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Strategies Towards Scenarios of Forest Damage Due to Air Pollution

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PREFACE

From mid 1970's, symptoms of a new forest decline which is spreading at an increasing speed have been detected in Central Europe. Today, many scientists share the opinion that the new symptoms are connected with air pollution, yet no single pollutant or damage mechanism has been proved as the single cause of forest dieback.

As a response to the need for greater understanding of the problem, scientists are gathering empirical evidence and developing new methods for treating the response of forests to external stress factors. Part of this development, mathematical modeling is becoming more important also in this field.

This paper reviews the current state of knowledge about the relationship between air pollution and forest damage in Europe in a mathematical modeling framework. It will serve as a basis for another submodel of the RAINS model (Regional Acidification and INformation Simulation).

Leen Hordijk

Acid Rain Project Leader

ABSTRACT

This report is a review of the requirements and possibilities of developing a regional scale, long term model for the response of forests to atmospheric pollution. A general input-output structure of such models is delineated, and the input, output and potential model structures are considered. With the objective of specifying the input variables, potential causes of forest damage are classified according to their dynamic properties. Forest damage is further specified, so as to define the output variables. Theoretical and empirical models for the response of forests to air pollution are reviewed, as well as some empirical evidence applicable to the models. The possibilities of constructing scenarios of damage over Europe are assessed, and the present theoretical and practical restrictions are evaluated.

STRATEGIES TOWARDS SCENARIOS OF FOREST DAMAGE DUE TO AIR POLLUTION

Annikki Mäkelä

1. INTRODUCTION

1.1. Forests in changing environment

From the middle of the 1970's, symptoms of a new forest disease have been detected in Central Europe. At least 1.5 million hectars of forest has so far been totally destroyed (Wentzel 1982), and much more is classified as "affected" (Walderkrankung, 1983). The disease first attacked silver fir (*Abies alba*), 80% of which has now been lost, and it is currently making progress in stands of Norway Spruce (*Picea abies*) (Walderkrankung, 1983). Recent observations indicate that the disease is also spreading out to Sweden (Kvist & Barklund, 1984).

Today, many scientists share the opinion that the emergency of the new symptoms was connected with a thorough change in the pollutant load. In Wentzel's (1982) classification, a new phase of air pollution was reached in

the early 1960's, when local and regional emission sources had gradually given way to global pollution with remote transport of chemicals and an ever-denser network of local sources such as industries and traffic. Simultaneously, the variable load with acute episodes had been replaced by a low but permanent pollution level.

1.2. Scenario analysis to assess alternative policies

The ratification of the Geneva Convention on Transboundary Air Pollution in 1983 showed that the European countries are deeply concerned about the problem and determined to take action that will reduce emissions. Since the aim is not only to reach an ecologically sustainable level of emissions, but also to do this at the lowest possible cost, rather detailed information about the expected consequences of alternative industrial policies should be available to the decision makers.

With the objective of increasing the background information, the IIASA Acid Rain Project is developing a model system (Regional Acidification INformation Simulation, RAINS) which produces scenarios about the consequences of alternative energy policies and emission control actions. The input to the model system is the pollutant emissions in Europe as a function of space and time, and it produces scenarios of (1) how the input strategy is reflected in the physical environment, e.g. air and soil, and (2) how the change in the physical environment affects different ecosystems, especially lakes and forests.

At present, the input comprises an ***energy pathway*** which depends upon selected energy sources and pollutant reduction policies in 27 European countries. From this input, the system first calculates the annual

emissions of sulphur over Europe. Then the atmospheric transport of sulphur and the deposition are calculated on an annual basis (Alcamo et al. 1984). The consequent soil acidification is determined using a model based on a buffer range theory (Kauppi et al. 1984) (See Figure 1). Further, a model of lake acidification is under preparation (Kämäri, 1984).

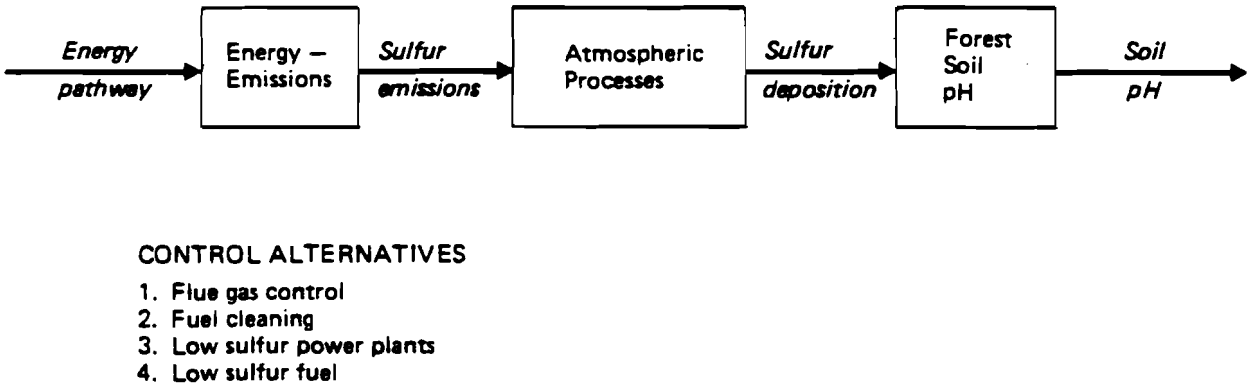

Figure 1. The present structure of the RAINS model.

The above concerns changes in the physical environment, termed as task (1) above. So far, the response of forests to this change has been considered assuming that soil acidity has an upper limit which cannot be exceeded without critically disturbing the ecosystem (Kauppi et al. 1984). The present objective is to delineate a strategy of how to further promote task (2), i.e. modelling the response of forests to the change in their physi-

cal environment.

1.3. Developing forest impact scenarios

Using an accurate language, to develop scenarios of forest damage over Europe means, to produce a spatially and temporally distributed estimate for the degree of damage as a function of air pollution, which is similarly distributed over space and time. In space, or geographically, the output should extend over Europe, and in time it should reach about 50 years forward. The time resolution of the existing submodels is from one month to one year, and the spatial grid net consists of rectangles with 50-150 km sides.

Since the existing atmosperic submodels of the RAINS model deal with the distribution and deposition of sulphur, a natural continuation would be to base the forest damage scenarios on sulphur derivatives. Furthermore, as the injurious consequences of soil acidification have already been considered (Kauppi et al. 1984), an obvious item for future work seems the impacts of airborne sulphur dioxide. However, as many other potential causes of damage have been gaining emphasis in the scientific discussion since the start of the project, it was regarded necessary to review the problem from a wider perspective. Section 2 treats the most frequently referred potential causes of damage, with the objective of choosing a suitable input for the scenario model.

The desired output of the model, the degree of forest damage, is problematic because it does not have a standard meaning, but a variety of perceptions exist. Further, if a qualitative meaning is specified, a selection of potential quantifications will remain. In order to specify a suitable output

variable, Section 3 considers different ways of defining and measuring damage. Section 4 aims at connecting output to input, and is devoted to both theoretical and empirical models for the response of forests to air pollution. Some empirical evidence are also reviewed. Section 5 deals with the possibilities of constructing scenarios of forest damage in Europe and evaluates the present theoretical and practical restrictions.

2. POTENTIAL CAUSES OF DAMAGE

Forest damage caused by air pollution is not a new phenomenon. In the early days of industrialization, it was not unusual that the neighbourhood of a smelter, for instance, suffered frequent exposures to high concentrations of pollutants, and acute symptoms of damage occurred. The first systematic studies of such events were written in the middle of the 19th century (Stöckhardt, 1850, 1871; Schroeder and Reuss, 1883). Later on, stacks became higher and the neighbourhood exposed to impurities enlarged. This began the period of regional air pollution and chronic damage. Both of these periods, classified as the first and the second phase of air pollution by Wentzel (1982), share the property that the cause of damage was relatively easy to track, the symptoms occurring over a limited area as well as during a restricted time. Further, the removal of the pollution source normally also removed the damage (Wentzel, 1982), a reason why there was apparently no need to consider the effects as accumulating or delayed. Until very recently, the most frequent explanation of forest damage was sulphur dioxide originating in energy combustion.

As mentioned above, a new phase of air pollution was reached in the early 1960's (Wentzel, 1982). Although severe concern of widespread dam-

age was expressed already in the 1960's (Knabe, 1966), the problem only gained wide publicity in the late 1970's when forest damage started to proceed rapidly. The explanation based on sulphur dioxide no more seemed to function, since (1) damage occurred in areas where concentrations of sulphur dioxide were far below expert recommendations, such as the 25 $\mu g / m^3$ recommended by IUFRO (Materna, 1983), and (2) the emissions of sulphur reduced in many European countries since the 1970's (Figure 2). Therefore, new hypotheses about the causes and mechanisms of the disease have entered the discussion. Ozone and soil acidification, in particular, have been brought on the list of potential agents of damage, but also other elements such as heavy metals, secondary photooxidants other than ozone, and even the radioactive release from nuclear power plants, as well as combinations of all these have been suggested.

From the point of view of modelling, it is illustrative to group the hypotheses on the basis of the dynamic response mechanisms they presume. Such a classification has already been presented by Kohlmaier et al. (1984), who distinguish between direct and delayed impacts. The delayed impacts can be further divided into delays in (1) physical environment and (2) biological response. Thus, the hypotheses concerning the potential causes of damage can be classified as follows:

[1] Direct (non-delayed) impacts.

The effect closely follows the exposure, i.e. the time development of damage follows that of the air pollutant concentration. What is actually meant by a delay - a second, a month, or a year - depends upon the time scale of the processes considered. As regards forests , the impact can

hardly be considered delayed if it occurs within one year after the exposure. The earlier, acute damage attributed to sulphur dioxide was consistent with this impact mechanism, but because of reasons mentioned above, this is no more the case. The hypothesis that ozone is the cause of damage, instead, gains its very support from the fact that in Western Europe, the concentrations of nitrogen oxides and their airborne derivatives, such as ozone, have increased in pace with the new forest disease (Guderian et al., 1984, Prinz et al., 1982, Prinz et al., 1984). Development of sulphur and NO_x emissions in some countries are depicted in Figure 2 (Figures 2a, 2b, 2c).

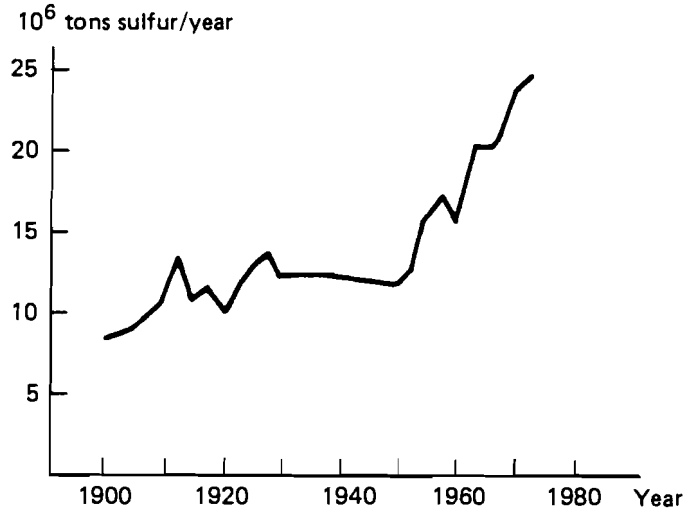

Figure 2a. Development of sulphur emissions in Europe (Forsurning ..., 1982).

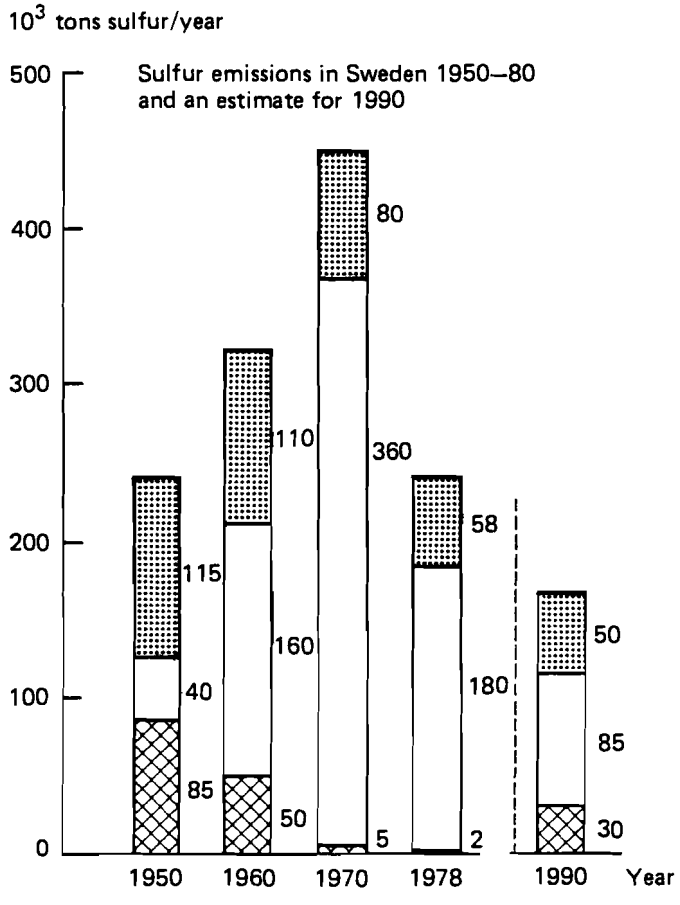

Figure 2b. Development of sulphur emissions in Sweden (Forsurning ..., 1982).

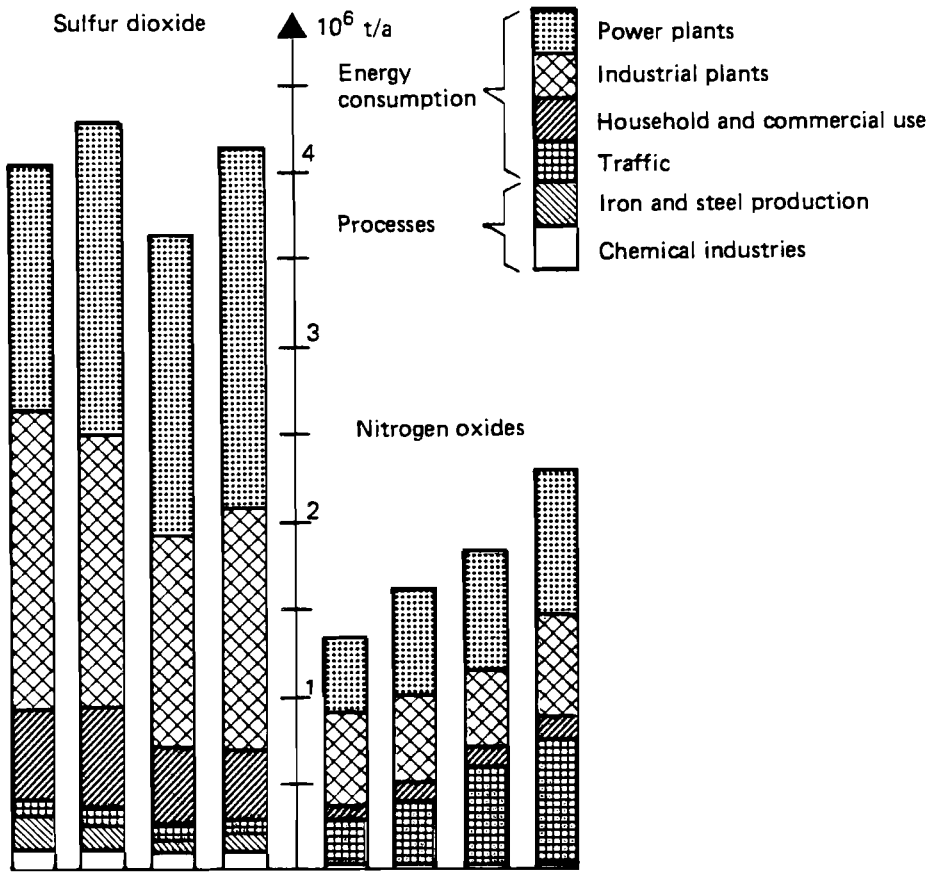

Figure 2c. Annual emissions of SO_2 and NO_x in the Federal Republic of Germany (UBA, 1977).

[2] Delay in the physical environment

The effect only occurs after a long time of exposure, because the toxification of the environment is a slow process. However, as soon as the environment has become toxified, the trees start to show damage. The soil acidification hypothesis presented by Ulrich (e.g. Ulrich, 1983) belongs to this group. Hence, sulphur can cause damage through a delayed accumulation process. However, since other substances have

not yet been investigated in this respect, they cannot be excluded either.

[3] Delay in the response

The damage is delayed, but the delay is connected with the response of the tree rather than its environment. The recent stress hypothesis (e.g. Wentzel, 1982, Schütt et al., 1983, Matzner and Ulrich, 1984) assumes an impact mechanism of this type. In its general form, the hypothesis claims that different stress factors can cause a similar chain of events that lead to the observed symptoms, including an interaction with natural stress factors. Therefore, it is important to get insight into the eco-physiological processes that are common to all the factors that lead to the disease. Certain substances have been pointed out as important stress factors, and the corresponding chains of injury have been sketched. Wentzel (1982) assumes that it is sulphur dioxide that initially launches the process, Schütt et al. (1983) describe a chain initiated by an air-pollutant imposed decrease in photosynthesis, and Matzner and Ulrich (1984) suggest that it is the acidified soil that imposes the fatal pollutant stress.

3. DAMAGE

3.1. Concept of damage

When the effect of pollutants on plants is studied from a scientific point of view, all reactions of plants to the abnormal factors are of interest. In decision making, however, only those changes that somehow reduce the value of the plant are important. To make a distinction between these two

types of impact, Guderian (et al. 1960; 1977) suggests that the terms "injury" and "damage" should be used in specific meanings. Though not necessarily harmful, all pollutant-imposed reactions should be called "injury", while "damage" should include those changes that reduce the intended value or use of the plant. In accordance with this definition, we shall use the term damage to indicate a harmful change in the plant's normal behaviour.

A more accurate definition of damage requires that the limit between accepted and harmful behaviour is specified. In the following, the area of accepted behaviour will be called the norm.

The characterization of the norm is a value judgement. What people regard accepted depends upon their point of view, experience and objectives. For instance, biochemists would look at disturbances in the vital reaction chains, eco-physiologists would measure decrease in photosynthesis and increase in leaf necrosis, foresters would perhaps point out declining timber yield, while forest hikers would be most interested in healthy looking canopies.

From the point of view of the present objective, the concepts of *visible injury* and *growth reduction* seem the most important. They concern trees and forests rather than organs and processes, and a great degree of measurement and monitoring has been based on these concepts. They are also in the interest of the general public and of forest industry.

3.2. Visible injury

Visible injury is a natural definition of damage if the forest basically serves a social function, especially providing recreation areas and contributing to the scenery. Also, in areas where the pollutant situation has already become serious it may be the only sensible way of viewing the problem. Hence, most of the recent surveys and reports of forest decline in Central Europe are based on this view of forest damage (Walderkrankung, 1983; Materna, 1983).

Visible injury occurs as leaf necrosis, immature leaf and needle death, and at a further stage, the death of branches and the fall-off of the top. When an individual tree is concerned, the reference norm is a tree that looks healthy, i.e. does not show any of the above symptoms. Since the occurrence of visible injury varies among individual trees, the definition of forest damage requires that one specifies how large a proportion of trees is accepted to show the symptoms before damage is reported.

A measure of the degree of visible damage of individual trees that has already become standard is the number of needle age classes in the crown. In the Federal Republic of Germany, for instance, three damage classes are separated, and standard examples of the classes have been published to unify the quantification procedure (Walderkrankung, 1983). The damage of deciduous trees is more difficult to quantify because their appearance changes over the growing season. The percentage of necrotic leaves has been used as an indicator.

When quantifying visible injury at forest level, it is a question of finding a measure that aggregates any distribution of individual tree damage to an index that expresses the seriousness of the damage. As this is by no means a straightforward task, it is understandable that a variety of

different usages exist. The official reports of the Federal Republic of Germany simply distinguish between affected and healthy areas, though an exact definition of an affected area is not always provided. Materna (1983) has developed some fairly exact definitions of the type "a stand is affected to a degree I if x % of the trees are damaged to the degree J", which have been used in studies of forest damage in Erztgebirge. Mixed stands impose some further problems if one of the species shows symptoms while the others look healthy. The German reports then take the percentage of trees belonging to the damaged species and agree that the same percentage of the area shall be reported as damaged.

Owing to the different usages in quantifying visible injury, the figures that are reported as regards the extent of damage are not properly comparable. Especially, a distinction should be made between the extent of tree and forest damage. The former measures the percentage of trees damaged in the whole growing stock, while the latter gives a figure for the "affected area", which also contains healthy trees. Hence, the figures given at the forest level give higher percentages than those concerning individual trees only. To solve these problems, it is important to develop consistent ways of defining the degree of damage.

3.3. Growth reduction

Since damage as reduction in growth relates to the economic value of the forest, it is of interest in particular for countries that practise forestry. It may be suitable for predictive purposes also, as a severe reduction in growth has been observed to proceed the appearance of visible symptoms (Figure 3).

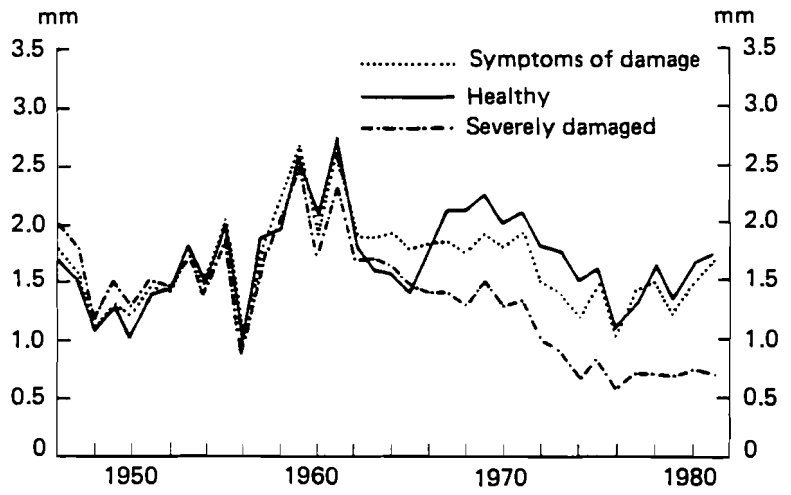

Figure 3. Growth development of healthy and diseased trees (Walderkrankung ..., 1983).

If damage is understood as reduction in radial growth, it is not as simple as in the case of visible injury to find a suitable norm. This is because the normal annual growth increment of trees varies as a function of several factors, even if the trees are growing under similar climatic and edaphic conditions. First, owing to the great within-stand variation, it is unjustified to refer to the normal growth of an individual tree. Instead, a whole stand should be chosen as a basic unit. Secondly, there is variation from year to year due to the stochastic variation in weather. This can be averaged out by looking at the growth increments over a period of time. Finally, since growth rate is age dependent, the norm becomes a function of tree age. The norm is hence a growth curve for a whole stand showing the average time

development of volume per unit area. This norm is species and site specific. Figure 4 depicts such curves for some species and growing sites in Finland (Koivisto, 1959).

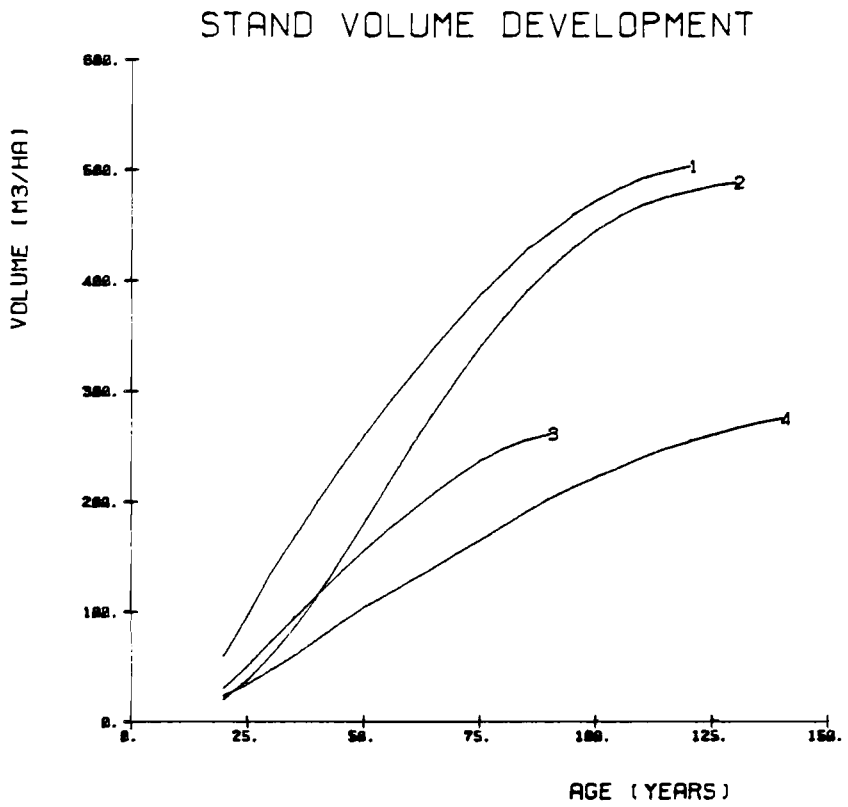

Figure 4. Stand volume development for some tree species and growing sites in Southern Finland according to Koivisto (1959).
1. Scots pine (*Pinus sylvestris*), *Myrtillus* type
2. Norway spruce (*Picea abies*), *Myrtillus* type
3. Silver birch (*Betula pendula*), *Myrtillus* type
4. Scots pine (*Pinus sylvestris*), *Cladonia*-type.

Detecting growth reductions may be difficult because small changes easily remain embedded. There are two kinds of methods for detecting injury from empirical data. Tree ring analysis provides a powerful method for detecting trends in diameter growth, and it has already been applied to

the search of air pollutant injury (e.g. McLaughlin et al., 1984; Hari et al., 1984). Data provided by forest inventories can also help to assess possible injury (e.g. Arovaara et al., 1984).

Measuring growth damage requires comparing and ordering different growth curves. As in the case of measuring visible injury at forest level, there is no unique way of defining a measure. One possibility is to compare the average annual yields over the rotation period in even-aged stands. In the National Acid Precipitation Assessment Program (NAPAP) carried out in the United States, species specific growth rates are being determined as a function of the air pollution load (Rosental,1984).

4. RESPONSE OF FOREST TO AIR POLLUTION

4.1. Conceptual model: stress theory

Air pollutants affect trees simultaneously with other environmental stresses. Trees react to this combination of stresses in a dynamic manner (e.g. Huttunen, 1975; Keller, 1978 ;Schütt et al., 1983; Matzner and Ulrich, 1984). A conceptual framework of how plants respond to environmental stress is provided by the concepts of strain and resistance, developed by Levitt (1972). Stress is defined as an environmental factor capable of inducing a potentially injurious strain in living organisms. Strain is any change in the normal behaviour of the organism, either physical or chemical. If the strain can be removed by removing the stress, it is elastic. Injury is equivalent with plastic or irreversible strain. The strain imposed by a specific stress depends upon the resistance of the organism. A measure for resistance is given by the magnitude of the lowest stress capable of imposing plastic strain.

Strain development and emergency of injury are dynamic processes. For instance, both strain and resistance can change slowly under constant stress, and the outbreak of injury may be delayed. Vice versa, if the stress is removed, the strain need not disappear immediately, but it may either stay the same or start decaying gradually over time. To clarify the dynamics, Kauppi (1984) distinguishes strain rate from accumulated strain. In these terms, air pollutant in the air could be identified with stress, the pollutant gas entering plant tissue would be strain, and the accumulated effects of this extraneous compound would become accumulated strain. Hence, constant stress imposes a constant rate of accumulation of strain, and it is the accumulated strain that is responsible for the potential injury.

Another dynamic response is that resistance changes under strain. Sometimes resistance increases, and then it is a question of acclimatization to an unfavourable environment. This kind of behaviour seems likely if the plant - as a species - has gone through a similar environmental change repeatedly in the lapse of evolutionary time; a mechanism capable of minimizing the disturbance can be expected to have developed.

Manion (1981) has classified different stresses on the basis of their dynamic nature. If the stress immediately brings about injury, it is called inciting. For instance, acute necrosis of needles caused by a sudden exposure to a high sulphur dioxide concentration is a response to an inciting stress. A stress that does not cause immediate injury but still harms the organism by either accumulating strain or decreasing resistance is called predisposing. A predisposing stress can gradually give way to a contributing stress factor: while the tree is exposed to one stress factor, its resistance to another factor can decrease. The final cause of the injury is

hence not the primary stress factor, but the contributing factor instead. A standard example is the invasion of insects to a forest weakened by, say, drought or frost.

Resistance to additional stress factors such as air pollution also varies geographically, owing to variation in climate and edaphic factors. Under ultimate conditions such as high elevations and northern areas resistance to additional stress factors is lower than in more favourable conditions (e.g. Huttunen, 1975; Materna, 1979; Materna, 1983). In terms of Manion's dynamic stress concepts, the harsh natural conditions could be understood as predisposing stress that decreases resistance to inciting air pollutant stresses.

4.2. Quantitative models

4.2.1. Quantification of stress theory

Although the stress theory seems to provide a good conceptual model for understanding how trees respond to air pollution, the quantification of the theory for the purpose of prediction is restricted by the fact that the concepts involved do not have a strict physical interpretation. First, strain and injury are defined as changes in "normal" behaviour which, owing to variation in both inherited properties and the environment, generally consists of a large and rather a fuzzy set of individual behaviours. To decide what is meant by a normal, healthy tree therefore always involves an arbitrary choice.

Secondly, since the concepts are very aggregate, it may be difficult to isolate a certain physical entity as a basis for a quantitative interpretation. In an attempt to do this, Taylor (1978) introduces an interpretation of the

concepts in the case of gaseous air pollutant stress. Stress is understood as the presence of a pollutant gas in the immediate neighbourhood of plant tissue, and strain is a result of the penetration of the extraneous substance into the tissue. However, this strain may launch a whole chain of events, where the strain may be counteracted by a resistance mechanism, this counteraction imposing a secondary strain that will be counteracted by a secondary resistance to yield a tertiary strain, and so on. For instance, the resistance mechanism against an extraneous substance in the plant tissue may involve some extra energy consumption which, in turn, imposes a secondary strain through the deficiency of metabolic products. If all these stages are isolated, so many strain and resistance variables are obtained that it hardly seems reasonable to build a predictive model upon them. Further, the dynamic interaction of strain and resistance processes makes it difficult to derive more aggregate variables from the basic processes in an adequate way.

The approach presented by Kauppi (1984) for the analysis of temperature stress on pine seedlings starts off with aggregate variables that are given an operational definition through measurements. If the stress theory is to be utilized, such an approach seems applicable for pollutant stress also. However, the stress theory still seems to have its major contribution in the conceptual understanding of pollutant impacts, while the existing models intended for prediction are based on different concepts and methodology.

4.2.2. Dose and response

Perhaps the simplest attempt to quantify the complicated impact of a long-lasting pollutant stress on a biological object is the so-called dose-response model, first presented already in 1922 by O'Gara, and further developed by, e.g.,Thomas and Hill (1935), Guderian et al. (1960), Zahn (1963) and Larsen and Heck (1976). This model has been based on the idea that damage first occurs after exposure to a threshold dose:

$$when\ D > D_0\ damage\ occurs \tag{1}$$

The dose depends both on pollutant concentration and exposure time.

In the standard form of the model, relationship (1) has a very special form: it is the product of the "effective" concentration and the "effective" exposure time, "effective" referring to the excess over a threshold time or concentration under which damage never occurs. Hence, the dose (D) is

$$D = (c - c_0)(t - t_0) \tag{2}$$

where c is concentration, t is exposure time and c_0 and t_0 are the respective threshold values. According to the model, damage can be brought about by all the combinations of c and t that make the product D exceed the threshold D_0. An n-fold increase in c implies that the time required for developing damage decreases by a factor of $\frac{1}{n}$.

A criticism that has been laid on the model is due to the observation that the threshold dose is not constant but depends on the exposure time. Therefore, attempts have been made to define the dose in another way such that a constant threshold value could be observed. For instance, Guderian et al. (1960) have suggested the following model:

$$D = (t - t_0)\, e^{a(c - c_0)} \tag{3}$$

which has fitted data better than the former one. According to this model, an n-fold increase in c will imply that the time required for developing damage can decrease more than by a factor of $\frac{1}{n}$.

Another version of the model with a more quantitative objective predicts the degree of damage as a linear function of the dose received (e.g. Thomas & Hill, 1935). Denoting the degree of damage by y, this can be expressed as follows:

$$y = aD + b \tag{4}$$

As opposed to Model (1) where damage is a binary variable with values "damage occurs" and "damage does not occur", this model provides the degree of damage as a continuous variable.

Larsen and Heck (1976) have further extended the idea of dose and response in their lognormal model, which includes the conventional dose-response model as a degenerate case. In this model dose is defined as

$$D = c\, t^p \tag{5}$$

where the exponent p has been observed to get values within the interval $0<p<1$ (in the dose-response model $p=1$). Degree of damage is expressed continuously as a function of dose, such that the following conditions are fulfilled:

[1] Degree of damage is proportional to the logarithm of dose, rather than the arithmetic value of the dose itself.

[2] There is a range of doses so low that no response is measured.

[3] There is a maximum response that will not be exceeded even if the dose is increased.

The function has been formulated with the aid of the statistical lognormal distribution. A two-variable function linear in ln*c* and ln*t* has been obtained, the parameters of which have been estimated on empirical basis utilizing the definitions of the lognormal distribution. Larsen and Heck (1976) have identified the model for 14 mainly agricultural species under ozone exposure and for 4 species under sulphur dioxide exposure. Degree of damage has been defined as percentage of leaf necrosis. The results are promising.

As the dose-response model does not consider strain and resistance as explicit variables, potential differences in resistance between species and growing sites should be incorporated in the parameters of the model. The applicability of the model hence largely depends upon the possibilities of identifying the parameters as functions of factors affecting stress resistance.

4.2.3. Growth damage

While the method of the above models is to predict ***damage*** directly, the following approach will yield as output ***growth under air pollution***. The method is built upon a simple dynamic growth model, the parameters of which are functions of the air pollutant level. ***Growth damage*** in the sense discussed in Section 3 is obtained when the model output is compared with a selected norm.

The model describes the time development of the dry weight of foliage, W (kg per hectar), by means of a differential equation:

$$\frac{dW}{dt} = \lambda \varphi f(W) W - \psi W \tag{6}$$

where $f(W)$ is a shading function with the property

$$\frac{\partial f(W)}{\partial t} < 0 \tag{7}$$

i.e. as the foliage grows, it gradually creates an increasing shade that decreases productivity. Further, λ is the fraction of growth increment allocated to foliage, φ is the maximum net production of dry weight per dry weight of foliage per year, and ψ is the foliage death rate.

This is a general form of a class of models the representatives of which can be distinguished on the basis of how they actually define the shading function $f(W)$ (e.g. Ågren, 1983; Kohlmaier et al., 1984; Mäkelä and Hari, 1984). The applicability of the model to the analysis of air pollutant impacts on forests is based on the fact that it includes as parameters the annual specific photosynthetic rate, φ (kg carbon per foliage area per year) and the senescence rate of the foliage, ψ (kg senescent foliage per kg total foliage per year), which are among the most important factors air pollution directly affects. By modelling the impact of air pollutants on these parameters the effects on canopy growth can be analyzed. Such an approach has been suggested for instance by Mäkelä et al. (1981), Kercher & Axelrod (1981) and Kohlmaier et al. (1984). Ågren and Kauppi (1983) applied a similar method for predicting possible changes in growth due to an anthropogenic nitrogen input to the soil.

Kohlmaier et al. (1984) discussed the behaviour of the equilibrium of the model under a pollutant load. The relative growth rate of the canopy is

$$R = \frac{1}{W}\frac{dW}{dt} = \lambda \varphi f(W) - \psi \qquad (8)$$

The system has an equilibrium, say W_0, when $R=0$. Property (7) implies that the equilibrium is stable, but its actual form largely depends upon the shading function $f(W)$.

It is easy to show on the basis of (7) that

$$\frac{\partial W_0}{\partial \varphi} > 0$$

$$\frac{\partial W_0}{\partial \psi} < 0$$

Hence, if either specific net photosynthesis decreases or specific senescence rate increases, as is assumed to happen under a permanent pollutant load, the maximum sustainable biomass decreases. This decrease can be quantified with the aid of the model, provided that the response of φ and ψ to air pollutants is known.

In general, it is not only the equilibrium, but also the other dynamic properties of the system that the model applies to. Hence it provides a means for analyzing the change in productivity as a response to pollutant impacts on photosynthesis and senescence. The use of the model for prediction involves, however, several problems. First, an extensive modelling requires that the values of the parameters are known over a range of species and environmental conditions. Even though certain data are available for the estimation, it seems rather unlikely that a satisfactory coverage can be achieved.

Secondly, the model does not provide any information on stemwood. The problem condenses in the partitioning of growth. If the partitioning coefficients were time constants, then stem growth would be simply a constant times total growth, i.e. $\lambda_s f(W) W$, λ_s denoting the partitioning coefficient of stemwood. There is strong evidence, however, that λ_s is not a constant but depends on several factors, including the size of the foliage, W. As the partitioning coefficients are interconnected, also λ_s is a function of W. This feedback, when incorporated in the model, can totally change its dynamic behaviour.

A more comprehensive model of growth under air pollution has been developed by Bossel et al. (1984a) (system BAUM). Though far more detailed, the model is similar to the above-described one in that it derives growth from physiological processes, such as photosynthesis and leaf senescence rate, and considers the impact of air pollutants on these processes. The model incorporates leaf, root, wood and assimilate biomass as state variables, and accounts for several physiological processes involved in tree growth. The "normal" driving variables comprise light conditions, day length, average temperature and soil nutrient status. Five important air pollutants are included, using empirical evidence on their effects on the physiological processes. The tree model is further linked with models describing the state of the soil, which allows soil acidification to be included in the dynamics. The authors emphasize, however, that the model should be regarded as an attempt to qualitatively understand the processes of pollutant impacts, rather than as a quantitative, predictive tool.

4.3. Empirical evidence

Empirical results have been published as regards the relationship between air pollution and forest damage. Some of them are reviewed here, with the objective of giving an idea of what kind of variables and results are available for model identification.

Wentzel (1983) has collected the results concerning the relationships between SO_2 concentrations and the damage observed that have been published in Europe. These studies are part of the IUFRO program Air Pollution. Figure 5 shows a summary of these results. The curves show the SO_2 immission concentrations of various central European forest-damage areas as connecting curves between actual measured annual average values and peak values. The numbers on the curves denote the responses of the forests, as they are reported in the quoted literature.

Materna (1984) has made some observations on the threshold times and concentrations as functions of the tolerance of the forest. He notes that a good measure for the tolerance is altitude from sea level. These results are shown in Table 1.

Guderian et al. (1984) have carried out a profound study in order to establish some dose-response relationships for the response of forests to ozone. Tolerance is incorporated by assuming that the threshold varies as a function of several variables affecting tolerance, e.g. the latitude of the forest, edaphic and climatic factors, etc.

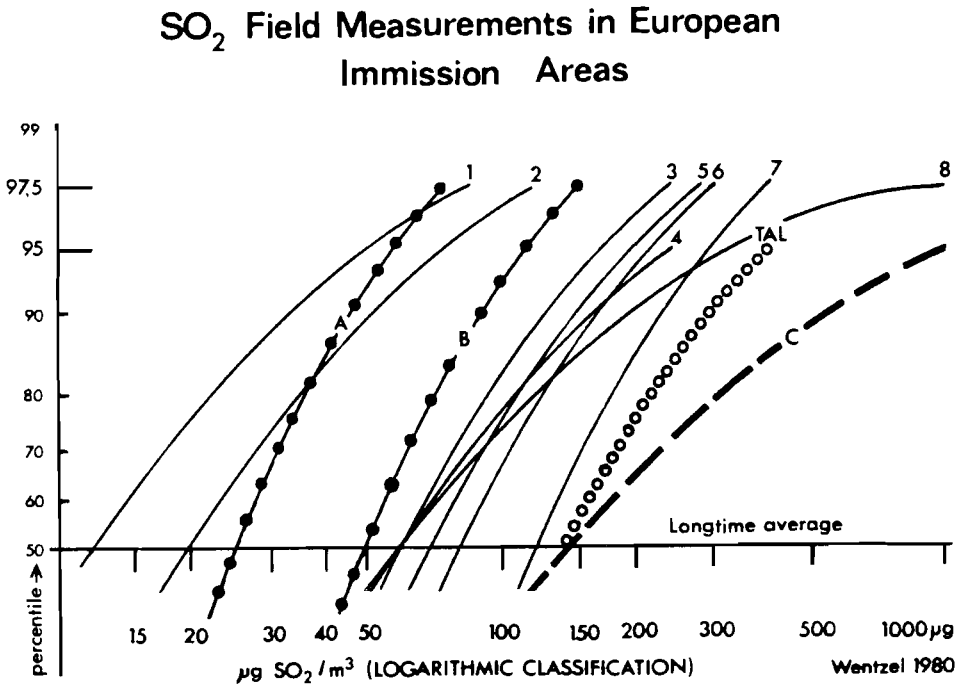

Figure 5. Relationships between SO_2 concentrations and the damage observed according to various sources (Wentzel, 1983).

1. Materna 1973 : High ranges of the Erzgebirge - resistance to frost and other secondary injuries diminished.
2. Materna 1972 : 20% loss of wood increment in the Erzgebirge.
3. Materna et al., 1969 : Dieback of Norway spruce stands in the Bohemian part of the Erzgebirge.
4. Wentzel 1979 : Good sites in Rhein-Main area sufficiently protected.
5. Stein a. Dässler 1968 : Moderate injury in the Saxonian part of the Erzgebirge.
   Lux 1976 : Situation comparable with that in Neiderlausitz.
6. Knabe 1970 : Wentzel 1971 : Severe growth losses in the Ruhr area.
7. Knabe 1972 : Economical forestry with Norway spruce and Scots pine made impossible in the Ruhr area.
8. Guderian a. Stratmann 1968 : Immission type Bierstorf = single immission source in mountain valley. Severe growth damage to nearly all species (young plants).

A relationship between the number of living needle age classes and sulphur content of the needles, found out for instance by Knabe (1981), pro-

Table 1. The time between the beginning of air pollution influence and the desintegration of Norway spruce stands.

SO_2 μgm^{-3}	-600	-900	-1050	1050+ height above the sea level
< 20				
20-30			30-40	20^x
30-50	50-60	20-30	20^x	
50-70	40-50	20^x	10^x	
70-90	30-40	10-15		
90+	20-30	< 10		

vides some empirical basis for determining the parameter values in Equation (6). (Figure 6). Similar results are available for other pollutant species also (e.g. Knabe, 1984).

The response of the potential photosynthetic rate, φ, to pollutant exposure has been studied in laboratory and field experiments by a number of authors (e.g. Keller, 1978; Katainen et al., 1983).

5. STRATEGIES TOWARDS FOREST IMPACT SCENARIOS

5.1. Preliminaries

The aim of this report is to search for a model structure that can be used in producing scenarios of forest damage due to air pollution in Europe.

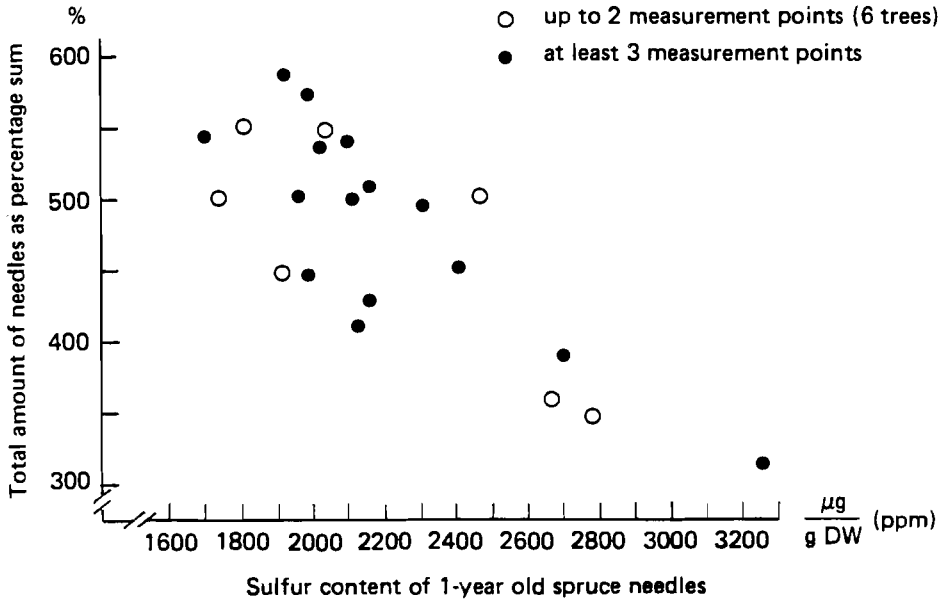

Figure 6. Relationship between sulfur content of 1-year old spruce needles and the total amount of needles on the tree expressed as a percentage of the 1-year old needle class.

The preceding sections show clearly that the problem we want to solve quantitatively, is at present rather poorly understood even qualitatively. Therefore, a stepwise strategy is preferable which allows gradual improvement of the model in pace with increasing knowledge. Secondly, it is of particular importance that the sources of uncertainty are understood, and means of increasing the reliability of the results are constantly developed.

5.2. Input

By Section 2, the evidence does not allow that any one of the reviewed potential causes of damage is excluded. On the contrary, it has been argued that several simultaneous mechanisms contribute to the damage, and that

the impact is deeply dynamic. The synergetic impacts are briefly understood, though, and their quantification requires a lot of guesswork. It is therefore considered more realistic to start with a model based on one pollutant at a time. The consequent results will obviously be applicable to areas where one pollutant dominates, but they can also be used to study how much this one factor can explain.

In Section 1, the suitability of sulphur dioxide concentration as an input variable was questioned. As mentioned in Section 2, it has been claimed that sulphur cannot be the main cause of damage unless the pathway through soil acidification is considered. This would require considering a delayed damage. According to stress theory, however, the delay can also be connected with the so-called direct impacts of sulphur dioxide, and this should therefore be regarded as one of the relevant potential causes.

In a summary, it is suggested that the first stage of the model should be based on airborne sulphur dioxide. As a further step, soil pH and airborne ozone should be considered.

5.3. Output

Model output should be simple enough to be projectable on a map. A simple classification of the output forest state is therefore preferable.

According to Section 3, visible injury is the most widely documented type of forest damage in Europe and hence provides the most realistic output variable for the scenario model. It should be possible at least to separate between two classes, injured and non-injured.

Since growth reductions may have economic significance even before visible injury appears, it would be informative if the class "hidden injury" could be distinguished. Another interesting border-line both economically and esthetically, is that between productive and non-productive land.

In a summary, a possible classification of damage is the following:

[1] no damage

[2] hidden damage, decreased productivity possible

[3] visible damage, decreased productivity

[4] disintegration, zero productivity

In terms of the dynamic behaviour of the system, categories [1]-[3] represent stable development, although classes [2] and [3] show reduced growth, whereas category [4] corresponds to unstable behaviour. Therefore, an important borderline is to distinguish class [4] from the others.

5.4. Model structure

As the model is intended for interactive use, an important criterion is to keep the simulation time short enough. Therefore, a multi-variable dynamic model cannot be chosen. Further, as was seen in Section 4, the rather simple models based on theoretical argument are not suitable for extensive quantitative use, because they either have some theoretical discrepancies, or they cannot be reasonably tested against data. Hence a more or less empirical, aggregated approach has to be taken, generalizing the implications of some individual data sets and the overall understanding of the forest damage process to cover a large area and a long period of time.

However, as there is relatively few data available for quantifying input-output relationships between air pollution and forest response, some theoretical understanding of the process would be helpful in constructing the above-mentioned model. Models describing the growth dynamics of trees under air pollutant load can well contribute to such information, integrating the current state of knowledge about individual response processes to the response of the whole tree.

5.5. Evaluation of uncertainty

The uncertainty of the results of an empirical model is connected with problems of generalization. In this case, both geographic and temporal extrapolation is necessary. If uncertainty is due to statistical unreliability, it can be decreased by increasing the size of the sample. This way for instance the growth and yield tables developed in forestry have become successful and practical. However, each experiment takes a long time up to several decades, and varying the input under field conditions is almost impossible.

A more difficult part of the uncertainty originates in the possibility that not all the important factors have been included in the model structure. Secondly, the dynamics of the impact may be more complicated than assumed, for instance some delay structures may not have become apparent by the data. When the model is applied to conditions different from the data, erroneous conclusions can be made if these possibilities are ignored.

There is no general method for assessing this kind of uncertainty. One possibility is to analyze how the results would change if the simplifying hypotheses were substituted with some more realistic ones. Obviously such

an analysis can only cover part of the problem at a time, since if it was possible to treat the whole problem this way, why should the model simplify at all? For instance, the dynamic behaviour of the model delineated here could be compared with that of the mechanistic models presented in Section 4, to assess the results qualitatively over limited areas. This would also contribute to the further development of forest impact scenarios, especially as regards synergistic effects of air pollutants.

5.6. Summary

It is suggested that modelling forest impacts proceeds from the simplest model towards more difficult tasks which can be built upon the former steps. Each stage utilizes an aggregated input-output model with geographically distributed parameters describing stress resistance. Such a model structure is depicted in Figure 7. This model will be constructed on the basis of empirical data and theoretical understanding of the dynamics of the process. More detailed "mechanistic" models will be used to extract information of forest damage dynamics to the aggregate input-output model. The consequent steps, specified by the choice of input and output, are listed in Table 2. The stepwise procedure is shown schematically in Figure 8. Evaluation of uncertainty and further steps and questions are also indicated.

Table 2. Model inputs and outputs to be applied in a stepwise manner as A1, A2, ..., B3.

input	output
A. ground concentration of SO_2 (annual average + statistical data)	1. binary-valued: damage - no damage
B. ground concentration of ozone (annual average + statistical data)	2. multi-valued: several stages of damage
	3. continuous, based on growth reduction

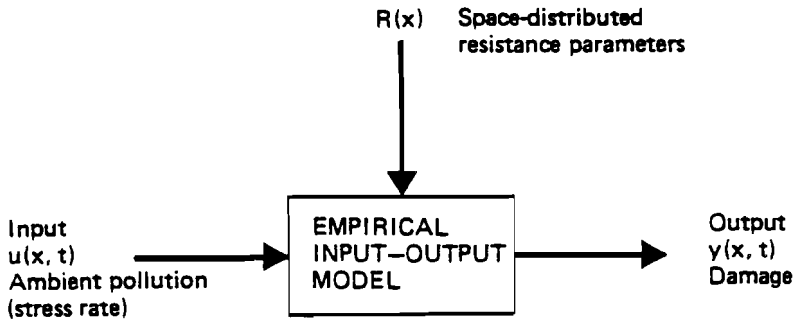

Figure 7. Structure of forest impact submodels.

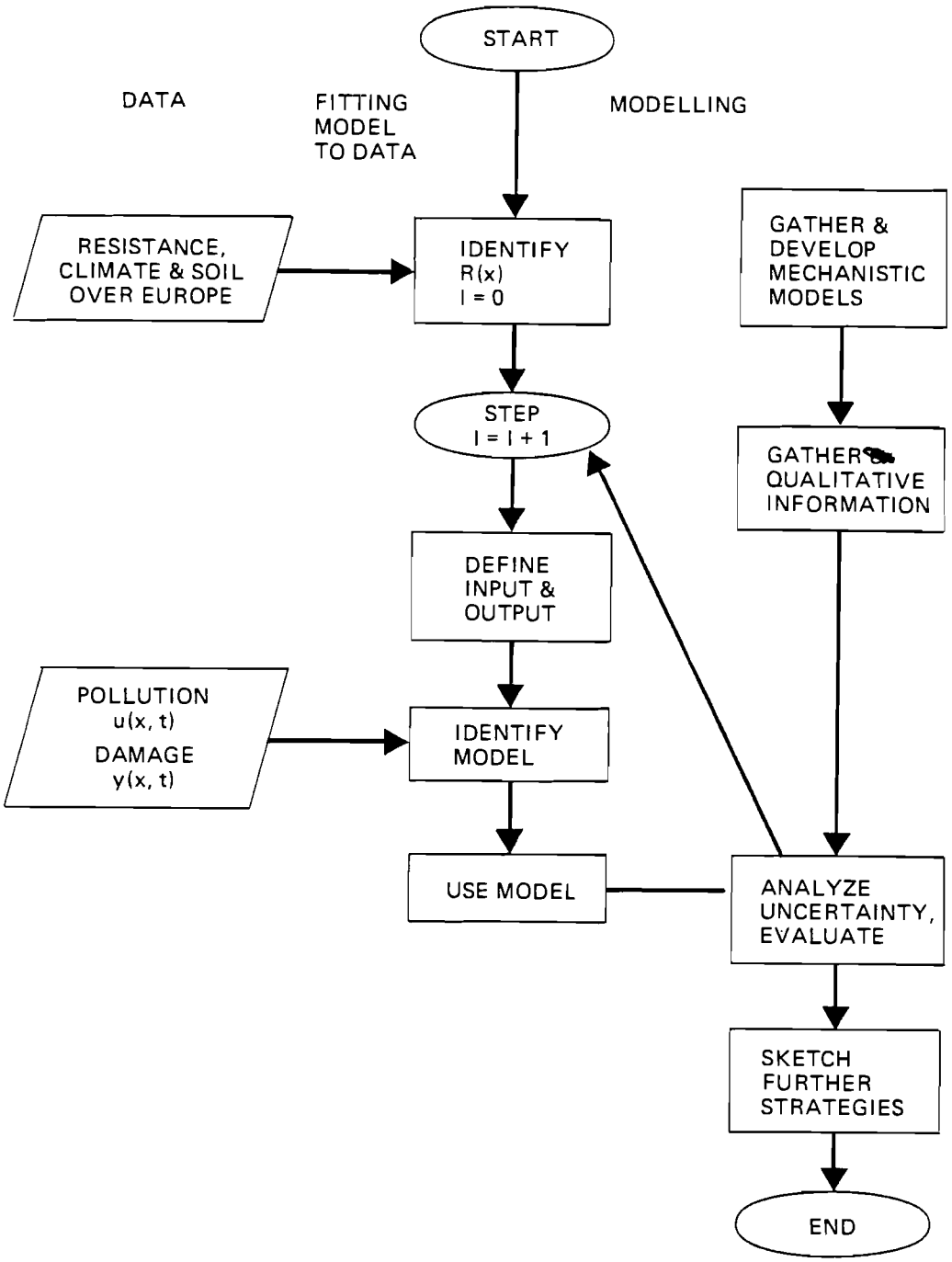

Figure 8. Suggested procedure for building forest impact submodels.

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