TREE MORTALITY IN GAP MODELS: APPLICATION TO CLIMATE CHANGE *

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Abstract. Gap models are perhaps the most widely used class of individual-based tree models used in ecology and climate change research. However, most gap model emphasize, in terms of process detail, computer code, and validation effort, tree growth with little attention to the simulation of plant death or mortality. Mortality algorithms have been mostly limited to general relationships because of sparse data on the causal mechanisms of mortality. If gap models are to be used to explore community dynamics under changing climates, the limitations and shortcomings of these mortality algorithms must be identified and the simulation of mortality must be improved. In this paper, we review the treatment of mortality in gap models, evaluate the relationships used to represent mortality in the current generation of gap models, and then assess the prospects for making improvements, especially for applications involving global climate change. Three needs are identified to improve mortality simulations in gap models: (1) process-based empirical analyses are needed to create more climate-sensitive stochastic mortality functions, (2) fundamental research is required to quantify the biophysical relationships between mortality and plant dynamics, and (3) extensive field data are needed to quantify, parameterize, and validate existing and future gap model mortality functions.

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1. Introduction

Forest gap models are a class of individual plant-based community models that have been used extensively to study successional dynamics for a wide variety of purposes and applications. Researchers and land managers have modified the original JABOWA (Botkin et al., 1972) and FORET (Shugart and West, 1977) computer models to create variants for specific applications in new ecosystems and under novel disturbance regimes (see Botkin and Schenk, 1996; Bugmann, 1996; Shugart, 1998; Urban and Shugart, 1992). Moreover, the gap model structure and design have been implemented in other forest and grassland models; comparisons have also been made between forest and grassland structure and dynamics using gap models (e.g., Bossel and Schafer, 1989; Coffin and Lauenroth, 1990; Coffin and Urban, 1993; Kimmins, 1993; Korol et al., 1995; Pacala et al., 1996; Zhang et al., 1994). The great success of gap modeling is chiefly that the simple protocols for estimating model parameters allow wide application while still producing realistic predictions. Yet, considering the widespread use of gap models, it seems somewhat surprising that few modelers have critically evaluated the limitations and shortcomings of the gap algorithms (but see, Loehle and LeBlanc, 1996; Pacala and Hurtt, 1993). This becomes especially relevant as gap models are used to explore community dynamics under changing climates and environments (Dale and Rauscher, 1994; Kellomäki and Kolström, 1992; Loehle and LeBlanc, 1996; Makipaa et al., 1999). Gap models must faithfully and comprehensively represent the basic processes that govern plant dynamics if they are going to be used in novel applications such as changing climates.

Most gap models place the vast majority of their emphasis, in terms of process detail, computer code, and validation effort, on tree growth. A model component that has received little critical attention is the simulation of plant death (i.e., mortality), which is strikingly similar across nearly all gap models (see Botkin and Schenk, 1996; Shugart, 1998). Much of the reason for the limited detail in the characterization of mortality comes from the greater availability of growth data (Pacala et al., 1993). Compared to the huge body of observations relevant to defining, evaluating, and parameterizing the growth equations, mortality is infrequently and inadequately sampled, and the precise causes of plant mortality are rarely documented because causes are difficult to identify in the field (Battaglia and Sands, 1998). With sparse data on the causes of mortality and the way that the causal mechanisms interact with environmental conditions and plant health, the mortality algorithms have been limited to very general relationships (Shugart, 1998). Yet, the mortality functions in gap models may be particularly critical for community dynamics in some applications, such as the explicit simulation of disturbance and climate regimes (Botkin and Schenk, 1996; Keane et al., 1990; Lexer and Hönninger, 1998). Models that simulate plant community composition changes as a consequence of interactions among and between plants and their environment should deal with all processes that govern plant life cycles at the same level of con-

sistency, especially as environments become altered (i.e., climate change) (Franklin et al., 1987). Interspecific differences in reproduction, growth, and mortality can all play critical roles in determining plant community structure and successional dynamics (Shugart, 1998).

In this paper, we review the treatment of tree mortality in gap models, evaluate the formulations used to simulate mortality in the current generation of gap models, and then assess the prospects for making improvements, especially for applications involving global climate change. We focus on trees in this paper because the vast majority of climate change applications of gap models are for forested ecosystems.

Three considerations motivate an evaluation of the mortality functions in gap models. First, advances in the science and experience with the models point to a number of areas where improvements are feasible, without changing the basic formulations. In cases where existing formulations have been parameterized with more attention to interspecific differences, the improvements in the simulation results can be substantial (Pacala et al., 1996; Wyckoff and Clark, 2000). For these improvements, the challenge is mainly one of parameterization – obtaining critical information necessary for accurately representing the responses of the individual species. A second motivation comes from the growing appreciation that the probability of mortality can vary by causal factors, as well as with the status of the plant. When mortality is caused by exogenous factors, such as fire, insect outbreaks, and hurricanes, it is clear that the nature of the causal agent plays a central role in determining how mortality varies across species, size classes, and vigor classes (Kercher and Axelrod, 1984a). Other kinds of abiotic factors, including drought, brief periods of heat or cold, or pollutant exposure, may also drive mortality in a manner that is fundamentally different from plant stress due to shading, the mechanism that underlies the mortality response in the current generation of gap models. The third and most important motivation comes from the prospect that ongoing global changes will disrupt or invalidate some of the implicit and mostly empirical relationships on which the mortality algorithms in gap models are based (Bugmann and Solomon, 2000; Burton and Cumming, 1995; Korzukhin et al., 1996; Loehle, 1996; Solomon, 1986). Current formulations are abstractions based on empirical relationships that appear robust for existing conditions. But in a world with higher atmospheric CO_2 concentrations, warmer temperatures, and modified precipitation, these relationships may be less robust or even wrong, as a result of fundamental changes in the way mortality varies with age or growth. This last consideration is the major impetus for this paper.

2. Background

Plant death is a complex process, influenced by physiology, environment, successional development, age, and, chance (Harcombe, 1987; Franklin et al., 1987). In general, trees die when they cannot acquire or mobilize sufficient resources to

recoup from stress, heal injuries or sustain life, or they are killed by some external factor (Waring, 1987). But the interactive and sequential nature of the complex mortality mechanisms precludes the definitive classification of what caused the ultimate death of a tree (Franklin, 1987). While a tree's death might ultimately have resulted from a negative carbon balance (i.e., respiration exceeds photosynthesis), the deficit might have been caused by an insect infestation brought about by poor vigor during a prolonged period of drought under low light conditions. Indeed, increasing a tree's life span might require increased energy investment in protective measures (e.g., thick bark, defensive chemicals; Loehle, 1987) or carbon reserves (Shigo, 1985), but there are always corresponding tradeoffs (e.g., carbon allocated to defensive chemicals cannot be used to grow additional roots). Ultimately, the physiological causes of tree death are still unknown and difficult to observe. It is the temporal and spatial complexity in tree mortality, coupled with the lack of information on the causal mechanisms of mortality, which prevents a thoroughly mechanistic treatment of plant mortality in most gap models.

Most mortality formulations in the current generation of gap models are simple and general (see Hawkes, 2000; Shugart, 1998). For the most part, they do not attempt to assign mortality to any particular cause, but rather, the formulations reflect evidence that agents of mortality tend to fall into three broad classes that are generally but not perfectly related to scale: *intrinsic mortality* is typically a tree-level event, *growth-dependent mortality* is a stand-level process, and *exogenous mortality* is a landscape-level process.

In his book, Botkin (1993) identifies two gap model mortality processes. The inherent risk of death, which we refer to as *intrinsic mortality*, is mortality expected to occur in favorable environments with or without competition from other trees. This mortality is simulated independently of a plant's health, age, or position in the canopy as a stochastic age-independent function of the maximum observed longevity of individuals of a species. Though not specifically identified in the models, this class might include non-epidemic diseases, lightning, windthrow, or ice. It might also include a number of agents that are not truly independent of age, size, or health, but for which the dependencies are unknown.

Competition-induced death is mortality due to poor growth (referred to as *growth-dependent mortality* in this paper) that is often due to competition for resources (i.e., suppression) and is frequently modeled as a stochastic function of diameter growth increment (see Hawkes, 2000; Botkin, 1993). This assumes the slowest growing trees are most likely to die because they are weaker and less able to defend against insect or disease attacks, or less prone to survive high wind events or other abiotic perturbations. Other agents of mortality, including insect pests, pathogens, drought, and nutrient limitation may also be likely to act on slower growing trees that may be predisposed to these agents (Botkin, 1993). The evidence for this, however, is far from conclusive, especially with regard to the question of whether probability of mortality from different agents scales uniformly with reductions in growth. Manion (1979, 1981) describes three factors that represent

parts of the growth-dependent mortality process. Predisposing factors reduce tree growth over time (e.g., climate), inciting factors act over short time periods on low vigor trees unable to recover (e.g., ice storm), and contributing factors are those agents that attack stressed trees and are the proximate cause of death (e.g., opportunistic insects).

We add a final class of mortality, called *exogenous mortality*, which results when an external factor sweeps into a patch or stand and kills some or all of the trees. The intensity and severity of exogenous mortality agents, such as fire, major pest outbreaks, or severe wind, may depend more strongly on conditions outside than inside a simulated patch (Keane et al., 1996b).

2.1. INTRINSIC MORTALITY

Intrinsic mortality is often modeled as an age-independent mortality routine (i.e., maximum-age dependent) that serves as a proxy for those causes of death that can happen at any time in the lifetime of a tree. This includes such factors as lightning strikes, falling trees and branches, animal browsing or girdling, local insect defoliation, and fungal infection. The assumption is that chance plays a major role in this type of mortality because the mortality is usually random and localized (Shugart, 1998). Most forest gap models deal with this chance by designing routines that assume a constant probability of death throughout the lifetime of the tree, usually ending with 1% or 2% (depending on model parameterization) of all trees of a species surviving to their maximum known age. The most commonly used equation for intrinsic mortality is:

$$P_m = 1 - e^{\left[-\frac{4.605}{\text{age}_{\max}}\right]}, \tag{1}$$

where P_m is the probability of intrinsic mortality and $\text{age}_{\max}$ is the maximum age observed for that species (yrs) (Botkin, 1993; Botkin et al., 1972; Bugmann, 1996; Shao et al., 1994). Because additional sources of mortality are present, much less than 1 or 2% of trees actually reach their maximum known species-specific age. Indirectly, the annual rate of mortality is scaled by species, in that each species has a different maximum age.

This algorithm (Equation (1)) has been implemented in many gap models (e.g., Busing and Clebsch, 1987; Friend et al., 1993; Kellomäki and Kolström, 1992; Krauchi and Kienast, 1993; Pastor and Post, 1986), but there are exceptions. The FORSKA line of models has no maximum age (Leemans and Prentice, 1989; Leemans, 1992). A constant rate of background mortality is included, scaled to each species by their shade tolerance. Because shade tolerance and species longevity are usually related, shade tolerance becomes a less empirical proxy for maximum age in FORSKA. Some gap models, such as Reed and Clark's (1979) SUCSIM, only simulate growth-dependent mortality. In TREEDYN3, Bossel (1994) simulates 'low' or intrinsic mortality as a constant value for each species and does not relate this value to age. Kimmins et al. (1999) use a smoothed tree density-age

curve to compute mortality rates for each tree age class. SORTIE kills a constant portion of stems per year (e.g., 1–2%) based on analyses of extensive field data (Pacala et al., 1993).

2.2. GROWTH-DEPENDENT MORTALITY

Growth-dependent mortality has a diverse representation in forest gap models. Early gap models, such as JABOWA (Botkin et al., 1972) and FORET (Shugart and West, 1977) increase the probability of growth-dependent mortality any time the tree diameter growth falls below a minimum threshold value, usually estimated from tree ring measurements. This approach attempts to mimic the response by trees to a severe stress in which slowed growth leads to enhanced probability of mortality, in some cases for several years following relief of the stress. It is assumed that growth rates below a specified threshold will predispose trees to insect and disease attacks or severe weather event damage, and could result in negative carbon balances. In the initial formulation, Botkin et al. (1972) specified that one year of stem radial growth below 0.1 mm by any tree of any species resulted in an increased probability of mortality during the succeeding 10-year period such that 30% of vulnerable trees did not survive. Survivors of the 10-year period were returned to the pool of non-vulnerable trees. This routine remained unchanged in several of the subsequent versions of JABOWA (e.g., FORTNITE, Aber et al., 1978; SILVA; Kercher and Axelrod, 1984b; FIRESUM, Keane et al., 1990) and FORET (e.g., FORAR, Mielke et al., 1978; BRIND; Shugart and Noble, 1981; BOREAL; Bonan, 1989; ZELIG; Urban et al., 1991).

The growth-dependent mortality routine approach in FORENA (Solomon and Shugart, 1984; Solomon, 1986) more closely approximates known mortality processes (i.e., Manion, 1979). First, tree species become vulnerable to higher mortality rates only after three or more consecutive years of below-threshold diameter growth are simulated. This change was designed to match the observations that a single year of stress rarely increases mortality in otherwise vigorous trees (McCune and Henckel, 1993; Waring and Running, 1998). Second, low-growth thresholds are defined as being species-specific to avoid the automatic elimination of those species capable of surviving slow growth. This especially applies to many shade- and drought-tolerant species. Differential species response is achieved by flagging trees when they achieve less than 10% of their optimum diameter growth for that specific year of their life (rather than 0.1 mm of growth at any age). Two or three consecutive years of flagged growth makes them vulnerable to the increased probability of mortality defined by Botkin et al. (1972). This modification also appears in several later models (i.e., LINKAGES, Pastor and Post, 1986; FORCLIM, Bugmann and Solomon, 2000; FORECE, Kienast and Krauchi, 1991).

The more physiologically based FORSKA gap model (Prentice, 1987; Leemans, 1992; FORSKA 2, Prentice et al., 1993) takes a different tack. It does not simulate enhanced mortality resulting from slow growth from which recovery

can be made. Instead, it simulates mortality at any age as a function of declining growth. Here, the inability of a tree to match or exceed its respiratory losses with annual photosynthetic production results in the most inefficient leaves being shed from the bottom of the leaf area cylinder. Loss of lower leaves and branches can reduce overall productivity such that additional leaves must be shed in subsequent years, until the tree finally dies because of a negative carbon balance. The annual mortality rate (X_m) is then based on relative growth efficiency (E_{rel}) as follows:

$$X_m = U_0 + \frac{U_1}{\left[1 + \left(\frac{E_{\text{rel}}}{q}\right)^r\right]}, \tag{2}$$

where U_0 is the intrinsic mortality rate, U_1 is a species-specific mortality rate due to suppression (i.e., light competition), q is a threshold value for a vigor index, and r is a species-specific shape parameter. The relative growth efficiency (E_{rel}) is defined as the ratio of realized growth efficiency to a maximum for that species and uses stem volume increment per unit leaf area (Prentice et al., 1993). Fulton (1991) also uses relative growth efficiency to simulate height class mortality as a step function in the FLAM model.

Other gap models modify the original JABOWA approach to more realistically simulate stress mortality. CLIMACS simulates only growth-dependent mortality using the ratio of diameter to maximum diameter stratified by different stochastic relationships depending on the species' role in the successional process (Dale et al., 1986). Although Jorritsma et al. (1999) simulate stress-caused tree mortality in FORGRA from equations in Botkin (1993), they model seedling mortality as a function of total seedling biomass. In SORTIE, Pacala et al. (1993) parameterized the following diameter growth-dependent function from probabilities calculated from ring width distributions of live and dead individuals:

$$P_m = e^{[-(ud)^v]}, \tag{3}$$

where P_m is the probability of mortality, u and v are species-specific constants, and d is average ring width (mm). Busing and Clebsch (1987) scale the threshold diameter growth rate to a proportion of the species' maximum rate. The SUCSIM model uses a threshold diameter growth and population level (tree density) to compute 'slow-growing' mortality (Reed and Clark, 1979).

Some modelers have added mortality functions derived from empirical, stand-level parameters to integrate all factors involved in intrinsic and growth-dependent mortality. Gertner (1990) computes individual tree survival from tree diameter and annual diameter growth statistical equations developed from stand basal area, tree density, and site index. The FORCYTE-11 model computes density-independent tree mortality from input stand data relationships while density-dependent mortality is based on shading (Kimmins, 1993). Probability functions in CLIMACS were developed from a chronosequence of stands in Washington and Oregon, U.S.A. (Dale et al., 1986). SORTIE mortality rates (Equation (3)) are based on extensive

mortality data gathered from the ecosystem being simulated (Pacala et al., 1993). Bossel (1994) parameterized 'crowding' mortality as a constant from extensive stand data.

2.3. EXOGENOUS MORTALITY

Inclusion of exogenous mortality in gap models came about mainly as a result of the long time periods being simulated. Modelers realized that major, landscape-level disturbances would eventually affect the successional dynamics of a patch (i.e., stand) given enough time (Battaglia and Sands, 1998; Glenn-Lewin and van der Maarel, 1992; Smith and Urban, 1988). For example, Urban et al. (1991) found that the dynamic coupling of the patterns of growth, exogenous mortality, and regeneration lead to unique emergent model behaviors where the exclusion of exogenous mortality created different simulation trajectories. Exogenous mortality had not been implemented in many gap models because: (1) it was previously thought to be unimportant to the dynamics of the simulated ecosystem, (2) there was little known about the spatial mechanisms of the disturbance process, (3) it was difficult to simulate because of extensive computer requirements, (4) there were very little data for parameterization, and (5) the simulated variables could not be related to exogenous disturbance effects.

Wildland fire and tree harvests are probably the most common exogenous disturbances included in gap models, but other exogenous mortality sources have also been simulated (Table I). The first generation of these models to include exogenous fire- or harvest-caused mortality simulated it by simply assigning the mortality probability a value of 1.0 for each tree in the simulated patch thereby killing all trees (Prentice et al., 1993). Little attention was given to differential mortality among species and size classes (Dale et al., 1986). And, because tree mortality was usually the only disturbance effect simulated, there was little treatment of the effect of the perturbation on other ecosystem processes, such as carbon cycling (i.e., fuel consumption) or water use (i.e., leaf area reduction from crown scorch) (Dale et al., 1986; Shugart and Noble, 1981). Some gap modelers assume that growth-dependent mortality, as simulated from the reduction in diameter growth indirectly caused by a perturbation, is sufficient to simulate the effect of that disturbance on the ecosystem (Phipps, 1979). For example, Chen and Twilley (1998) have a growth reduction factor for the effect of salinity pulses on mangrove trees in the FORMAN model.

The first routine that incorporated differential fire-caused mortality (FORAR, Mielke et al., 1978) was quite simple, with random fire occurrences causing three classes of fire intensity that in turn prescribe three classes of species-specific fire-caused mortality as derived from field data. Other models (Keane et al., 1990, 1996a) have since simulated fire mortality from empirically derived stochastic

Table I
Gap models that simulate exogenous disturbance mortality

Wildland Fire	BRIND	Eastern Australia	Shugart and Noble (1981)
	CLIMACS	Pacific Northwest, U.S.A.	Dale et al. (1986)
	EDEN	Australia	Pausas et al. (1997)
	FIRESUM	Northern Rockies U.S.A.	Keane et al. (1990)
	FORAR	Southeastern U.S.A.	Mielke et al. (1978)
	MIOMBO	Zambezian Africa	Desanker and Prentice (1994)
	SILVA	California, U.S.A.	Kercher and Axelrod (1984a)
	ZELIG	Sierra Nevada, U.S.A.	Miller and Urban (1999)
Browsing	FORSUM	Central Europe	Krauchi and Kienast (1993)
	FORECE	Southern Central Europe	Kienast and Kräuchi. (1991)
Grazing	FORGRA	Netherlands	Jorritsma et al. (1999)
Air pollution	SILVA	California, U.S.A.	Kercher and Axelrod (1984b)
	FORANAK	Great Smokey Mtns, U.S.A.	Busing and Clebsch (1987)
Harvest	CLIMACS	Pacific Northwest, U.S.A.	Dale et al. (1986)
	FLAM	Central Sweden	Fulton (1991a)
	FORCAT	Cumberland Plateau, U.S.A.	Walthrop et al. (1986)
	FORCYTE	Canadian forest	Kimmins (1993)
	FORSKA	Central Sweden	Prentice et al. (1993)
	FORTNITE	New England, U.S.A.	Aber et al. (1978, 1982)
	KOPIDE	Korea-China Border	Shao et al. (1994)
Hurricane	FORICO	Puerto Rican Forests	O'Brien et al. (1992), Doyle (1981)
Chestnut blight	FORET	Tennessee, U.S.A.	Shugart and West (1977)
Bark beetle	PICUS	Central Europe	Lexer and Honninger (1998)
Flooding	FORFLO	Southeastern U.S.A.	Phipps (1979)
	SWAMP	Southeastern U.S.A.	Pearlstine et al. (1985)
River Flow	FORFLO	American Midwest, U.S.A.	Liu and Malanson (1992)
Salinity	FORMAN	Caribbean Mangrove	Chen and Twilley (1998)

equations that predict the probability of tree death one year after the fire (P_{fire}) from:

$$P_{\text{fire}} = \frac{1}{1 + e^{[-1.941+6.32(1-e^{[\text{bt DBH}]})-0.000535\ \text{CK}^2]}}, \tag{4}$$

where P_{fire} is the probability of fire-caused mortality, DBH is tree diameter (cm), bt is a species-specific bark thickness coefficient (cm bark cm^{-1} diameter), and CK is percent crown volume scorched (Ryan and Reinhardt, 1988; Ryan et al., 1987). The independent variables of diameter, bark thickness, and percent crown scorched act as surrogates for the actual causes of fire mortality: root, cambium, and foliar kill (Ryan et al., 1987). Percent crown scorched is calculated from the scorch height, which is a function of windspeed (m s^{-1}) and fire intensity (kW m^{-1}), the only fire behavior characteristics needed to predict tree mortality. Differential fire-caused

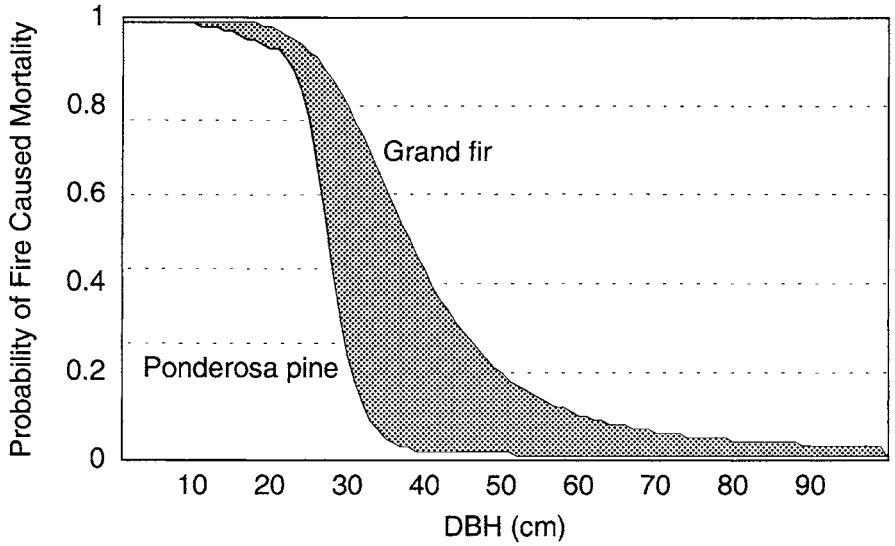

Figure 1. Probability of fire-caused mortality one year after fire for *Pinus ponderosa* (Ponderosa Pine) and *Abies grandis* (Grand fir) when the scorch height of the fire is 10 meters. The red area shows the difference in mortality between the two species.

mortality rates as simulated from Equation (4) can result in unique post-fire stand compositions and structures (Figure 1).

There are very few spatially explicit gap models that simulate the growth of fire as it progresses across the landscape (e.g., Keane et al., 1996a). Fire behavior (i.e., fire spread and intensity) is often computed as if the fire started and ended in the stand rather than as it moved across a landscape. Furthermore, it is assumed the fire burned all portions of the stand at the same intensity. This is important because fire intensity, and therefore fire mortality, are influenced by the spatial expressions of topography, weather, and fuel connectivity. Fire-BGC linked a fire growth model FARSITE (Finney, 1998) to a gap model to simulate the full range of fire effects on forest succession (Keane et al., 1996b). Miller and Urban (1998) implemented ZELIG in a spatial application to simulate fire spread using cellular automata.

3. Evaluation of Mortality Factors

3.1. INTRINSIC TREE MORTALITY

The inclusion of intrinsic tree mortality in gap models raises two major questions. First, is the explicit consideration of intrinsic tree mortality justified or even necessary? And second, is the classical approach of parameterizing intrinsic mortality from species longevity appropriate?

It often has been argued that intrinsic mortality is meant to account for the unexplained or unidentified contributions to mortality a tree experiences as it ages (Botkin, 1993; Shugart, 1998). By combining intrinsic with growth-dependent mortality, a U-shaped mortality function is formed where the highest mortality occurs early (growth-dependent mortality) or late (intrinsic mortality) in life (Figure 2). The rationale for this underlying shape of the composite mortality function in gap models is based on theoretical considerations, but results from a tree mortality modeling study in Norway spruce (*Picea abies* (L.) karst.), based on an extensive, large-scale forest inventory data set, support this hypothesized U-shape over the traditional J-shape (Monserud and Sterba, 1999). Yet, several authors suggest intrinsic mortality should not be considered as a separate cause of tree death (Prentice et al., 1993; Williams, 1996) because it cannot be described using mechanistic explanations. Pacala et al. (1996) consider an intrinsic risk of tree death but did not derive it from maximum age or maximum tree size. The argument that intrinsic mortality is more or less a 'fudge factor' to prevent trees from living indefinitely defies any physical evidence, especially if the growth-related mortality is treated realistically by accounting for tree architectural features, such as the dependence of hydraulic conductance on tree height (see Landsberg and Gower, 1997). Maximum age is, by assumption, the age when a tree reaches its maximum size (Botkin, 1993; Shugart and West, 1977). Thus, intrinsic mortality is independent of the actual tree age, stand density, as well as the environment where the tree grows. It is important to note that the interpretation of intrinsic mortality as dependent on actual tree age is somewhat erroneous. Rather, it is exponentially linked to maximum attainable tree age.

There are several problems with the current formulation of intrinsic tree mortality in the gap models. First, since maximum age is used to derive the intrinsic probability of death for an individual tree, background mortality can be different for each species. It is questionable if this assumption is universally correct. Reconsidering the probable causes of mortality covered by the intrinsic tree mortality algorithm, it might be questionable whether the probability of death by a lightning strike or from windthrow is lower for *Abies alba* (high maximum age) compared to *Acer pseudoplatanus* (intermediate maximum age), for instance. Studies on uneven-aged forests revealed that age might be of limited value as a predictor of a tree's fate (i.e., growth, mortality) (Hamilton, 1986; Peet and Christensen, 1987).

The value of the maximum age parameter is also questionable. There is strong evidence that maximum attainable age of an individual is related more to growth history than site characteristics, and thus cannot be treated as an independent variable (Franklin et al., 1987; Loehle, 1987). This may be of particular importance under changing environmental conditions. The substitution of maximum tree size for maximum age also doesn't seem to be useful in modeling intrinsic tree mortality, but size characteristics of simulated individuals are frequently included as variables in computing growth- or stress-induced mortality. The problem is empirical estimates of size or age mortality parameters assume an unchanging

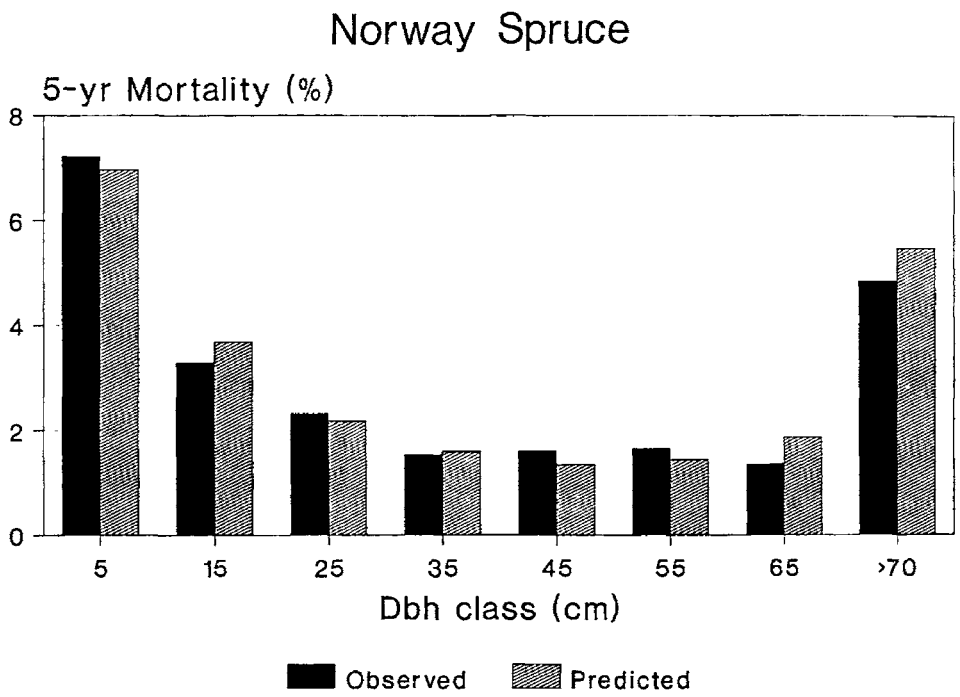

Figure 2. The U-shaped mortality curve from Monserud and Sterba (1999). This curve supports the inclusion of intrinsic and growth-dependent mortality in gap models.

relationship between climate, growth, and mortality (a situation Hawkes (2000) describes as temporal inflexibility) and this can essentially invalidate the model's use in climate change applications (Loehle and LeBlanc, 1996).

The assumption that the probability of intrinsic mortality is static for long simulations or under changing climates is also suspect. The endemic mortality factors that define the unexplained variation in intrinsic mortality will surely change over long time spans or as climate changes. Michaels and Hayden (1987) mention that future climates will have dramatically different types and rates of gap-scale tree mortality phenomenon, such as hailstorms, blowdown, and ice damage. Moreover, these factors, along with local drought or crown damage from weather events such as snowstorms, early frosts, or high winds, could easily predispose trees to additional insect and pathogen mortality. So, the assumption that the elements that comprise intrinsic mortality remain constant as climate changes will be wrong for many situations (Price and Rind, 1994).

Considering that tree mortality is a highly stochastic process that is difficult to predict (Monserud, 1976; Lee, 1971), explicitly including intrinsic tree mortality in gap models might be justified for some climate change applications, especially if tree species actually senesce or if environmental factors favor large tree mortality (e.g., lightning, wind, snowbreak). However, if the explanatory power of models

is not improved by such a simple approach, intrinsic and growth-related mortality probably should be combined to keep gap models simple and consistent (Loehle and LeBlanc, 1996). Such approaches have already been included in gap models as presented by Prentice et al. (1993) and Williams (1996).

3.2. GROWTH-DEPENDENT MORTALITY

From their beginning, gap-models have assumed a relationship between stress and mortality (Botkin et al., 1972). Borrowing from forest growth and yield simulators (Monserud, 1976), gap-models have included the additional assumption that growth rate integrates the stresses experienced by an individual tree (Botkin and Schenk, 1996). Growth-mortality functions thus serve as a surrogate for estimating stress-related mortality. Owing to a lack of data, gap modelers have often assigned the same low growth tolerance to every species, or made some other subjective estimates regarding species differences in tolerance of low growth rates. In recent years, considerable effort has gone into parameterization of the growth-mortality relationship via actual field data for a variety of species (Buchman, 1983; Buchman et al., 1983; Kobe et al., 1995; Kobe, 1996; Kobe and Coates, 1997; Pacala et al., 1996; Wyckoff and Clark, 2000). For many species, however, empirical mortality functions remain unavailable, and, for those species that have been studied, changes in the growth-mortality relationship with environment and throughout the life-cycle remain largely undocumented. Many forestry-related stand-level studies fail to collect data on the early stages of stand development, to the detriment of fitting robust mortality models (Sievanen and Burk, 1994).

One major question concerning growth-mortality functions is the ability of diameter increment to comprehensively (across many species) and robustly (across many environmental conditions) predict stress-caused mortality. Some species can tolerate extremely slow growth indefinitely, while others can only tolerate growth suppression when young (Loehle and LeBlanc, 1996). Loehle (1996) noted that the simulation of growth-dependent mortality is not realistic in some gap models because they fail to represent the growth responses of species to fluctuating climatic conditions, which amplifies the rate of mortality from simulated climate warming. This is because the climate response functions in the model variants are being parameterized from species range extremes and not from basic ecophysiological processes (see Pacala and Hurtt, 1993).

Another problem with estimating mortality from diameter increment is in the way diameter increment is computed. In most gap models, a maximum diameter increment is reduced by a series of reduction factors that represent the influence of important environmental factors (Botkin, 1993). The selection of which reduction factors to include depends on the ecosystem (e.g., water stress) and application (e.g., ozone concentration effects), which seems somewhat arbitrary and subjective. It's possible that factors important to mortality are not included in the growth increment calculation. This is important to climate change applications in that the

influence of a growth reduction factor will change as the trees respond to climate change (Loehle, 1996).

Some other index that is more explicitly connected to the amount of respiring biomass, such as basal area or volume growth, might better reflect changes in tree growth rates across various life stages. Waring and Running (1998) and Waring (1983) suggested some ratio of stem volume increment (m^3 yr^{-1}) per unit leaf area (m^2) might improve simulation of growth-dependent mortality. Growth efficiency based on annual volume increment per unit absorbed radiation might also provide an index of stress (Kaufmann, 1990). Since the relationship between diameter growth and volume/biomass is age and size related, diameter growth *per se* might be a poor indicator of tree vigor or of a tree's susceptibility to death. Vose and Swank (1990) suggested that effects of mortality could be modeled mechanistically by reductions in leaf area index (m^2 leaf area m^{-2} corresponding ground area). There is also evidence that height increment may better predict mortality risk than diameter increment for smaller trees, but since height growth stops long before trees attain maximum diameters and age, it could not be applied across all trees in the model (Franklin et al., 1987). However, it appears that the annual carbon balance (photosynthesis – respiration) may predict stress mortality poorly because: (1) trees can store carbon from previous years, (2) trees can alter physiological and morphological characteristics to reduce respiration, (3) trees may use carbon for other processes besides growth (e.g., reproduction, defense), and (4) there is very little research relating carbon budgets to tree mortality (Waring and Running, 1998; Franklin et al., 1987).

New understanding of interspecific differences in low growth tolerances has not been incorporated into many gap models (Pacala and Hurtt, 1993), and the need for additional field data remains (Harcombe, 1987; Pacala et al., 1996). This is especially true if gap models are to address rapid environmental change or are to be used across many ecosystems. A major question is whether growth-mortality relationships remain constant for species under changing climates? Do species possess the ability to change physiological or morphological characteristics to reduce minimum growth thresholds in response to long-term environmental change? For example, Pacala et al. (1993) suggested that just because a species has high mortality at low growth rates does not mean it will also have high mortality at low light conditions. Knowledge of intraspecific variation in the growth-mortality relationship along gradients of environmental stress is almost non-existent. A few studies have begun to address this issue (Kobe, 1996; Pedersen, 1998a,b), but more effort is needed. This is important considering that when gap models have been modified to simulate effects on growth of new environmental conditions, such as soil moisture, solar radiation, and biogeochemical cycles, these additional processes have yet to be directly linked to mortality (Korol et al., 1991; Levine et al., 1993; Leemans, 1992; Sievänen et al., 1988). There is also the question of whether low growth thresholds can change throughout the development of individ-

ual trees. Are seedlings able to withstand long periods of low growth better than mature individuals?

A larger question still remains; is growth-rate an adequate integrator of all non-catastrophic stresses a tree might face? If not, what stresses elude the growth-mortality relationship and thus must be modeled separately? Here we are truly speculating, but expert speculation may be the tool needed to guide future field efforts. There is evidence that severe drought sometimes kills fast-growing, rather than slow-growing individuals (Clinton et al., 1993). Fast-growing trees may also be more susceptible to winter desiccation and severe frost events. Wind, lightning, and ice disturbances (discussed in *Exogenous Mortality*) may stress large, vigorous trees, more than suppressed, slow growing individuals because of canopy position, morphology, and size. LeBlanc (1990) found large canopy emergent individuals suffered more from 'red spruce decline' than smaller trees because their size caused higher exposure to atmospheric stresses. Is this differential stress captured in growth increment?

Even when a stressing agent is reflected in growth rate, it is not clear that all causes of stresses will impact growth equally. Traditionally, growth-related mortality in gap models has represented mainly light (i.e., decreased radiation) stress. Do other stresses impact growth in the same way? Wyckoff and Clark (2000) found that dogwood anthracnose disease in a population of *Cornus florida* resulted in a very strong growth-mortality relationship. This relationship, however, is likely different from the light-stress induced growth-mortality relationship found in a population of shade tolerant *Cornus florida* not facing epidemic disease. Trees can adjust physiological and morphological characteristics to mitigate progressive mortality agents by shading. Another question is whether growth stresses are multiplicative or additive in their influence on growth-mortality relationships. Perhaps some variant of Liebig's law is more appropriate where mortality is only computed for the most stressful agent.

The large time steps (i.e., monthly to annual) often used in modeling growth in most gap models may preclude consideration of extreme, short-lived events (hours to days), such as severely cold or hot periods and temporary water stresses, in mortality formulations. These stresses, while sometimes included in gap models as sources of exogenous mortality, may induce relatively instantaneous mortality. Such mortality might defy prediction via an annual growth increment, and the stress itself might be missed in simulations where monthly average climate data are employed. Some predictions of future climates point to an increase in the occurrence of short-term, severe temperature and moisture anomalies, so the need to capture such daily events in gap-models will only increase. In addition, the influence of phenology on mortality, such as frost damage, winter desiccation, or early bud burst, also begs a daily time step solution in gap models (Burton and Cumming, 1995; Hanninen, 1995; Lieth, 1974).

Some variations of gap models were created to investigate effects of critical ecosystem processes on tree growth, but these models rarely link those added

processes to mortality. Mineral cycling was extensively simulated in LINKAGES (Paster and Post, 1986), FORTNITE (Aber et al., 1982), and FORNUT (Weinstein et al., 1982). Yet, the only connection of nutrient and carbon dynamics to tree mortality is through depression of diameter growth rate. Moreover, since most nutrient cycling gap models were designed to simulate the effect of declining availability of nutrients on tree growth, can these models then be used to evaluate effects of increasing availability, such as nitrogen saturation, on tree mortality (Aber et al., 1997)? More work is needed to evaluate the direct contribution of important ecosystem dynamics to basic mortality processes. For example, high leaf nitrogen concentrations may heighten the damage from herbivory but lead to high growth rates.

In the end, as long as mortality risk is linked to growth in gap-models, the ability of the model to accurately predict mortality is entirely dependent on an accurate description of growth. If the growth simulation is deficient, then the mortality submodel will also be inadequate.

3.3. EXOGENOUS MORTALITY

For simplicity, simulation of mortality from exogenous disturbances can be represented by three phases: initiation, spread, and effects. The initiation phase is the start of a disturbance in time and space, spread is the advancement of the disturbance agent across the simulation landscape or stand, and the effects phase is the direct and indirect consequences of the disturbance for the ecosystem components. A comprehensive simulation of all three phases is essential to predict tree mortality from exogenous disturbances. This section deals mostly with wildland fire and insects because they are common natural agents of exogenous mortality and probably represent the most complex vegetation-mortality interactions.

3.3.1. *Initiation*

More information is available on the spread and effects of a disturbance, depending on the agent, than on disturbance initiation. This is because initiation for most disturbances is a highly variable process governed by many fine to coarse scale factors. For example, the primary ignition of a wildland fire depends on ignition source (e.g., lightning), fuel loading (amount and type of burnable biomass), fuel condition (i.e., moisture content), and ambient weather (e.g., temperature, wind), all of which are highly variable in time and space. The obvious exceptions are man-caused disturbances such as harvesting that are mostly dependent on easily predicted vegetation characteristics. The onset of natural disturbances is also highly dependent on the condition of the entire landscape. For example, a landscape dominated by balsam fir stands may have a higher probability of experiencing a spruce budworm or tussock moth epidemic than a landscape with a mixed hardwood forest (Stoszek et al., 1981).

Climate change will have a direct impact on disturbance initiation (Crutzen and Goldammer, 1993; Flannigan and van Wagner, 1991; Torn and Fried, 1992; Gardner et al., 1996; Ryan, 1991). For example, fires may be more frequent under future climates because of increased lightning (Price and Rind, 1994), longer and drier fire seasons (Flannigan and van Wagner, 1991; Wotton and Flannigan, 1993), higher fuel loadings (Keane et al., 1996a; Kasischke et al., 1995), and higher winds (Torn and Fried, 1992). But, a mechanistic treatment that relates climatic parameters to disturbance initiation, while desirable, might be difficult given the large number of factors involved, the high variability associated with each factor, and the paucity of data needed to simulate these factors and their interactions. Therefore, initiation is usually simulated using stochastic functions parameterized from historical data (Keane et al., 1996b). A better approach may be to construct stochastic functions that link climate and ecosystem characteristics as independent variables to simulate the initiation of a disturbance (Torn and Fried, 1992). However, this approach will require abundant data and complex algorithms that are difficult to obtain and build for many applications.

A common problem with a stochastic approach for wildland fire initiation is that the simulated fire regimes often don't match observed fire regimes for many landscapes. For example, simulation of numerous ignitions that all result in large fires may overpredict burned area (Baker, 1989; Baker et al., 1991). Some models treat fire frequency, size, and pattern as inputs so simulated fire regimes are predestined to match observed regimes, but this effectively eliminates any influence of weather, topography, and vegetation on the fire regime which would not be desirable for climate change simulations (Baker et al., 1991). Fire synchronicity is another problem in stochastic simulations of fire initiation (Keane and Long, 1998). It is critical that climate and fire ignitions be linked so that the occurrence of fires will be synchronous with dry years, for example. Without this link, the interaction of landscape pattern with fire ignition and spread will be limited (Keane and Long, 1998).

In many regions, vegetation development during the transient phase of climatic change may be most heavily influenced by large-scale tree mortality caused by the initiation of insect and disease outbreaks. The rationale behind this expectation is that drought periods may occur more frequently in future climates and thus many tree species vulnerable to drought will suffer from a lack of vigor (e.g., Waring and Pitman, 1980). Consequently, they will be more susceptible to infestations by pathogens and insects. This feedback will be amplified by favorable environmental conditions for many insects whose life cycles are temperature-dependent (Schwerdtfeger, 1981; Führer, 1993). Despite these potential impacts, a very limited number of gap models explicitly include insect-induced mortality events. One reason for this might be the highly stochastic nature of the host-pathogen system and the initiation of an epidemic. Another reason is that classical gap models cannot account for phenomena that occur on scales larger than the simulated stand (usually between 0.01 and 0.1 ha).

3.3.2. *Spread*

The spread of an exogenous disturbance can be adequately modeled using physical relationships or stochastic algorithms. Often, spread can be ignored if the disturbance agent spreads rapidly and homogeneously across the landscape. However, if the disturbance creates patterns that are important to species dispersal, regeneration, and growth, then a more spatially explicit simulation of disturbance spread is warranted (Urban et al., 1991). Because disturbance spread typically occurs over a short time (hours to weeks), it is probable that climate change will only affect those factors that control spread, and not the process itself, so many current spread algorithms may be used in climate change applications.

3.3.3. *Effects*

Disturbance effects represent the critical feedback of the disturbance to changes in vegetation composition and structure, as well as other ecosystem properties and processes. Effects can be direct, such as plant mortality (see Equation (4), Figure 1), or indirect, such as high regeneration success caused by extensive litter consumption that exposes a desirable mineral soil seedbed (Boyce, 1985). It is important to simulate the entire suite of disturbance effects on the ecosystem because they can directly influence growth-dependent mortality. Most algorithms of disturbance effects are empirical (very few are process-driven or ecophysiologically based), but the independent variables are usually plant or stand biophysical properties that probably will not change under different environments. For example, the relationship of bark thickness to tree diameter, a primary predictor of fire-caused tree mortality, will probably remain unchanged with warming climates.

Our current understanding of the ecophysiological basis of the exogenous mortality relationships is not sufficient as yet to include them in gap models (e.g., Ryan, 1996). Ryan (1996) attempted to quantify fire mortality from ecophysiologically based parameters more closely linked to climate, but found that the high variation in the physiological measurements precluded accurate prediction equations. Specialized growth and reproductive adaptations to disturbances may also complicate process-based mortality prediction, or its complement, survival. For example, severe fires can kill stems of eucalypts, but some species can regenerate from lignotubers to create altered age-size class structures (Gill, 1997). But simulating the heat pulse from fire to the lignotubers to predict mortality might be complicated and problematic (Hungerford, 1990). Empirical fire effects relationships are available for tree mortality, duff consumption, woody fuel reduction, and smoke generation for many North American ecosystems (Reinhardt et al., 1997). Some fire effects, such as fuel consumption, are ready for inclusion in climate change applications because these new formulations replace the narrow empirical equations with process-driven, mechanistic algorithms (e.g., BURNUP; Albini and Reinhardt, 1998).

Numerous empirical models link site and stand characteristics to insect susceptibility (Daniels et al., 1979; Hedden, 1981; Stoszek et al., 1981; Schenk et al.,

1980). As with fire, spread and effect must be distinguished for insect exogenous mortality. Usually empirical models differentiate between infestation probability (i.e., whether a stand is damaged at all) and intensity of the damage (Stage and Hamilton, 1981). Thus, the linkage of empirical insect mortality models with gap models in a spatial domain is a promising approach. However, because of their empirical basis, the extrapolation of these models to changing climates is limited. Most current empirical models defy any mechanistic link to either tree susceptibility or population density of the damaging insect. In a recent simulation study, Lexer and Hönninger (1998) coupled an empirical two-stage stand risk model of Norway spruce susceptibility to bark beetle infestations (*Ips typographus* and *Pytiogenes chalcographus*) with a spatially explicit 3D-gap model. The probability of an insect infestation and damage are estimated from several stand and bioclimatic variables. In this approach, the effect of climate on the insect mortality is represented by the bioclimatic variables, such as soil moisture, used to determine infestation initiation and severity. A thermoenergetic model of bark beetle development that calculates the potential number of insect generations per year is also contained in the model and is linked to climate. Ecophysiological detail devoted to the carbon balance of individual trees might improve the identification of trees that are susceptible to a beetle attack.

4. Future Challenges

A mechanistic treatment of tree mortality in gap models is probably not possible at this time because most causal mechanisms are unknown, complex, and difficult to measure. Although major ecophysiological processes (e.g., photosynthesis, respiration) are well documented, the quantitative relationships between and among mortality processes and environmental variables must often be assumed (Zhang et al., 1994). For instance, very little is known on how the onset and duration of physiological stress affect carbon allocation patterns and eventually plant mortality (Landsberg and Gower, 1997; Loehle, 1987). And, the complexity of the interactions between mortality and environmental conditions, insect attacks, abiotic perturbations, and stand characteristics might be so great that their simulation may be difficult for conventional gap models due to parameter and computer limitations (Dale and Rauscher, 1994).

One wonders whether the inclusion of a mechanistic simulation of poorly understood mortality processes will improve the ability of gap models to explore the effects of climate change on ecosystem dynamics. Often, the simplest structure that meets the user's requirements of resolution and accuracy will be most appropriate (Battaglia and Sands, 1998). Korzukhin et al. (1996) suggested that models should not be overloaded with unnecessary processes, and the processes that are added should be tested in a stepwise manner. Errors resulting from inaccurate ecosystem process representation can be propagated throughout the model, creating faulty

but seemingly believable predictions (Deutschman et al., 1999). Dale et al. (1985) noted that it might be difficult to add physiological detail without greatly increasing computational cost and confounding complexity.

There may be several approaches for improving the simulation of mortality in gap models while waiting for fundamental research on tree mortality to be completed. One option may be to base the computation of mortality on empirically derived formulations. Elegant empirical formulations of tree mortality have been created for many growth and yield forestry models (Monserud, 1975; Hamilton, 1986), and these could be adapted for inclusion in some gap models (Hamilton, 1990). Tree life tables and stand tables that link birth and death rates to environmental drivers may provide a means of encapsulating mortality for use in climate change gap models providing there is an explicit description of the biophysical setting (Harcombe, 1987; Michaels and Hayden, 1987). Another approach would involve modifying the growth module to include a more explicit consideration of a tree's carbon budget. Mortality could be modeled as a function of a tree's ability to maintain a positive carbon balance, among other mortality factors. Burton and Cumming (1995) demonstrated that phenological enhancements to gap models are essential to simulate forest response to climate change.

It is certain that comprehensive mechanistic field and simulation studies investigating the role of physiology in plant death need to be completed before an accurate, physiologically based mortality algorithm can be added to gap models. In addition, it may be essential to expand the size and sophistication of the database of mortality observations to best address the challenge of building and testing better mortality algorithms in gap models (Michaels and Hayden, 1987). The USDA Forest Service's Forest Inventory and Assessment (FIA) provides some critical information towards this end. New studies of tree mortality can be designed along the framework used to study human mortality in medicine. These techniques allow the investigation to focus on individuals that died and compare them with an appropriately chosen sample of living individuals. A gradient approach could also be used to create this database where mortality data are collected from stands along major environmental gradients, such as temperature and evapotranspiration, so that mortality processes can be linked to climatic drivers. Inferring causes of mortality will be another major undertaking. Determining the proximate and ultimate causes of mortality for a large multi-species, multi-age sample of trees, based on post mortem analyses, will be impractical or even impossible, unless currently un-imagined science is developed.

Future gap models should probably be spatially explicit to account for the effects of spatial relationships among competition, regeneration, and resource availability with respect to plant mortality and other life cycle properties (Huston, 1991; Roberts, 1997; Sharpe et al., 1986; Shugart and Seagle, 1985). Comprehensive simulation of important exogenous factors in gap models nearly always requires a spatially explicit approach (Michaels and Hayden, 1987; Turner et al., 1995). As with fire, spatially explicit simulations of vegetation are essential for

improved simulations of the effects of insects on forest dynamics for two reasons (Li et al., 1996). First, landscape structure (i.e., pattern) plays a major role in the initiation and spread of insect epidemics. For example, high radiation loads near open forest edges may enhance insect epidemic mortality (Stoszek et al., 1981; Lexer, 1998). Second, to realistically capture the effects of an insect infestation (e.g., from phloem-feeding bark beetles) on vegetation development (i.e., regeneration development following a disturbance) the heterogeneous site conditions (i.e., light) originating from bark beetle-caused mortality need to be considered (Lexer and Hönninger, 1998).

A factor that has rarely been taken into account in tree population modeling is genetic variability. Under constant climates, variability in plant traits might not be an important process to simulate. But, variability in individual responses to disturbance and climate change will heavily influence future landscape composition and structure (see Scarascia-Mugnozza et al., 1994; Lieth, 1974). Rehfeldt et al. (1999) found that *Pinus contorta* survival was dramatically different across the species' range and that the functional niche was much broader than the realized niche, or natural populations have different climatic optima but occupy suboptimal environments. So, as climates change, species survival will depend on the position of its realized niche in relation to the fundamental niche. Abrams (1994) identified high genetic variation in stress adaptations of trees at the biome and community levels. Genetic variability has been cited as a limitation to gap modeling in other studies (Host and Isebrands, 1994; Keane et al., 1990).

The observed distribution or range of a species represents the realized environmental niche of that species resulting from physiological limits, competitive interactions, and the impacts of herbivores and pathogens. However, gap models, in their simulation of growth and growth-dependent mortality, require the estimation of this fundamental response prior to competition (Malanson et al., 1992). There are no obvious ways that all physiological components of the fundamental response of a plant can be recognized a priori. The physiological control of the lower temperature limit of *Tilia cordata* in northern Europe provides a critical counter-intuitive example for the common assumption that species limits are adequate to determine growth-dependent mortality. Evidence from Pigott (1981, 1989) and Pigott and Huntley (1989) suggests that the northern extent of *Tilia* is controlled by low temperatures that cause the failure of the pollen tube to reach the ovule due to inadequate growth. *Tilia* will live and grow at lower temperatures but without significant sexual reproduction. Improvements in the definition of this fundamental response can be achieved by increasing the explicit recognition in gap-models of the different stages in the life cycle of plants (i.e., seeds, vegetative parts and reproductive parts) (Price et al., 2001). Physiological limitations specific to each stage of the life cycle are known for many species (e.g. photoperiod, chilling and heating effects for flowering), but data are often limiting for tree species especially with regard to plant mortality. Incorporation of these life cycle properties could provide

a sufficient approximation to the fundamental requirements to support growth and mortality simulations in the next generation of gap-models (Malanson et al., 1992).

4.1. VALIDATION

While there is an extensive literature on how to evaluate simulation models, gap-models have yet to be subjected to the full range of testing these methods, especially with respect to mortality (Botkin, 1993; Shugart and West, 1980). Comparative analyses of different gap-model formulations are now being actively pursued (Yaussy, 2000). However, validation of models by comparison with real data on stand composition and structure is needed. Dahlman (1985) noted that gap models are potentially powerful tools for simulating community-level responses to CO_2 increase, but only if properly parameterized and validated. Otherwise, they are just exploratory exercises.

There is the question whether appropriate mortality data are available or can ever be collected to extensively validate the representation of plant death in gap models, especially spatial gap models. Many gap models are validated by initializing the model with historical data and then the model is run to the present to compare outputs with contemporary conditions. Unfortunately, there are probably very few historical validation data sets that can be used to test the predictive power of gap models because of the compounding errors of sampling, synthesis, and extrapolation (Deutschman et al., 1999).

An alternative is to validate model components, such as mortality, separately using appropriate data sets that have been tested for accuracy. This will require an extensive database of field observations documenting the conditions of plant death mentioned above, which will be costly and difficult to compile. Another option is to simulate the error in input data from the output predictions to create response surfaces for model mortality parameters and evaluate if the surface contains sampled reference conditions. A comprehensive sensitivity analysis can also provide ad hoc validation of many model components in lieu of readily available comparison data (van der Voet and Mohren, 1994).

5. Conclusions

Climate change will probably render the generalized treatment of plant mortality in gap models ineffectual. But, we conclude that the current treatment of mortality in gap models is probably the best that can be done given the lack of comprehensive data linking mortality to physiological and environmental conditions. Because the assumptions behind current mortality algorithms will not necessarily be applicable under changing climates, these algorithms must be improved based on extensive new field data to more accurately simulate the interactions of ecosystem processes and climates on mortality. Listed below are some other general recommendations.

- Stochastic mortality functions must be developed that use process-based, mechanistic relationships as predictive variables. For example, fire ignition probabilities could be simulated from climate-based variables.
- Research should be expanded that attempts to mechanistically understand the relationship between ecophysiological processes and plant mortality.
- A comprehensive field database is needed to design, parameterize, and validate gap model mortality algorithms. The proximate and ultimate cause of tree death should be recorded in this database, and tree seedlings and saplings need to be intensively sampled.

Some specific recommendations on modifying gap models for climate change applications are listed below by the three types of mortality.

5.1. INTRINSIC MORTALITY

The intrinsic factors that cause the mortality should be explicitly simulated and not lumped into one stochastic function. Those factors that influence intrinsic mortality, such as fine-scale disturbances and senescence, can be simulated as separate processes when that science matures. Gap model time steps may need to be shortened to days or weeks to account for short-term phenomena, such as frost damage or blow down, that directly affect tree mortality. Meanwhile, the general tree characteristics used in the computation of intrinsic mortality, such as age and height, should be replaced with more suitable variables that correlate closely with those factors that influence old tree mortality, such as diameter or biomass. Intrinsic mortality could be lumped into growth-dependent mortality unless its independent simulation improves the predictive power of the model.

5.2. GROWTH-DEPENDENT MORTALITY

Growth-dependent mortality, above all others, should be modeled as a mechanistic, ecophysiologically based process in gap models to realistically simulate effects of climate change (Norby et al., 2001). But, the lack of scientific understanding and available data preclude a process-based formulation. It is recommended that new research investigate causal mechanisms of tree mortality and that databases be augmented (e.g., FIA database) or created that can support and test this science. For now, diameter increment should be assessed for its ability to determine growth mortality, and it should be replaced, if needed, with species-specific metrics that more realistically reflect the changing growth patterns of a tree, such as amount of respiring biomass.

5.3. EXOGENOUS MORTALITY

Exogenous mortality must be included in gap models if appropriate for the ecosystem or time spans being simulated. Gap models must be made spatially explicit

because exogenous disturbances often affect areas much larger than the simulated patch and other important spatial landscape processes are directly affected by exogenous disturbances (e.g., seed dispersal, hydrology). The creation of a generalized exogenous mortality algorithm, applicable to all ecosystems and exogenous disturbances (e.g., fire, insects, disease), is not currently possible because of differences in the effects, initiation, and spread between the types of disturbances. The initiation phase of many exogenous disturbances is probably the most influenced by changing climates and environments, and it is important that its simulation is closely linked to climate drivers to model future vegetation response. A comprehensive mechanistic simulation of disturbance initiation is currently impracticable because of the inherent complexity of the process across time and space scales, but a stochastic approach that integrates climate variables in the probability functions may suffice until research becomes available. For example, fire frequency could be related to some part of the biogeochemical cycle such as soil moisture, integrated plant moisture stress, or drought-days, for example.

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