*). However, our comparisons clearly showed the opposite pattern (Table 2). Even when the entire population of BCI trees (including all subcanopy species, with their high proportion of slowly growing trees) was compared to the entire population of Pacific States trees, BCI's higher community-wide

TABLE 2. Between-forest comparisons of comparable life-history (LH) groups.

| Comparison | Absolute difference in mortality rates | Proximate causes | | Differences in proportions of trees that are: | |
|---|---|---|---|---|---|
| | | Proportions (%) | Mortality (%) | Growing slowly (%) | Growing rapidly (%) |
| BCI tolerant canopy (1.68% per year) vs. PS tolerant canopy (1.18% per year) | +0.50% per year | −28 | +128 | −4 | +142 |
| BCI intolerant canopy (2.60% per year) vs. PS intolerant canopy (1.32% per year) | +1.28% per year | −19 | +119 | −26 | +260 |

*Notes:* Definitions: Growing slowly, diameter growth rate of −2 to 2 mm/yr; growing rapidly, 6 to 40 mm/yr. Values in the columns under "Proximate causes" show the percentage of the absolute difference in LH-group-specific mortality rates between the two forests (preceding column) that can be attributed to differences, respectively, in GS-group proportions and GS-group-specific mortality rates. Values in the columns under "Differences in proportions of trees that are:" show the relative percentage difference in proportions of trees in the indicated growth-rate classes at BCI relative to the Pacific States; the differences expressed in these columns are statistically significant (Fisher's exact test, $P < 0.0002$). Absolute differences in LH-group-specific mortality rates differ somewhat from those shown in Fig. 3 because trees from Oregon and Washington that were <5 cm dbh at census 1 could not be included (Appendix C). Both of the absolute differences between forests in LH-group-specific mortality rates were statistically significant ($P < 0.0001$).

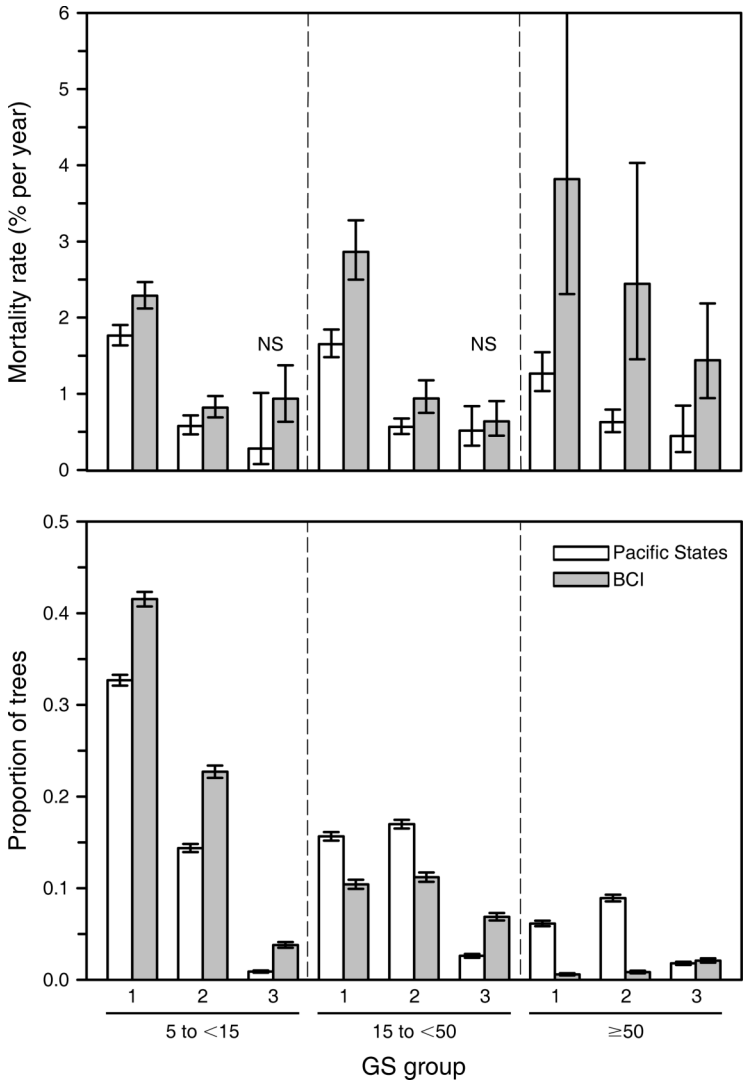


FIG. 4. Mortality rates and proportions of trees belonging to shade-tolerant canopy species, by growth-rate and size classes (GS groups), at the Pacific States (white bars) and BCI (gray bars). Each of the nine GS groups is defined by size class (5 to <15, 15 to <50, and ≥50 cm in diameter at breast height) and growth rate (1, 2, and 3 indicating, respectively, diameter growth rates of −2 to <2, 2 to <6, and 6 to 40 mm/yr). Binomial 95% confidence intervals are as in Fig. 3. For any given GS group, mortality rate is higher at BCI than in the Pacific States; the difference is significant for seven of the nine pairwise comparisons ($P < 0.05$, Fisher's exact test; "NS" indicates the nonsignificant comparisons). Proportions differ significantly for all GS groups (lower panel).

mortality rate remained entirely a consequence of higher GS-group-specific mortality rates, not differences in GS-group proportions. Conversely, in the Pacific States, proportionally more trees suffer the elevated probability of mortality associated with slow growth than at BCI. An interesting corollary is that, at least for the tree size classes we examined (≥5 cm in diameter), competition may be a relatively more important source of tree mortality in the Pacific States than at BCI, contrary to some theoretical expectations (cf. Goldberg et al. 1999, Grime 2001).

*Apparently major contribution from enemies and little or none from broad-scale growth–defense or reproduction–persistence trade-offs, but evidence is limited.*—Three of the remaining hypotheses invoke two distinct mechanisms leading to increased GS-group-specific mortality rates (locations 7 and 8 in Fig. 2): increased pressure by herbivores, pathogens, and agents of decay (the enemies hypothesis), or decreased defenses or structural integrity (the growth–defense and reproduction–persistence hypotheses). We first examine enemies, then defenses.

During typical conditions in old forests of the Pacific States, leaf area loss to folivory is quite low, averaging ~0–2% among gymnosperm species (Schowalter 1989, 1995, Shaw et al. 2006). Within the California subset of our Pacific States plots (in which trained field personnel have taken detailed notes on the pathology of each tree

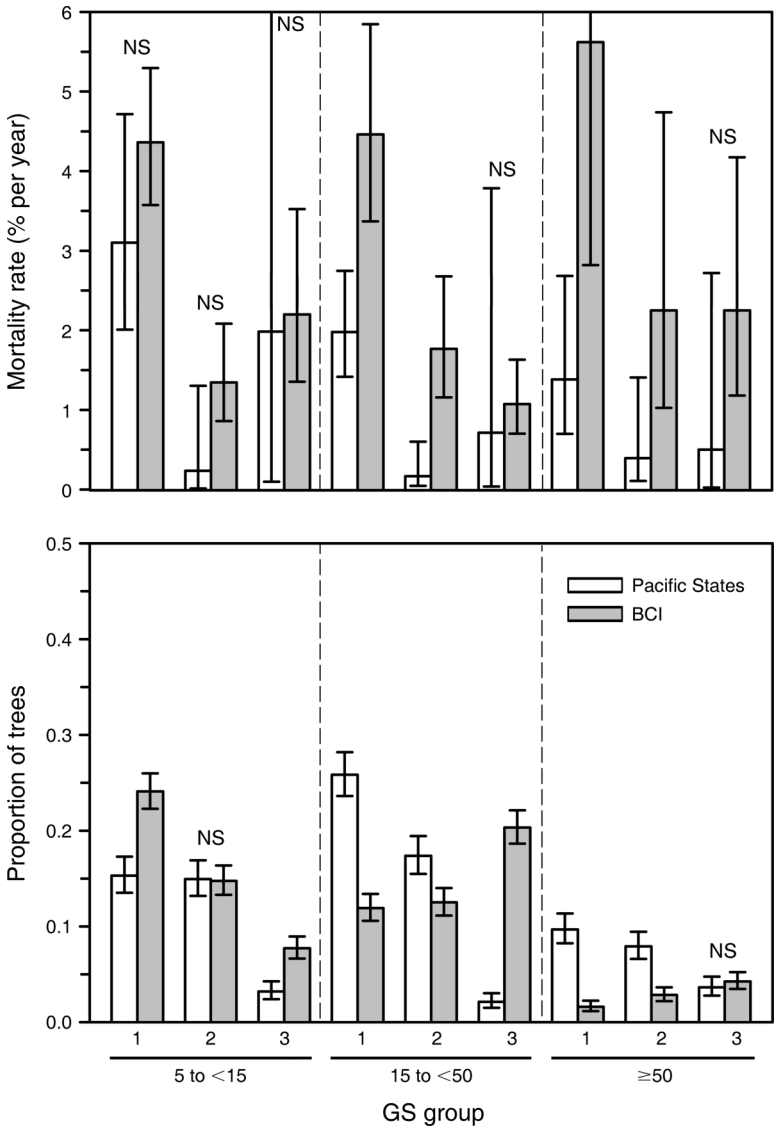


FIG. 5. Mortality rates and proportions of trees belonging to shade-intolerant canopy species, by growth-rate and size classes (GS groups), at the Pacific States (white bars) and BCI (gray bars). Axes and binomial 95% confidence intervals are as in Fig. 4. For any given GS group, mortality rate is higher at BCI than in the Pacific States; the difference is significant for four of the nine comparisons (at $P < 0.05$, Fisher's exact test; "NS" indicates the nonsignificant comparisons). Proportions differ significantly for all but two GS groups (lower panel).

for 13 consecutive years), only 0.4% of tree mortality has been attributed directly to defoliation. Folivory, usually of limited extent and occurring one to several years before tree death, was recorded as a possible indirect contributing factor in only an additional 2% of tree deaths. Since an individual tree's defoliation often must exceed 30%, an amount easily observed by field personnel, before a noticeable increase in probability of mortality occurs (Wickman 1963, Dobbertin and Brang 2001), it is unlikely that we have underestimated the role of folivory when we conclude that its contribution to tree mortality in our Pacific States plots is, at best, minor.

Average leaf loss to folivory at BCI and vicinity is higher, with estimates ranging from 4% to 21% or more (Coley 1996, Leigh and Windsor 1996, Leigh 1999:166, Van Bael et al. 2004, Van Bael and Brawn 2005, Kalka et al. 2008). At least some of BCI's greater folivory is a consequence of leafcutter ants (Leigh and Windsor 1996, but see Barone 2000), a defoliator not found in the Pacific States. In addition to folivory, pathogens contribute substantially to leaf area loss at BCI (Coley and Barone 1996, Barone 1998), and the photosynthetic capacity of the remaining leaf area can be significantly reduced by epiphylls (mostly lichens) (Coley et al. 1993, Coley and Kursar 1996). (While we lack quantitative

estimates of leaf area affected by pathogens and epiphylls in our Pacific States plots, our observations indicate it is much lower.) Agricultural species host, on average, ten times more diseases when grown in tropical rather than temperate climates (Wellman 1968; see also Gilbert 2005), consistent with the possibility that BCI's high leaf pathogen load is at least partly an intrinsic function of the environment. However, even though high endemic levels of leaf loss at BCI can contribute substantially to seedling mortality (e.g., Howe 1990), effects on the larger trees of our case study remain unclear. For example, during a defoliator outbreak in one of BCI's most abundant canopy species (*Quararibea asterolepis*, comprising 2% of trees in our BCI data set) about half of the trees experienced >30% crown defoliation and 20% experienced >90% defoliation (Wong et al. 1990), yet mortality rate of the species was not discernibly affected in the subsequent census interval (Condit et al. 1995).

Under typical conditions, the overwhelmingly dominant biotic agents of tree mortality in the Pacific States are the insects and pathogens that attack tree boles and roots, not foliage (e.g., Ferrell 1996, Hansen and Goheen 2000, Hawkins and Henkel 2011). For example, bark beetles (subfamily Scolytinae) directly or indirectly contributed to nearly half of all tree mortality in our intensively studied California plots. Fungal pathogens of boles and roots (such as *Armillaria*) were recorded as contributing to 27% of tree deaths. The latter value almost certainly underestimates the actual importance of pathogens; for example, we suspect that many of our trees that died without discernable signs or symptoms nonetheless suffered from root pathogens.

Although we are unaware of comparably intensive community-wide analyses of biotic agents of tree mortality at BCI or in other tropical forests, it is clear that, as in the Pacific States, biotic attacks on boles and roots are an important source of tropical tree mortality. Bark beetles are more diverse and can have broader host ranges in tropical than in temperate forests (Beaver 1979). Tropical bark beetles and other bole-feeding invertebrates (such as wood borers and termites, the latter being particularly abundant in the tropics [Cornwell et al. 2009]) can cause extensive damage and contribute substantially to tropical tree mortality (Bultman and Southwell 1976, Apolinário and Martius 2004, Nair 2007, Werner and Prior 2007). Although fungal and other pathogens of boles and roots have been poorly studied in the tropics (Gilbert 2005, García-Guzmán and Morales 2007), it is evident that they, too, contribute to tree mortality at BCI (e.g., Gilbert et al. 1994, Mangan et al. 2010). In the BCI plot, a survey of 869 randomly selected trees of ten species showed that 7% of living trees had fruiting bodies of wood-decaying polypore fungi on their boles (Gilbert et al. 2002). In contrast, in our intensively studied California plots only 0.3% of living trees showed fruiting bodies of any native fungi. (This contrast would likely have been even more dramatic if, like our California data, the BCI data had included all native fungi, not just polypores.) While these last observations do not prove that a greater proportion of trees suffer pathogenic fungal attack at BCI than in the Pacific States (because not all fungi—polypore or otherwise—are pathogenic, and not all trees infected by fungi show visible fungal fruiting bodies), they are certainly suggestive. Finally, two years after the boles of Bolivian humid forest trees of seven species (including three that produced abundant latex) were experimentally wounded, ~99% of trees showed decay in the wounds (Romero and Bolker 2008). In contrast, decay was found in only one half of bole wounds averaged over six of our Pacific States gymnosperm species (Vasiliauskas 2001), even though the wounds were generally orders of magnitude larger and much older, providing greater opportunities for fungal infection. We therefore expect that bole wounds, a frequent consequence of falling trees and limbs in both tropical and temperate forests, become sites of decay more rapidly and in a greater proportion of wounded trees at BCI than in the Pacific States.

Comparisons of the rates, not just incidence, of wood and root decay provide further insights, because higher decomposition rates (1) may lead to higher tree mortality rates due to accelerated structural weakening and failure (e.g., Loehle 1988, McCarthy 2001, Larson and Franklin 2010), and (2) may more generally reflect environmental favorability to higher attack rates by other plant enemies. For several common gymnosperms of our Pacific States plots, the half-life of dead trees (the time for half of the original bole mass to decay) ranges from 14 to 230 years, averaging >80 years (Harmon et al. 1986). The half-life of dead trees in moist neotropical forests is more than an order of magnitude less, ranging from <1 to 69 years and averaging only four to six years (Chambers et al. 2000, van Geffen et al. 2010, Hérault et al. 2011). At BCI, it is not uncommon for large dead trees to have half-lives of <1.5 years (Lang and Knight 1979), and some large trees die and decompose completely during the five years between censuses (Condit et al. 1995). Similarly, root decomposition rates are much higher in tropical than in temperate latitudes (Silver and Miya 2001). Some of these differences in decomposition rates may be related to differences in wood properties; when exposed to a common environment, gymnosperm wood decomposes at roughly half the rate of angiosperm wood (perhaps due to gymnosperms' lower nutrient and higher lignin contents, differences in fine wood structure, etc. [Cornwell et al. 2009, Weedon et al. 2009]). However, broad-scale studies using standard wood and root substrates still show much more rapid decomposition in tropical than in temperate environments (Wong et al. 2004, Parton et al. 2007, González et al. 2008; M. E. Harmon, B. Fasth, H. Chen, W. J. Parton, J. Sexton, I. C. Burke, W. S. Currie, and Long-term Intersite Decomposition Experiment Team, *unpublished manuscript*), and indicate that

the majority of the order-of-magnitude difference in decomposition rates is a consequence of the environments themselves, not differences in wood or root properties.

Turning to defenses, we note that most studies comparing tropical and temperate defenses have focused on leaves, and their interpretation has been confounded by at least two issues. First, leaves generally conform to a global leaf economics spectrum ranging from short-lived, poorly defended leaves to long-lived, well-defended leaves (Coley et al. 1985, Reich et al. 1997, 1999 Wright et al. 2004). However, leaf lifespan varies by nearly two orders of magnitude within latitudinal zones: much greater than its variation between latitudinal zones (e.g., Wright et al. 2005). Studies that do not control for leaf lifespan therefore risk obscuring any latitudinal variation in defenses with variation due to differences in leaf lifespans. (Indeed, the lower folivory rates observed in the Pacific States might partly be a consequence of greater defenses associated with the longer leaf lifespans of the dominant species there.) A large global study and a global meta-analysis that did not control for leaf lifespans found no clear latitudinal trend in defenses (Moles et al. 2011*a*, *b*), whereas an earlier comparison of leaves of comparable lifespans found that tropical leaves were much better defended than temperate leaves (Coley and Aide 1991). Second, an implicit assumption in many studies is that leaf defenses reflect whole-plant defenses. At least for trees, however, the strength of leaf defenses may have little relation to the strength of bole or root defenses (cf. Chave et al. 2009*a*, Baraloto et al. 2010). Because the biotic agents of tree mortality predominantly appear to be those that attack boles and roots, not leaves (see the preceding paragraphs), we conclude that regardless of the potentially confounding issue of leaf lifespans, our ability to distinguish among the hypotheses is almost certainly better served by examining bole and root defenses (cf. Loehle 1988, Poorter et al. 2008).

However, data allowing comparisons of bole and root defenses between tropical angiosperms and temperate gymnosperms are quite limited (e.g., see Chave et al. 2009*a*, van Dam 2009). On the one hand, bole wood of gymnosperms may be better protected than that of angiosperms by having lower nutrient and higher lignin contents, different lignin chemistry, and a wood structure less favorable to fungal growth (Cornwell et al. 2009, Weedon et al. 2009). Similar trait differences may also occur in roots (e.g., Silver and Miya 2001). Collectively, however, these traits are unlikely to be indicative of broad-scale growth–defense or reproduction–persistence trade-offs (location 8 in Fig. 2); gymnosperm wood is less dense than angiosperm wood and is thus almost certainly cheaper to make per unit volume (Weedon et al. 2009). On the other hand, BCI heartwood may contain higher average concentrations of chemical defenses, as indicated by heartwood color (cf. Chave et al. 2009*a*); red or brown heartwood is more common in BCI taxa (species or genera) than in the Pacific States taxa, and white or gray heartwood is less common (Leavengood 1998, Wheeler et al. 2007 [InsideWood database]). (In fact, the dominant taxa in our Pacific States data disproportionately include those with white or gray heartwood, particularly *Abies*.) At least among the dicotyledonous angiosperms, silica bodies and crystals (such as calcium oxalate) are more common in tropical than in temperate woods (Wheeler et al. 2007), perhaps indicating enhanced defenses in the tropics (cf. Hudgins et al. 2003); however, we are unaware of comparable comparisons that include gymnosperms. Although we are also unaware of quantitative comparisons of resin and latex defenses between tropical and temperate forests, such defenses are common in both (e.g., Guariguata and Gilbert 1996, Turner 2001:73, Franceschi et al. 2005). Finally, in common-environment trials in Puerto Rico, heartwood from 43 angiosperm taxa found at BCI showed no difference in mean resistance to the dry-wood termite *Cryptotermes brevis* compared to heartwood from seven gymnosperm taxa found in the Pacific States ($P = 0.53$, $t$ test; data from Wolcott [1950]), suggesting no net difference in defenses against wood-boring invertebrates.

While much further work is needed, we believe the weight of available evidence favors the enemies hypothesis over the growth–defense and reproduction–persistence hypotheses (locations 7 and 8 in Fig. 2). Rates of biotic attack appear to be intrinsically higher at BCI and other moist tropical forests than in the Pacific States, and those higher attack rates are likely a consequence of the tropical forests' greater favorability to plant enemies (presumably through elevated humidity and year-round warmth; cf. Givnish 1999, Gilbert 2005). While the limited data on latitudinal differences in defenses are equivocal, we find no clear indication that defenses are substantially reduced at BCI compared to the Pacific States. Even if further study were to reveal that tropical angiosperm wood is intrinsically more susceptible to biotic attack than temperate gymnosperm wood (a between-latitude comparison that, to our knowledge, has rarely been made [cf. Wolcott 1950]), the difference (1) would not be enough to account for the much higher attack rates in the tropics (e.g., as indicated by studies that transported standard wood and root substrates between latitudes), and (2) would probably not be indicative of growth–defense or reproduction–persistence trade-offs (since gymnosperm wood is less dense and thus almost certainly cheaper rather than more costly to make per unit volume than angiosperm wood).

*Apparently little or no contribution from growth–hydraulic-safety trade-offs, but evidence is limited.*—To the extent that environments prone to prolonged droughts or freezing temperatures limit potential for productivity, the first of the conditions postulated by the growth–hydraulic-safety hypothesis (i.e., that more productive environments select for more efficient tree

hydraulic architectures, potentially supporting faster tree growth) seems likely to be met. Stressful environments often select for, among other things, species with xylem conduits that are more resistant to drought- or freezing-induced embolism (e.g., Hacke and Sperry 2001, Maherali et al. 2004, Sperry et al. 2008). These safer hydraulic architectures, in turn, sometimes (but not always) limit whole-tree water conductance and therefore potential for growth (Maherali et al. 2004, Sperry et al. 2008, Poorter et al. 2010, Russo et al. 2010). Indeed, estimated whole-tree water conductance in a number of Panamanian angiosperms substantially exceeds that of Pacific States gymnosperms (Meinzer et al. 2005, McCulloh et al. 2010).

However, very few studies have shed light on whether the more efficient hydraulic architectures found in benign environments contribute to higher mortality rates in those environments. Three points are particularly relevant. First, the apparently safer but less efficient hydraulic architectures found in some stressful, unproductive environments do not necessarily mean that trees in those environments experience less hydraulic failure than those in productive environments. For example, twigs of some conifers growing at the cold alpine treeline may regularly experience up to 100% loss of conductivity due to winter embolism, much of which is subsequently repaired (Mayr et al. 2006). Species with the safest hydraulic architecture can experience the greatest, not the least, hydraulic failure during drought because they do a poor job of regulating water loss through stomatal control (Hoffmann et al. 2011). Second, it is not yet known whether, in any forest experiencing the typical (non-extreme) climatic conditions that are the topic of this paper, hydraulic failure is a significant contributor to tree mortality relative to other causes. (Even the relative contribution of hydraulic failure to tree mortality during extreme drought remains unclear [e.g., McDowell et al. 2008, Brodribb and Cochard 2009, Sala et al. 2010, Hoffmann et al. 2011, McDowell 2011].) Trees across a broad range of environments exhibit a variety of mechanisms for avoiding or repairing conduit embolism under conditions typical of those environments (Hacke and Sperry 2001, Johnson et al. 2009, Meinzer et al. 2009). Finally, the only two studies of which we are aware that directly correlated tree mortality rates to aspects of their hydraulic architecture under apparently typical field conditions found no significant correlations between the two (Poorter et al. 2010, Russo et al. 2010). While these two studies offer important preliminary evidence that is inconsistent with the growth–hydraulic-safety hypothesis, additional studies will likely be needed before confident generalizations can be made.

*Other factors.*—Climate also appears to be responsible for the much greater abundance of lianas (woody climbers) in tropical than temperate forests (Schnitzer and Bongers 2002). Trees with heavy liana loads at BCI experience reduced growth and increased mortality rates (Putz 1984, Ingwell et al. 2010), a consequence of competition for both light and belowground resources (Schnitzer and Bongers 2002, Schnitzer et al. 2005, Ingwell et al. 2010). All else being equal, our approach to distinguishing among the hypotheses should be robust to determining whether competition—regardless of its origins in lianas, free-standing trees, or both—contributes to higher mortality rates at BCI relative to the Pacific States, and we earlier concluded that the overall effects of competition may be greater in the Pacific States. However, the possibility remains that infestation of tree crowns by lianas could sometimes proceed so rapidly that the associated increase in competition is not evident in tree growth rates calculated for the preceding 5-year growth interval, a possibility meriting further investigation.

Finally, we note that the higher LH-group-specific mortality rates at BCI compared to the Pacific States is unlikely to be explained by greater mechanical stresses on (or lesser structural integrity of) BCI trees, though these possibilities merit further investigation. BCI is outside of the hurricane zone; for any given location, the return time for winds strong enough to fell $\geq$1 ha of forest is estimated to be 1000 to 5000 years (Leigh et al. 2004). Additionally, although BCI trees can experience heavy loads of epiphytes and lianas, trees in the Pacific States endure substantial winter snow loads. Wetter soils at BCI might contribute to tree falls (cf. Givnish 1999), but it is unclear whether the rate would be higher than in the Pacific States plots, especially considering that the Pacific States trees typically grow on steeper slopes potentially subject to more erosion, rolling rocks and logs, and snow movement. Finally, we note that average bole wood density is roughly 40% greater for BCI angiosperms (0.54 g/cm$^3$; Chave et al. 2003) than for Pacific States gymnosperms (0.38 g/cm$^3$; data from Chave et al. 2009*b*). All else being equal, this difference should translate into greater potential for tree death by structural failure in the Pacific States, not at BCI (cf. King et al. 2006*b*, Chave et al. 2009*a*).

## Discussion

### *Case study*

Regardless of the ultimate causes of BCI's higher community-wide mortality rate compared to the Pacific States, our case study yields a particularly interesting empirical result. An assumption at the heart of individual-based forest models is that for trees within a given species or functional group, tree growth rate and probability of mortality are inversely related (Fig. 6A; Bugmann 2001, Keane et al. 2001). Our case study suggests that, at least in some cases, the nature of this inverse relationship may change along productivity gradients (cf. Kobe 1996, Wunder et al. 2008). Specifically, at any given growth rate a tree's probability of mortality can be higher in a productive environment than in an unproductive environment (Fig. 6B). It is thus possible for community-wide mortality rate to

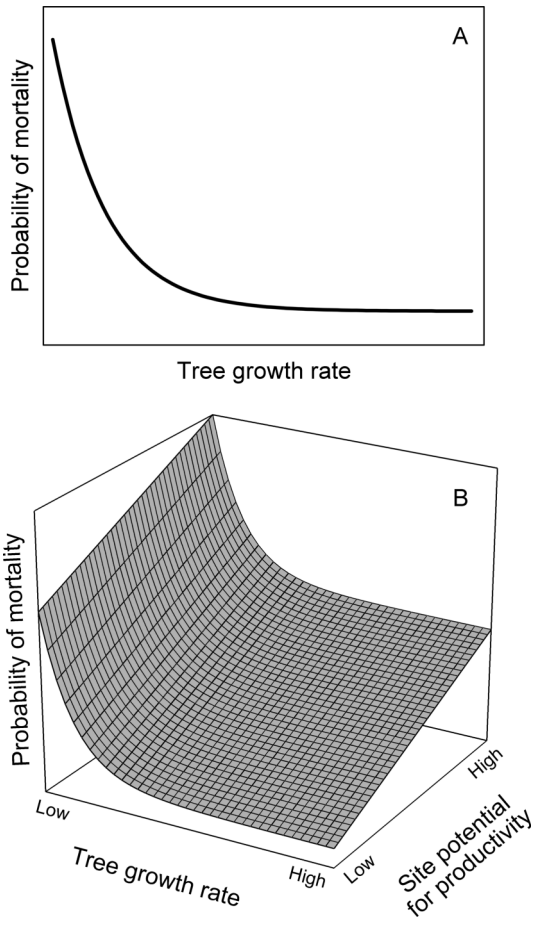


FIG. 6. Relationships among tree growth rate, site potential for productivity, and probability of tree mortality. (A) Contemporary individual-based forest models assume that, for trees within a given species or functional group, tree growth rate and probability of mortality are inversely related. (B) Concepts and results we have presented suggest that, at least in some cases, the nature of this inverse relationship may change along productivity gradients. Specifically, at any given growth rate, a tree's probability of mortality can be higher in a productive environment than in an unproductive environment.

increase even as individual tree growth rates also increase (cf. Laurance et al. 2009). Conversely, a higher community-wide mortality rate does not automatically imply that a greater proportion of trees is growing slowly, as would be implied by the assumptions of individual-based forest models.

As informative as this empirical result may be, ultimately we wish to understand its causes. We concluded that, within a given life-history group (such as shade-tolerant canopy species), BCI's higher mortality rate was most likely a consequence of differences in environmental favorability to plant enemies, and not a consequence of trait differences related to defenses, hydraulic architecture, or wood density. However, we cannot rule out the possibility that other trait differences, unidentified by us and potentially taxonomically linked, could have contributed to the differences in mortality rates. We note, however, that when angiosperm and gymnosperm trees grow in similar environments, their mortality rates may differ little or not at all (Stephenson and van Mantgem 2005, Martínez-Vilalta et al. 2010). This implies that any otherwise unidentified taxonomically linked trait differences are unlikely to be dominant contributors to the large differences in mortality rates we observed between these tropical angiosperm and temperate gymnosperm forests (Stephenson and van Mantgem 2005).

In light of growing interest in understanding properties of plant communities in terms of plant functional traits (e.g., McGill et al. 2006, Westoby and Wright 2006), our case study highlights two important points. First, factors unrelated to functional or life-history traits may contribute significantly to broad-scale differences in mortality rates among forest communities. For example, environmental favorability to plant enemies appears to be a major contributor to BCI's higher tree mortality rate, independent of species' trait differences between BCI and the Pacific States. Second, a sharp distinction sometimes must be made between the alpha (within-community) and beta (among-community) contributions of species' traits to mortality rates (cf. Ackerly and Cornwell 2007). In particular, relationships between species traits and mortality rates that are found within a forest community may not hold between forest communities. For example, the high resource availability found in gaps within forest communities tends to favor species expressing a relatively predictable suite of traits associated with shade intolerance (see *Background and theory*); those traits generally include low wood density and reduced defenses, and are associated with high mortality rates relative to other species in the community. In contrast, the between-community comparison between BCI and the Pacific States suggested that although the high-resource environment at BCI was indeed associated with higher mortality rates, species at BCI had higher bole wood density and may not have been any less well defended than those in the Pacific states: a pattern differing from that seen within communities.

### *Possible generalizations about mechanisms*

Our synthesis of the literature, coupled with results of our case study, leads us to conclude that of our seven hypothesized mechanisms, at least four are likely to contribute to geographic variation in background tree mortality rates. We further conclude that the relative importances of these four mechanisms at least partly depend on whether differences in site potential for forest productivity are determined climatically or edaphically (Table 3). Specifically, one mechanism (enemies) may most commonly dominate along climatic productivity gradients; two (growth–defense trade-offs and competition) may most commonly dominate along edaphic productivity gradients, and one (canopy/subcanopy proportions) may occur on both. This proposed

TABLE 3. Proposed dominant mechanisms driving the positive correlation between tree mortality rates and forest NPP.

| Class of mechanism | Cause of productivity gradient | |
|---|---|---|
| | Climatic | Edaphic |
| Life-history trade-offs | canopy/subcanopy proportions | canopy/subcanopy proportions, growth–defense trade-offs |
| Ecological interactions | enemies | competition |

*Note:* Refer to *Background and theory* for explanations of the mechanisms.

segregation of mechanisms according to the drivers of productivity gradients may largely be a consequence of plant enemies responding more strongly to climatic than to edaphic gradients.

*Mechanisms along climatic productivity gradients.*—Along latitudinal climatic gradients, we propose that the two dominant mechanisms identified in our case study might dominate more generally, contributing to higher mortality rates in tropical than temperate forests at broad scales. Available evidence indicates that, globally, tropical forests have a greater relative abundance of trees belonging to subcanopy species than do temperate forests, and that subcanopy species usually have higher mortality rates than canopy species (see *Background and theory*). Although consensus is currently lacking regarding the nature of latitudinal gradients in herbivory and defenses (e.g., see the conflicting conclusions of recent reviews and analyses in Schemske et al. [2009] and Moles et al. [2011*a*, *b*]), we propose that at least some of the lack of consensus is a consequence of most studies having focused on leaves, so that patterns related to leaf lifespan may confound those related to latitude (see *Ultimate causes of BCI's higher mortality rate*). Although much more work is needed, we further suggest that the lines of evidence that led us to favor the enemies hypothesis in our case study may prove to hold more generally. On the surface, evidence favoring the enemies hypothesis would seem to be at odds with the existing body of evidence supporting the growth–defense hypothesis (see *Mechanisms along edaphic productivity gradients*) which, all else being equal, would predict that plants in more productive tropical climates have higher mortality rates primarily because they are more poorly defended than those in less-productive temperate environments. However, support for the growth–defense hypothesis comes from soil fertility and light gradients within latitudinal (climatic) zones, not from climatic gradients spanning latitudinal zones (Endara and Coley 2011). Thus, evidence supporting the enemies hypothesis carries an important implication: the greater humidity and year-round warmth of moist tropical forests creates conditions so favorable to plant enemies that those enemies exert a strong selective pressure, sufficient to at least partly (if not fully) overcome the resource-driven selection for decreased plant defenses that is otherwise predicted by the growth–defense hypothesis (e.g., Coley and Aide 1991, Coley and Barone 1996, Hallam and Read 2006).

We note that in some cases, extremes of soil fertility between latitudinal zones might lead to reversals of expected climatically driven patterns of tree mortality. For example, temperate angiosperm forests, which usually occur on relatively fertile soils, have slightly higher average mortality rates than tropical angiosperm forests growing on infertile soils (Stephenson and van Mantgem 2005). A possibility to be investigated is whether the tropical angiosperms growing on infertile soils are sometimes so much better defended than the temperate angiosperms growing on fertile soils (cf. Fine et al. 2004, 2006) that they more than compensate for increased pressure from plant enemies in the tropics.

The same mechanisms implicated between latitudinal zones may also act to varying degrees along climatic productivity gradients within latitudinal zones. For example, proportions of trees belonging to subcanopy species vary relatively widely among tropical forests, perhaps at least partly due to differences in rainfall (see *Background and theory*). Plant enemies are also likely to play a role along climatic gradients within latitudinal zones. For example, our California data came from evergreen gymnosperm forests arrayed along a steep elevational gradient, along which community-wide mortality rate increases nearly four-fold with decreasing elevation (Stephenson and van Mantgem 2005). Differences in proportions of trees belonging to subcanopy species cannot explain this trend; such trees are virtually absent. Instead, the increase in community-wide mortality rates parallels a significant increase in tree deaths attributable to insects and pathogens (Stephenson, *unpublished data*), consistent with the warmer climates at lower elevations being more favorable to plant enemies. Similarly, levels of herbivory suffered by herbaceous species commonly increase with decreasing elevation (summarized in Scheidel et al. 2003), as does wood decomposition rate (e.g., Kueppers et al. 2004). In Panama, reciprocal transplant experiments showed that attack by herbivores and pathogens increased along a gradient of increasing rainfall (Brenes-Arguedas et al. 2009). Across the tropics, standard litter substrates demonstrated that decomposition rates increased with increasing rainfall (Powers et al. 2009).

*Mechanisms along edaphic productivity gradients.*—Independent of climate, fertile soils may also favor increased dominance by subcanopy species (see *Background and theory*). Thus, a relative poverty of trees belonging to subcanopy species might sometimes contribute to the substantially lower mortality rates of

tropical forests growing on nutrient-poor soils (Phillips et al. 2004). Additionally, at least some of the difference in mortality rates between tropical forests growing on fertile and infertile soils appears to be driven by a growth–defense trade-off, with tree species found on infertile soils growing more slowly and being better defended than those on fertile soils (Fine et al. 2004, 2006). Thus, while we suggest that the growth–defense hypothesis may not generally apply along climatic gradients (because of the counteracting effects of climate on attack rates by plant enemies, resulting in strong selection for defenses), it may apply to forests along soil fertility gradients within regions sharing similar climates, and thus similar potential for attack by plant enemies (Endara and Coley 2011).

Importantly, the positive correlation between mortality rates and soil fertility often occurs not just among species, but also within species (e.g., Eid and Tuhus 2001). Although a growth–defense trade-off conceivably could exist between genetically distinct populations within a species, it cannot explain the positive correlation between mortality and soil fertility observed both in fertilization experiments and in experiments that control for (or arbitrarily plant) different genotypes (Shen et al. 2001, Álvarez González et al. 2004). Instead, higher mortality rates on the more fertile sites apparently are at least partly a consequence of enhanced asymmetric competition, a manifestation of the Sukatschew effect (e.g., Turnblom and Burk 2000).

*Other mechanisms*.—More study is needed to determine whether the three remaining mechanisms (tolerant/intolerant proportions, growth–hydraulic-safety trade-offs, and reproduction–persistence trade-offs) play dominant roles in controlling tree mortality rates, and if so, under what circumstances. All else being equal, forests that have high mortality rates in canopy trees should have high rates of gap formation and therefore more opportunities for establishment of shade-intolerant species. Higher proportions of shade-intolerant species, in turn, should lead to higher community-wide mortality rates and therefore higher gap formation rates (the tolerant/intolerant proportions hypothesis), potentially contributing to a positive feedback process (cf. Chao et al. 2008, 2009). However, we suspect this mechanism is not a major contributor to broad-scale patterns in mortality rates. Higher mortality rates also mean that fewer trees reach sizes capable of creating large gaps when they fall; for example, the density of canopy trees ($\geq$50 cm dbh) at BCI is only about one fourth that of the Pacific States. Gap formation rates are relatively low worldwide, with median values $\leq$1% per yr (McCarthy 2001). The limited available data suggest that gap formation rates are higher in tropical angiosperm forests than in temperate gymnosperm forests (McCarthy 2001), but rates of forest development within gaps (and presumably succession from shade-intolerant to shade-tolerant species) are almost certainly more rapid in the tropics (e.g., Anderson et al. 2006). The net effect might be that proportions of trees belonging to shade-intolerant species are roughly comparable among old forests and, most important, those proportions are too small to have a major effect on community-wide mortality rates (see *Ultimate causes of BCI's higher mortality rate*). However, further work is needed to test this possibility.

We are unaware of existing studies capable of shedding additional light on the reproduction–persistence hypothesis. Recall that this hypothesis refers to trade-offs found *within* a given life-history group across broad productivity gradients, not to trade-offs found between life-history groups growing sympatrically (the latter is expressed in the canopy/subcanopy proportions hypothesis). Using a global database, Moles et al. (2009*b*) reported increasing proportions of NPP devoted to seed production along a gradient from high to low latitudes; however, results were not segregated by tree life-history groups. We further note that tree growth rates generally increase from high to low latitudes, at least superficially suggesting that any reproduction–persistence trade-off may not be strong enough to significantly increase mortality through reductions in growth and perhaps defenses. However, much more work is needed to elucidate trade-offs among growth, reproduction, and persistence along both climatic and edaphic productivity gradients.

Finally, even though stressful, unproductive environments sometimes select for intrinsically safer but less efficient tree hydraulic architectures, we know of no evidence supporting the second requirement of the growth–hydraulic-safety hypothesis: that the more efficient hydraulic architectures found in productive environments contribute to higher mortality rates under conditions typical of those environments (see *Ultimate causes of BCI's higher mortality rate*). Although some recent studies offer evidence against the second requirement (e.g., Poorter et al. 2010, Russo et al. 2010, Hoffmann et al. 2011), additional studies will likely be needed before confident generalizations can be made.

### *Implications of the correlation between NPP and mortality rates*

An important implication of this paper is that the positive correlation between NPP and mortality rate does not automatically imply that NPP itself directly controls mortality rate. For example, our case study suggests that the higher community-wide mortality rate at BCI is not a consequence of enhanced asymmetric competition induced by higher NPP; rather it is a consequence of environmental selection for species exhibiting certain life-history trade-offs (subcanopy species) and, most likely, favorability to plant enemies. Thus, tree mortality rates would still be high at BCI even if its forest density were reduced until its NPP was lower than that of the Pacific States (cf. Ferry et al. 2010).

We propose that the higher mortality rates of trees of all growth rates and sizes at BCI (Figs. 4 and 5), a

probable consequence of high attack rates by plant enemies, reduce dominance by larger trees, eliminating the possibility for enhanced asymmetric competition relative to the Pacific States (cf. Goldberg et al. 1999). Even though tree density at BCI is about twice that of the Pacific States, the forest's total basal area (a more accurate index of potential for competition [Uriarte et al. 2004, Canham et al. 2006]) is only one-third that of the Pacific States.

However, even in those cases when high mortality rate is not a direct consequence of high NPP, mortality undoubtedly influences NPP. Specifically, mortality rates affect forest size structure, density, and tree growth rates, all of which can affect NPP (Mattson and Addy 1975, Castello et al. 1995, Ferry et al. 2010). Thus, while the potential upper limits of forest NPP may be determined by temperature and resource availability (bottom-up control), realized NPP is almost certainly further influenced by independent environmental controls on mortality rates, which can include enemies (top-down control). We emphasize an important distinction between this mechanism and the more traditional view of consumer (enemy) influences on NPP. Consumer influences on forest NPP may depend less on the proportion of living plant mass consumed, which is relatively small in any forest (McNaughton et al. 1989, Cebrian 1999), than on the effects of enemies on population processes, particularly mortality rates (cf. Mattson and Addy 1975, Castello et al. 1995). Pathogens might consume only a few grams of their host, but in the process they can bring down trees weighing hundreds of tons.

The positive relationship between NPP and mortality helps shed light on why increased NPP often is not associated with increased forest biomass, and can even be associated with decreased forest biomass (Keeling and Phillips 2007, Litton et al. 2007, Körner 2009). Our case study provides a concrete example. Compared to the Pacific States, mortality rates are higher in all tree size classes at BCI, resulting in a lower density of trees achieving large size even though potential maximum growth rate is higher at BCI. The lower density of large trees, in turn, contributes to lower forest biomass. Specifically, compared to old forests in the Pacific States, BCI has only one-fourth the density of trees $\geq$50 cm dbh (74 and 19 trees/ha, respectively) and distinctly lower community-wide aboveground biomass ($<$500–784 Mg/ha and 281 Mg/ha, respectively; Pacific States biomass estimated from Smithwick et al. [2002], Van Tuyl et al. [2005], Hudiburg et al. [2009], averaged for the Pacific States regions included in this study and assuming a 2$\times$ conversion from C mass to biomass [Harmon et al. 2004]; BCI biomass from Chave et al. [2003]). Similarly, across Amazonia, Lewis (2006) has shown a negative relationship between forest turnover rate (mortality and recruitment) and aboveground biomass. However, high mortality rates can also contribute to greater availability of certain limiting resources, such as nutrients (due to more rapid input from dead trees), light, and space (Mattson and Addy 1975, Castello et al. 1995). These, along with greater climatic favorability (such as year-round warmth and humidity), may contribute to higher recruitment densities (e.g., 763 trees/ha that are 5 to $<$15 cm dbh at BCI, compared to only 207 in the Pacific States) and allow more trees to grow rapidly (e.g., 93 trees/ha at BCI with annual diameter growth rates of 6–40 mm, compared to only 24 in the Pacific States). The net result is higher community-wide NPP at BCI, even though mortality rates are higher and standing biomass is lower; aboveground NPP is estimated to be 18 $Mg{\cdot}ha^{-1}{\cdot}yr^{-1}$ at BCI (Chave et al. 2003) compared to an estimated 5–13 $Mg{\cdot}ha^{-1}{\cdot}yr^{-1}$ in old forests of the Pacific States (data from Harmon et al. 2004, Van Tuyl et al. 2005, Hudiburg et al. 2009). Compared to the Pacific States, more of BCI's NPP is concentrated in small trees and in rapidly growing trees, as might be expected for a forest more heavily dominated by young trees. For example, in the Pacific States only 36% of annual basal area increment (a crude index of biomass increment) is found in trees that are small (5 to $<$15 cm, all growth rates) or growing rapidly (6–40 mm/yr, all sizes), compared to 81% at BCI (Fig. 7).

### *Conclusions*

Across forested ecosystems, the broad-scale correlation between tree mortality rates and forest NPP cannot be explained by a single mechanism. Rather, at least four mechanisms appear to be at play, with the dominant mechanisms depending on whether the underlying productivity gradients are climatically or edaphically determined (Table 3). Two of the mechanisms are consequences of environmental selection for species exhibiting certain combinations of life-history traits and trade-offs, and the other two are consequences of environmental influences on ecological interactions.

These conclusions have several implications. For only one of the four mechanisms can high mortality rates be considered to be a relatively direct, bottom-up consequence of high NPP, by way of enhanced asymmetric competition. The remaining mechanisms demand that we adopt a different view of causality, in which tree growth rates and probability of mortality can vary with at least some degree of independence (Fig. 6B). A corollary of this independence is that in many (perhaps most) cases, rather than being a direct cause of high mortality rates, NPP may remain high in spite of high mortality rates. The independent influence of plant enemies (top-down control) and other factors on mortality rates helps explain why forest biomass can show little correlation, or even negative correlation, with forest NPP. However, if mortality rates increase enough, NPP ultimately is likely to decline (cf. Ferry et al. 2010). Additionally, in the absence of direct causal relationships between forest NPP and tree mortality rates, the transient responses of NPP and mortality to environmental changes may be decoupled. A final critical

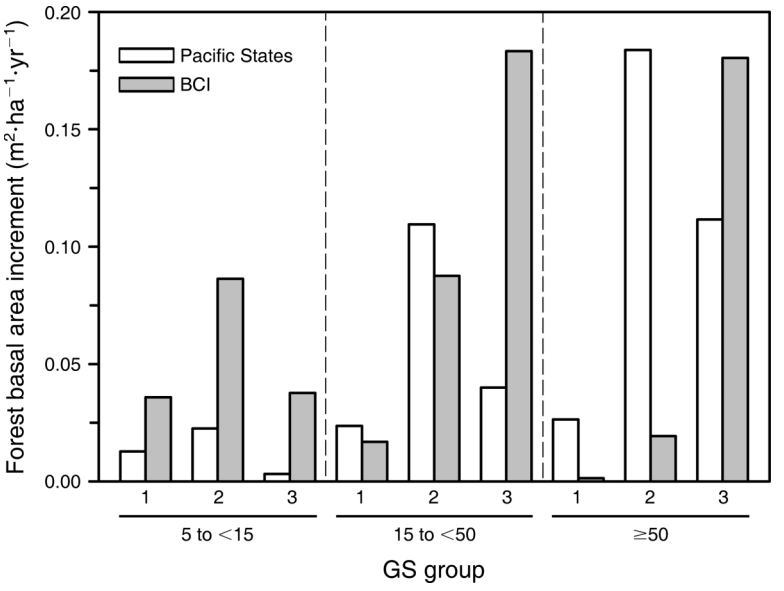


FIG. 7. Annual basal area increment by growth-rate and size classes (GS groups) at the temperate Pacific States (white bars) and tropical BCI (gray bars). GS groups are as in Figs. 4 and 5.



implication of our synthesis is that species' functional traits may not always be the dominant determinants of community-wide mortality rates; other strong determinants include environmental influences on the nature of ecological interactions.

Several avenues for future research are evident. Validation of our hypotheses and proposed generalizations will require many more targeted studies of life-history traits and the nature of ecological interactions along local and (especially) broad-scale productivity gradients, both climatic and edaphic. In particular, our ability to distinguish among the hypotheses will benefit greatly from detailed, community-wide studies of causes of tree death, supported by frequent assessments of the pathology of each living tree in a plot. We suspect that such studies will more broadly support our contention that enemy attack on boles and roots is a greater source of tree mortality than folivory in most forests, and that comparative studies of bole and root defenses therefore are of particular importance. Validation will also require more analyses similar to those of our case study, systematically applied across a number of climatic and edaphic productivity gradients worldwide. Data requirements are daunting, underscoring the importance of large, long-term data sets from permanent forest plots. Fortunately, qualifying data sets are likely to become more abundant over the next decade, especially from the dozens of large (20–50 ha) permanent forest plots recently established (or being established) in association with the Smithsonian Tropical Research Institute's Center for Tropical Forest Science, following a common set of protocols in both tropical and temperate forests worldwide. Finally, these analyses will benefit from improved approaches to partitioning proximate causes of difference in mortality rates. Ideally, such improvements would allow us to work with smaller sample sizes, would allow many plots to be analyzed simultaneously, and would treat life-history traits as continua rather than as discrete classes.

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## APPENDIX A

Determining relative contributions of proximate causes to differences in mortality rates (*Ecological Archives* M081-019-A1).

## APPENDIX B

Characteristics of the study plots (*Ecological Archives* M081-019-A2).

## APPENDIX C

Case study methods (*Ecological Archives* M081-019-A3).