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ACID DEPOSITION AND CATASTROPHES IN FORESTS: A TREE-NUTRIENT MODEL

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Abstract. The paper investigates the influence of acidic deposition on the dynamics of forest ecosystems by means of a simple mathematical model having tree biomass, nutrient concentration in the trees and nutrient concentration in the soil as state variables and exogenous nutrient inflow as driving force. The existence and stability of equilibria are analyzed for three different mechanisms of tree damage: direct effects on exposed tissue, increased soil acidity, and excessive nutrient accumulation. It is shown that when the nutrient inflow from acid precipitation is increased, the second and third mechanisms imply the sudden collapse from a viable equilibrium to tree extinction. More precisely, a fold catastrophe, as it is called within the relevant theory, is generated.

Introduction

Acidic deposition has raised wide concern in the last decade both in Europe and in North America. Several international institutions started and in some cases completed programs for monitoring, modeling and evaluating its causes and effects. It is widely recognized that one major impact of acid precipitation both dry and wet is on forests. Recent episodes, like the most publicized one in the Black Forest, and many others in the USA, Canada, Germany, Poland, etc., have thrown this issue into the political arena, while public opinion in several countries is becoming increasingly sensitive to the problem of forest damage from "acid rain", as the phenomena is commonly termed.

A large body of scientific literature is now available with regard to the effects of air pollution on forests: as an example we cite the books by Smith (1981), Treshow (1984) and Linthurst (1984). This literature is mainly concerned with the careful and detailed identification of the processes that could directly or indirectly affect the vegetation. The emerging evidence is that there are many ways in which plant growth and survival can be influenced by acidic deposition and, quoting from Smith (1981), sometimes "forests are influenced by air contaminants in a subtle manner". This paper's contribution is quite different and may be considered somewhat simple-minded and crude. In fact, we condense a variety of complex phenomena into a simple mathematical model of tree and nutrient dynamics, which, however, is appealing in its abstraction. This simplicity itself allows us to analyze the stability properties of a hypothetical forest subject to acidic deposition and to pinpoint some possible key mechanisms that may determine the long-term fate and possible collapse of forest ecosystems. It should be clear that the aim of this work is basically conceptual and qualitative and that our model is not meant to rival, on quantitative ground, the detailed simulation models of forest and/or soil dynamics that have been and are currently being built (see Shugart, 1984, for a review).

The focus of this paper will be on the intrinsic nonlinear nature of the vegetation response to acidic deposition which might play an important role in causing unexpected and severe damages to forests. The collapses we shall consider are those corresponding to so-called catastrophic transitions in dynamical systems (Thom 1972), namely discontinuities in the behavior of a system due to very small changes of a strategic allogenic parameter (a very brief account on the concept of catastrophe will be given in the next section). We contemplate three possible mechanisms of forest disruption from acidic deposition: (i) through direct effects upon vegetation (such as physical damage to tissues), (ii) through increased soil acidity, which *inter alia* entails the release of toxic amounts of aluminium and manganese, (iii) through accumulation in the tree biomass of excessive amounts of nutrients, which may be harmful to the plants. We analyze the three corresponding models and demonstrate that the second and third mechanisms give rise to a so-called fold catastrophe. Obviously, all this does not imply that the two above mechanisms are the only potential or the most important causes of forest collapse. In fact, the most frequent collapses in forests are associated with storms, fires, droughts and pests, which, nevertheless, being large-scale allogenic disturbances, do not require a catastrophe-theoretical framework.

The concept of catastrophe

The description of catastrophes has recently become quite fashionable in many fields of natural, physical and social sciences. With regard to forest dynamics a remarcable example is the long study conducted by Holling and colleagues on the interaction between the

the spruce budworm and the coniferous forest of eastern North America (Clark et al. 1978, Ludwig et al. 1978, Casti 1982).

Altough a formal theory of catastrophes was developed more than a decade ago, mainly by Thom (1972), we do not attempt to give any kind of general review of the theory, but only a very simple and brief presentation of what is meant by catastrophe in this specific context.

Let us suppose that we are interested in discussing the influence on forest dynamics of a particular allogenic parameter such as the inflow of nutrient from external sources. If, hypothetically, this input is constant over time, as well as all other exogenous biotic and abiotic factors, the state of the system, *i.e.* the variables characterizing the problem (biomasses of different tree species, nutrient concentrations in the soil and in the vegetation, etc.) will converge to a stable equilibrium (in the case there are multiple stable equilibria, the convergence to one or another will depend on the initial conditions of the forest ecosystems). If the nutrient inflow - call it W - is smoothly and slowly growing, e.g., due to an increase of acid precipitation, also the equilibria will in general vary according to a smooth pattern. Sometimes, however, a small (infinitesimal) perturbation of W from a critical value $\overline{W}$ may entail the appearance or disappearance of an equilibrium. In other cases, an equilibrium that is stable for $W < \overline{W}$ may become neutrally stable for $W = \overline{W}$ and unstable for $W > \overline{W}$ or vice versa (at this point the reader should not try to understand why this may occur, but simply be content that examples will be given in the following). Now, if one imagines that a forest is in a stable steady state for a nutrient inflow $W < \overline{W}$ and that this equilibrium disappears or becomes unstable for $W > \overline{W}$ one can say that a catastrophe will occur. In fact, as soon as the input exceeds the threshold $\overline{W}$ the state of the ecosystem moves toward a different stable equilibrium which can be far from that existing before the perturbation. This transient from one to another equilibrium is a catastrophic transition.

Fig. 1 gives a simple and pictorial representation of the process described above with reference to the tree biomass T of a hypotetical forest in the case there are two critical values ($\overline{W}$ = W, and $\overline{W}$ = W'') of the nutrient inflow. In a forest like this there is a single stable equilibrium for sufficiently low (W < W') and for sufficiently high (W > W'') nutrient inflows. On the contrary, in the range W' < W < W'' two stable equilibria are possible. In which of the two equilibria the system lies depends upon the past history of the forest. In particular, a storm or a fire in a healthy forest (upper branch of the graph in Fig. 1) will give rise to a sudden drop of tree biomass followed either by a recovery to the pristine equilibrium or by a transient toward the lower equilibrium biomass if the disturbance is very severe. But one should remark that a switch from a healthy to a depressed forest can also be originated by an infinitesimal increase of W around W''. This latter switch is a genuine catastrophic transition. The graph in Fig. 1 also shows that the process can be reversed by reducing the nutrient inflow below the threshold W'. For these reasons, this kind of graph is called reversible fold catastrophe.

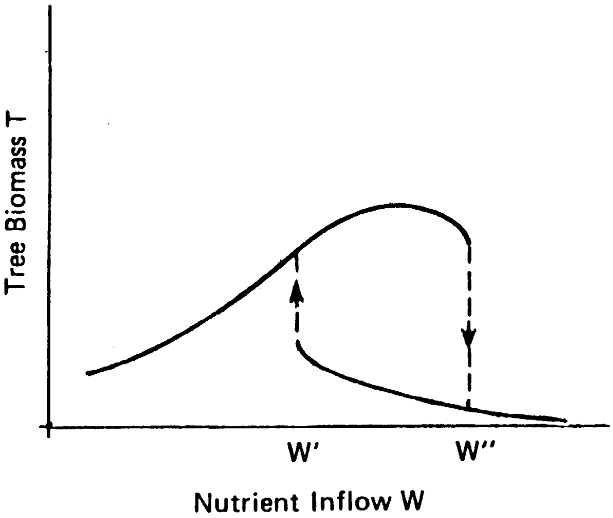

Fig. 1. Hypothetical representation of the tree biomass T corresponding to stable equilibria, as function of the nutrient inflow W. The pattern is that of a typical catastrophe with hysteresis.

A model of tree and nutrient dynamics

This section is devoted to the presentation of the mathematical model that is the basis of our analysis. We also attempt to make a clear statement of the assumptions that underlie the equations so as to let the reader appreciate the limitations of the theory set forth.

The first basic hypothesis is that the state of the forest endangered by acidic deposition can be represented by the total tree biomass T in a given area, without further detailing the structure of the plant population. This amounts to assuming that our attention is focused on a forest landscape, consisting of a mosaic of patches, at a very coarse scale. In this case the use of very simple models is realistic as shown by Shugart (1984) in his Chapter 6 on the biomass response of landscapes (see specially his Fig. 6.4 which is obtained from the sum of 50 plots, each simulated by a gap model).

The second basic assumption is that the soil nutrient pool can be described by the concentration N of one nutrient which is homogeneously distributed in the soil. This hypothesis is crude, because in general there are more than one limiting factor that act differentially on different segments of a forest. However, it is widely recognized that in many a case basically only one element limits productivity. Most often in temperate and boreal forests this limiting nutrient is nitrogen (Cole and Rapp 1981, Agren 1983, Vitousek 1984, Vitousekd and Matson 1985). Nitrogen is also one of the most important components of anthropogenic emissions into the atmosphere (about one third of sulphur emissions, Söderland 1977, OECD 1979).

We further assume that the nutrient is taken up by trees and used for the processes of biomass production. The rate of synthesis of new biomass is thus supposed to depend, in an increasing and saturating fashion, upon the concentration S of nutrient in the tree biomass. On the other hand, the nutrient contained in the dead biomass is at least in part returned, via decomposition, to the nutrient pool.

If we now introduce the total amount Q = TS of nutrient stored in the standing biomass, the basic model can be stated as follows.

$$\frac{dT}{dt} = -mT + e\,(S)\,h\,(T)\,T$$

$$\frac{dQ}{dt} = \beta NT - mTS \qquad (1)$$

$$\frac{dN}{dt} = W - \alpha N - \beta NT + \eta mTS$$

where

t = time

T = tree living biomass

S = nutrient concentration (per unit biomass) in the trees

Q = amount of nutrient stored in the standing biomass = TS

N = nutrient concentration in the soil

m = tree mortality

h (T) = maximum production of new biomass per unit biomass; a decreasing function of T

e (S) = efficiency of production, *i.e.* the percentage of maximum production which is actually achieved; an increasing function of S

β = rate of nutrient uptake by one unit of tree biomass per unit of nutrient concentration

W = input to the nutrient pool from sources other than tree decomposition

α = coefficient of nutrient decay due to leaching losses from the forest watershed or to uptake by vegetation other than trees

η = percentage of dying tree biomass that returns to the nutirent pool through decomposition.

Model (1) contains many further assumptions that need explanation and comment. As far as the production of new biomass is concerned, we take into account some form of density dependence, namely the fact that an increasing total tree biomass entails a decreasing production of new biomass by each unit of standing biomass. This is obviously due to increase shading, root competition, etc., which limit the amount of energy captured from the surrounding environment by each tree unit (for a detailed account of these phenomena see Silvertown 1982). Therefore the function h (T) is assumed to be decreasing with T (as for instance in Fig. 2 which is based on the elaboration of data on balsam fir reported by Sprugel 1984). As a result, the net primary productivity h (T) T is either increasing (like, *e.g.*, in Sprugel's data on balsam fir) or unimodal (which seems to occur in the majority of cases - for a review see Sprugel, 1985, Section IIc). Actually h (T) is the primary productivity per unit biomass under optimal conditions of the nutrient concentration S in the trees. If S is too low the productivity will be less than the maximum achievable at that given biomass T. Thus h(T) is multiplied by an efficiency e (S) which is increasing from 0 and saturating to 1 when the concentration S is above a certain threshold.

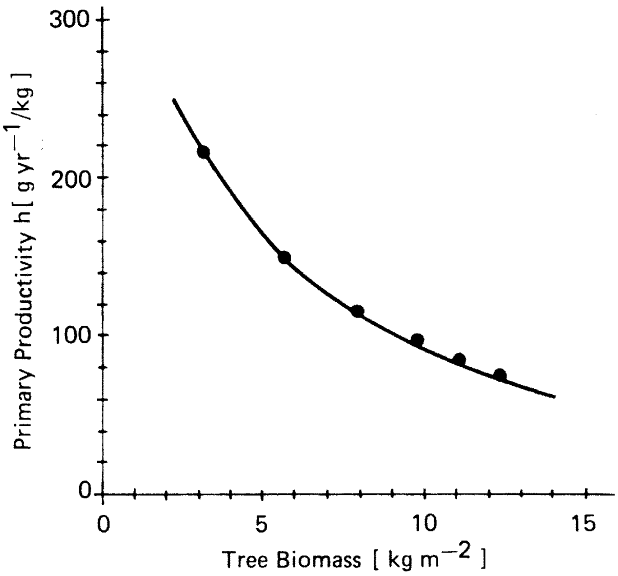

Fig. 2. Maximum production h of new tree biomass per unit biomass as a function of tree biomass. The graph is actually obtained from data on Balsam fir reported by Sprugel (1984).

The nutrient uptake from the soil by each tree biomass unit is considered to increase linearly with the nutrient concentration (βN). This is equivalent to assuming that the root system is proportional to the aboveground standing biomass and that each root pumps up a constant amount of water where the nutrient is dissolved with concentration N. We therefore suppose that a tree cannot withstand an excessive and potentially harmful quantity of nutrient by limiting its uptake. This is at least partially unrealistic, since a tree can develop roots in a soil layer which is temporarily free from an excessive nutrient concentration. However, with ever-increasing nutrient loads - and this paper is mainly concerned with the long-term consequences arising from this occurrence - the nutrient is more or less homogeneously distributed in the whole soil and the assumption of purely passive uptake becomes less crude under this perspective.

The dying tree biomass (mT) contains a certain amount of nutrient (mTS). In the model, this is subtracted from the nutrient storage in the living biomass and a fixed fraction η is transferred back to the soil by decomposition. The remainder $1-\eta$ is not decomposable or is washed off the forest ecosystem. We make the strong assumption that decomposition is fast, since the flow of nutrient from the decomposable dead biomass (ηmTS) enters the balance of the soil pool without delay. It should be remarked, however, that the components of the dead biomass which are most readily decomposable (foliage and branches) are usually richest in nutrient content (see Sprugel 1984 for relevant data on balsam fir). For these components mineralization is achieved with a delay which is small when compared with tree lifetime.

In the balance of the soil nutrient pool, besides the positive contribution of decomposition and the negative one of tree uptake, there are two other terms. One is of course the external inflow W, which comes mainly from the atmosphere in the case of nitrogen and sulphur (Abrahamsen 1980); acid precipitation goes together with an increased supply W of nutrient. The other term ($-\alpha$N) is negative and takes into account both the leaching losses (which at least for nitrogen seem to be proportional to the nutrient concentration in the soil, Abrahamsen 1980) and the uptake by the remaining vegetation. This vegetation is assumed to be more or less constant so that its uptake is again proportional to N and its decomposition is part of W.

The final and most important comment concerns tree mortality m. So far and purposely, we have not specified any kind of functional relationship for this key factor of our model. If there were no adverse effects of acidic deposition, only the natural mortality would be present and might be assumed to be a constant coefficient. But, of course, acidic deposition does influence the mortality rate of trees. There are several ways in which a forest suffers from acidic deposition and the different sources of damage are in general present at the same time. Therefore, mortality is likely to be a function of all the variables related to acidic deposition, notably of W, N and S. In spite of all this, we will take into consideration three basic mechanisms of forest damage that lead to three different types of functional dependence for mortality, and separately analyze the consequences of each mechanism on the tree dynamics as though only one mechanism at a time were operating. This procedure is limiting on one hand, but on the other helps us to assess which of the three mechanisms is responsible for catastrophic effects.

One type of impact is the direct, immediate effect of air contaminants on vegetation. There can be foliar damage, influence on photosynthesis and respiration, enhancement of microbial pathogens, increased leaching of vital elements from the foliage, etc. (Smith 1981). In some cases there is a positive correlation between the nutrient inflow W, the magnitude of this direct damage, and the intensity of acid precipitation (notice that also the formation of photoxidants, which, in addition to acid rain, seem to play a central role in damaging crops and forests (*e.g.*, Skärby and Selldén 1984) is related to nitrogen, hence possibly to nutrient inflow). Thus the first mechanism we assume is that mortality is a function of W only. This function is roughly constant and equal to the natural mortality up to a certain point and then sharply incrasing.

The second mechanism is linked to the increase in soil acidity. There are several buffering reactions in the forest soils that can partially counteract the effect of acidic deposition (Ulrich 1983), but when pH falls below 5 the solubility of aluminum increases sharply and if pH is less than 4.2, which occurs in many forest soils in Central Europe, the aluminum buffer range is reached: aluminum ions are present in high concentrations and can be toxic to bacteria and plant roots. Simultaneously the leaching of calcium, magnesium and possibly potassium, which are vital, though usually monlimiting factors, is enhanced. With even lower pH values most heavy metals are mobilized and can damage the trees. As soil acidity is positively correlated with nutrient concentration in the soil, the assumpbion that follows from this second mechanism is that mortality is constant up to a certain value of N, beyond which it increases very sharply with N.

The last mechanism is another indirect effect. Acid precipitation, bringing about an increase in the inflow W, can cause an accumulation of nutrient in the trees. Usually this is not harmful; on the contrary, it enhances the primary productivity, as already pointed out. However, when very high levels of concentration are reached, tissue injury can result. As for nitrogen, this threshold concentration is about 2% of dry weight for conifers (Ingestad 1979) and about 4% for deciduous species (Ingestad 1981). As a consequence, we assume that, if this mechanism of tree damage is operating, the mortality rate m is a function of the tree nutrient concentration S and, as usual, is constant over a wide range and then sharply increases.

Analysis of the model

This section is devoted to analyzing the tree dynamics as predicted by model (1) under different nutrient loads W and in the three cases described above. Before proceeding further, it is convenient to restate equations (1) by taking into account that $Q = TS$, hence $\frac{dS}{dt} = \frac{1}{T}\frac{dQ}{dt} - \frac{S}{T}\frac{dT}{dt}$ · Thus

$$\frac{dT}{dt} = [-m + e(S)\,h(T)]\,T$$

$$\frac{dS}{dt} = \beta N - e(S)\,h(T)\,S \tag{2}$$

$$\frac{dN}{dt} = W - \alpha N - \beta NT + \eta mTS$$

and we are left with the three state variables T, S and N only.

Direct tree damage. In this case the mortality rate is assumed to be a function of the nutrient input W only: $m = m(W)$. It is further hypothesized that $m(W)$ is practically constant up to a certain point, after which it sharply increases. Given a constant W, the corresponding steady states of model (2) can be found by imposing the time constancy of T, S and N, or equivalently the simultaneous vanishing of $\frac{dT}{dt}$, $\frac{dS}{dt}$ and $\frac{dN}{dt}$ ·

There are two possibilities:

i) $T = 0$ and consequently $N = W/\alpha$, $S = \hat{S}$ where $\hat{S}$ is the unique solution of the equation

$$e(S)\,S = \frac{\beta W}{\alpha h(0)}\ \cdot$$

This equilibrium corresponds to the extinction of trees.

ii) $e(S)\,h(T) = m(W) = \beta N/S$ from the first and second of equations (2). Hence

$$N = m(W)\,S/\beta$$

and

$$T = h^{-1}\,(m(W)/e(S)) \tag{3}$$

where h^{-1} is the inverse function of h. Since h (T) is a decreasing function of T and e (S) an increasing and saturating function of S, the right-hand side of equation (3) is an increasing and saturating function of S. From the last of equations (2) and substituting for N one gets

$$T = \frac{W}{m(W)(1-\eta)\,S} - \frac{\alpha}{\beta\,(1-\eta)}\ \cdot \tag{4}$$

If the curves given by equations (3) and (4) intersect in the first quadrant, there is a nontrivial equilibrium characterized by $T > 0$, $S > 0$, $N > 0$. However, this does not occur for all values of W. In view of the assumptions on m (W) it follows that W/m(W) is a bell-shaped function, hence the situation is as portrayed in Fig. 3. When W is low ($W = W_1$ in figure) there is no intersection. As soon as W takes on the critical value W_2 intersections start appearing and can be found for a range of intermediate W values (W_3, W_4, W_5). When W equals W_4 the curve described by equation (4) begins moving leftward, so that intersections finally disappear for W equal to the critical value W_6. For very high nutrient loads (W_7) no intersection is possible. It is worthwhile to remark the smooth pattern - i.e., without jumps in T or S - of intersection appearance-disappearance.

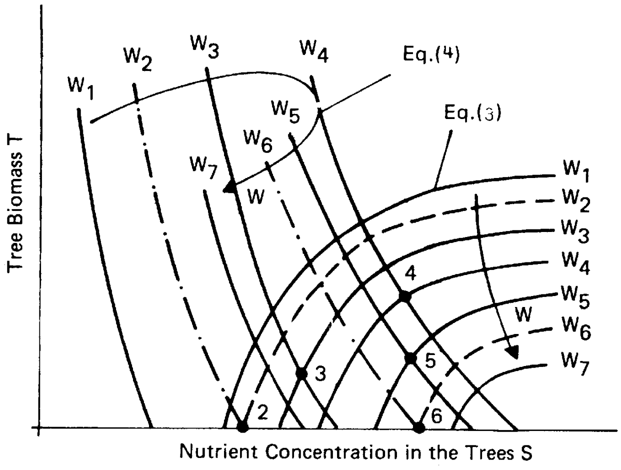

Fig. 3. The two families of curves obtained from equations (3) and (4) by letting W vary. Intersections of curves with equal W (dots labelled as 2, ... 6) give values of T and S at nontrivial equilibrium.

We can summarize by saying that when W is too low or too high only tree extinction is a feasible equilibrium, while for intermediate nutrient inputs there exists also a nontrivial equilibrium.

Stability of these equilibria can be studied via linearization (see Appendix). It is thus possible to show that tree extinction is stable when it is the unique steady state, whereas, for the W values that permit the existence of a viable equilibrium, this latter becomes stable and extinction unstable.

In conclusion, when W is very low (insufficient nutrient inflow, hence poor forest soil) or very high (heavily polluted precipitation) the forest is driven to tree extinction, while trees can survive when W is intermediate. These two regimes are smoothly joined, without any catastrophic event, as shown in Fig. 4, which displays the qualitative behavior, of T, S and N as function of W.

Increased soil acidity. This mechanism implies that mortality is a function of the nutrient soil concentration N only: $m = m(N)$. As usual, mortality is suppo-

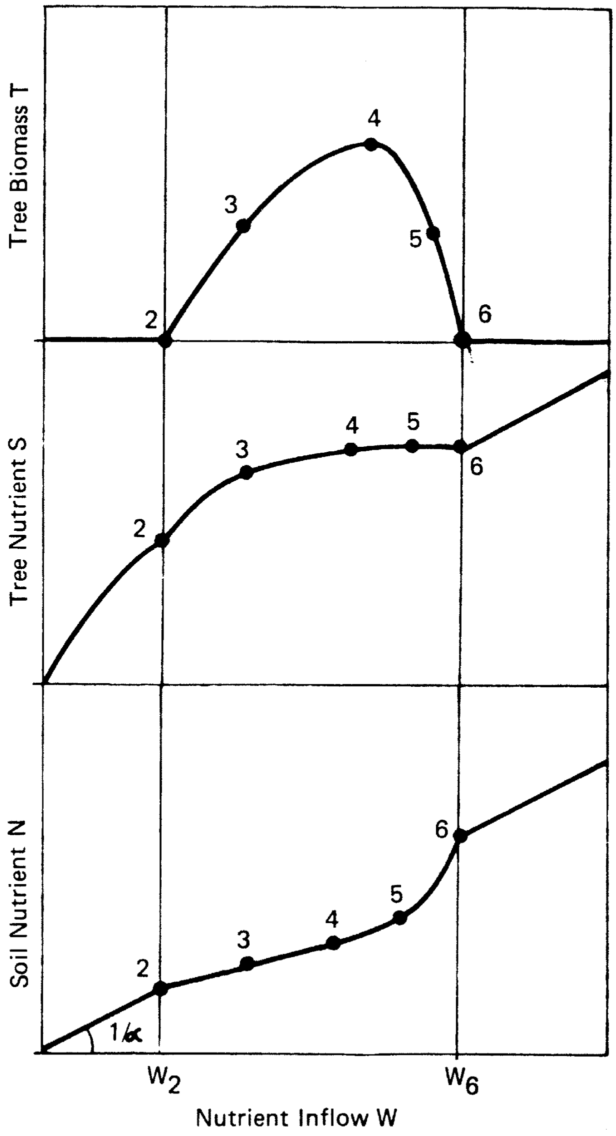

Fig. 4. Pattern of variation of the stable steady state as a function of W when the first mechanism of tree damage is operating. Points 2, ... 6 make reference to those in Fig. 3.

sedly constant up to a threshold, beyond which it rapidly increases.

The equilibria corresponding to a constant inflow W can be found by setting $\frac{dT}{dt} = \frac{dS}{dt} = \frac{dN}{dt} = 0$ in equations (2). Again, there are two possible outcomes i) tree extinction, namely $T = 0$, $N = W/\alpha$, $S = S$ with S being the unique solution of the equation

$$e\,(S)\,S = \frac{\beta W}{\alpha h\,(0)}$$

ii) $e\,(S)\,h\,(T) = m\,(N) = \beta N/S$ from the first and second of equations (2). It follows that the nutrient concentration S in the trees is related to the one in the soil N by

$$S = \beta N/m\,(N)$$

which, owing to the assumptions on the mortality rate, is a unimodal function of N. Substituting for S one also obtains

$$T = h^{-1}\,[m\,(N)/e\,(\beta N/m\,(N))]. \qquad (5)$$

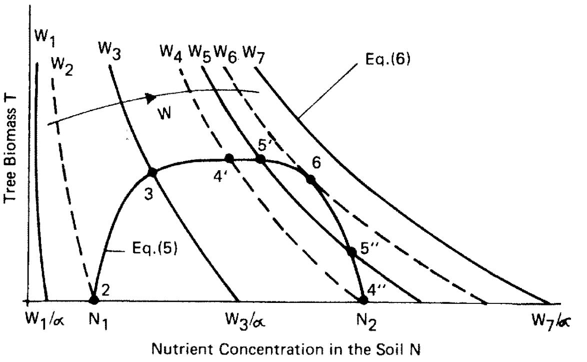

Fig. 5. The curve described by equation (5) and the family of curves obtained from equation (6) by letting W vary. Intersections yield values of T and N at nontrivial equilibrium. When double intersection occurs, distinction is made between stable (4' and 5') and unstable (4'' and 5'') equilibria.

Since e (S) is increasing and saturating, e ($\beta N/m\,(N)$) is dome-shaped, possibly with a flat top. Therefore, as h (T) is decreasing, the right-hand side of equation (5) is unimodal, positive in the interval (N_1, N_2) and may have a flat top (see Fig. 5). Notice that it does not depend upon W.

From the last of equations (2) and replacing S by its expression as a function of N it is easy to obtain

$$T = \frac{1}{\beta\,(1-\eta)}\,[\frac{W}{N} - \alpha]. \qquad (6)$$

Thus there exist nontrivial equilibria if there are intersections between the curves described by equations (5) and (6). Fig. 5 displays the most interesting situation which arises when the curves given by equation (6) are not too steep (this occurs when the leaching coefficient α is not too large). When the nutrient inflow is low ($W = W_1$ in figure) no intersection occurs. At W equal to the critical value $W_2 = \alpha N_1$ intersection begin showing up. For intermediate values of W, only one intersection is present (3 in Fig. 5), but as soon as the nutrient load reaches the critical value $W_1 = \alpha N_2$ a second intersection appears and stays on for a range of high W values ($W = W_5$). When W equals the critical value W_6 (the one for which the curve described by equation (6) is tangent to the curve described by equation (5)) the two intersections collapse into one. At even higher nutrient inputs no intersection occurs.

The complex and articulate pattern emerging from the above analysis can be summed up as follows. When the nutrient input is very low or very high ($W < W_2$ or $W > W_6$) only tree extinction is a feasible steady state. Intermediate-low nutrient loads ($W_2 < W < W_4$) imply the existence of the trivial and of one nontrivial equilibrium. For intermediate-high W's ($W_4 < W < W_6$) there exist the trivial and two nontrivial equilibria.

It is very important to assess the stability of all these equilibria. When this is accomplished via linearization (see Appendix), the following results are obtained:

$W < W_2$ or $W > W_6$	tree extinction is stable
$W_2 < W < W_4$	the unique viable equilibrium is stable; tree extinction is unstable
$W_4 < W < W_6$	the viable equilibrium with higher tree biomass (5' in Fig. 5) is stable; the other (5'' in Fig. 5) is unstable; tree extinction is stable.

Therefore, when there is insufficient nutrient inflow or heavy acidic deposition, trees are doomed to extinction. For intermediate-low nutrient inputs the forest is healthy and trees can survive, but under the burden of intermediate-high loads the trees can be attracted either to viability or to extinction depending on the initial conditions: if the tree biomass is impoverished, but not too much, *e.g.* by a storm or a fire, the forest can regrow; if it is severaly depleted, the trees become extinct.

It is just this flip-flopping pattern of equilibria which occurs in a certain W range that determines the essentially catastrophic impact of increased soil acidity. This is clarified in Figure 6 which qualitatively shows how W affects the T, S and N values corresponding to stable steady states. It should be remarked that the curve of the tree biomass has qualitatively the same shape as in Fig. 1. Thus all the relevant discussion on catastrophes also pertains here. When acid precipitation becomes so intense that the nutrient load even slightly exceeds the threshold W_6, trees inevitably collapse fron an equilibrium characterized by a normal standing biomass to extinction. Moreover, the mere decrement of the nutrient load below W_6 is not sufficient to get the trees out of the extinction trap. Recolonization can occur only after a substantial decrease (below W_4) of nutrient inflow. In this case a reversed catastrophe (regeneration) takes place. This hysteresis pattern is typical of a so-called fold catastrophe.

Excessive nutrient accumulation. This last impact is translated into a mortality $m = m(S)$ that is constant up to a threshold and thereafter rapidly increases. The analysis of equilibria and their stability very much resembles the one performed with the previous mechanism. So, we do not go into much detail.

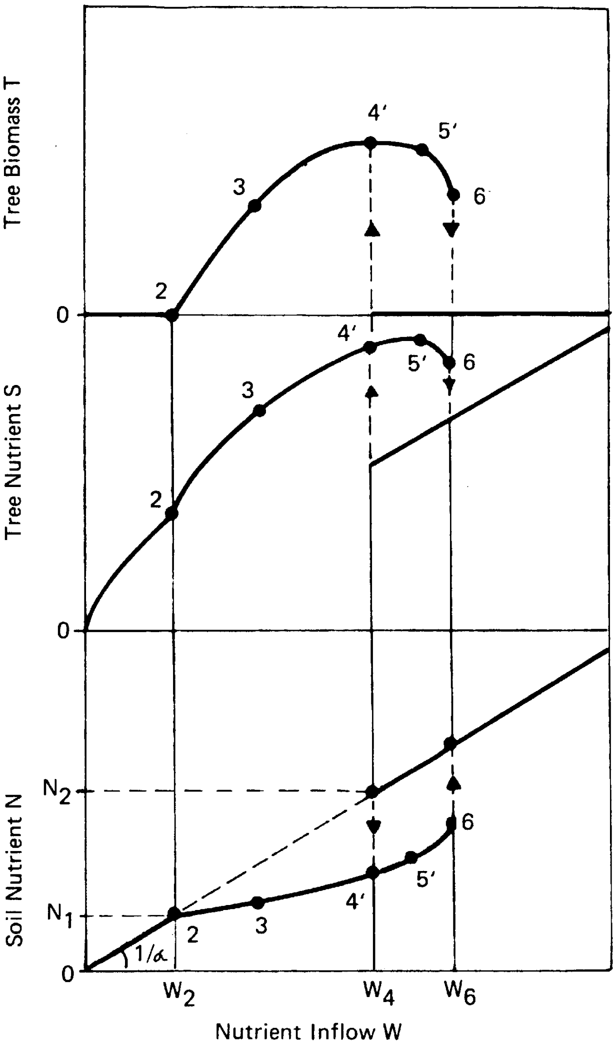

Fig. 6. Pattern of variation of the stable steady states versus nutrient inflow when the second mechanism of tree damage is operating. Point labelled 2, ... 6 correspond to those in Fig. 5.

Besides the usual trivial extinction equilibrium, the nontrivial ones satisfy the following relationships

$$N = \frac{m(S)\,S}{\beta}$$

$$T = h^{-1}\left(\frac{m(S)}{e(S)}\right) \qquad (7)$$

$$T = \frac{W}{(1-\eta)\,m(S)\,S} - \frac{\alpha}{\beta(1-\eta)}\,. \qquad (8)$$

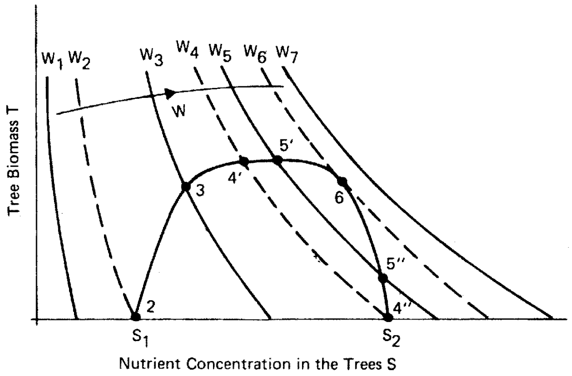

Fig. 7. The curve described by equation (7) and the family of curves obtained from equation (8) by letting W vary. Intersections give values of T and S at nontrivial equilibrium. When double intersection occurs, distinction is made between stable (4' and 5') and unstable (4'' and 5'') equilibria.

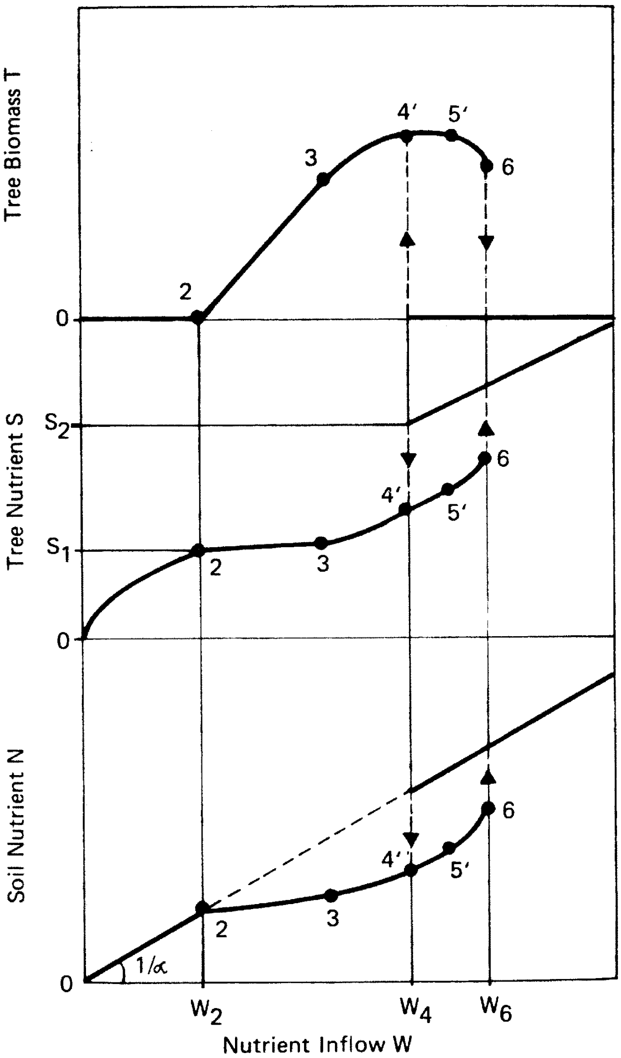

Fig. 8. Pattern of variation of the stable equilibria as functions of W when the third mechanism of tree damage is operating. Points labelled 2,... 6 correspond to those in Fig. 7.

If there is any intersection in the first quadrant of the plane S-T between the curves given by equations (7) and (8), then viable steady states exist. Since the threshold at which m (S) sharply increases is conceivably higher than the threshold at which the efficiency e (S) saturates, the function m (S)/e (S) is bowl-shaped with a flat bottom. Consequently the right-hand side of equation (7) is unimodal, positive in the interval $(S_1, S_2$ and has a flat top (see Fig. 7). Moreover it is independent of W. The curve given by equation (8) obviously decreases with S and depends on W, so that the situation is as represented in Fig. 7. The relevant discussion is not given, because it is totally analogous to that for the previous mechanism (Fig. 5). Also the stability analysis leads to very similar results, which are summarized in Fig. 8. When the nutrient inflow is too low, only tree extinction is stable; by increasing W, one obtains a positive and increasing tree biomass. Further increments of the nutrient load, leading to a harmful accumulation in the trees, induce first a decrease of the standing biomass and then a dramatic collapse, when the threshold W_6 is exceeded. As in the previous case recovery is possible only if the nutrient inflow is substantially decreased (below W_4).

The remark that is worth making is that, even if trees are capable of successfully withstanding the direct damage from soil acidity and only the indirect impact from excessive nutrient accumulation is operating, catastrophes still occur.

Conclusions

We have illustrated how three possible impacts of acidic deposition on tree survival and growth could affect the temporal dynamics of tree biomass. From the analysis of equilibria and their stability two different patterns have emerged. The direct injuries to vegetation, which are not only the most conspicuous, but also the most important impact of acidic deposition, cannot give rise, formally speaking, to a catastrophe, while the other two mechanisms can. This fact should cause no surprise since increased soil acidity and excessive nutrient buildup in the trees entail the accumulation in the forest ecosystem of a stress that does not show up immediately, but explodes when a breakpoint is reached.

This result should not be taken as an advice to neglect the direct impact on vegetation. This mechanism, as we have shown, causes the decline and possibly the extinction of the trees subject to increased acid precipitation in any case. The course of these unpleasant events can be quite fast, thus becoming a catastrophe in practice, though not on a mathematical basis. Therefore, if there is a small lesson that may emerge from this paper, is rather not to neglect the other two mechanisms which, albeit indirect and thus given less attention, can in the long run cause the unexpected collapse of forest ecosystems.

Our analysis has been purely qualitative, since the model is simplistic and the values of many parameters are in practice unknown and could hardly be estimated. However, we believe that, before refining the present model in order to make it a useful tool for operative forecast and real management, it would be worthwhile to analyze other major mechanisms of impact with the same aim and style used in this paper. Along this line, we have already investigated the interplay between acidic deposition and forest exploitation (Gatto and Rinaldi, 1987), while interesting preliminary results have been obtained with two other models, one (Muratori and Rinaldi, 1988) describing the influence of acidic deposition on host-parasite interactions and the second detailing the role of soil biochemistry.

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APPENDIX

This appendix is devoted to explaining how the stability analysis of the equilibria of model (2) can be performed. The method we use is called linearization, which consists of replacing the dynamics of equations (2) in the neighborhood of each equilibrium by the approximate dynamics of an equivalent linear model. More precisely, if we denote by $\overline{T}$, $\overline{S}$, $\overline{N}$ the values of the state variables at equilibrium and by $\Delta x\,(t)$ the vector whose components are $T\,(t)-\overline{T}$, $S\,(t)-\overline{S}$, $N\,(t)-\overline{N}$, then the time evolution of Δx is approximately given by the vector linear differential equation

$$\frac{d\Delta x}{dt} = \overline{J}\,\Delta x$$

where $\overline{J}$ is the Jacobian matrix of model (2) evaluated at $\overline{T}$, $\overline{S}$, $\overline{N}$. If the matrix $\overline{J}$ has eigenvalues with negative real parts, the corresponding equilibrium of model (2) is stable.

We can apply this procedure to the analysis of the first mechanism where the mortality is given by m = m (W). In this case the Jacobian matrix is

$$J = \begin{bmatrix} -m(W)+e(S)h(T)+e(S)T\frac{dh}{dT} & Th(T)\frac{de}{dS} & 0 \\ -e(S)S\frac{dh}{dT} & -h(T)(S\frac{de}{dS} + e(S)) & \beta \\ -\beta N + \eta mS & \eta mT & -\alpha - \beta T \end{bmatrix}$$

By evaluating J at the trivial equilibrium T = 0, $S = \tilde{S}$, N = W/α, one gets

$$\bar{J} = \begin{bmatrix} -m(W) + e(\tilde{S})h(0) & 0 & 0 \\ -e(\tilde{S})\tilde{S}\frac{dh(0)}{dT} & -h(0)(\frac{de(\tilde{S})}{dS}\tilde{S} + e(\tilde{S})) & \beta \\ -\beta W/\alpha + \eta m(W)\tilde{S} & 0 & -\alpha \end{bmatrix}$$

Since the eigenvalues of J are, in this case, the elements on the diagonal, the trivial equilibrium is stable if $m(W) > e(\tilde{S})h(0)$. It is easy to understand that this last condition is equivalent to the non-existence of the viable equilibrium. In fact, nontrivial steady states exist only when the intersection S_3 of the curve given by equation (3) with the S axis lies on the right of the intersection S_4 of the curve given by equation (4) (see Figure 3). Since

$$S_3 = \frac{\beta W}{\alpha m\ (W)}$$

$$e\ (S_4) = m\ (W)/h\ (0)$$

$$e\ (\tilde{S})\ \tilde{S} = \frac{\beta W}{\alpha h\ (0)}$$

it turns out that

$$e\ (S_4)\ S_3 = e\ (\tilde{S})\ \tilde{S}.$$

Therefore, from the fact that e (S) is increasing, it follows that either $S_3 \le \tilde{S} \le S_4$ or $S_4 \le \tilde{S} \le S_3$.

On the other hand, the inequality

$$m\ (W) > e\ (\tilde{S})\ h\ (0)$$

is equivalent to

$$e\ (S_4) > e\ (\tilde{S}),$$

hence to

$$S_4 > \tilde{S} > S_3 .$$

The procedure is more complex when dealing with the nontrivial equilibrium. In this case the Jacobian matrix is

$$\bar{J} = \begin{bmatrix} e(\bar{S})\frac{dh(\bar{T})}{dT}\bar{T} & \frac{de(\bar{S})}{dS}h(\bar{T})\bar{T} & 0 \\ -e(\bar{S})\bar{S}\frac{dh(\bar{T})}{dT} & -h(\bar{T})(\frac{de(\bar{S})}{dS}\bar{S} + e(\bar{S})) & \beta \\ -(1-\eta)m(W)\bar{S} & \eta m(W)\bar{T} & -\alpha-\beta\bar{T} \end{bmatrix}$$

and the eigenvalues cannot explicitly be computed. However, there exists a condition on the elements of $\bar{J}$ which guarantees the negativity of the eigenvalues, real parts. If we indicate the trace of $\bar{J}$ by tr $\bar{J}$, the determinant by det $\bar{J}$ and the sum of the second order leading minors by Σ then this condition is

$$\mathrm{tr}\ \bar{J} < 0,\ \det \bar{J} < 0,\ \Sigma\ \mathrm{tr}\ \bar{J} < \det \bar{J}$$

As for the trace, it results

$$\mathrm{tr}\ \bar{J} = e\ (\bar{S}) \frac{dh\ (\bar{T})}{dT} \bar{T} - h\ (\bar{T})(\frac{de\ (S)}{dS} \bar{S} + e\ (\bar{S})) - \alpha - \beta\bar{T}$$

and, since h (T) is a decreasing function and e (S) an increasing one, the first inequality is satisfied. As for the determinant, one has

$$\det \bar{J} = e\ (\bar{S}) \frac{dh\ (\bar{T})}{dT} \bar{T}\ (\alpha m\ (W) + (1-\eta)\ \beta m\ (W)\ \bar{T}) -$$

$$- \frac{de\ (\bar{S})}{dS} h\ (\bar{T})\ \bar{T}\beta\ (1-\eta)\ m\ (W)\ \bar{S}$$

Since h (T) is decreasing, e (S) increasing and $\eta < 1$, the negativity of the determinant is also verified. The proof of the third inequality is straightforward but cumbersome and is not reported here.

The linearization procedure can also be applied to the stability analysis for the second and third mechanisms of tree damage. The relevant computations are standard algebra, though long and boring, and are not given here.

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