Feedback loop dominance analysis of two tree mortality models: relationship between structure and behavior

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Summary Tree mortality is the least understood process of a tree’s lifecycle. Two hypotheses on how mortality progresses in a tree are proposed in the literature: Manion’s gradual decline hypothesis and Bossel’s sudden death hypothesis. Bossel formulated a mechanism in his model, BAUMTOD, as the cause of sudden death phenomena. BAUMTOD, however, cannot be used to generate a causal understanding of Manion’s hypothesis. Therefore, we postulated a causal mechanism for the gradual decline pattern advocated by Manion and modified the BAUMTOD accordingly. The modified model is called BAUMTOD-M. The suggested mechanism concerns the internal imbalance of respiration demand and available photosynthetic supply. We then employed a novel approach to analysis of structure–behavior relationships in mechanistic models, called eigenvalue elasticity analysis (EEA), to pinpoint how the simulated tree responds to various stresses. Specifically, we applied EEA to BAUMTOD and BAUMTOD-M to study the likely structural causes behind tree mortality according to Bossel’s and Manion’s hypotheses, respectively. The analyses of both models suggest that, in the absence of a significant amount of stress, a tree functions as an integrated organism. Growth rates in foliage, feeder roots and respiring permanent biomass are synchronized; however, this coordinated structure is lost if a tree is seriously affected by stress factors. The analyses further suggest that the inability to supply respiration demand plays a crucial role at the onset of mortality in both models. Differences in assumed mechanisms regarding the impact of this inability result in different paths to mortality in each model. This may mean either (1) Bossel’s hypothesis is a special case of Manion’s hypothesis, or (2) there are truly different mechanisms at work in various observed mortality cases. More data and research are needed to clarify these points.

Keywords: dynamic modeling, eigenvalue elasticity analysis, environmental stress, mechanistic models, tree physiology.

Introduction

Identification of the impact of environmental factors on tree mortality is problematic (Franklin et al. 1987). One reason is the difficulty in identifying the role of interacting stresses affecting a tree throughout its lifetime (Kozlowski et al. 1991); another reason is the scarcity of knowledge on how a tree as a physiological system responds to such external pressures (Keane et al. 2001). The potential role of endogenous factors such as genetic potential or age of trees further complicates the matter (Manion 1981). These difficulties are reflected in the models of tree and forest dynamics. Even the most advanced mechanistic forest models treat mortality either as a deterministic or stochastic algorithm (Hawkes 2000). There are, however, exceptions (Keane et al. 2001). For instance, the FORSKA gap model takes a more physiologically based approach by modeling mortality as a function of declining growth (Prentice et al. 1993).

Despite the relatively poor understanding of tree mortality processes, two hypotheses of individual tree mortality are proposed in the literature (Manion 1981, Bossel 1986). Manion (1981) proposed the gradual decline hypothesis, where tree mortality is a process that involves a combination of stress factors. His hypothesis identifies three main types of stresses (predisposing factors, inciting stresses and contributing factors) that lead to changes in tree vigor prior to death. The long-term stresses, called predisposing factors, such as poor soil quality (Johnson 1989, Shen et al. 2001, Moore et al. 2004), weaken a tree and make it susceptible to various inciting stresses. An inciting stress is a short-term event such as a severe drought (Mäkinen et al. 2001, Bigler et al. 2006). Such short-term stresses radically impact the physiological functioning of a tree and severely reduce its already weakened vigor. If tree vigor is sufficiently high, the tree may recover its pre-inciting stress vigor following termination of the inciting stress. If not, the tree enters a death course, during which, it becomes susceptible to contributing factors, such as pathogens. According to Manion, these contributing factors may be viewed as indicators of eventual tree death.

Manion describes seven cases of tree decline progressions that may be explained by his hypothesis. However, as he notes, causal relationships have not been shown in any of the examples. Nonetheless, several recent studies provide evidence of interactions between predisposing and inciting stress factors as causes of tree mortalities (Arriaga 2000, Akema and Futai...
The second mortality hypothesis was suggested by Bossel (1986) to explain the sudden death of trees due to persistent environmental stresses, which he observed while studying how impairment of essential functions of tree growth, such as photosynthesis and feeder root renewal, affects tree growth. Bossel’s hypothesis predicates that long-term stresses of sufficient intensity eventually cause trees to die quickly. Bossel makes his case with simulations of two models of tree dynamics that incorporate a mechanistic formulation of mortality (Bossel 1986). Bossel’s hypothesis differs from Manion’s in three aspects: First, it does not require a short-term stress for a tree to die; second, death occurs after a rapid breakdown of tree health, characterized by its respiring permanent biomass, whereas death occurs as a result of a slower process according to Manion’s hypothesis; and third, Bossel formulated a causal explanation by building mechanistic models, whereas Manion’s description of his hypothesis was mostly qualitative, though supported by data from seven case studies.

Keane et al. (2001) argue for research initiatives to investigate cause mechanisms of tree mortality to establish the role of physiological processes underlying tree mortality and to obtain a physiologically based mortality algorithm to be included in models. Bossel already formulated a mechanism in his models as the cause of sudden death phenomena. His models, however, cannot be used to provide a causal understanding of the gradual decline in Manion’s hypothesis. Therefore, our first objective was to suggest a causal mechanism for the gradual decline pattern advocated by Manion. Our suggested mechanism concerns the internal imbalance of respiration demand and available photosynthate supply. This imbalance was incorporated into the simpler of Bossel’s models, BAUMTOD. The modified BAUMTOD model, representing Manion’s hypothesis, is called BAUMTOD-M. The second objective was to understand how a tree’s physiological structure leads to mortality as a result of the effects of various stresses. To this end, we used the two mechanistic models, representing the two mortality hypotheses, to study mechanisms that might influence the progression of mortality. We applied a novel methodology, based on eigenvalue elasticity analysis, to build a formal connection between each model’s structure and behavior.

Methods

Models

BAUMTOD portrays the essential processes of a tree (i.e., photosynthesis, leaf and root turnover, wood growth through production, and utilization of photosynthate) with a minimal system structure and a minimal set of data. Structural relationships in BAUMTOD represent accepted knowledge about the various processes occurring in a tree (Bossel 1986). Bossel formulated this mechanistic model for conifers based on a more complex model parameterized with data for spruce (Picea abies (L.) Karst.), called SPRUCE (Bossel 1986). Although originally not included, short-term stresses can easily be incorporated into the model formulation (Pedersen 1998a). Thus, in our study, BAUMTOD forms the basis for the two mechanistic models we used to analyze the two tree mortality hypotheses. The models are built in Vensim©, a software program for developing and analyzing high quality mechanistic models (Vensim, U.K.). We used the original model (i.e., BAUMTOD) to study Bossel’s sudden death hypothesis; and we used its slightly modified version, BAUMTOD-M, to study Manion’s gradual decline hypothesis. The main components and parameters of BAUMTOD are listed in Table A1; rate variables that change the masses of main tree components and other variables are given in Table A2. Their abbreviations, dimensions and numerical values are also provided. The equations of the model are given in Appendix A. Details of BAUMTOD and its dynamics can be found in Bossel (1986). A brief description of the model is given in the following two paragraphs.

The three main components of the tree model are LEAF, ROOT, and BIOM (foliage mass, feeder root mass, and respiring permanent biomass, respectively). BIOM consists of the live tissues in the stem, branches, and coarse roots. The amount of photosynthate the simulated tree can produce depends on foliage mass, the specific photosynthetic efficiency of the leaves, and the availability of water and nutrients provided by the feeder roots. Environmental stresses may reduce photosynthetic efficiency resulting in decreased photosynthate production. Water and nutrients are supplied by the feeder roots in proportion to their mass. If the supply is less than the demand, actual photosynthate production will be further decreased. The photosynthate produced is first allocated to meet the respiration requirements of the tree. If the photosynthate supply is inadequate to meet this demand, the tree dies. If, however, the photosynthate supply is adequate, any remaining photosynthate is used for the growth of foliage and feeder root mass and for fructification. If the remaining photosynthate is inadequate to meet these demands, it is allocated in proportion to the demand of each of the three components. If photosynthate is still available, however, it is directed to the production of additional permanent biomass. The model simulates an evergreen tree; hence the foliage is subject to continuous turnover: one-eighth of the foliage is normally replaced each year. However, this turnover rate is increased if the feeder root mass is inadequate to meet the water and nutrient demands of the foliage. Complete annual root turnover is assumed. Similar to the foliage and root turnover, a certain portion of respiring permanent biomass is lost annually but with a much lower rate of 1%.

The respiring permanent biomass, BIOM, may be regarded as an indicator of tree vigor (Pedersen 1998a, Keane et al. 2001). The critical assumption in Bossel’s hypothesis is that when the photosynthate produced becomes less than the respiration demand, the tree immediately dies. Therefore, it is impossible to obtain a gradual decline in respiring permanent
biomass using the original BAUMTOD formulation. Although Pedersen (1998a) reports results similar to gradual decline from simulations of BAUMTOD, they are, in fact, artifacts of the yearly timestep used in his simulations. Because of the importance of an imbalance in respiration demand and available photosynthetic supply in leading a tree to mortality, we considered an alternative assumption regarding the impact of this imbalance on the simulated tree. The new assumption allows the tree to live even after the photosynthetic produce becomes less than the total respiration demand; however, because those tissues whose respiration demand is not met die, the strain on the tree intensifies as the discrepancy between respiration demand and available photosynthetic widens. A simple formulation reflecting this asphyxiation assumption is presented in Equation 1. The biomass loss rate, now the sum of the background loss rate and the loss rate due to asphyxiation which equals the asphyxiation factor except for a unit conversion, increases if respiration demand is not met. If the respiration demand is not satisfied at all, the factor has a value of 1, and if there is no photosynthetic shortage for respiration, it is equal to zero; thus, the corresponding biomass loss rates are 1.01 and 0.01 (i.e., the background loss rate), respectively.

\[
\text{asphyxiation factor} = \begin{cases} 
1 & \text{if (assi produced < dresp),} \\
(1 - \frac{\text{assi produced}}{\text{dresp}}) & \text{else}
\end{cases} 
\]

where assi produced is the actual amount of photosynthetic produced, and dresp is the assimilate demand for respiration.

We modified BAUMTOD to reflect this change and named the modified model BAUMTOD-M. The new loop set resulting from the addition of Equation 1 is shown in Table 1. Three new feedback loops emerged from the introduction of an asphyxiation factor: L17, L18, and L19. These replace loops L13, L14, L15, and L16 in the analysis loop set of BAUMTOD-M.

### Eigenvalue elasticity analysis

Eigenvalue elasticity analysis (EEA) is used to identify structural sources of observed behavior in dynamic mechanistic models. Feedback loops between different components of a model are regarded as the main building blocks of the model structure. The methodology was originally developed in the context of control engineering (Porter and Crossley 1972). Its early applications were limited to linear systems; however, since then, the efficiency of the approach has been demonstrated for several case studies including nonlinear models. These applications range from models of national economies to population dynamics (Saleh 2002, Gonçalves et al. 2005, Güneralp 2005). In addition to mechanistic models, the eigenvalue elasticity concept has been used in matrix population model studies (van Groenendael et al. 1994, Caswell 2001).

In dynamic modeling, the structural sources of behavior can be traced in the changing dominance of the model’s underlying feedback loops (Ford 1999, Richardson 1999). To analyze these structure–behavior relationships relevant hypotheses are formulated and numerical simulation experiments are performed. Nevertheless, formal analysis tools are needed to support this procedure. Eigenvalue elasticity analysis is such a formal tool to uncover those feedback loops that influence model behavior. Consequently, one of the main tenets of this methodology concerns sensitivity analysis. By revealing what parts of the system structure are most influential on certain behavior patterns, the methodology facilitates a more efficient sensitivity analysis. An overview of the methodology is presented in Figure 1 and a more complete explanation is provided in Appendix B.

The basic ideas in calculating feedback loop elasticities are the same as for loop analysis in population studies (van Groenendael et al. 1994). However, there is an important difference between dynamic models and life-history models: the number of feedback loops in a dynamic model can be quite large. Fortunately, there is no need to consider all feedback loops in the model for such analysis. Using concepts from graph theory and network theory, a “shortest independent loop set” that contains every variable and every causal link in the model can be formed (see Oliva 2004 for details of this procedure).

To understand the roles played by different parts of the tree

<table>
<thead>
<tr>
<th>Loop no.</th>
<th>Variable sequence within the loop</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1</td>
<td>BIOM, BMLOS</td>
</tr>
<tr>
<td>L2</td>
<td>LEAF, LFLOS</td>
</tr>
<tr>
<td>L3</td>
<td>ROOT, RTLOS</td>
</tr>
<tr>
<td>L4</td>
<td>LEAF, mprod, wdem, LFLOS</td>
</tr>
<tr>
<td>L5</td>
<td>BIOM, dresp, assr, abiom, BMNEW</td>
</tr>
<tr>
<td>L6</td>
<td>BIOM, dfru, demnd, abiom, BMNEW</td>
</tr>
<tr>
<td>L7</td>
<td>BIOM, dleaf, demnd, abiom, BMNEW</td>
</tr>
<tr>
<td>L8</td>
<td>ROOT, wtrc, assi produced, assr, cass, sroot, RTNEW</td>
</tr>
<tr>
<td>L9</td>
<td>LEAF, mprod, assi produced, assr, cass, sleaf, LFNEW</td>
</tr>
<tr>
<td>L10</td>
<td>LEAF, mprod, root, droot, demnd, cass, sleaf, LFNEW</td>
</tr>
<tr>
<td>L11</td>
<td>ROOT, wtrc, LFLOS, LEAF, mprod, root, droot, sroot, RTNEW</td>
</tr>
<tr>
<td>L12</td>
<td>LEAF, mprod, assi produced, assr, abiom, BMNEW, BIOM, dleaf, sleaf, LFNEW</td>
</tr>
<tr>
<td>L13</td>
<td>LEAF, mprod, root, droot, demnd, cass, sroot, RTNEW, ROOT, wtrc, LFNEW</td>
</tr>
<tr>
<td>L14</td>
<td>LEAF, mprod, assi produced, assr, abiom, BMNEW, BIOM, dfru, demnd, cass, sleaf, LFNEW</td>
</tr>
<tr>
<td>L15</td>
<td>LEAF, mprod, root, droot, demnd, abiom, BMNEW, BIOM, dleaf, sleaf, LFNEW</td>
</tr>
<tr>
<td>L16</td>
<td>BIOM, dleaf, sleaf, LFNEW, LEAF, mprod, root, droot, sroot, RTNEW, ROOT, wtrc, assi produced, assr, abiom, BMNEW</td>
</tr>
<tr>
<td>L17</td>
<td>BIOM, dresp, asphyxiation factor, BMLOS</td>
</tr>
<tr>
<td>L18</td>
<td>BIOM, dleaf, sleaf, LFNEW, LEAF, mprod, assi produced, asphyxiation factor, BMLOS</td>
</tr>
<tr>
<td>L19</td>
<td>BIOM, dfru, demnd, cass, sroot, RTNEW, ROOT, wtrc, assi produced, asphyxiation factor, BMLOS</td>
</tr>
</tbody>
</table>

1 These loops belong to the set of BAUMTOD and not that of BAUMTOD-M.
2 These loops belong to the set of BAUMTOD-M and not that of BAUMTOD-M.
in leading it to mortality under the two hypotheses, we performed an EEA on the simulation output from the two mechanistic models. In the analysis of the two models, each state variable (i.e., each main component of the tree) is treated separately. Thus the behavior modes that compose the behavior of each component (foliage, feeder root, and respiring permanent biomass) can be identified. Then feedback loops that strongly influence the behavior of each component are identified. These loops represent the tree’s dominant physiological processes. Initially, we studied two scenarios with both models: first, when there is no predisposing stress corresponding to normal growth mode of trees (= no stress scenario), and second, when there is moderate predisposing stress leading to stagnation or very slow growth or decay (= moderate stress scenario). These conditions are simulated by setting leaf photosynthetic efficiency (eff) to 1 and 0.7, respectively. Then, Bossel’s hypothesis is analyzed on BAUMTOD by setting the efficiency to 0.55. This eff value represents significant predisposing stress (= breakdown scenario). On the other hand, Manion’s hypothesis is studied on BAUMTOD-M by setting the efficiency to 0.8 (representing the effect of predisposing stress except throughout the fifth year in which it is 0.4 to represent the effect of an inciting stress (= gradual decline scenario). The eff values selected are based on values used in earlier studies (Bossel 1986, Pedersen 1998a).

Results

The resulting dynamics and dominant processes under the no stress and moderate stress scenarios are the same for both models. Hence, the results are shown only on the original model, BAUMTOD. There are 16 loops in the shortest independent loop set (SILS) of the model (Table 1).

The behaviors exhibited by the main components of the tree under no stress (eff = 1), moderate stress (eff = 0.7), and breakdown scenarios (eff = 0.55) are given in Figure 2. To illustrate a typical output of the method, the pattern of behavior modes under the breakdown scenario (eff = 0.55) is presented in Appendix C. Their relative contributions to the behavior of foliage (LEAF) and the resulting loop dominance dynamics over time are given in Appendix C. The presentation of the results and the following discussion are focused on the effects of the dominant physiological processes (i.e., the feedback loops) on the behavior of each main component under different stress conditions and different mortality models.

No stress scenario (eff = 1) All components of the tree show growth. The production of photosynthate is limited by the feeder root mass (ROOT). Hence, the foliage (LEAF) mass has no direct influence on the amount of photosynthate produced (assr produced). In the normal growth scenario, all components are influenced by the same loops (Figure 3A). In Figures 3 and 5, boxes represent the main components of the simulated tree; double-lined arrows with values add to or subtract from these components. Black and gray arrows are dominant and non-dominant causal links, respectively. Hence, the loops that are most influential at one time or another during simulation are depicted with black arrows. Loops L2 and L3 are responsible for the turnover of leaves and feeder roots, respectively. Despite the counteracting influence of turnover of foliage and feeder roots, there is steady growth in all parts of the tree driven by loop L16. An otherwise serious problem in the formulation of BAUMTOD is the absence of a self-regulating mechanism that would potentially bring the state of the tree to an equilibrium in the future. This drawback maybe disregarded in this study, as we are interested in understanding the internal dynamics eventually leading to death under the influence of stress factors. It is a positive feedback loop and includes all three main components.

Moderate stress scenario (eff = 0.7) Tree components cannot grow under conditions of moderate stress (Figure 2). Feeder root mass, apparently higher than the tree could support under predisposing stress, shrinks. This is caused by its turnover loop (L3) (Figure 3B). On the other hand, foliage and respiring permanent biomass (BIOM) are driven by the turnover loop of foliage (L2), respiration demand (L5) loop, and loop L12. Initially, biomass decreases slowly and the respiration demand (dresp) decreases too. Everything else being equal, this would increase the remaining photosynthate (assr) that would be used toward more foliage and root growth, more fructifica-
tion and, through loop L5, possibly more permanent biomass growth. However, under the influence of its turnover loop (L2), foliage decreases. This leads to a decrease in photosynthate produced (assi produced) that is larger than the decrease in the respiration demand (dresp). Thus the interplay of loops L5 and L12 causes less remaining photosynthate at each turn for foliage growth and biomass growth (Figure 3B). As a result, both respiring permanent biomass and foliage mass decrease until about the ninth year of the simulation. Then the second phase begins in which contraction of feeder root mass slows. During this phase, coordination between the components of the simulated tree is restored and their masses diminish slowly. Loop L1, which drives the background decay of respiring permanent biomass, plays no significant role in the first two scenarios.

**Breakdown scenario (eff = 0.55)** The results of this scenario reveal the dominant processes leading to death based on the assumptions of Bossel’s hypothesis. There are two phases leading to the sudden death of the tree (Figure 2). In the first phase, foliage mass restricts photosynthate production. Foliage is slowly diminishing in this phase partly because of turnover (loop L2) (Figures 2A and 5A). However, both inefficient production and biomass decay also contribute to the decrease in
foliage mass: the former, through the influence of loop L9; the latter through the influence of respiring permanent biomass decay loop L1. Detached from the other two, feeder root mass decays under the influence of its turnover loop, L3. In time, feeder root mass drops so low that it, rather than foliage, becomes the limiting component in photosynthate production. Then, loop dominances shift abruptly marking the beginning of the next phase. This happens sometime between the second and third years of the simulation (Appendix C; Figure C3).

The decay in foliage and feeder root mass accelerates in this phase. For foliage, this happens toward the fourth year of simulation, partly because of the decrease in photosynthate production (assi produced). The decrease shows its effect on new foliage and feeder root growth through the way assimilate is distributed (cass), because loop L8 acts on the growth of these two components through actual supplies of assimilate to the foliage and feeder roots (sleaf and sroot), respectively (Figure 5A). Its turnover loop (L3) diminishes the existing feeder root mass, causing further reduction in photosynthetic output. The sharp increase in the rate of decrease in foliage mass toward year four of the simulation is caused by reduced transpiration flow (wtrc), which, in turn, is caused by dwindling feeder root mass. This reduction eventually leads to an insufficient supply of water and nutrients to foliage, resulting in increased foliage loss rate (LFLOS) (see model equations in Appendix A).

In the absence of available photosynthate, respiring permanent biomass is decoupled from the rest of the system from the beginning. It slowly decays under the influence of its own decay loop (L1). However, when photosynthate production (assi produced) drops so low that it cannot satisfy the respiration demand (dresp), the tree dies suddenly (Figure 2).

Gradual decline scenario (eff = 0.4 in Year 5; 0.8 otherwise)

The simulation of BAUMTOD-M generates dynamics that are markedly similar to Manion’s hypothesis as suggested by Johnson (1989) (Figure 4). Initially, foliage mass and respiring permanent biomass exhibit slow growth, whereas feeder root mass decays. This continues until year five of the simulation, after which the combined effects of predisposing and inciting stresses become manifest. Thus, although respiring permanent biomass begins to decrease with the initiation of the inciting stress, the sharp drop as predicated by Manion’s hypothesis occurs only after the inciting stress has terminated. Such delayed responses are not uncommon in tree physiology (Kozlowski et al. 1991).

The loop dominance analysis shows that, during the initial slow growth phase, the tree components are under the influence of the same loops that were influential in the moderate stress scenario (eff = 0.7) (Figures 3B and 5B). The relative influences of the loops differ, however, between the two scenarios. In the gradual decline scenario (eff = 0.8/0.4), the relative influence of loop L12 is slightly higher than those of the turnover loops. This results in slow but steady increases in all three components. In case of ROOT, these are decrease initially because its initial value is too high relative to the demands of the other two components under the predisposing stress. Thus, it is...
under the influence of its turnover rate initially. In time, however, it synchronizes with the two other components and begins to slow increase as well. During year five of the simulation, the interactions between the components virtually fail. There is just enough photosynthate production to satisfy the respiration demand and for a little growth of the tree components. However, the new growth is insufficient to compensate for the losses from turnover. Consequently, each component is virtually isolated and decreases under the influence of its turnover loop. The loss of feeder root mass is the most prominent because it has the highest turnover rate (Figure 4). Unlike the other components, foliage is under the influence of loop L9 as well (Figure 5B). This positive loop essentially drives new foliage growth based on the availability of photosynthesize after respiration demands are met, reflecting the fact that photosynthesize production is limited by foliage during this phase. In the presence of an inciting stress, loop L9 initiates a decline in foliage mass causing it to decline faster and faster. In summary, the lower the production of photosynthesize, the lower the productivity of new foliage; these results in lower foliage mass, which in turn leads to even lower production.

Shortly after the inciting stress has terminated, the continuing decrease in photosynthesize production leads to a shortage in supplying the respiration demand. At this point, asphyxiation occurs and loop L17 becomes dominant (Figure 5B). This accelerates the death of permanent live tissues, reflected as a faster decrease in respiring permanent biomass (Figure 4). Because all production is directed to satisfying respiration demand, there is no new growth in any component. Furthermore, because leaf efficiency (eff) is restored to its pre-inciting stress value, the water and nutrient demands of foliage increase. However, feeder roots with their much lower mass cannot meet this demand. Consequently, the foliage turnover rate increases. Thus both root turnover loop L3 and foliage turnover loop L2 influence the drop in foliage mass. This results in an even faster decrease in foliage mass (Figure 4). Feeder root mass, in contrast, is unaffected by the other components and slowly approaches zero, driven by its turnover loop L3 (Figure 5B).

Discussion
The three components of the simulated tree are fully coordinated only when there is little or no stress. Under sufficiently stressful conditions, however, this integrity is threatened, and the coordination among components degrades as the simulated tree undergoes increasing stress.

We compared two aspects of the two hypotheses on how mortality progresses in a tree. The first aspect on which the two hypotheses were compared is the likely causal mechanisms leading to mortality. Although the imbalance between production of photosynthesize and respiration demand has a large effect in both models, the difference is in the way the tree is assumed to respond to this shortage in each model. In the breakdown scenario (eff = 0.55), any shortage in photosynthesize to supply respiration demand is deemed to be so critical for the tree’s survival that it leads to sudden death. However, a different outcome results when it is assumed that the impact on the tree of the imbalance between respiration demand and available photosynthesize supply is proportional to the extent of this imbalance. In this scenario, the simulated tree exhibits dynamics that are in accordance with what Manion’s mortality hypothesis seems to imply.

Manion argues that pathogens, secondary insects or other agents may play an active role as contributing factors during this phase, effectively leading to the tree’s death. However, our study suggests that an internal imbalance between respiration demand and available photosynthesize supply may, in large part, be responsible for the slow but steady approach to mortality after an inciting stress (Pedersen 1998b, Keane et al. 2001). This finding, if correct, supports the idea of Mueller-Dombois (1987) that contributing factors may not be necessary to cause tree death. Nevertheless, the validity of our finding needs to be further scrutinized, because the particular species, the timing, and even the location would certainly affect the outcome of the response to this imbalance.

The second aspect on which the two hypotheses were compared is the relative importance of predisposing and inciting stresses in triggering mechanisms that lead to mortality. The analyses imply that the severity of inciting stress required to cause tree mortality depends on the severity of the predisposing stress. Moreover, an inciting stress may not be required if there is a sufficiently severe predisposing stress. Pedersen (1998a) was the first to make the case for the possible interaction between predisposing and inciting stresses based on a mechanistic model (i.e., BAUMTOD). He showed that the same inciting stress may cause mortality of a tree under a sufficiently severe predisposing stress, but not of another tree subject to a less severe predisposing stress. Evidence from recent field studies directed to better understand the causes of mortality in forest stands tends to support this conjecture. For example, in a study on Picea abies (Norway spruce) in southern Finland, trees on nutrient-rich soils regained their vigor after a long period of drought, whereas tree on nutrient-poor soils continued to decline until mortality (Mäkinen et al. 2001). In another study, Wyckoff and Clark (2002) report anecdotal evidence that diseased individuals of shade-intolerant Cornus florida L. (flowering dogwood) growing beneath closed canopies are more predisposed to mortality than those growing in high-light environments. van Mantgem et al. (2003) report the role of fire as an inciting stress in burned stands of Abies concolor (Gord. and Glend.) Lindl. (white fir) in the Sierra Nevada of California. They also show that knowledge of pre-fire growth rate is a useful indicator of whether a fire event will ultimately lead to mortality. Further evidence of this interplay can be found in several other studies (Jimenez et al. 1985, Pfensham 1998, Arriaga 2000, Lwanga 2003, Suarez et al. 2004, Akema and Futai 2005).

The literature suggests that the dynamics of forest decline differ among species as well as among regions (Waring 1987, Kobe and Coates 1997, Karev 2003). For example, the duration of growth decline prior to mortality seems to differ among species (Wyckoff and Clark 2002). These differences should
be taken into account in multi-species forest simulation models. As an early example of this approach, in the FORENA gap model, mortality is modeled as a growth-dependent process in which tree species become increasingly vulnerable to mortality if diameter growth remains below a species-specific threshold for three or more consecutive years (Solomon 1986). Similar approaches for representing mortality have been adopted in studies with the FORCLIM model (Bugmann and Solomon 2000). However, it would be unreasonable to conclude that a single mortality hypothesis is applicable for every situation (Pedersen 1998a). Similarly, the two mortality hypotheses evaluated in this study should not be considered exhaustive. Nevertheless, they provide alternative explanations for understanding the mechanism of tree mortality under different assumptions about the impacts of external stresses on trees and the response of trees to these stresses. There is evidence of weakened trees dying soon after an inciting stress (Lwanga 2003). There is also evidence of weakened trees gradually dying, the duration of which seems to vary depending on the species and other characteristics of the specific incident (Pedersen 1998b). In addition, there is evidence suggesting that a predisposing stress that is severe enough could cause mortality without an inciting stress (Moore et al. 2004). On the whole, it seems that, in the field, there is a continuum between the two hypotheses. Moreover, these hypotheses and the corresponding models can serve as frameworks to further analyses on causes and mechanisms of tree mortality. Improved understanding of the physiological responses of trees to environmental stresses would also help in effectively linking mortality to new environmental conditions associated with climatic change (Keane et al. 2001).

We have laid out structural causes underlying two possible explanations of tree mortality that have been proposed in the literature. We speculate that the structural mechanism suggested in BAUMTOD-M is more realistic than the sudden death mechanism suggested in BAUMTOD. If this is the case, Manion’s gradual decline hypothesis may be a general framework, which also includes Bossel’s hypothesis. For example, if a tree undergoes a series of very severe inciting stresses, even with a mechanism in BAUMTOD-M, it may quickly die.

Storage of carbon (assimilate) is not represented in the models we considered. Its exclusion may not matter much for two reasons. First, carbon storage of a tree under predisposing stress would probably already be low at the onset of an inciting stress (van Mantgem et al. 2003). Second, the result of having a carbon stock would probably only delay the occurrence of mortality (Hanson and Weltzin 2000) or would change the required combination of predisposing and inciting stresses for the tree to be driven to death, or both. Still the availability of assimilate supply for defense is shown to be important in some empirical studies (Loehle 1988). Therefore, a separate assimilate stock could be considered in more elaborate studies of tree mortality in the future.

Although a single inciting stress-related event is simulated in this study, the frequency and intensity of the short-term signals may differ from place to place. These variations need to be incorporated in application-oriented simulation studies, as well as future theoretical studies (Fensham 1998, Monserud and Sterba 1999). However, there is also need to be wary about including unnecessary processes in models (Kozlowski et al. 1996). The simplicity of the models used in this study should be viewed in this light.

Conclusions

Two simulation models are used as mechanistic representations of two hypotheses on tree mortality. Their analyses improved our understanding on what causal mechanisms might be most influential at the onset of tree mortality. A novel method for analysis of dynamic feedback models was used to uncover the physiological dynamics of a tree in the presence of varying degrees of stress under the assumptions of two models. The differences as well as the similarities in progression of tree mortality between the two models were examined.

We found that imposing stress on a tree disturbs its otherwise integrated functioning. The failure of different components of a tree to grow synchronously can, therefore, be seen as an onset of mortality. The findings suggest that the unavailability of sufficient photosynthate to supply respiration demand is fatal in both mortality models. The models differ in their assumptions on how this shortage acts on the tree. Bossel’s sudden death hypothesis assumes that any shortage brings immediate death to a tree. Manion’s gradual decline hypothesis, however, posits that the tree lives for a few years after the damaging effect of an inciting stress. Although the shortage of assimilates to supply respiration demand is not explicitly mentioned in Manion’s hypothesis, the dynamic model used to represent his mortality hypothesis made use of this shortage. The resulting dynamics suggest that the way this imbalance impacts the tree may be the cause of gradual decline (Kozlowski et al. 1991). The exact nature of the impact and the relationship between the two hypotheses need to be further scrutinized. There are also other factors that are likely to be important (e.g., assimilate supply for defense) and should be included in future studies to reach a more comprehensive understanding of the causal mechanisms underlying tree mortality.

Models such as those considered here are crucial for developing a theoretical understanding of the dynamic response of a tree’s structure under normal and stressed conditions. The increased availability of relevant field data coupled with the development of better models will generate a deeper understanding of the physiological processes leading to tree mortality.

Acknowledgments

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References


Appendix A. Definitions and equations of the models

Table A1. State variables (main tree components) and parameters for the models.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Value (dimension)</th>
<th>Parameter</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIOM</td>
<td>1 (assimilate)</td>
<td>Respiring permanent biomass</td>
</tr>
<tr>
<td>LEAF</td>
<td>1 (assimilate)</td>
<td>Foliage mass</td>
</tr>
<tr>
<td>ROOT</td>
<td>1 (assimilate)</td>
<td>Feeder root mass</td>
</tr>
<tr>
<td>eff</td>
<td>1 (–)</td>
<td>Leaf photosynthetic efficiency</td>
</tr>
<tr>
<td>nbml</td>
<td>0.01 (1/year)</td>
<td>Normal loss constant</td>
</tr>
<tr>
<td>ndl</td>
<td>8 (year)</td>
<td>Number of years a given need remains on the tree</td>
</tr>
<tr>
<td>nrtto</td>
<td>1 (–)</td>
<td>Natural root turnover constant</td>
</tr>
<tr>
<td>rdam</td>
<td>1 (–)</td>
<td>Normal root decay constant</td>
</tr>
</tbody>
</table>

1 For the state variables, the value shown is the initial value.

Table A2. Rates and other variables for the models.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description of rate or other variable (dimension)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMNEW</td>
<td>Respiring permanent biomass growth rate (assimilate/year)</td>
</tr>
<tr>
<td>BMLOS</td>
<td>Respiring permanent biomass loss rate (assimilate/year)</td>
</tr>
<tr>
<td>LFNEW</td>
<td>Foliage growth rate (assimilate/year)</td>
</tr>
<tr>
<td>LFLOS</td>
<td>Foliage loss rate (assimilate/year)</td>
</tr>
<tr>
<td>RTNEW</td>
<td>Feeder root growth rate (assimilate/year)</td>
</tr>
<tr>
<td>RTLOS</td>
<td>Feeder root loss rate (assimilate/year)</td>
</tr>
<tr>
<td>abiom</td>
<td>Photosynthetic remaining for biomass growth (assimilate)</td>
</tr>
<tr>
<td>asphyxiation factor</td>
<td>Respiring permanent biomass loss factor due to shortage in assimilate supply to meet respiration demand (–)</td>
</tr>
<tr>
<td>assi produced</td>
<td>Actual amount of photosynthate (i.e., assimilate) produced (assimilate)</td>
</tr>
<tr>
<td>assr</td>
<td>Remaining assimilate (assimilate)</td>
</tr>
<tr>
<td>cass</td>
<td>Assimilate distribution factor (–)</td>
</tr>
<tr>
<td>demnd</td>
<td>Total demand on renewal (assimilate)</td>
</tr>
<tr>
<td>dfru</td>
<td>Assimilate demand for fructification (assimilate)</td>
</tr>
<tr>
<td>dleaf</td>
<td>Assimilate demand for leaf growth (assimilate)</td>
</tr>
<tr>
<td>dresp</td>
<td>Assimilate demand for respiration (assimilate)</td>
</tr>
<tr>
<td>droot</td>
<td>Assimilate demand for feeder root growth (assimilate)</td>
</tr>
<tr>
<td>mprod</td>
<td>Photosynthetic production capacity (assimilate)</td>
</tr>
<tr>
<td>root</td>
<td>Required feeder root mass (assimilate)</td>
</tr>
<tr>
<td>sfru</td>
<td>Actual assimilate supply for fructification (assimilate)</td>
</tr>
<tr>
<td>sleaf</td>
<td>Actual assimilate supply for leaf growth (assimilate)</td>
</tr>
<tr>
<td>sroot</td>
<td>Actual assimilate supply for feeder root growth (assimilate)</td>
</tr>
<tr>
<td>wdem</td>
<td>Normalized water (and nutrient) demand (assimilate)</td>
</tr>
<tr>
<td>wtrc</td>
<td>Actual (nutrient and water) transport capacity of the root system (assimilate)</td>
</tr>
</tbody>
</table>

Table A3. Equations of BAUMTOD. The time-step is 0.02 years; and the integration method is Euler.

State variables (Main tree components)

BIOM = INTEG (BMNEW – BMLOS)
LEAF = INTEG (LFNEW – LFLOS)
ROOT = INTEG (RTNEW – RTLOS)

Rates of change (flows) of state variables

BMNEW = abiom * 0.075
BMLOS = nbml * BIOM
LFNEW = sleaf * (1 / ndl) * (1 / 0.15)
LFLOS = If ((wdem / wtrc)>1.2) then (LEAF * ((1 / ndl) + (1 – (wtrc / wdem)))) else (LEAF / ndl)
RTNEW = sroot * (1 / 0.065)
RTLOS = rdam * nrtto * ROOT

Auxiliary variables

abiom = If (assr>= demnd) then (assr – demnd), else 0
assi produced = If ((wtrc / wdem)<1) then wtrc else mprod
assr = If ((assi produced – dresp)>0) then (assi produced – dresp), else 0
cass = If (assr>= demnd) then 1, else (assr / demnd)
demnd = dfru + dleaf + droot
dfru = 0.085 * BIOM
defu * 0.3 * BIOM
root = rdam * nrto * root * 0.065
mprod = LEAF * eff
root = mprod
sfru = dfru * cass
sleaf = dleaf * cass
sroot = droot * cass
wdem = mprod
wtrc = ROOT

Appendix B. Mathematical foundations of Eigenvalue elasticity analysis

Any dynamic model based on differential equations can be represented in matrix form (Equation B1). Each entry in the matrix represents a compact link between the state variables of the model. Specifically, each matrix entry is a compact net gain that represents the slope of the relationship between the net rate of the state variable \( p \) and the state variable \( q \), i.e., the change in the net rate of the state variable \( p \) in response to a change in the level of the state variable \( q \), \( \frac{\Delta p}{\Delta q} \). If the model under study is linear, the matrix is constant. If nonlinear, the entries of the matrix may change throughout the simulation. At sufficiently small time intervals (e.g., the simulation time step), however, the entries of the matrix of a nonlinear model can be assumed to be constants. In other words, the dynamics of the nonlinear model is approximated by a series of linear models with varying entries in their matrices. Then the EEA can be applied to these series of matrices produced by simulation (Saleh 2002, Güreralp 2006).
The eigenvalues of the matrix $G$ represent behavior modes the system is capable of generating. These behavior modes that may be present in a model are: first, monotonic convergent behavior mode (i.e., a real negative eigenvalue); second, monotonic divergent behavior mode (i.e., a real positive eigenvalue); third, sustained oscillatory behavior mode (i.e., a complex conjugate eigenvalue pair with zero real parts); fourth, convergent oscillatory behavior mode (i.e., a complex conjugate eigenvalue pair with negative real parts); and fifth, divergent oscillatory behavior mode (i.e., a complex conjugate eigenvalue pair with positive real parts).

The model behavior is a linear combination of all behavior modes represented by the eigenvalues of the system (Equation B2). During the simulation, the dominance of each behavior mode may change over time. These changes, in turn, are reflected in the overall behavior pattern of the system. Because behavior modes can be assumed to be linearly independent at small time intervals, the contributions of behavior modes on the overall behavior at any instant can be examined separately.

\[
G = \left( \begin{array}{cccc}
\frac{\delta x_1}{\delta x_1} & \cdots & \frac{\delta x_1}{\delta x_n} \\
\vdots & \ddots & \vdots \\
\frac{\delta x_n}{\delta x_1} & \cdots & \frac{\delta x_n}{\delta x_n}
\end{array} \right) \quad (B1)
\]

where $\Delta s_i$ is the contribution of $i$th behavior mode to the behavior of interest.

The entries of the matrix $G$ are determined by the model variables. Therefore, a change in the value of a particular variable changes values of certain entries. This, in turn, modifies the behavior modes of the model (i.e., changes the eigenvalues of the model). Thus, in the EEA, eigenvalues and eigenvectors characterize the complete relation between the model structure and behavior (Figure 1).

The contribution of a behavior mode to the behavior of interest (Equation B3) is the slope vector, $r_i$ is the right eigenvector associated with the $i$th behavior mode, and $\alpha_i$ is the coefficient of the $i$th behavior mode.

\[
s = \alpha_1 r_1 + \cdots + \alpha_n r_n \quad (B2)
\]

where $s$ is the slope vector, $r_i$ is the right eigenvector associated with the $i$th behavior mode, and $\alpha_i$ is the coefficient of the $i$th behavior mode.

The contribution of a behavior mode to the behavior of interest is construed by calculating the change in the slope component of the behavior of interest along the associated eigenvector of the behavior mode between two consecutive analysis times. Normalizing the individual contributions by the sum of the absolute values of all contributions gives the relative contributions (Equation B3).

\[
c_i = \frac{\Delta s_i}{\sum_{i=1}^{n} |\Delta s_i|} \quad i = 1..n \quad (B3)
\]

where $\Delta s_i$ is the contribution of $i$th behavior mode to the behavior of interest.

The entries of the matrix $G$ are determined by the model variables. Therefore, a change in the value of a particular variable changes values of certain entries. This, in turn, modifies the behavior modes of the model (i.e., changes the eigenvalues of the model). Thus, in the EEA, eigenvalues and eigenvectors characterize the complete relation between the model structure and behavior (Figure 1). In Figure 1, $e_{ij}$ is the elasticity of behavior mode $i$ to feedback loop $k$, $c_i$ is the contribution of behavior mode $i$ to the behavior of interest, which may be the behavior of any variable in the model. In particular, causal links with large elasticities are significant. Most influential feedback loops in the model are those formed by such causal links. These loops define a dominant subset of the model structure (Forrester 1982).

The sensitivity matrix $S$, of the eigenvalue is equal to the product of the $i$th left eigenvector and the $i$th right eigenvector of the gain matrix, $G$ (Equation B4) (Caswell 2001, Saleh 2002). A formulation of elasticity of eigenvalue to matrix entry $g_{pq}$ is then given in Equation B5.

\[
S_i = 1, r_i' \quad (B4)
\]

\[
e_{pq,i} = \frac{l_i(p) r_i(q) g_{pq}}{\lambda_i} \quad (B5)
\]

where $l_i(p)$ is the $p$th element of the $i$th left eigenvector ($1 \times n$ vector) and $r_i(q)$ is the $q$th element of the $i$th right eigenvector ($1 \times n$ vector).

The elasticities to matrix entries are then related to the elasticities to feedback loops using a membership matrix. The matrix, also called directed cycle matrix, contains information on which links are located on which loops. The relative contributions computed in Equation B3 are then used as weights in computing the weighted elasticity which measures the overall influence of a feedback loop on the behavior of interest (Equation B6) (Figure 1).

\[
oe_k = \sum_{i=1}^{n} c_i e_i' \quad k = 1..K \quad (B6)
\]

where $e_i'$ is the elasticity of $i$th behavior mode to loop $k$. 
Appendix C. Output of the methodology for the breakdown scenario

Figure C1. Pattern of behavior modes over time under breakdown conditions (Eigenvalues in the figure represent three behavior modes).

Figure C2. Relative contributions of behavior modes to the behavior of foliage, LEAF (Cont'ev 1: contribution of behavior mode 1 to behavior of LEAF; Cont'ev 2: contribution of behavior mode 2 to behavior of LEAF; Cont'ev 3: contribution of behavior mode 3 to behavior of LEAF).

Figure C3. Loop dominance dynamics acting on behavior of foliage, LEAF.