Feedback loop dominance analysis of two tree mortality models

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Abstract

Tree mortality is the least understood process of a tree's life-cycle. 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 already formulated a mechanism in his model, BAUMTOD, as the cause of sudden death phenomena. His model, however, cannot be used to generate a causal understanding to Manion's hypothesis. Therefore, a causal mechanism for the gradual decline pattern, advocated by Manion, is suggested. BAUMTOD is modified accordingly and the modified model is called BAUMTOD-M. The suggested mechanism concerns the internal imbalance of respiration demand and available photosynthate supply. Then, the eigenvalue elasticity analysis (EEA) is employed to pinpoint how a simulated tree responds to various stresses. It is applied 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. Growths 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 cause 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 is needed to clarify these points.

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

1. Introduction

The 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); the other is the scarcity of knowledge on how a tree as a physiological system responds to such external pressures (Keane et al. 2001). These difficulties reflect on the modeling of tree/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).

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 that lead to tree vigor change prior to death. First, called predisposing factors, are the long-term stresses such as poor soil quality (Johnson 1989, Moore et al. 2004). They weaken a tree and make it susceptible to various other 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 the tree is healthy enough (i.e., its vigor is sufficiently high) it may recover its pre-inciting stress vigor. If not, the tree basically 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 the eventual tree death.

Manion gives an account of 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, recently, studies presenting evidence of interaction between predisposing and inciting stress factors as causes of tree mortalities at several localities appear in the literature (Magnussen et al. 2005). Pedersen (1998*b*) found evidence of growth reductions at the onset of the mortality of overstory oaks (*Quercus* spp. L.) in the midwestern United States. Mäkinen et al. (2001) report decade-long growth decline before the incidence of mortality in damaged Norway spruce stands in southern Finland.

The second mortality hypothesis was suggested by Bossel (1986) to explain the sudden death of trees due to persistent environmental stresses. His aim was to study 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 quickly die. 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 trees to die; second, the death occurs after a rapid break-down of the tree health, characterized by its respiring permanent biomass, whereas the 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, while Manion's description of his hypothesis was mostly qualitative, though supported by data from several case studies.

Keane et al. (2001) argue for research initiatives to investigate causal mechanisms of tree mortality to properly establish the role of physiological processes in tree mortality and 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 to the gradual decline in Manion's hypothesis.

Therefore, the first objective of this paper is to suggest a causal mechanism for the gradual decline pattern advocated by Manion. The suggested mechanism concerns the internal imbalance of respiration demand and available photosynthate supply. It is incorporated by modifying the simpler of Bossel's models, BAUMTOD, accordingly. The modified model is called BAUMTOD-M. The second objective is to understand how a tree's physiological structure may lead to mortality due to the effects of various stresses. To this end, the two mechanistic models, representing the two mortality hypotheses, are used to study those mechanisms that might be influential on the progression of mortality. Eigenvalue elasticity analysis (EEA) is used to build a formal connection between each model's structure and behavior (Güneralp and Gertner 2006).

2. Methods

2.1. Eigenvalue elasticity analysis

The eigenvalue elasticity analysis (EEA) is used to identify structural sources of observed behavior in dynamic mechanistic models. The methodology has originally been developed in the context of control engineering (Porter and Crossley 1972). However, its early applications have been limited to linear systems. Since then, the efficiency of the approach is demonstrated for several case studies including nonlinear models (Kampmann 1996, Saleh 2002, Gonçalves et al. 2005, Güneralp 2005). In addition to mechanistic models, the eigenvalue elasticity concept is used in matrix population model studies (van Groenendael et al. 1994, Caswell 2001).

The basic ideas in calculating feedback loop elasticities are the same as that of 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, one does not 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 the details of this procedure). In the following, mathematical foundations of the EEA are presented. For a comprehensive review of the basics of the methodology refer to Kampmann (1996), Saleh (2002), and Güneralp (2005). In the application of the EEA in this study, the procedure proposed in Güneralp (2005) is adopted.

Any dynamic model based on differential equations can be represented in matrix form (Eq. 1). 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, $\partial \dot{x}_p / \partial x_q$. 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, Güneralp 2006).

$$\mathbf{G} = \begin{bmatrix} \partial \dot{x}_1 / \partial x_1 & \cdot & \cdot & \partial \dot{x}_1 / \partial x_n \\ \cdot & \cdot & & \cdot \\ \cdot & & \cdot & \cdot \\ \partial \dot{x}_n / \partial x_1 & \cdot & \cdot & \partial \dot{x}_n / \partial x_n \end{bmatrix} \quad (1)$$

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 (Eq. 2). 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. Since 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.

$$\mathbf{s} = \alpha_1 \mathbf{r}_1 + .. + \alpha_i \mathbf{r}_i + .. + \alpha_n \mathbf{r}_n \quad (2)$$

where **s** is the slope vector, $\mathbf{r}_i$ is the right eigenvector associated with the i^{th} behavior mode, and α_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 (Eq. 3).

$$c_i = \frac{\Delta s_i}{\sum_{m=1}^{n} |\Delta s_m|} \qquad i = 1...n \quad (3)$$

where Δs_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_{ik} 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 $\mathbf{S}_i$ of the eigenvalue λ_i is equal to the product of the i^{th} left eigenvector and the i^{th} right eigenvector of the

gain matrix, **G** (Eq. 4) (Caswell 2001, Saleh 2002). A formulation of elasticity of eigenvalue λ_i to matrix entry g_{pq} is then given in Eq. 5.

$$\mathbf{S}_i = \mathbf{l}_i \cdot \mathbf{r}_i' \tag{4}$$

$$e_{pq,i} = \mathbf{l}_i(p) * \mathbf{r}_i(q) * \frac{g_{pq}}{\lambda_i} \tag{5}$$

where $\mathbf{l}_i(p) \equiv$ the p^{th} element of the i^{th} left eigenvector ($1\times$n vector)

$\mathbf{r}_i(q) \equiv$ 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 Eq. 3 are then used as weights in computing the weighted elasticity, which measures the overall influence of a feedback loop on the behavior of interest (Eq. 6) (Figure 1).

$$oe_k = \sum_{i=1}^{\text{n}} c_i e_k^i \qquad k = 1..\text{K} \tag{6}$$

where e_k^i is the elasticity of i^{th} behavior mode to loop k.

To understand the roles played by different parts of the tree in leading it to mortality under the two hypotheses, the EEA is carried out 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, two scenarios are studied 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/decay (= moderate stress scenario). These conditions are simulated by setting the 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 efficiency level 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 efficiency levels are based on values used in earlier studies (Bossel 1986, Pedersen 1998*a*).

2.2. 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 taking place 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*), called SPRUCE (Bossel 1986). Although originally not intended, short-term stresses can easily be incorporated into the model formulation (Pedersen

1998*a*). Thus, in this study, BAUMTOD forms the basis for the two mechanistic models to be used in the analytical treatment of the two tree mortality hypotheses. The original model (i.e., BAUMTOD) is used in studying Bossel's sudden death hypothesis; and its slightly modified version, BAUMTOD-M, is used to study Manion's gradual decline hypothesis. The main components of BAUMTOD and parameters are in listed 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 also given in Appendix A. The 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 model variable names are in *italics* in the text.

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. The photosynthetic efficiency may be reduced due to environmental stresses causing lower photosynthate production. Water and nutrients are supplied by the feeder roots in proportion to their mass. The supply must match the demand of the foliage for the production to take place. If the supply is less than the demand, actual photosynthate production will be even lower. 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 by 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 slower rate of one per-cent.

The respiring permanent biomass, *BIOM*, may be regarded as an indicator of tree vigor (Pedersen 1998*a*, 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 not possible to obtain a gradual decline in respiring permanent biomass using the original BAUMTOD formulation.[1] Noting the importance of an imbalance of respiration demand and available photosynthate supply in leading a tree to mortality, an alternative assumption regarding the impact of this imbalance on the simulated tree is considered. The new assumption allows the tree to live even after the photosynthate produced becomes less than the total respiration demand; however, the strain on the tree intensifies as the discrepancy between the respiration demand and available photosynthate widens. In other words, as there is insufficient assimilate supply to meet the respiration demand of all live permanent tissues, those tissues whose respiration demand is not met would die. A simple formulation reflecting this asphyxiation assumption would be as in Eq. 7. The biomass loss rate, now the sum of the

[1] Although Pedersen (1998*a*) reports results similar to gradual decline from simulations of BAUMTOD, they are, in fact, artifacts of the yearly timestep used in his simulations.

background loss rate and the asphyxiation factor, increases if respiration demand is not met. If the respiration demand is not satisfied at all, the factor takes on a value of 1, and if there is no photosynthate 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.

$$\textit{asphyxiation factor} = \text{IF } (\textit{assi produced} < \textit{dresp}) \text{ THEN } (1 - \textit{assi produced} / \textit{dresp}) \text{ ELSE } 0 \quad (7)$$

where *assi produced* is the actual amount of photosynthate (i.e., assimilate) produced, and *dresp* is the assimilate demand for respiration.

BAUMTOD is modified to reflect this change; the modified model is called BAUMTOD-M (Figure 2*B*). The new loop set resulting from the addition of the new formulation is in Table 1. Note the three new feedback loops that emerged from the introduction of asphyxiation factor: *L17*, *L18*, and *L19*. These replace loops *L13*, *L14*, *L15*, and *L16* in the analysis loop set of BAUMTOD-M.

3. Results

First, the no stress and moderate stress scenarios are presented. The resulting dynamics and dominant processes under these two 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 3. To illustrate a typical output of the methodology, the evolution of behavior modes under the breakdown scenario (*eff* = 0.55) is presented in Appendix B. Their relative contributions to the behavior of foliage (*LEAF*) and the resulting loop dominance dynamics over time are also given in Appendix B. The presentation of the results and the following discussion are focused on 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 shows growth. The production of photosynthate is limited by the feeder root mass (*ROOT*). Hence, the foliage (*LEAF*) mass has no direct influence over the amount of photosynthate produced (*assi produced*). In the normal growth scenario, all components are influenced by the same loops (Figure 4*A*).[2] Two of these loops are loops *L2* and *L3* that are responsible for the turnover of leaves and feeder roots, respectively. In spite of the counteracting influence of the turnover in foliage and feeder roots, there is steady growth in all parts of the tree driven by the loop, *L16*.[3] It is a positive feedback loop and includes all three main components.

Moderate stress scenario (*eff* = 0.7): Tree components cannot grow at this stress level (Figure 3). Feeder root mass, apparently higher than the tree could support under predisposing stress,

[2] In Figures 1 and 4, the loops that are most influential at one time or another during simulation are depicted with darker arrows.

[3] 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 level into the future. This drawback may be disregarded in this study, as we are interested in understanding the internal dynamics eventually leading to death under the influence of stress factors.

shrinks. This is caused by its turnover loop (*L3*) (Figure 4*B*). 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, as biomass decreases very slowly 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 fructification 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 4*B*). As a result, both respiring permanent biomass and foliage mass decrease until about the ninth year. Then the second phase begins in which the contraction of feeder root mass slows down. During this phase, coordination between the components of the simulated tree is restored and their masses are slowly diminishing. It is worth noting that loop *L1* that drives the background decay of respiring permanent biomass does not play a significant role under the first two scenarios.

Breakdown scenario (*eff* = 0.55): The results of this scenario reveal the dominant processes leading to death under the assumptions of Bossel's hypothesis. There are two phases leading to the sudden death of the tree (Figure 3). In the first phase, foliage mass restricts photosynthate production. Foliage itself is slowly diminishing in this phase partly because of the turnover (loop *L2*) (Figures 2*A*, 3*A*). However, both inefficient production and biomass decay take part in the decrease of foliage mass as well: 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 low enough so 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 some time between the second and third years of the simulation (Figure C3).

The decay in foliage and feeder root mass accelerates in this phase. In the case of foliage, this happens towards the fourth year of simulation. The reason is partly due to the decrease in photosynthate production (*assi produced*). The decrease shows its effect on the new foliage and feeder root growth through the way assimilate is distributed (*cass*). In other words, loop *L8* acts upon the growth of these two components through actual supplies of assimilate to the foliage and feeder roots (*sleaf* and *sroot*), respectively (Figure 1*A*). Its turnover loop (*L3*) diminishes the existing feeder root mass. This causes further reduction in photosynthetic output. The sharp increase in the rate of decrease in foliage mass towards year four is caused by the reduced transpiration flow (*wtrc*), which, in turn, is caused by dwindling feeder root mass. This reduction eventually leads to an insufficient supply of required water and nutrients to foliage. The result is increased foliage loss rate (*LFLOS*) (see model equations in Appendix A).

Having no 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 the photosynthate production (*assi produced*) drops so low that it cannot even satisfy the respiration demand (*dresp*), the tree suddenly dies (Figure 3).

Gradual decline scenario (*eff* = 0.4 during 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 5). Initially, foliage mass and respiring permanent biomass

exhibit slow growth while feeder root mass decays to a lower level. This continues until year five, after which the combined effects of predisposing and inciting stresses show themselves. Note the delayed response of respiring permanent biomass: although it 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 is gone. 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 also influential in the moderate stress scenario (*eff* = 0.7) (Figures 2*B*, 4*B*). Their relative influences are, however, different 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 turnover loops. This results in slow but steady increase in all three components.[4]

During year five, the interactions between the components virtually fail. There is just enough photosynthate production to satisfy the respiration demand and for the little growth of the tree components. However, the new growth is far from sufficient to afford the losses from turnover. Consequently, each component is virtually isolated and decreases under the influence of its turnover loop. The loss of the feeder root mass is the most prominent because it has the highest turnover rate (Figure 5). Unlike others, foliage is under the influence of loop *L9* as well (Figure 2*B*). This positive loop essentially drives the new foliage growth based on the availability of photosynthate after respiration demands are met. This reflects the fact that photosynthate production (*assi produced*) is limited by foliage during this phase. In the presence of an inciting stress, loop *L9* initiates a decline in the foliage mass causing it to drop faster and faster. In other words, the lower the production of photosynthate is, the less the new foliage growth; this results in lower foliage mass, which in turn leads to even lower production.

Shortly after the inciting stress is over, the continuing decrease in photosynthate production leads to a shortage in supplying the respiration demand. At this point, asphyxiation occurs and loop *L17* becomes dominant (Figure 2*B*). This accelerates the death of permanent live tissues, reflected as a faster decrease in respiring permanent biomass (Figure 5). Since all production is directed to satisfying respiration demand as much as possible, there is no new growth in any component. Furthermore, since leaf efficiency (*eff*) is restored to its pre-inciting stress level, the water and nutrient demands of foliage increase. However, feeder roots with their much lower mass cannot cope with 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 5). Feeder root mass, in contrast, is not affected by the other components. It slowly approaches zero driven by its turnover loop *L3* (Figure 2*B*).

4. Discussion

The three components of the simulated tree are fully coordinated only in the absence or at low levels of stress. In other words, there is constant interaction between all components in the absence of significant levels of stress. Under sufficiently stressed conditions, however, this integrity is threatened. The coordination between the components degrades as the simulated tree undergoes increased levels of stress.

[4] In the case of *ROOT*, there is decrease initially because its initial level is too much compared 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 other two and begins to slowly increase as well.

The two hypotheses are compared in two aspects. The first is the likely causal mechanisms leading to mortality. Even tough the imbalance between the production of photosynthate and the respiration demand has a dramatic effect in both models the difference is in the way the tree is assumed to respond to this shortage under each model. In the breakdown scenario (*eff* = 0.55), any shortage in respiration demand supply is deemed to be so critical for the tree's survival that it leads to sudden death of the tree. However, assuming the impact on tree of the imbalance between respiration demand and available photosynthate supply is proportional to the level of this imbalance results in a different outcome. Then the simulated tree exhibits dynamics that is on par 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, this study suggests that an internal imbalance of respiration demand and available photosynthate supply may, in large part, be responsible for the slow but steady approach to mortality after an inciting stress (Pedersen 1998*b*, 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. At any rate, the validity of this finding needs to be further scrutinized in future field and laboratory experiments. It is worth noting that 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 are compared is the relative importance of predisposing and inciting stresses in triggering mechanisms that lead to mortality. The analyses imply that the inciting stress level required for tree mortality to occur depends on the severity of the predisposing stress. Moreover, an inciting stress may not even be required in the presence of sufficiently heavy predisposing stress. Pedersen (1998*a*) is the first to make the case for the possible interaction between predisposing and inciting stresses on a mechanistic model (i.e. BAUMTOD). He showed that the same amount of inciting stress may cause mortality of a tree under sufficient level of predisposing stress but not of another under lower levels of predisposing stress. Evidence from recent field studies directed to better understand the causes of mortality in forest stands tends to support this conjecture. In a study on *Picea abies* (Norway spruce) in southern Finland, trees on better soils regained their vigor after a long period of drought while those on poor soils continued to decline until mortality (Mäkinen et al. 2001). In another study, Wyckoff and Clark (2002) bring up anecdotal evidence that diseased individuals of shade-intolerant *Cornus florida* (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* (white fir) in the Sierra Nevada of California. They also show that knowledge of prefire growth rate is a useful indicator of whether a fire event would ultimately lead to mortality. Further evidence of this interplay can be found in several other studies (Jimenez et al. 1985, Lwanga 2003, Suarez et al. 2004).

The extensive literature on the subject suggests that the dynamics of forest decline differ among species as well as from region to region (Waring 1987, Karev 2003). For example, the length of growth decline prior to mortality seems to differ between species (Wyckoff and Clark 2002). These differences should be properly taken into account in multispecies 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 to representing mortality are adopted in some recent

modeling studies as well (FORCLIM: Bugmann and Solomon 2000). However, it would be unreasonable to imagine a single mortality hypothesis to be applicable for every situation (Pedersen 1998*a*). Similarly, the two mortality hypotheses evaluated in this study should by no means be considered exhaustive. Still, they serve as alternative explanations to understanding the mechanism of tree mortality under different assumptions regarding impacts of external stresses on trees and the response of trees to these stresses. There is certainly evidence of weakened trees dying soon after an inciting stress (Lwanga 2003). There is also evidence of such trees gradually dying, the duration of which seems to change depending on the species and other characteristics of the specific incident (Pedersen 1998*b*). In addition, there is evidence suggesting that a predisposing stress that is severe enough would cause mortality without an inciting stress (Moore et al. 2004). On the whole, it seems that there is a continuum of what can be observed in the field between the two hypotheses. Moreover, these hypotheses and the corresponding models can be thought of as suitable frameworks to further the analyses on causes and mechanisms of tree mortality. Improved understanding of trees' physiological responses to environmental stresses would also help in effectively linking new environmental conditions due to climatic change to mortality (Keane et al. 2001).

Two possible explanations on tree mortality are put forward in the literature. This study laid out the likely structural causes behind these two hypotheses. More research should be focused on revealing which of these processes are more likely to be realistic. It is the authors' opinion that the structural mechanism suggested in BAUMTOD-M seems to be 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 tree undergoes a series of very severe inciting stresses, even with a mechanism in BAUMTOD-M, it may quickly die. To make these points clearer, however, more data and research is certainly needed.

Storage of carbon (assimilate) is not represented in the models considered in this study. Its inclusion may not matter much because of the following 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) and/or would change the required combination levels of predisposing and inciting stresses for the tree to be driven to death. Still the availability of assimilate supply for defense is shown to be of high importance 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 should be suitably incorporated in application-oriented simulation studies, as well as future theoretical studies. Having said that, one needs to be wary not to include unnecessary processes in models (Korzukhin et al. 1996). The simplicity of the models used in this study should be viewed in this light.

5. Conclusion

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 methodology for analysis of dynamic feedback models is used to uncover a tree's physiological dynamics in the presence of varying stress levels

under the assumptions of two models. The differences as well as the similarities in progression of tree mortality between the two models are also examined.

This study shows that imposing stress on a tree disturbs its otherwise integrated functioning. The failure of different components of 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 upon 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 more after the damaging effect of an inciting stress. Although the shortage in supply of 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 actual cause of gradual decline (Kozlowski et al. 1991). The exact nature of this impact and the relation between the two hypotheses need to be scrutinized in future lab or field experiments. There are also other factors that are likely to be of high importance. They, such as assimilate supply for defense, should be included in future studies to reach a more comprehensive understanding of the causal mechanisms behind tree mortality.

Models such as the ones considered in this study are crucial for developing a theoretical understanding of the dynamic response of a tree's structure under normal and stressed conditions. The findings distilled from theoretical studies on mechanistic models may well serve as a basis for further analysis of environmental stress effects on specific locations and time periods, provided that specific data is available on the nature of the stress factor(s) and the exposure patterns (Monserud and Sterba 1999, Keane et al. 2001). We hope the findings and arguments put forward in this study will prove useful in motivating future field, lab, and simulation studies. The increased availability of relevant field data coupled with the development of better models will, without a doubt, generate a deeper understanding of the physiological processes leading to mortality of a tree.

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Appendix A: Table of definitions and equations of the models

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

Abbreviation	Value (dimension)	Parameter
BIOM[§]	= 1 (assimilate)	Respiring permanent biomass
LEAF[§]	= 1 (assimilate)	Foliage mass
ROOT[§]	= 1 (assimilate)	Feeder root mass
eff	= 1 (—)	leaf photosynthetic efficiency
nbml	= 0.01 (1/year)	normal loss constant
ndl	= 8 (year)	number of years a given needle remains on the tree
nrto	= 1 (—)	natural root turnover constant
rdam	= 1 (—)	normal root decay constant

[§] For the state variables, Value indicates the initial value.

Table A2. Rates and other variables for the models used.

Abbreviation	Description of rate or other variable (dimension)
BMNEW	respiring permanent biomass growth rate (assimilate/year)
BMLOS	respiring permanent biomass loss rate (assimilate/year)
LFNEW	foliage growth rate (assimilate/year)
LFLOS	foliage loss rate (assimilate/year)
RTNEW	feeder root growth rate (assimilate/year)
RTLOS	feeder root loss rate (assimilate/year)
abiom	photosynthate remaining for biomass growth (assimilate)
asphyxiation factor	respiring permanent biomass loss rate due to shortage in assimilate supply to meet respiration demand (—)
assi produced	actual amount of photosynthate (i.e., assimilate) produced (assimilate)
assr	remaining assimilate (assimilate)
cass	assimilate distribution factor (—)
demnd	total demand on renewal (assimilate)
dfru	assimilate demand for fructification (assimilate)
dleaf	assimilate demand for leaf growth (assimilate)
dresp	assimilate demand for respiration (assimilate)
droot	assimilate demand for feeder root growth (assimilate)
mprod	photosynthetic production capacity (assimilate)
rroot	required feeder root mass (assimilate)
sfru	actual assimilate supply for fructification (assimilate)
sleaf	actual assimilate supply for leaf growth (assimilate)
sroot	actual assimilate supply for feeder root growth (assimilate)
wdem	normalized water (and nutrient) demand (assimilate)
wtrc	actual (nutrient and water) transport capacity of the root system (assimilate)

Equations of BAUMTOD

TIME STEP = 0.02 years; Integration method: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 * nrto * 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

dleaf = 0.15 * BIOM

dresp = 0.3 * BIOM

droot = rdam * nrto * rroot * 0.065

mprod = LEAF * eff

rroot = mprod

sfru = dfru * cass

sleaf = dleaf * cass

sroot = droot * cass

wdem = mprod

wtrc = ROOT

Appendix B: Output of the methodology for the breakdown scenario

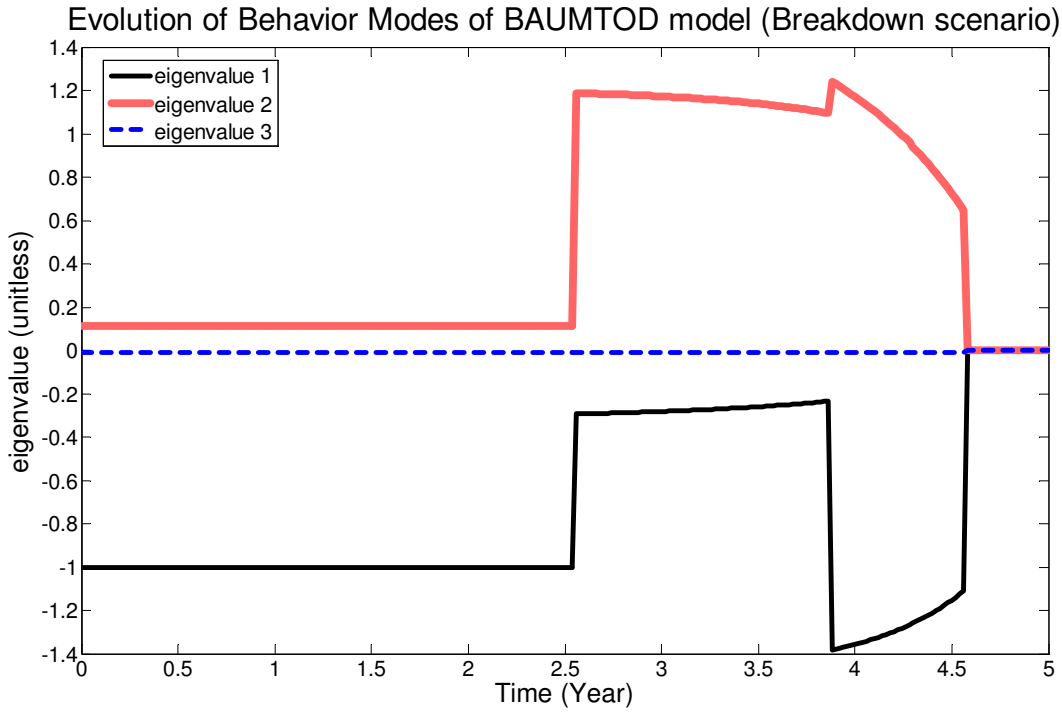


Figure B1. Evolution of behavior modes over time under breakdown conditions (Eigenvalues in the figure represent three different behavior modes).

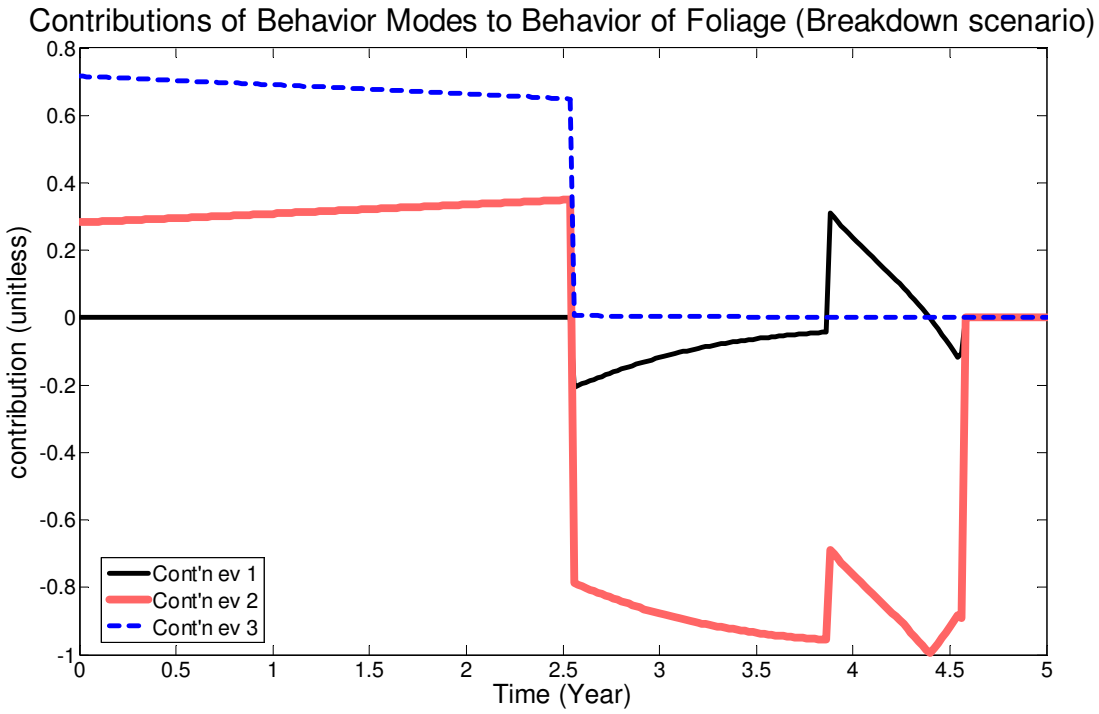


Figure B2. Relative contributions of behavior modes to the behavior of foliage, *LEAF* (Cont'n ev 1: contribution of behavior mode 1 to behavior of *LEAF*; Cont'n ev 2: contribution of behavior mode 2 to behavior of *LEAF*; Cont'n ev 3: contribution of behavior mode 3 to behavior of *LEAF*)

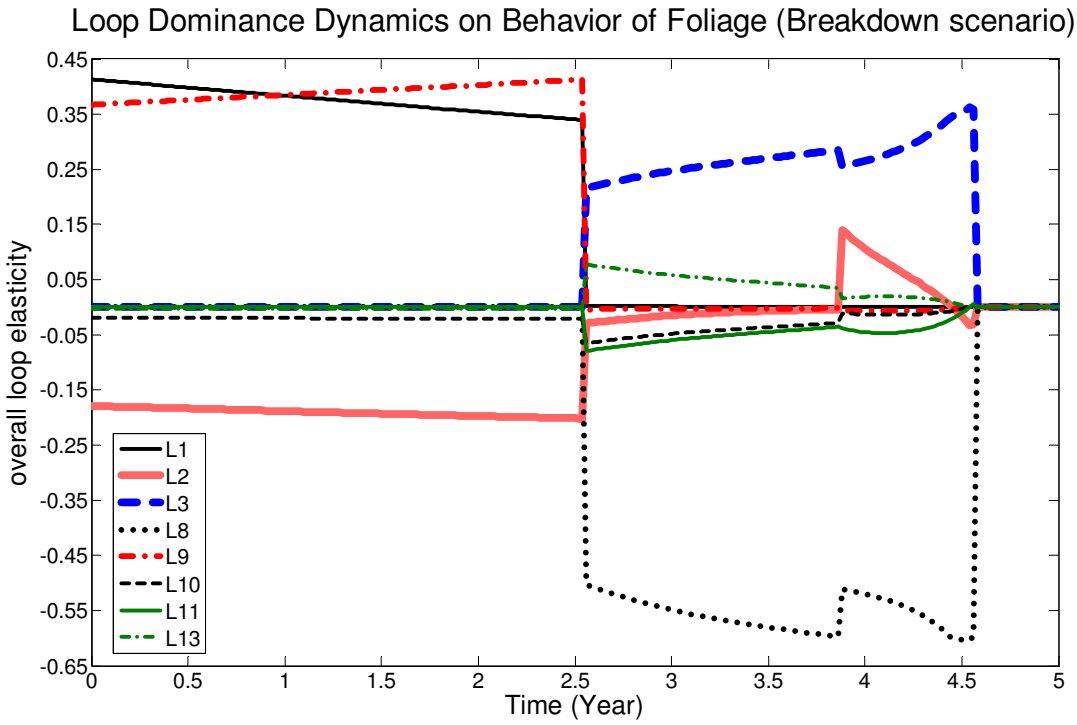


Figure B3. Loop dominance dynamics acting upon behavior of foliage, *LEAF*.

Tables

Table 1. Feedback loops in the Shortest Independent Loop Sets of the models.

Loop no	Variable sequence within the loop
L1	BIOM, BMLOS
L2	LEAF, LFLOS
L3	ROOT, RTLOS
L4	LEAF, mprod, wdem, LFLOS
L5	BIOM, dresp, assr, abiom, BMNEW
L6	BIOM, dfru, demnd, abiom, BMNEW
L7	BIOM, dleaf, demnd, abiom, BMNEW
L8	ROOT, wtrc, assi produced, assr, cass, sroot, RTNEW
L9	LEAF, mprod, assi produced, assr, cass, sleaf, LFNEW
L10	LEAF, mprod, rroot, droot, demnd, cass, sleaf, LFNEW
L11	ROOT, wtrc, LFLOS, LEAF, mprod, rroot, droot, sroot, RTNEW
L12	LEAF, mprod, assi produced, assr, abiom, BMNEW, BIOM, dleaf, sleaf, LFNEW
L13†	LEAF, mprod, rroot, droot, demand, cass, sroot, RTNEW, ROOT, wtrc, LFNEW
L14†	LEAF, mprod, assi produced, assr, abiom, BMNEW, BIOM, dfru, demand, cass, sleaf, LFNEW
L15†	LEAF, mprod, rroot, droot, demand, abiom, BMNEW, BIOM, dleaf, sleaf, LFNEW
L16†	BIOM, dleaf, sleaf, LFNEW, LEAF, mprod, rroot, droot, sroot, RTNEW, ROOT, wtrc, assi produced, assr, abiom, BMNEW
L17‡	BIOM, dresp, asphyxiation factor, BMLOS
L18‡	BIOM, dleaf, sleaf, LFNEW, LEAF, mprod, assi produced, asphyxiation factor, BMLOS
L19‡	BIOM, dfru, demnd, cass, sroot, RTNEW, ROOT, wtrc, assi produced, asphyxiation factor, BMLOS

† These loops belong to the set of BAUMTOD and not that of BAUMTOD-M.

‡ These loops belong to the set of BAUMTOD-M and not that of BAUMTOD.

Figures

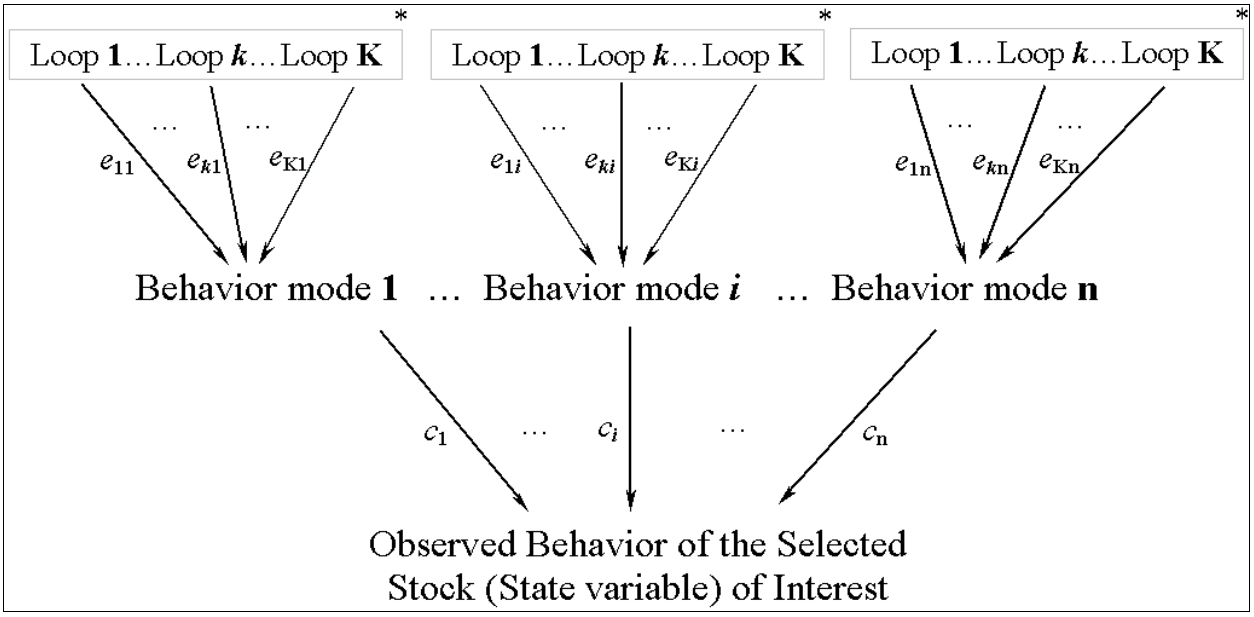


Figure 1. A diagrammatical representation of the EEA methodology (c_i is relative constribution of behavior mode i on behavior of interest; e_{ki} is elasticity (normalized sensitivity) of behavior mode i to feedback loop k). *Loops in Shortest Independent Loop Set (SILS).

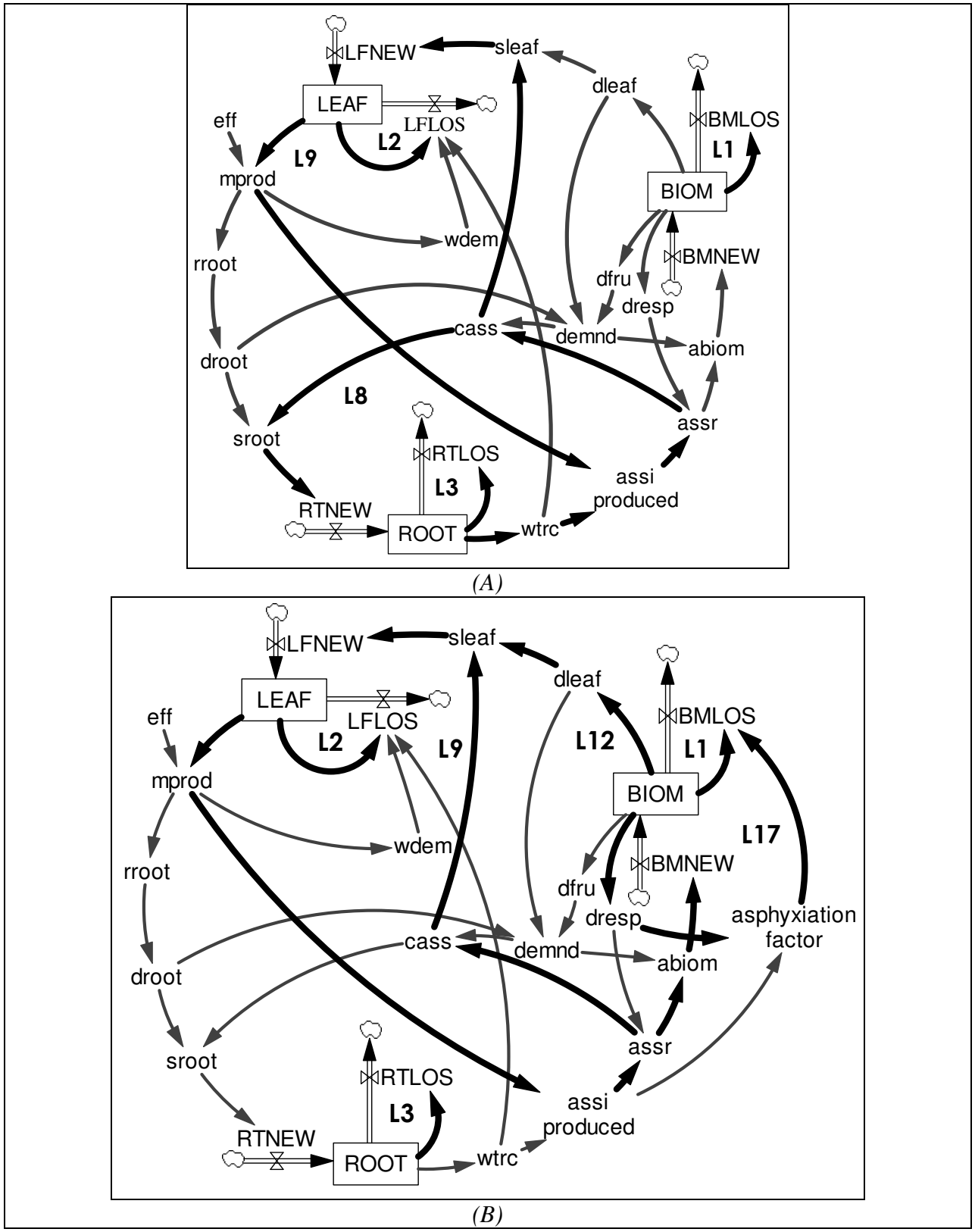


Figure 2. Stock-flow diagrams for BAUMTOD and BAUMTOD-M. Dominant feedback loops of breakdown (*eff* = 0.55) *(A)* and gradual decline (*eff* = 0.8/0.4) *(B)* scenarios are also shown (see Section 5). Refer to Table 1 and Tables A1-2 for loop and variable information, respectively.

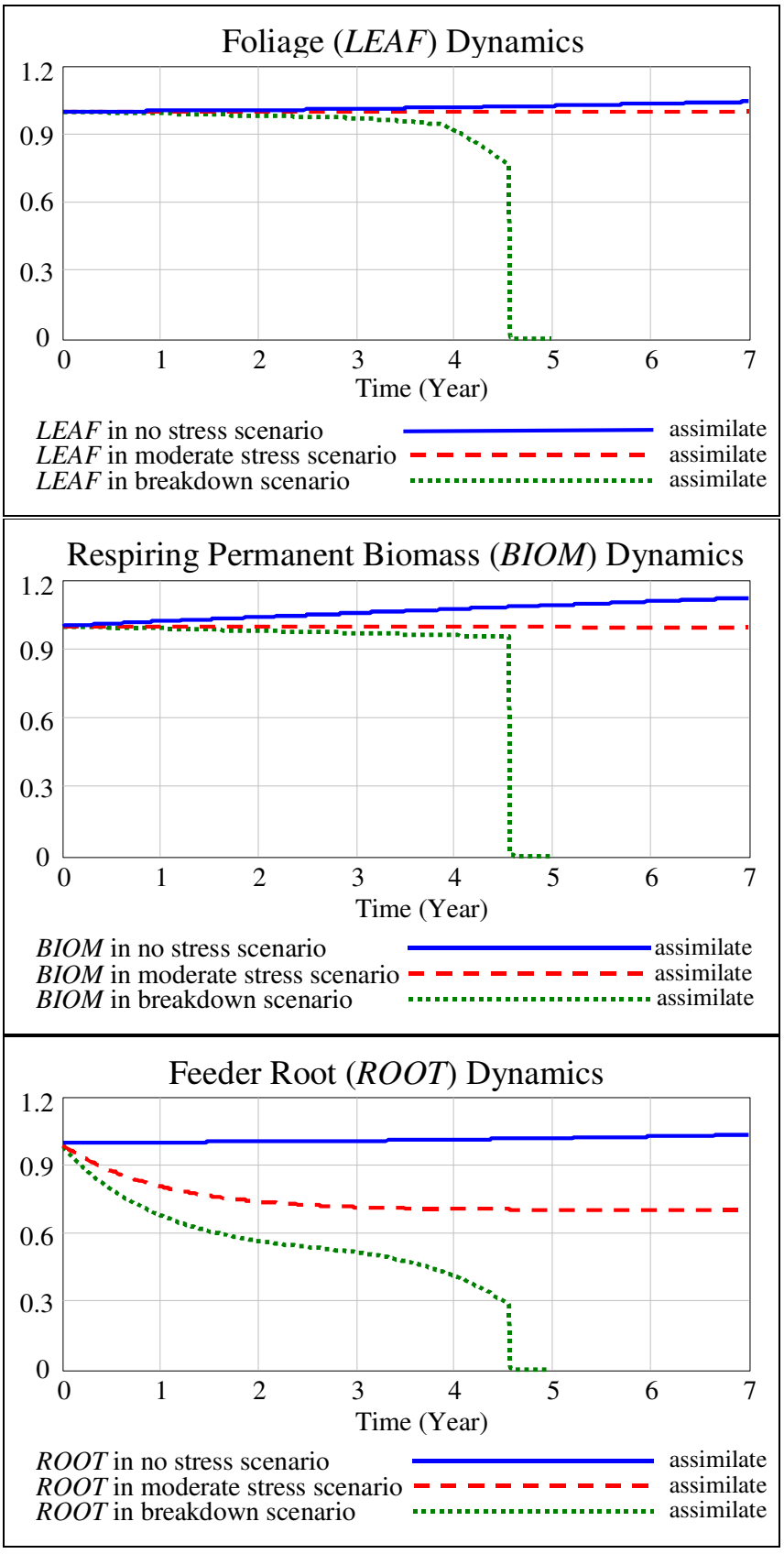
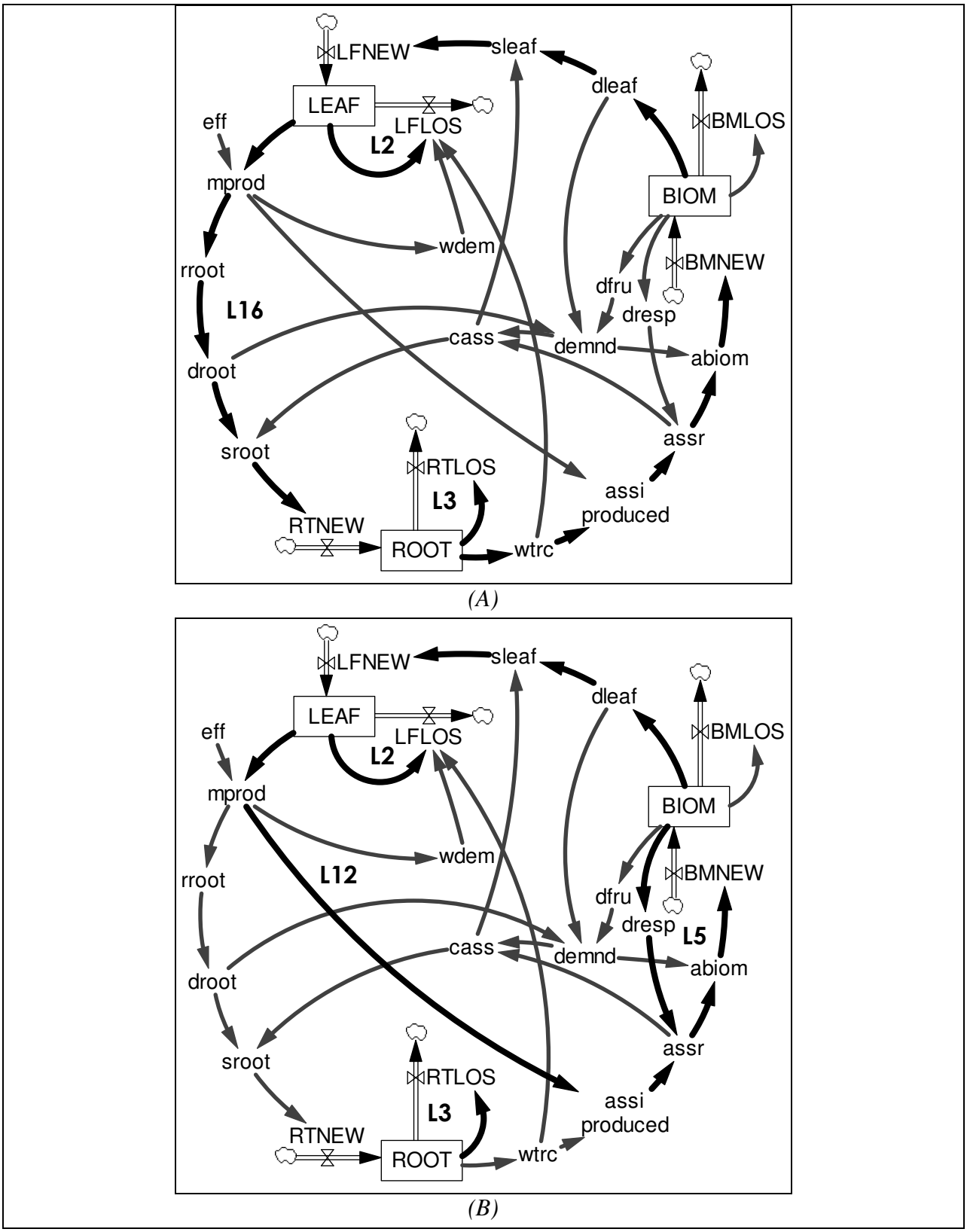


Figure 3. Dynamics of main components (foliage, *LEAF (A)*; respiring permanent biomass, *BIOM (B)*; feeder roots, *ROOT (C)*) in no stress (*eff* = 1), moderate stress (*eff* = 0.7), and breakdown (*eff* = 0.55) scenarios.



1 Figure 4. Dominant feedback loops under no stress (*eff* = 1) *(A)* and moderate stress (*eff* = 0.7)

2 *(B)* scenarios. Refer to Table 1 and Tables A1-2 for loop and variable information, respectively.

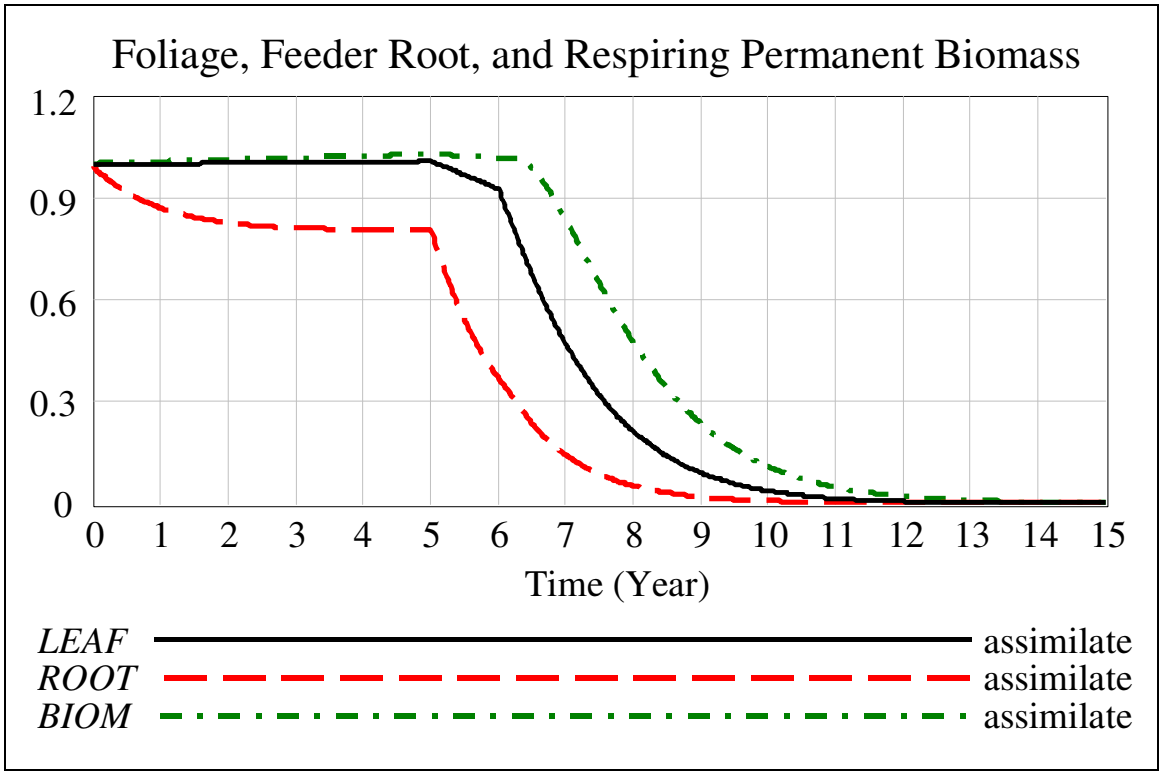


Figure 5. Behaviors of main components (foliage, *LEAF*; feeder roots, *ROOT*; and respiring permanent biomass, *BIOM*) under the gradual decline scenario (*eff* = 0.8/0.4) with BAUMTOD-M. Dynamics are similar to the scheme in Johnson (1989) regarding Manion's hypothesis.