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Epidemiological Evaluation of the Development of Waldsterben in Germany

According to several authors (1,28,29), a large-scale decline of many different forest ecosystems has taken place in central Europe since 1979–1980. This alleged general decline collectively has been called Waldsterben and was believed to stand apart from ordinary diseases of trees because many tree species were thought to be involved and numerous ecosystems were expected to be destroyed. Thus, Waldsterben was considered a complex disease of the forest ecosystem rather than an accumulation of distinct diseases of individual trees or particular species. Air pollutants, in a broad sense, were suggested to be the most important causal factors (27,35), whereas abiotic stresses (e.g., climate, malnutrition) were considered as predisposing factors and biotic agents (e.g., pathogens, insects) as secondary or contributing factors (27,28). These suggestions are in line with Houston's concept (6) of stress-triggered diseases and decline.

The time span until most or all of the forests would be destroyed was predicted to range from a few years to a few decades. In the case of spruce and pine, the period of time between first symptoms and the final stage of damage was predicted to range from a few weeks to 3 years (28), and all spruce and fir trees at the permanent observation plots in Baden-Württemberg in the southwest of Germany were expected to die by 1990–1992 (26).

New observations and experimental results recently accumulated do not support many of the early predictions and have opened new perspectives (2,9). Among the numerous activities initiated by the Waldsterben discussion have been the annual forest damage surveys started in the Federal Republic of Germany in 1983 and later adopted by several other European countries. These surveys have supplied the most relevant epidemiological data sets on the spatial and temporal course of forest condition. In this paper, I wish to:

1. Compare the actual course of forest condition, as evidenced by the annual surveys conducted during 1983–1988, with the development predicted by the advocates of Waldsterben in the early 1980s;
2. Discuss the implications of the discrepancy between the predicted and the actual development for current hypotheses on the causes of Waldsterben;
3. Discuss alternative views to help understand the observed variable health condition of trees based on tested phytopathological approaches for analyzing plant diseases of complex etiology (12,36); and
4. Encourage phytopathologists studying diseases of forest trees to intensify research on diseased or debilitated trees so far lumped together under the ill-defined term "Waldsterben."

Symptoms and Measure of Waldsterben

The description of symptoms of Waldsterben has been rather vague. Almost every type of symptom ever attributed to any kind of disease or damage of trees has been listed among the so-called *neuartige Waldschäden* (new types of forest damage) thought to be typical of Waldsterben: increment decrease, different kinds of growth anomalies, root damage, discoloration, loss of foliage, etc. Symptoms also have been reported as heterogeneous from one region or forest stand to another, even within species (27–29). All kinds of symptoms have been lumped together, and the resulting mixture has been declared as typical of a new complex disease of forest ecosystems caused by an undefined mixture of pollutants. This careless habit is totally contrary to the detailed symptom analysis procedure that has been practiced successfully for more than a century in classical phytopathology. Hence, the new approach has often been criticized as being inappropriate to solve the problems of forest decline (7–9,13). Manion (13) emphasizes the point when he states: "The 'fruit salad' of deterioration, decline, and dying trees should not all be squeezed into a generic 'fruit juice.'"

The development of the "new complex disease" defined by such vague symptoms was categorized by a very general description of damage classes based on foliage deficit and discoloration. In the annual surveys started in 1983, the damage classes are defined by the percentage of foliage deficit, with 0 = healthy (<10%), 1 = slightly damaged (11–25%), 2 = distinctly damaged (26–60%), 3 = heavily damaged (>60%), and 4 = dead (100%). Foliage discoloration can increase the damage class of a particular tree, depending on the percentage of affected foliage. The number of trees in the various damage classes is converted to the corresponding forest area they cover. Thus, the annual forest damage reports published by the German government give the percentage of the total forest area covered by trees of the particular damage class. (Although

"foliage class" would be the more correct term, the official term "damage class" will be used throughout this paper to avoid confusion.)

Dynamics of Waldsterben

These crude survey methods are of little value for defining distinct syndromes or for elucidating the etiology of any disease, but the series of six annual survey reports available from 1983 to 1988 helps discern the dynamics of Waldsterben and may answer some fundamental epidemiological questions. For example, is there a steady progression in damage that may finally lead to the destruction of the forest? Is there a spatial extension of damage that may indicate an epidemic? Does injury in the various tree species show the same pattern of development and spatial distribution? Are the spatial and temporal distributions of the damage correlated with the supposed causal abiotic factors, e.g., air pollutants?

The cumulative development for Waldsterben in the total forest area of the Federal Republic of Germany (all tree species studied; Fig. 1A) and for single tree species over smaller areas (e.g., spruce in Bavaria; Fig. 1C) does not correspond to the predicted progress of mortality (26,28). The undamaged forest area decreased from 1983 to 1984 but has remained constant since 1984 (Fig. 1A), and progression from lower to higher damage classes, expected for a syndrome leading toward death, has not been seen. The actual curves are at odds with the model curves (Fig. 1B), which assumed an annual damage progression of 20% among classes. An annual progression of 20% was chosen to meet the initial slope of the actual curves, since the damage progression from 1983 to 1984 had been the basis of earlier predictions and scenarios (26,28). The model curves could be modified to match the annual survey data beyond 1984 more closely if different or varying rates of progression were assumed for the individual damage classes or if hypothetical regeneration phases were introduced. Complete matching, however, cannot be achieved without abandoning the tree death scenario.

In public discussions, the absence of the predicted continuous rise in classes 3 and 4 is often attributed to increased harvesting of declining trees. However, the regular annual tree harvest corresponds to only 1–1.5% of the total forest area. Harvest of trees supposed to be affected by Waldsterben is only 3–6% of the total cut (9). Thus, the "extra cut" corresponds to only 0.02–0.09% of the total forest area, and as such it does not alter significantly the curves shown in Figure 1. If a substantial extra cut had been harvested, the excess of available timber should have led to a sustained fall in timber prices, as predicted by economists in the early days of the Waldsterben discussion (9), but changes in prices did not exceed the usual fluctuation (2,9).

It is important epidemiologically to compare the progression of the higher damage classes in the various tree species in different areas (Fig. 2). Air pollution should cause relatively uniform damage, since the main sources of air pollution (power stations and vehicles using fossil fuels) are qualitatively the same in all areas and average concentrations of major pollutants have been consistent during the period in question (9). Furthermore, tree species should reflect different sensitivities to prevailing pollutants, or mixtures thereof, and responses should remain the same unless concentration or kind of pollution changes significantly. According to the surveys, however, damage to species varies from area to area and over time (Fig. 2). Similar differences are found in much smaller areas, such as different districts of the states involved (9). Such behavior is not compatible with a general pollution hypothesis but is more consistent with effects of spatial and temporal variations in climatic conditions combined with partly reversible diseases of biotic origin.

Crown Condition in Conifers

The high regenerative capability of trees after biotic diseases and abiotic (e.g., climate, malnutrition) damage is well known. Changes in ratios among damage classes can be understood when one recognizes that trees do not remain in a single damage class. Three examples are documented by a series of photographs. In the first example, each of four heavily damaged spruce trees photographed in 1959 at the Dreiländereck in the Bavarian Forest (Fig. 3) behaved differently during the following 27 years. In 1959, tree 1 would have been in damage class 1 and trees 2–4 would have been in class 3. By 1986, the condition of tree 1 had worsened but tree 2 had improved slightly and trees 3 and 4 had regenerated almost normal upper crowns. Air pollution, soil, or climatic conditions are unlikely causes for the different fates of these neighboring trees. Most probably, root and heart rot, found by core inspection, became more acute or less acute or was suppressed by the oscillating tree/pathogen interaction.

The second example shows recovery of three spruce trees at Rachel Diensthütte in the Bavarian Forest over

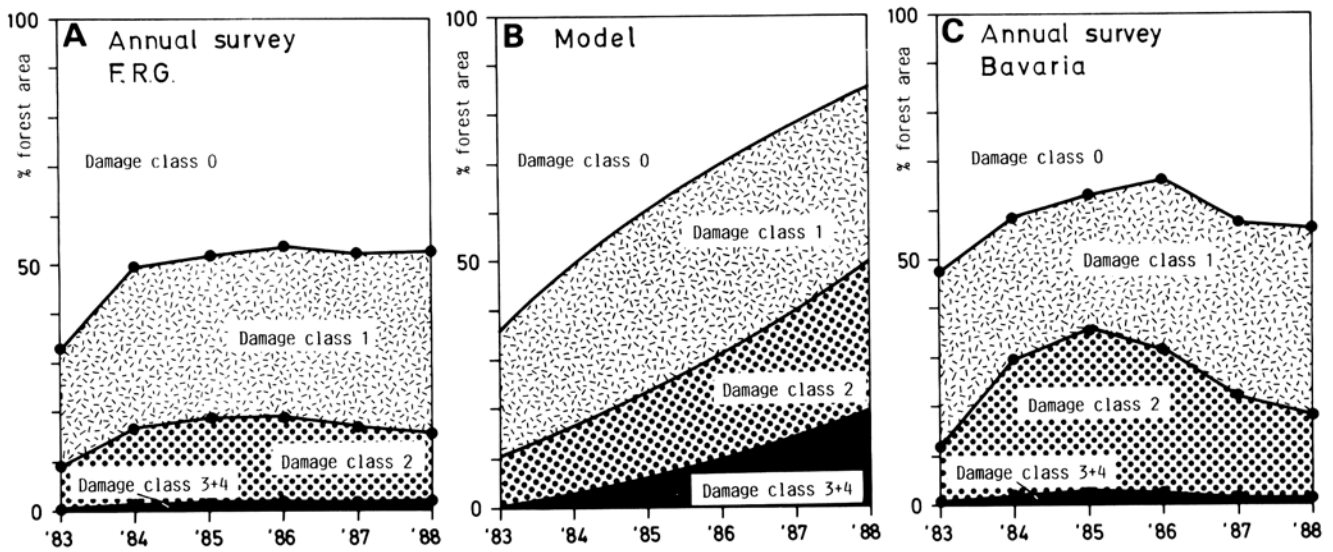


Fig. 1. (A) Percentage of forest area in damage classes (0 = healthy [<10%], 1 = slightly damaged [11–25%], 2 = distinctly damaged [26–60%], 3 = heavily damaged [>60%], and 4 = dead [100%]) for 1983–1988 for all tree species in the Federal Republic of Germany; class 4 is 0.1–0.2%. (B) Model curves of damage classes that assume an annual increase of damage or progression of 20% based on the levels of 1983. A progression of 20% was chosen to meet the initial damage progression from 1983 to 1984. (C) Percentage of forest area in damage classes for 1983–1988 for spruce trees in the forests of Bavaria.

20 years (Fig. 4). In 1966, the trees would have been in damage class 3 because of distinct needle loss and dead tops. In 1986, much denser foliage and regenerated tops place them in classes 1 and 2. Suppression of root and stem rot by compartmentalization (34) followed by regeneration of fine roots probably led to recovery, since neither air pollution levels nor soil conditions improved during that period.

The third example shows recovery of a diseased spruce tree (FK-1) within a few years (Fig. 5). Compared with two healthy trees (FK-2 and FK-3) growing only 10 to 20 m away, annual growth increment for FK-1 started to decrease in 1969 and was minimal in the dry summer of 1976 (Fig. 6), when needle loss began. Recovery of incremental growth was delayed until 1980, but needle loss was greatest (class 3) in 1982. In following years, more needles were produced than were shed, and in 1987 the tree returned to class 1 or 2. When the tree was cut in the spring of 1989, *Heterobasidion annosum* (Fr.) Bref. was found to have affected the deteriorated heartwood up to a height of 8 m. Most older roots were infected and deteriorated by *H. annosum* and by *Armillaria mellea* (Vahl:Fr.) Kummer sensu lato (*unpublished*). The latter had killed about one-fourth of the cambium at the base of the bole in 1978. Many new roots that had formed since 1980 led to recovery of the crown.

These three examples show the different time courses of oscillating crown conditions associated with root and heart rot and the different effects of drought on healthy and diseased trees (Fig. 6). Root and heart rots are the most common endemic tree diseases in European forests. In the 1960s, a joint research project among several groups of forest pathologists supported by the Deutsche Forschungsgemeinschaft showed that in many areas, 20% to almost 100% of spruce trees in stands older than 50 years had been infected with *H. annosum*, a common root and heart rot fungus (24). In 1988, only 1–2% of spruce trees were reported to have fungus infections in the official German forest damage report, but in these surveys, only fungal diseases recognizable by viewing crowns with binoculars are reported. All other fungal infections—and specifically root rots—are neglected. Thus, crown transparency and discoloration caused by acute root and heart rot diseases (18) or by pathogens suppressing mycorrhizae and rhizosphere organisms that may lead to mineral deficiency (20) are included among those registered as Waldsterben. Mechanisms of fungal attack and tree responses have been excellently described by Shigo (34), but details of the orderly succession of invading organisms and the biochemistry of fungal attack and plant response are still unknown.

In addition to root and heart rot, many other diseases of known and unknown etiology cause needle loss and discoloration. Since the beginning of the Waldsterben discussion, the so-called needle-reddening disease, whose etiology is not fully understood, has been an epidemic in southern Germany (19). Reddening of needles starts in the fall, and needles are shed in early winter. Older segments of secondary branches that hang down from the main branches become defoliated and in recurrent epidemics become the famous "silver tinsel" symptom (28), which remains visible for several years. Eventually, the tinsels are hidden by new growth of well-foliated axillary branches. The distinct recovery of crown conditions of spruce trees in Bavaria (Fig. 1C) is at least partly due to this phenomenon.

The so-called Tannensterben (fir death) is another disease of unknown etiology that has spread in epidemic waves across central Europe since the middle of the last century (8,37). This disease of silver fir (*Abies alba* Miller) is characterized by abrupt, long-lasting (up to a few decades) increment reduction and by defoliation and dying of the branches, beginning from the bottom and ascending to the top. The tree top is flattened (stork's nest formation) and remains green until late in the syndrome, when many trees die (36). Others recover and show an abrupt increase in incremental growth.

The most recent epidemic of Tannensterben began in the 1960s or as early as the 1950s (8,9) and culminated in the 1970s, when symptoms were augmented by dry summers. Recovery of incremental growth began about 1980, but improvement of crown conditions was delayed 4–6 years. Almost 200 papers on Tannensterben since the beginning of this century show clearly that it existed long before the Waldsterben discussion. Unfortunately, however, both phenomena are lumped together in current discussions. The etiology of Tannensterben still is unknown in spite of many research

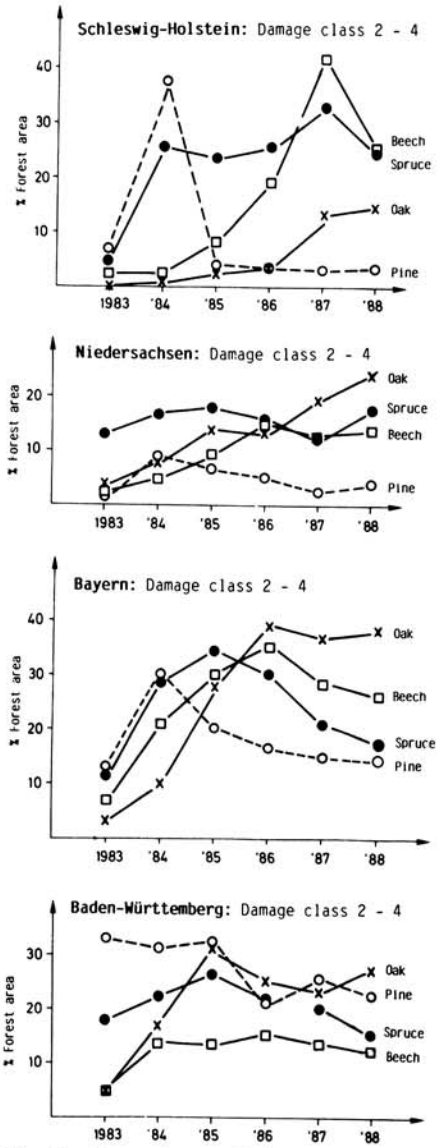


Fig. 2. Percentage of forest area in damage classes 2–4 for four tree species in four states of the Federal Republic of Germany for 1983–1988.

Fig. 3. Changes in the condition of four diseased Norway spruce trees at Dreiländereck in the Bavarian Forest occurring between 1959 and 1986. Note tree canopy. (Photographs by Riemenschneider and Kandler)

efforts and proposed hypotheses and remains a continuing challenge to forest pathologists.

Crown Condition in Hardwoods

The foliage of hardwoods is attacked by more insects and pathogens (15) than is that of conifers. The extent of infestation or infection depends largely on climatic conditions and thus shows a considerable annual variation. In the annual forest damage surveys, the extent of injuries caused by insects and pathogenic fungi, recognized simply by viewing the crowns with binoculars, is recorded separately. The extent of such injuries correlates poorly with damage classes and varies with species. In 1988, for example, 44% of beech (*Fagus sylvatica* L.) in damage class 0 and 54% in class 3 showed injuries by insects and pathogenic fungi. In the two oak species *Quercus robur* L. and *Q. sessiliflora* Salisb., the biotic injuries were 53 and 83% for damage classes 0 and 3, respectively. The high percentage of biotic injuries in beech and oak in damage class 0 indicates that the main symptoms—crown thinning and discoloration—on which damage classes are based differ from recognized biotic injuries. However, the latter may contribute to a higher grouping, as indicated by the higher percentage of infested and infected trees in damage class 3 than in damage class 0.

Crown transparency in beech and oak ascribed to Waldsterben is often accompanied by discoloration (usually yellowing of leaves) without detectable pathogens and distinct, long-lasting growth inhibition of terminals. Annual growth of shoots may be reduced to a few millimeters, compared with about 10–25 cm in healthy trees. Single branches or the whole crown eventually may die. Such symptoms are not new (8); they occur in various combinations and represent a mixture of different and as yet poorly described or undefined types of decline. However, one type of oak decline, reported to have spread from southeast to central Europe during the last decade, was studied in detail in Rumania (16). This decline is characterized by reduction of annual shoot growth and leaf size and by yellowing, wilting, and loss of leaves, which often show necrotic margins resembling symptoms of potassium deficiency. In the phloem of such declining trees, mycoplasmalike organisms (MLOs) have been detected by electron microscopy. When the MLOs were transferred to *Vinca rosea* L. via *Cuscuta campestris* Yunck, chlorosis and reduction of leaves and flowers were observed, suggesting that this particular type of oak decline may be caused by MLOs. However, these findings have not been confirmed for central European oak decline with similar symptoms.

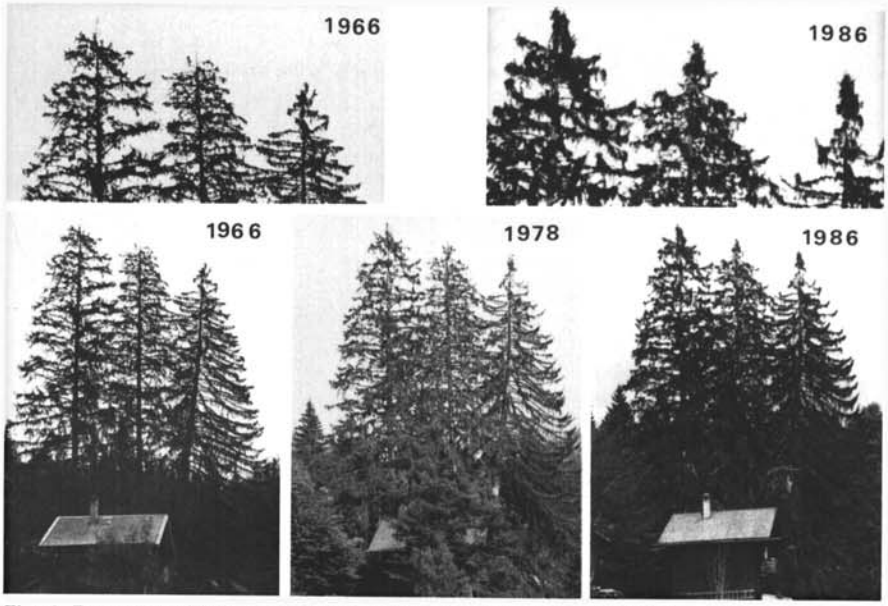


Fig. 4. Recovery of foliage and regeneration of apical shoots in three diseased Norway spruce trees at the Rachel Diensthütte in the Bavarian Forest. (Photographs by Liedl and Kandler)

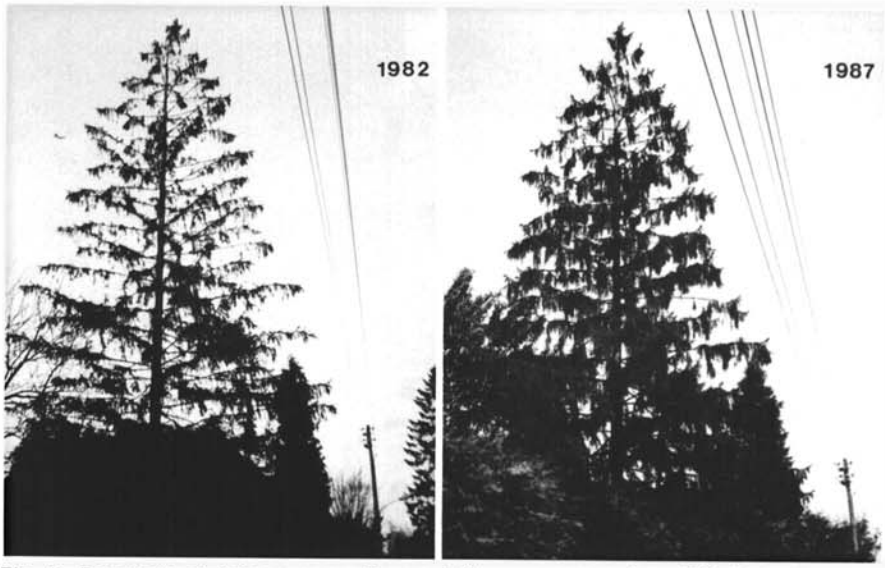


Fig. 5. Recovery of foliage on a diseased Norway spruce tree (FK-1) in Munich-Untermenzing between 1982 and 1987. (Photographs by Kandler)

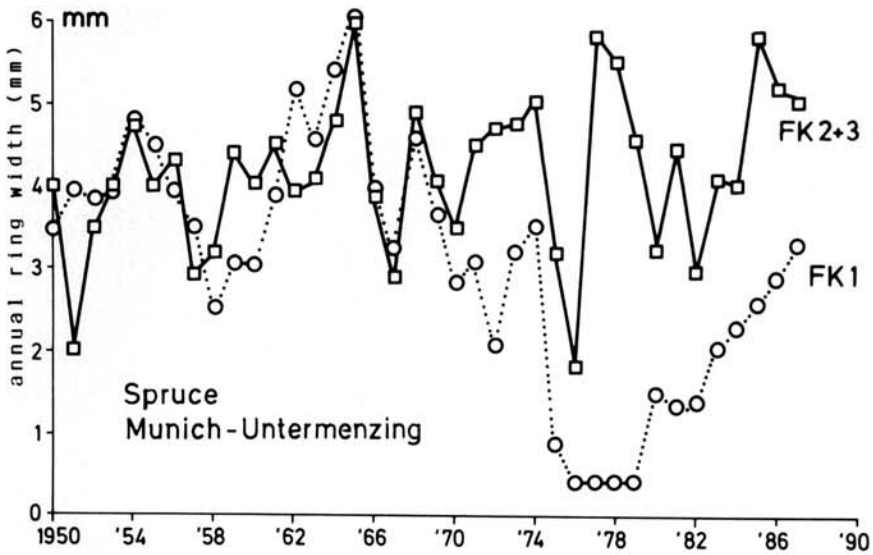


Fig. 6. Annual increment of the diseased Norway spruce tree (FK-1) shown in Figure 5 and two neighboring healthy Norway spruce trees (FK-2 and FK-3).

Similar decline was observed in many other, economically less important or even unimportant hardwood species. A partly defoliated black alder (*Alnus glutinosa* (L.) Gaertner) with stunted terminal branches was depicted by Schütt et al (27) as typical of Waldsterben. However, Seemüller and Lederer (31,32) demonstrated by fluorescence and electron microscopy that alder decline (Fig. 7A and B) is associated with MLO infection of the phloem (Fig. 8A). Diseased trees show early yellowing of the leaves, reduced growth, and frequent dying of the terminals. Trees survive by forming axillary branches. The disease may become latent, but often trees die as depicted in Figure 7A.

Alder decline is widespread in Germany. In some areas, about 50% of the trees are diseased and dying. MLO-associated decline also has been found (31,32) in *Populus tremula* L. (Figs. 7C and 8B), *Crataegus monogyna* Jacquin (Fig. 7D), *Sambucus racemosa* L. (Fig. 7E and F), *S. nigra* L. (Fig. 8C), and *Corylus maxima* Mill., with symptoms similar to those in alder. In *Populus*, *Crataegus*, and *Sambucus*, however, pronounced leaf reddening occurs in the early stages of the disease (Fig. 7C, D, and F). In *Crataegus*, dying has not been observed. Because grafting or other transmission experiments have not been performed, the importance of MLO infection as the cause of the observed symptoms is undetermined, and the identity of the organisms found in the various tree species is unknown.

The ecological and phytopathological implications of these findings are evident. Whereas previously the colder climate in central Europe was said to prevent the spread of MLOs and limit the number of pathovars to a few exceptional cases (e.g., blueberry stunt), MLOs now must be considered as potentially common pathogens of hardwood in central European forests. However, MLOs have not been detected in conifers so far.

Dynamics of Acute Yellowing in Spruce Trees

Among the various syndromes ascribed to Waldsterben, the so-called acute yellowing (8,10) of spruce is the best defined. It is characterized by yellowing of older needles of both young (Fig. 9A) and old (Fig. 9B) trees. The current year's needles remain green (Fig. 9C) until new growth commences the following spring. The syndrome is accompanied by distinct magnesium deficiency in the yellow needles and is restricted to soils with low magnesium supply, usually originating from silicate rocks (granite, sandstones, etc.). Acute yellowing has spread rapidly since the early 1980s (8,10) and is associated with about 30% less annual diameter increment. Mortality is low (<3%) but may be enhanced by diseases such as root rot (10). Regreening follows fertilization with magnesium (39) and can occur spontaneously. In both cases, regreening is accompanied by restored pigment and magnesium content (10,39). Spontaneous regreening may occur in two modes: 1) The yellowing of the previous year's needles that usually occurs during the new flush of growth may not occur and the yellowed older needles may become green again, and 2) older needles remain yellow but the previous year's needles do not yellow, and thus acute yellowing is halted. If this process continues for several years, tree color becomes normal.

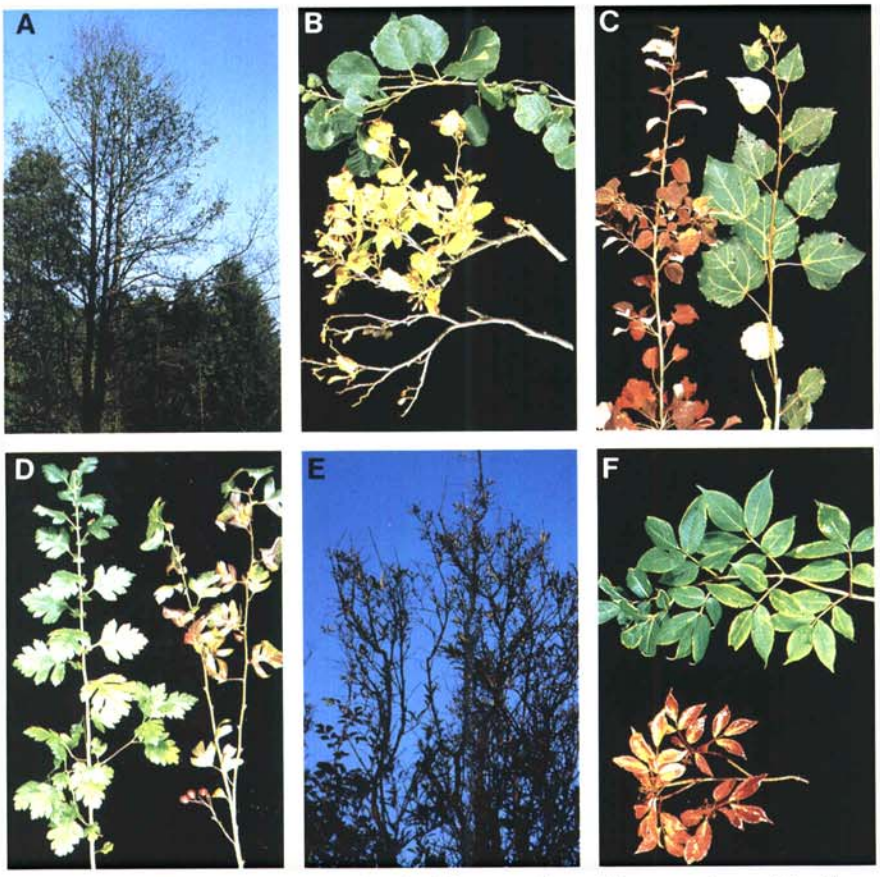


Fig. 7. Trees with diseases associated with mycoplasmalike organisms: (A) *Alnus glutinosa*, severely diseased, at the Grosse Ohe (Bavarian National Park). (B) Healthy (top) and diseased branches of *A. glutinosa*. (C) Healthy (right) and diseased (left) branches of *Populus tremula*. (D) Healthy (left) and diseased (right) branches of *Crataegus monogyna*. (E) Severely diseased *Sambucus racemosa* near Heidelberg. (F) Healthy (top) and diseased leaves of *S. racemosa*. (Photographs by Kandler [A] and Seemüller and Lederer [B–F])

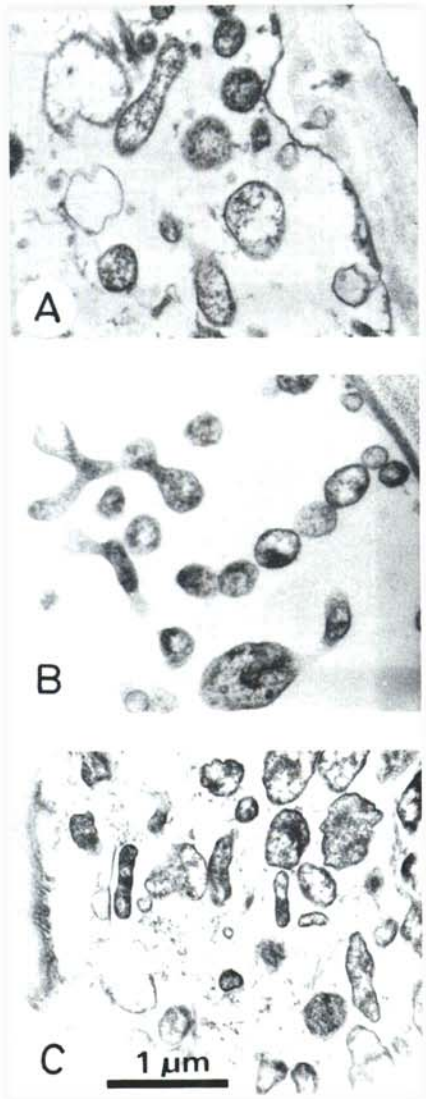


Fig. 8. Electron micrographs of mycoplasmalike organisms in sieve tubes of (A) *Alnus glutinosa*, (B) *Populus tremula*, and (C) *Sambucus nigra*. (Photographs by Seemüller and Lederer)

An example of this is a spruce tree in which stagnation of yellowing began in 1986 (Fig. 9D). The needles formed in 1985 did not become yellow as expected, while the needles of the 1984 and earlier seasons remained yellow and were still yellow in spring 1989 (Fig. 9E).

The population dynamics of acute yellowing were studied (10) in two plots, Spiegelhütte I and II, of naturally regenerated spruce (about 2,000 young trees) in the Bavarian Forest by monitoring yellowing from 1984 to 1988. All phases of acute yellowing—onset, progression, stagnation, regreening—occurred in both plots at the same time. In some cases, even neighboring trees with overlapping root systems showed opposite development of the disease.

The disease started in plots I and II in 1982 and 1984, respectively, reached a maximum of 50 and 14% yellowed trees in 1985, and decreased to about 10 and 2% yellowed trees, respectively, in 1988. A similar onset is revealed when the percentages of yellowed needles in the annual surveys of Bavaria, the Black Forest, and the Neckar region are plotted (Fig. 10). Maximum incidence of acute yellowing in 1985 also was observed in other mountain areas of Germany, for instance, Fichtelgebirge and Hils Mountains. Thus, synchronization of yellowing and recovery extended to widely separated areas. Exceptions were seen in all areas in individual trees, groups, and stands, with yellowing beginning later or still progressing, even in 1989.

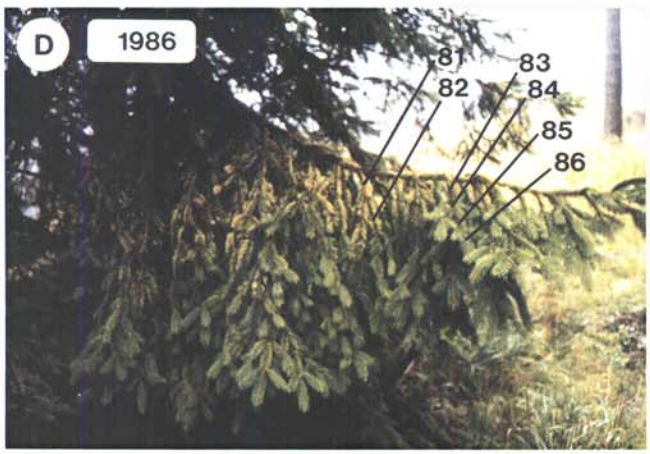
It is uncertain whether, and in what time spans, the recurrence and recovery are periodically repeated, since the etiology of acute yellowing has not been elucidated unequivocally. A common early hypothesis stated that pigment destruction and enhanced magnesium leaching are caused by ozone-mediated direct photooxidation (5). This theory lacks supporting data, and several lines of evidence are contrary: 1) Ozone treatment did not reproduce the symptoms of acute yellowing (10), 2) spruce is largely ozone-resistant (33), and 3) carotene, the most photosensitive chloroplast pigment, is similarly or less affected than chlorophyll in yellowed needles (10,33), whereas photooxidation caused by high light intensity and elevated oxygen or ozone concentrations affects carotene much more than chlorophyll (10,23,33). Histological studies on spruce needles show distinct differences between the damage caused by ozone treatment and that associated with acute yellowing (4).

Because acute yellowing is unequivocally associated with magnesium deficiency, the availability of magnesium in the soil is the decisive factor. However, acute yellowing resembles disease epidemics in its episodic nature and rapid geographic expansion and in the small-scale mosaic of healthy, diseased, and regreening trees within the population of even single stands. Such diseases might limit uptake or translocation of minerals from the soil at the level of the rhizosphere, mycorrhizae, root, or vascular bundles (18,20), thus leading to the characteristic symptoms of magnesium deficiency at sites where magnesium availability nears deficiency for healthy trees. This view is supported by recent experiments where spruce plantlets were grown in treated (e.g., application of steam or fungicides, inoculation with mycorrhizal fungi) soils from healthy and diseased stands. The experiments indicate the presence of a noxious microbial factor in the soil from diseased stands that interferes with the rhizosphere or ectomycorrhizae, or both, and inhibits mineral uptake (3).

Thus, acute yellowing is most likely a syndrome of complex etiology. In extreme cases, it may be caused solely by too little plant-available magnesium in the soil or by severely restricted magnesium uptake resulting from root diseases or antagonists of rhizosphere flora or mycorrhizae. In most cases, both determinants are probably involved in various proportions.

The Waldsterben Hypothesis and Growth Increment

Growth increment is one of the most important criteria for measuring tree vitality. The width of annual growth rings should show clearly if vitality has



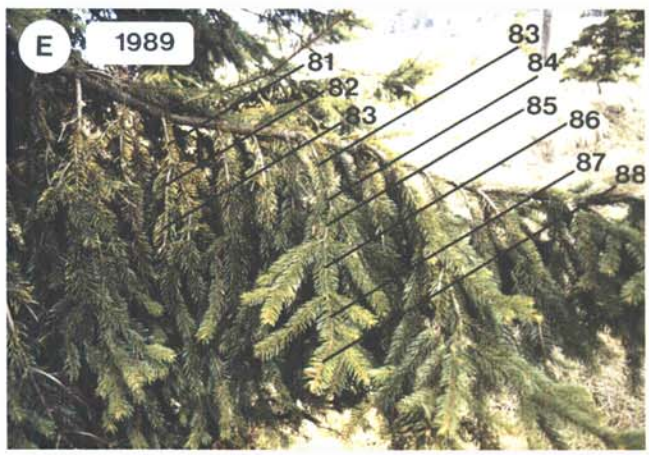


Fig. 9. At Dreisessel in the Bavarian Forest, (A) young and (B) old spruce trees show acute yellowing; (C) acute yellowing of branch. At the Schneeberg in the Fichtel Mountains, spruce branches pictured in (D) August 1986 and (E) May 1989 show recovery by continuous stagnation of yellowing since 1985; only needles of 1984 and of earlier seasons are still yellow. (Photographs by Kandler and Miller)

decreased recently, as suggested by the Waldsterben hypothesis. A study of representative collections of trees (with the exception of fir affected by Tannensterben) from various regions of Germany revealed no general decrease in increment during the last decade but rather an average or above-average growth rate compared with the century as a whole (9,11). In spruce, 30- to 60-year-old stands grew faster than did 90- to 120-year-old stands at the same age (11,25). Why did forests grow faster in the second half of this century than they did in the first? Nitrogen input by deposition of air pollutants (e.g., nitrate, ammonia) may be one of the reasons for improved growth. However, the elevated CO_2 level, a slight increase in precipitation and temperaure in central Europe, and improved forest management also must be considered (9,11).

Some stands do have poor growth and substantial tree death, but they are restricted primarily to orographically extreme sites having unfavorable climatic conditions, poor mineral supply, endemic diseases (e.g., root and heart rot), insufficient forest management, or combinations of these. Spruce decline at the rim of the Ore Mountains in Czechoslovakia is a well-known example of the disastrous effect of man-made air pollution in highly industrialized areas and surroundings (38). It is caused by high (70–100 $\mu g/m^3$ annual average), but not extreme, SO_2 concentrations from the numerous neighboring coal mines and power plants. Only spruce is dying, and except for *Lecanora conizaeoides* Nyl. ex Crombie, a species of lichen highly resistant to SO_2, the lichen flora is destroyed. The more SO_2-resistant hardwoods, e.g., beech and *Sorbus aucuparia* L., appear healthy, but hardwoods also show increasing damage along the steep pollution gradient in the immediate vicinity of the power plants. These natural and anthropogenic declines are site- and species-specific problems that have been known for a long time (8,38) and do not indicate a new type of general forest decline. This is underlined by the fact that recolonization of formerly highly polluted areas of central Europe by lichens and conifers has taken place immediately upon reduction of SO_2 concentrations (9).

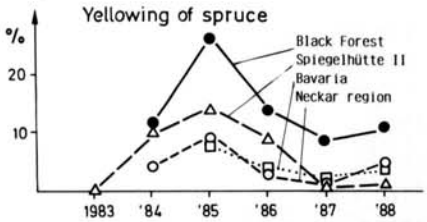


Fig. 10. Percentage of yellow needles on spruce trees older than 60 years in the Black Forest, Neckar region, and Bavaria and of yellowed young trees in a natural regeneration of Norway spruce at Spiegelhütte II in the Bavarian Forest (total trees = 1,600).

A Problem of Awareness?

Annual forest damage surveys lack comparable data from former periods, because the present system of classification of forest condition was not used before 1983. The traditional forest descriptions (*Forsteinrichtungswerke*), which are updated every 10 years, do not utilize the degree of crown transparency but describe semiquantitatively the occurrence of biotic (e.g., root and heart rot, insect damage) or abiotic (e.g., frost damage, windthrow) calamities. Trees showing very severe needle loss (beyond approximately 75%) are mentioned as "declining trees," but their proportion has never been estimated.

The only known early attempt to quantify the degree of crown transparency was made by Rebel (17), who in the early 1920s observed very poor crown conditions in conifers and hardwoods and called this phenomenon "heat disease." He thought that a series of drought years had caused this condition, and according to his judgment, 21–51% of the forest area of Bavaria was "endangered" or "irreversibly diseased," depending on the growth area. The damage caused by this "heat disease" may have been similar to the present Waldsterben. Although the symptom description is vague for "heat-diseased" trees, recent photographs documenting Waldsterben are similar to those of trees and stands found in old scientific journals, photograph albums, and postcards. From such photographs it is obvious that crown transparency is not new but occurred very frequently in earlier times. For instance, on about 90 photographs published by Rubner during 1936–1939 (21,22), almost none of the spruce depicted as typical of particular races would today appear in class 0 (healthy) but would be put in classes 2 and 3. Some of these photographs (Fig. 11A) are almost indistinguishable from those recently showing the "new symptoms" of Waldsterben (Fig. 11B). Schweingruber (30) recently compared crown conditions of spruce shown on postcards collected from the Swiss Alps and the Jura area from 1900 to 1925 and from 1975 to 1987. About 2,000 clearly recognizable trees were classified by four independent experts according to the presently used system of the annual forest damage surveys. Depending on the district where the photographs were taken, the proportion of damage classes 2 and 3 ranged between 16 and 41% in the old set of postcards and between 16 and 21% in the new set. Swiss Sanasilva reports (official annual forest damage reports of Switzerland) show that during 1986–1988, 18% of the spruce trees were in damage classes 2 and 3 in these areas. Thus, the two series of postcards reveal the same proportion of crown transparency in spruce as the current forest damage surveys, with a tendency toward higher damage in the first quarter of the century.

Comparison of old and new photographs, growth ring chronologies, and the development of the damage classes during the period from 1983 to 1988 all suggest the same conclusion: General dying of forests (Waldsterben) or even an unprecedented decline in central European forests during the 1980s has not occurred, but similar patterns of crown conditions and incremental growth caused by fluctuations in site, by weather conditions, and by diseases

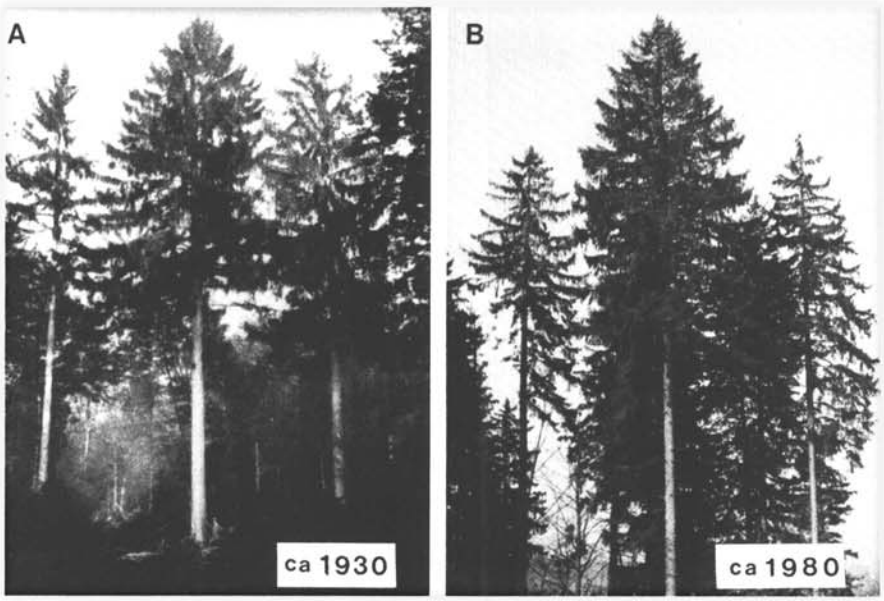


Fig. 11. Two groups of spruce trees photographed (A) in the early 1930s as an example of normal *Kammfichten*, a special form of spruce (22), and (B) in the early 1980s as an example of a healthy tree (foreground) contrasted with two trees (background) showing the symptoms of Waldsterben (29).

clearly have occurred in the past. This view is in accordance with Mueller-Dombois' (14) statement: "Stand-level dieback in forests is by no means a new phenomenon." This statement applies in both natural and managed forests, although under management, the natural succession cycle is interrupted but not abolished. In managed stands, accumulation of dead wood and decaying trees, which may reach 50% in unmanaged forests (9), is largely prevented by the rotation of stands and regular thinning. Thus, fluctuations of the very low proportions of damage classes 3 and 4 in the current annual surveys depend mainly on the quality and intensity of forest management—not the natural decline and mortality rate.

Waldsterben may be understood as a problem of awareness: Forest conditions that were believed to be "normal" in earlier times became suddenly a symbol of the growing fear of the destructive potential of human activities on environment. Of course, this new feeling also involves numerous different emotional and even political aspects, which will not be discussed here.

The Consequences for Forest Pathology and Phytopathologists

After the emotions about Waldsterben, defined by its advocates as an unsolvable complex lethal ecosystemic disease, have calmed and more facts have become known, forest pathologists may be encouraged by new public awareness of "sad-looking" trees to conceive new research projects necessary to unravel the old, often discussed decline phenomena. Very general Waldsterben hypotheses proposed in the past will be of little help in elucidating the causal biotic and abiotic factors in particular cases of decline. These must be analyzed symptom by symptom, species by species, and site by site according to the classical principles and more recent concepts (12,36) of phytopathology.

The inevitable adoption of advanced methods supplied today by molecular biology, biochemistry, and environmental chemistry will require a substantial extension of facilities as well as manpower. While pollution research has been stimulated as a first consequence of the new awareness of forest conditions, forest pathology may eventually flourish in the long run.

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(Extended lists of additional references may be found in references 8, 9, and 10.)