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Short communication

Modeling tree mortality in response to short- and long-term environmental stresses

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Abstract

An understanding of how environmental stresses contribute to tree mortality is essential for predicting the impact of these stresses on forests. Utilizing a qualitative, mechanistic model of tree resource acquisition and allocation, I evaluated a conceptual model of the process of tree mortality. The conceptual mortality process model posits that tree mortality may result from short-term environmental stresses (e.g. drought or insect defoliation) acting on trees that have been predisposed to injury by long-term environmental stresses (e.g. air pollution or competition). A previous field study identified oaks that died in a manner consistent with this conceptual model and the mechanistic model provides further support for the conceptual model. When subjected to simulated short- and long-term stresses, the mechanistic model predicts changes in tree vigor that are similar to the changes in vigor predicted by the conceptual mortality process model. The simulation results also suggest a mechanism by which short-term environmental stresses affect tree physiology prior to death. © 1998 Elsevier Science B.V.

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1. Introduction

Understanding how environmental stresses contribute to tree mortality is essential for predicting forest growth rates and forest responses to these stresses (Franklin et al., 1987; Miner et al., 1988). However, establishing the role of environmental stresses in tree mortality is difficult because of the multitude of interacting stresses affecting trees throughout their lifetimes (Franklin et al., 1987; Manion, 1981).

To determine the role of environmental stresses in the mortality of overstory oaks (*Quercus* spp.

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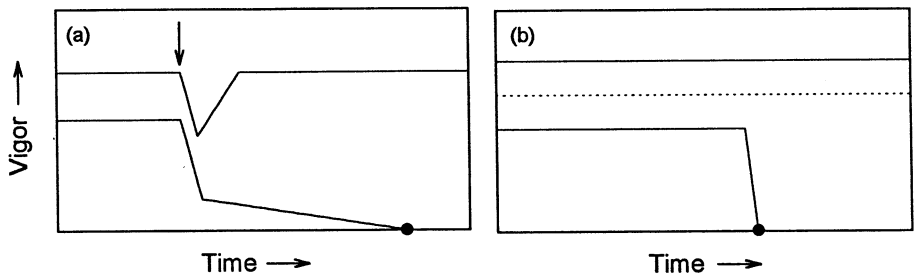

Fig. 1. Alternative models of the tree mortality process. The upper line in each diagram represents a surviving tree and the lower line represents a dying tree. Tree death is indicated by ● (a) Manion's model (schematic after Johnson (1989), who labeled the ordinate 'carbon available for defense and repair'). The lower initial vigor of the dying tree is due to long-term predisposing stress. The inciting stress, indicated by the arrow, is an extreme, short-term stress. According to Manion (1981), the vigor decline following the inciting stress is due to long-term contributing stress; (b) Bossel's model. Schematic adapted from Bossel (1986) where the ordinate was permanent biomass (live tissues in the stem, branches, and coarse roots). Trees with low vigor due to long-term stress eventually enter a breakdown mode and then quickly die.

L.) in the Midwestern USA, Pedersen, 1998 (in press) examined the growth patterns of oaks prior to their deaths. Tree-ring growth measurements from recently dead oaks from seven sites were utilized as long-term records of tree vigor prior to mortality. He found that 76% of the dead trees experienced vigor changes prior to mortality that were consistent with a conceptual model of the tree mortality process proposed by Manion (1981); Fig. 1a. These dead trees experienced one or more sudden, permanent growth declines, or interventions, during years of environmental stress. The median basal area growth rate decline was 38% and, with one exception, these trees never regained their prior rates of growth. In Manion's model, the interventions are attributed to inciting stresses, which are extreme, short-term stresses such as droughts. Pedersen (in press) found that the probability of a tree experiencing an intervention increased with the severity of environmental stress and 40% of the interventions occurred during years of notable drought. After interventions occurred, the median time until tree death was 20 years. Prior to experiencing interventions, the now dead trees were growing an average of 18% slower than comparable surviving oaks. In Manion's model, the low growth rates prior to interventions are evidence of predisposing stresses, long-term stresses that increase tree susceptibility to inciting stresses.

Bossel (1986), examining the impact of air pollution stress on trees, presents what may be considered an alternative conceptual model of the tree mortality process. According to this model, when trees are subjected to long-term stresses of sufficient intensity, they eventually enter a breakdown mode and then quickly die (Fig. 1b). In contrast to Manion's model, no extreme, short-term stress is involved. Bossel's conceptual model of the tree mortality process is based on simulations with two mechanistic computer models of tree resource acquisition and allocation (Bossel, 1986).

Analyzing the oaks described above, Pedersen (1992) found no evidence that Bossel's conceptual model of the tree mortality process applies to these trees. However, Bossel's mechanistic tree growth models provide an opportunity to seek a mechanistic basis to Manion's conceptual mortality process model. Accordingly, I modified the simpler of Bossel's two mechanistic tree growth models (BAUMTOD) so the simulated tree could be subjected to predisposing and inciting stresses as defined in Manion's conceptual mortality process model.

2. Method

Bossel's (1986) qualitative, mechanistic model of tree growth simulates an evergreen tree with

three components: foliage biomass, fine root biomass, and permanent biomass. The latter includes the live tissues in the stem, branches, and coarse roots. The model tree's foliage annually produces an amount of photosynthate based on the foliage biomass, availability of water and nutrients provided by the fine roots, and photosynthetic efficiency. The fine roots annually acquire amounts of water and nutrients in proportion to their biomass. Each year the model allocates the photosynthate produced. The first allocation priority is to meet the respiration requirements of the permanent biomass. If the photosynthate supply is inadequate to meet this demand, the tree dies. The second allocation priority is to replace foliage and fine root biomass lost to annual turnover, and to meet reproductive requirements (this priority implicitly includes the respiration requirements of the foliage, fine roots, and reproductive tissues). If the supply of photosynthate remaining is inadequate to completely satisfy these demands, the available photosynthate is allocated in proportion to the demand by each of these three sinks. If photosynthate still remains, however, it is allocated for production of additional permanent biomass. In the absence of imposed stresses, 40% of the photosynthate is available for producing additional permanent biomass. Complete fine root turnover occurs each year. The model tree retains foliage for 8 years so one-eighth of the foliage is normally replaced each year. However, if the fine root biomass is inadequate to meet the water and nutrient demand of the foliage, the foliage turnover rate is increased. Permanent biomass is lost at the rate of 1% per year to represent, for example, self-pruned branches. Based on Bossel's (1986) description of this model, the model was implemented in Microsoft QuickBASIC (version 4.0; the computer code is in Pedersen, 1992).

In Bossel's (1986) analysis, the impact of long-term, constant stresses on the model tree were simulated by reducing photosynthetic efficiency or increasing the rate of fine root turnover (both of these stress responses were considered possible consequences of stresses such as air pollution (Bossel, 1986)). In the analysis presented here, only reductions in photosynthetic efficiency were considered. But in addition to simulating the impact of long-term (predisposing) stresses, I simulated the impact of short-term (inciting) stresses by further reducing photosynthetic efficiency in particular years.

3. Results and discussion

The new implementation of Bossel's (1986) mechanistic model reproduced Bossel's original results. With no imposed stress, the model tree's permanent biomass (live stem, branch, and coarse root tissues) increases each year (Fig. 2a; because the model is qualitative, the simulated permanent biomass is only an indicator of tree vigor). At intermediate levels of constant stress, the permanent biomass increases at a slower rate or gradually declines. At extreme levels of stress, the permanent biomass eventually drops precipitously and mortality occurs. The greater the degree of extreme stress, the less time passes prior to mortality.

The mechanistic model also exhibits behavior consistent with Manion's conceptual model of the tree mortality process. An inciting stress in 1 year is sufficient to cause mortality, but the model tree continues to survive for many years after the inciting stress (Fig. 2b). When the model tree is subjected to an inciting stress and a predisposing stress, mortality results from a less severe inciting stress (Fig. 2c). Because the mechanistic growth model simulates permanent biomass rather than tree vigor, it was not expected that the change in permanent biomass would exactly mimic the vigor change shown in Fig. 1a. But the key features of Manion's model are demonstrated by this simulation (Fig. 2c): long-term stresses predispose trees to injury by short-term stresses and mortality occurs several years following the short-term stress.

Manion (1981) attributed the continuing decline in tree vigor following an inciting stress to contributing stresses, long-term stresses such as weak pathogens (Fig. 1a). But since the mechanistic model does not simulate such stresses and mortality still occurs, this suggests that contributing stresses may not be necessary to cause tree death. This is consistent with the view of Mueller-Dom-

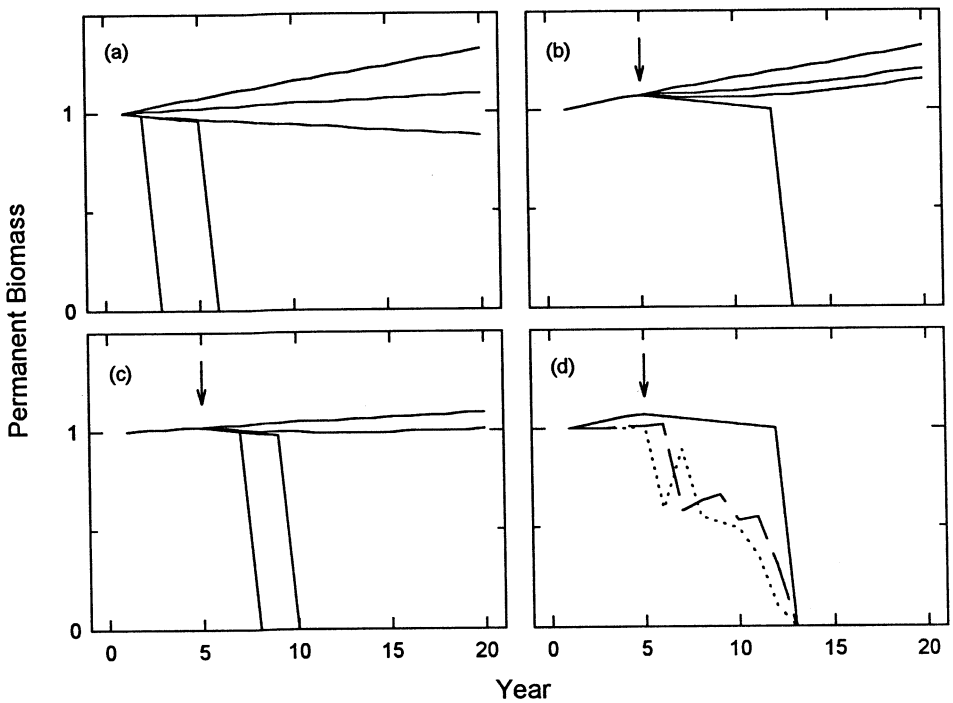

Fig. 2. Mechanistic tree growth model simulation results. Because the model is qualitative, the simulated permanent biomass results are only an indicator of tree vigor: (a) model behavior in response to constant stress. The five lines represent separate simulations with constant photosynthetic efficiencies of, from top to bottom, 1.00, 0.80, 0.60, 0.56, and 0.40. Only the lowest two photosynthetic efficiencies result in mortality (permanent biomass = 0) during the 20 year simulations, with the lower photosynthetic efficiency leading to more rapid death; (b) model behavior in response to an inciting stress, indicated by the arrow, that was simulated by setting photosynthetic efficiency during year 5 to, from top to bottom, 1.00, 0.80, 0.70, and 0.60. Photosynthetic efficiency was 1.00 in all other years. Only the most severe inciting stress led to mortality, but mortality did not occur until 8 years after the inciting stress; (c) model behavior in response to predisposing and inciting stresses. The predisposing stress was simulated by setting photosynthetic efficiency to 0.80 for all simulations in all years except year 5. The inciting stress, indicated by the arrow, was simulated by setting photosynthetic efficiencies in year 5 to, from top to bottom, 0.80, 0.64, 0.56, and 0.48 (these photosynthetic efficiencies represent inciting stresses equivalent to those simulated in (b): 100%, 80%, 70%, and 60% of the pre-inciting stress value). With the predisposing stress, mortality resulted when photosynthetic efficiency was 70% of the pre-inciting stress photosynthetic efficiency, rather than 60% for the case where there was no predisposing stress (see (b)), evidence of interaction between the predisposing and inciting stresses; (d) simulation results by tree component: solid line, permanent biomass (live stem, branch, and coarse root tissues), dashed line, foliage biomass, and dotted line, fine root biomass. There was no predisposing stress; photosynthetic efficiency was 1.00 in all years except year 5. The inciting stress, indicated by the arrow, was simulated by setting photosynthetic efficiency in year 5 to 0.60. The model tree's foliage and fine root components respond rapidly to the inciting stress, alternately exceeding each other, but generally declining until tree death occurs.

bois (1987) who said that contributing stresses are simply precursors to decomposition.

Bossel (1986) attributes tree death that occurs in a manner consistent with his conceptual mortality process model to two positive feedback loops. First, the stress-caused reduction in photosynthetic efficiency lowers photosynthate production. Since the curtailed supply of photosynthate is inadequate to replace the foliage biomass lost to turnover and meet the respiration requirements of the foliage, the foliage biomass declines, and photosynthate production drops further. Second, the reduced supply of photosynthate is inadequate to replace the fine root biomass lost to turnover and meet the respiration requirements of the fine roots, so the fine root biomass declines. This reduces the supply of water and nutrients to the foliage, in-

creasing the rate of foliage turnover, and further reducing photosynthate production.

These positive feedback loops are also part of the tree mortality process when an inciting stress is involved, although in a more complex manner. When the model tree is subjected to an inciting stress sufficient to cause mortality, the foliage and fine root components respond within 2 years of the inciting stress even though tree death does not occur for 8 years (Fig. 2d). The fine root biomass is affected first because it must be replaced each year (the fine roots turn over annually). But the reduced supply of photosynthate (due to the inciting stress) is adequate to only partially replace the fine root biomass. In the second year after the inciting stress, fine root biomass increases with the recovered photosynthetic efficiency, but now foliage biomass declines because the previous loss of fine root biomass led to reductions in the water and nutrient supply, increasing the rate of foliage turnover. In the third year after the inciting stress, foliage biomass partially recovers with the increased water and nutrient supply but fine root biomass falls again due to the previous decline in foliage biomass and, hence, photosynthate production. In sum, after an inciting stress the foliage and fine root system capacities exceed each other in alternate years, but decline overall, until mortality occurs. The excessive capacity in either the photosynthesis, or water and nutrient uptake systems results in some of the already stress-reduced supply of photosynthate being utilized unproductively to meet the respiration demands of tissues that are ready to acquire resources that cannot be utilized. These results suggest a mechanism by which inciting stresses may impact tree physiology between an inciting stress and tree death.

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