Peter Schütt
University of Munich, West Germany

Ellis B. Cowling
North Carolina State University, Raleigh

Waldsterben, a General Declin Symptoms, Developmen

During the past 5 years a large-scale regional decline of many different forest ecosystems has taken place in Germany, Belgium, France, Italy, Austria, and other countries of central Europe. These decline phenomena, collectively called Waldsterben, include several scientifically unprecedented features:

- A simultaneous and rapid decrease in the health and vigor of both softwood and hardwood forests has affected many different species of trees (including four important conifers and six important angiosperms) growing under a wide range of soil, site, and climatic conditions.
- The phenomenon has developed very rapidly since 1979 or 1980. The area of forest affected in West Germany increased from about 8% of the total in 1982, to about 34% in 1983, to about 50% in 1984.
- The syndrome stands apart from ordinary diseases of trees because many forest ecosystems as we have known them appear likely to be destroyed.
- Symptoms are of three general types—growth-decreasing (hypoplastic), abnormal-growth (hyperplastic), and water-stress. Growth-decreasing symptoms include loss of foliar biomass, loss of feeder root biomass, decrease in diameter increment, premature senescence and death of older needles and leaves, increased susceptibility to secondary root and foliar pathogens, death of affected trees, and death of herbaceous vegetation. Abnormal-growth symptoms include active casting (abscission) of green leaves and green shoots, altered branching habit and abundant production of adventitious shoots, altered morphology of leaves, repeated abnormal crops of seeds and cones, and abnormal allocation of the products of photosynthesis.
- The stress factors inducing the Waldsterben syndrome are not known, but it is widely assumed (and we believe correctly so) that atmospheric deposition of toxic, nutrient, acidifying, and/or growth-altering substances is involved.
- Six general hypotheses have been advanced to explain one portion or another of the syndrome: acidification/aluminum toxicity, ozone effects, magnesium deficiency, general disturbance of physiological function, excess nutrient (especially nitrogen) deposition from the atmosphere, and air transport of growth-altering organic substances.

This paper is adapted from an invited lecture presented by the first author at a conference entitled "Acid Rain and Forest Resources," sponsored by the Canadian Forestry Service and the USDA Forest Service at Quebec City, Canada, in June 1983. Portions of the symptom descriptions and certain of the ideas about possible causes of Waldsterben were developed during field excursions and consultations by both authors in West Germany and the United States in September 1983 and May and June 1984. A part of these consultations was completed during the German-United States Scientific Exchange on Forest Decline sponsored by the Bundesministerium für Forschung und Technologie in West Germany and the Environmental Protection Agency in the United States.

First Awareness of the Problem

During 1979 and 1980, foresters in the Bavarian Forest and other parts of southern Germany first realized that a certain change was taking place in the appearance of old Norway spruce trees (*Picea abies*). The trees were showing symptoms never seen before in the mountains to the east and south or in low-elevation forests on the plains surrounding Munich. The symptoms progressed, sometimes very rapidly, from partial to total defoliation and eventually to death of the trees.

At that time, one of the main problems in German forests was white fir (*Abies alba*) decline (Tannensterben), a somewhat mysterious disease that had occurred periodically on the north side of the Alps during the preceding 250 years (22). Around 1970, Tannensterben became evident once again, this time very widespread and rather intense. A research program was established in the mid-1970s to find the cause(s).

By 1979, a new disease of spruce—which occupies 40% of the total forest area in West Germany, whereas white fir occupies only 2% (Table 1)—began to dominate the attention of both forest pathologists and practical foresters. Because the symptoms on spruce were very similar to those on fir, many forest pathologists began to wonder if the stress factors inducing Tannensterben were also inducing the new disease on spruce (27). From 1980 to 1984, symptoms became evident in spruce forests in much of central Europe, southern Scandinavia, northern Italy, parts of the Balkan countries, and (probably) large areas in eastern Europe.

Before long, Scots pine (*Pinus sylvestris*) showed similar symptoms, and since 1981 we have observed an even more mysterious disease syndrome in European beech (*Fagus sylvatica*) (32). The disease in beech included a few new symptoms. But once again, the disease was widely dispersed geographically and, in some cases, the time between first observation of symptoms and death of affected trees was extremely short. Symptoms in beech now occur over large parts of central Europe—in the Alps, in the Appennin Mountains of northern Italy, and in southern Scandinavia.

During 1984, the forest disease situation in Europe grew worse. Symptoms are now found on almost every species of forest tree (Table 1) and on several shrubs and herbs. Fortunately, no agricultural crops appear to be affected.

Waldsterben has become a major problem not only in forestry but also in public discussions. It is now an important political issue in West Germany. As a consequence, a diverse array of survey,

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research, and regulatory activities has been initiated. In this article, however, we emphasize the botanical and pathological aspects of the disease syndrome.

Definition of the Term

Waldsterben is a collective term for the widespread and substantial decline in growth and the change in behavior of many softwood and hardwood forest ecosystems in central Europe. The phenomenon is characterized by a variety of growth-decreasing (hypoplastic), abnormal-growth (hyperplastic), and water-stress symptoms (Table 2). Waldsterben often leads to death of the affected trees and sometimes to death of associated herbaceous vegetation. Although the common symptoms of Waldsterben include increased susceptibility to insects, foliage and root pathogens, drought, frost, and other stress factors, Waldsterben should be understood as distinct from the diseases induced by the familiar biotic forest pathogens—fungi, bacteria, nematodes, insects, and viruses. Waldsterben is also distinct from the typical injuries to forest induced by drought and frost or by the toxic gaseous air pollutants such as ozone, sulfur dioxide, and hydrogen fluoride.

Table 1. Percentage of West German forests (total area = 7.4 million ha) showing Waldsterben symptoms in 1984[a]

	Species/percentage of total forest area in each species						
State	Spruce/40 (*Picea abies*) (%)[b]	Pine/20 (*Pinus sylvestris*) (%)[b]	Fir/2 (*Abies alba*) (%)[b]	Beech/17 (*Fagus sylvatica*) (%)[b]	Oak/8 (*Quercus robur*, *Q. petraea*) (%)[b]	Other/13 (%)[b]	Total (%)[b]
Schleswig-Holstein	55	72	98	10	8	14	27
Niedersachsen	40	36	45	43	40	22	36
Nordrhein-Westfalen	40	78	33	38	34	36	42
Hessen	27	66	36	49	42	23	42
Rheinland-Pfalz	37	64	97	47	42	23	42
Baden-Württemberg	65	78	89	64	66	44	66
Bayern	58	64	86	59	51	36	57
Saarland	26	41	0	42	33	13	31
West Germany	51	59	87	50	43	31	50

[a] Data developed by Bundesministerium für Ernährung, Landwirtshaft und Forsten (2).
[b] Percentage of forest area affected by Waldsterben.

Table 2. The common symptoms of Waldsterben

Growth-decreasing (hypoplastic) symptoms	Abnormal-growth (hyperplastic) symptoms	Water-stress symptoms
Discoloration and loss of foliar biomass	Active casting (abscission) of green leaves and green shoots	Altered water balance
Loss of feeder root biomass	Stork's nest formation in white fir	
Decreased annual increment	Altered branching habit and greater-than-normal production of adventitious shoots	
Premature senescence of older needles in conifers	Altered morphology of leaves	
Increased susceptibility to secondary root and foliar pathogens	Altered allocation of photosynthate	
Death of affected trees	Excessive seed and cone production	
Death of herbaceous vegetation		

We believe that an accurate and comprehensive understanding of the *many different disease symptoms shown by the trees themselves* provides the most rational foundation for identification of causal factors and possible management strategies. We hope this paper will attract the intellectual resources of some of the world's most able plant pathologists, soil scientists, forest ecologists, and atmospheric scientists. Your help is needed because we must learn more about the pathogenesis and primary causes before we will have a realistic chance to stop a unique disease phenomenon that includes several features without precedent in science. We consider the Waldsterben phenomenon one of the most remarkable forest disease problems of the 20th century.

Chronology and General Aspects of Waldsterben

A number of special facts and observations make the Waldsterben syndrome a pathological phenomenon standing apart from ordinary diseases of trees. Although some are difficult to interpret, these facts and observations lead to the impression that the syndrome may destroy or seriously impair the forest ecosystems of central Europe as we have known them. Furthermore, the disease syndrome is developing so rapidly, on so many species of trees, on so many soil and site conditions, and on so large a geographic scale that major ecological and economic consequences appear very likely.

The following features are of special significance:

- Visible symptoms of Waldsterben started at about the same time in many different parts of Europe.
- Within 4 years, symptoms were found over large parts of the continent.
- Waldsterben is still expanding in intensity and geographic distribution and is associated with an increasing frequency of secondary stress factors, including insects, needle and root fungi, and climatic influences such as frost.
- Waldsterben affects almost every tree species in central Europe, both native and exotic, including the four most important conifers (spruce, fir, pine, and larch) and six of the important angiosperms in forest commerce (beech, birch, oak, ash, maple, and alder) (31).
- Various species of shrubs and herbs are also affected.
- Symptoms are heterogeneous from one region or stand to another and even within the same species.
- Most affected tree species show a variety of growth-decreasing and abnormal-growth symptoms.
- Waldsterben occurs with similar intensity on rich or poor, acid or basic, wet or dry soils, independent of their geologic origin (23).
- Waldsterben is independent of climatic differences and rather independent of the direction of slope in hilly or mountainous terrain.
- Although symptoms are often most severe in high-mountain forests, similar symptoms have been observed in middle- and low-elevation forests.
- Differences in forest management practices or in the structure of the forest (single species vs. mixed stands, planted vs. natural stands) do not have important influences on the amount of damage.
- Young trees are generally less affected than older ones, but exceptions occur, especially at high elevation.
- Some of the typical symptoms have never been described before, especially in beech, spruce, larch, and some deciduous trees of lesser importance.

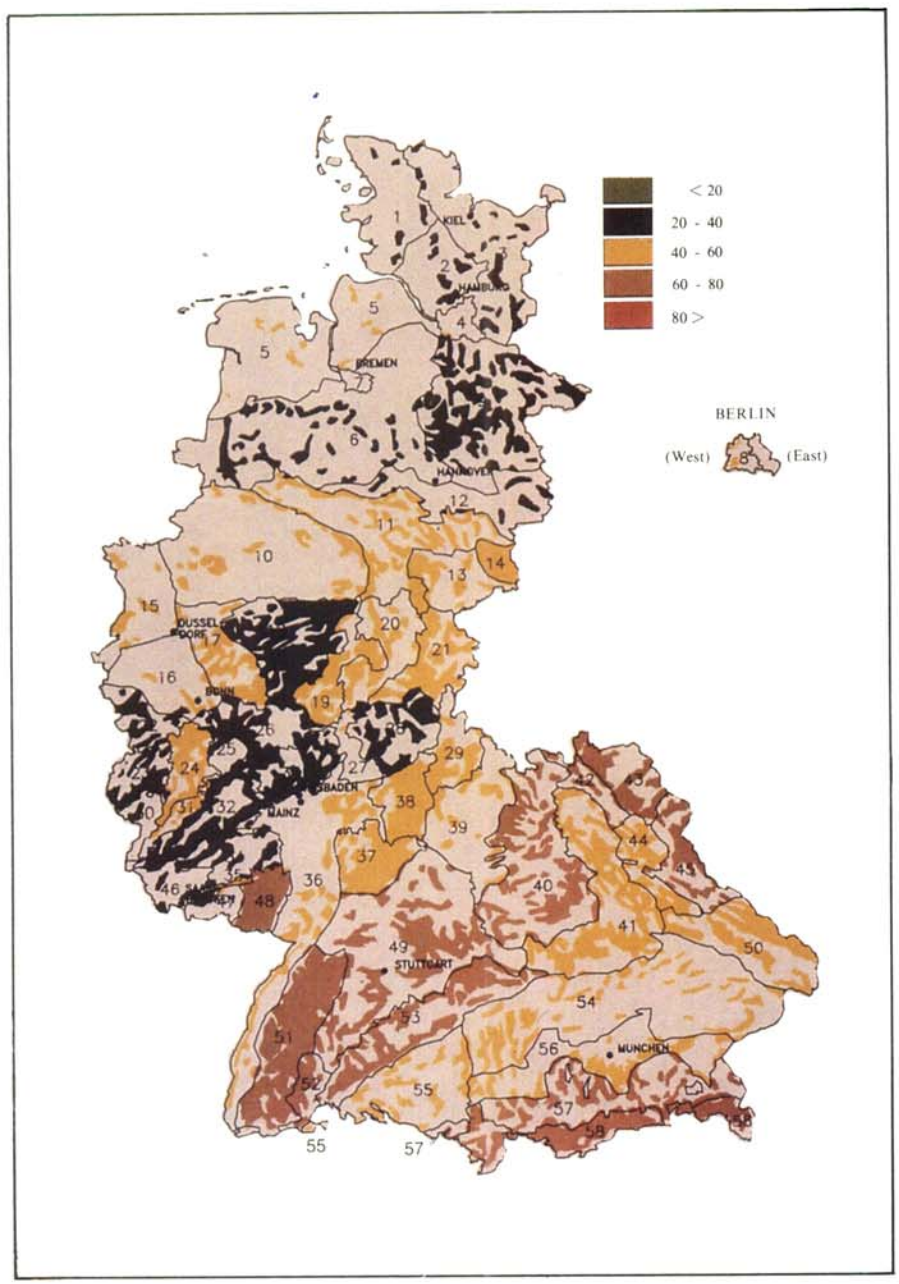

Fig. 1. Distribution of Waldsterben in West Germany in 1984. Damaged area in percent of total forest area: green <20%, black 20–40%, yellow 40–60%, brown 60–80%, red >80%. (Data developed by the Bundesministerium für Ernährung, Landwirtschaft und Forsten, Bonn [2]. Reproduced with permission)

Fig. 2. Sparse needles from Waldsterben-affected white fir (right) compared with needles from healthy tree (left). Similar symptoms are seen in Norway spruce, Douglas-fir, and Scots pine.

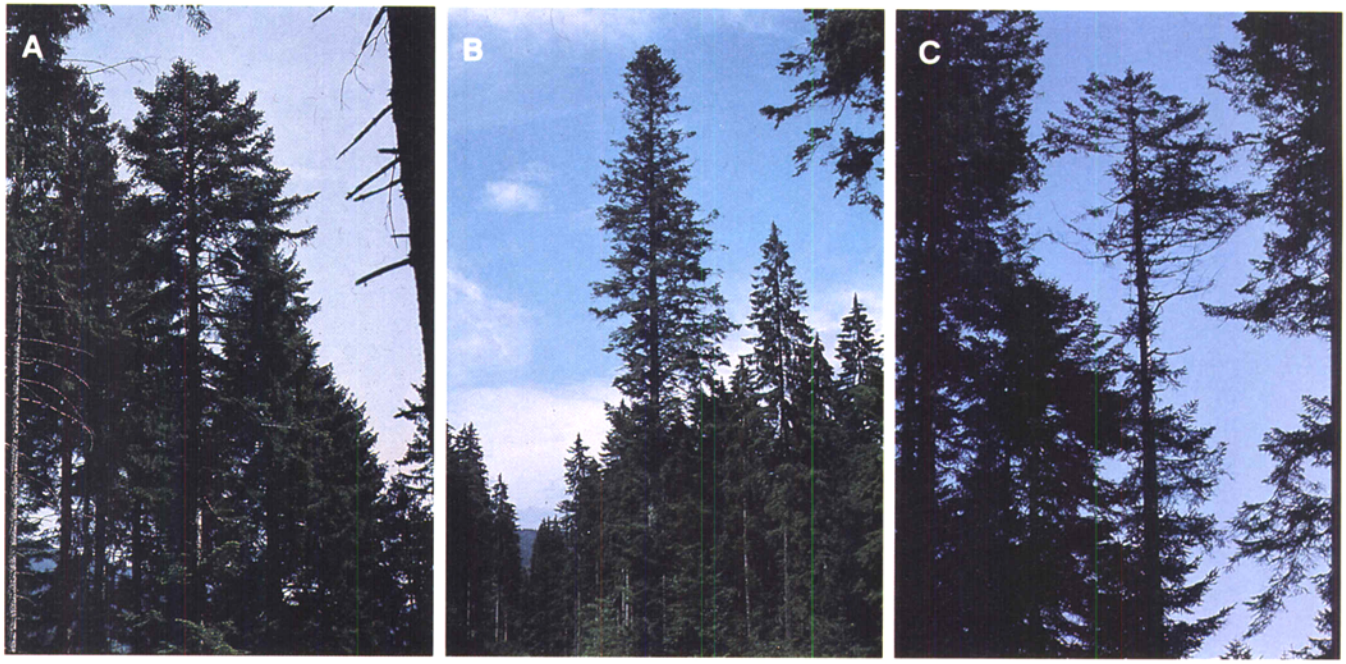

Fig. 3. Typical crown symptoms of Waldsterben in white fir: (A) Dense needle development in healthy fir compared with increasing transparency of the crown at (B) moderate and (C) advanced stages of the disease. Symptoms are similar in Norway spruce, Scots pine, and several broad-leaved species.

• At present, the primary causal factors of Waldsterben are not known.

The distribution pattern of affected trees or stands and the symptoms of pathogenesis give only some rough hints about the probable causes of Waldsterben. The widely held assumption that atmospheric deposition of toxic, nutrient, acidifying, or growth-altering substances is involved is still only a hypothesis that cannot be confirmed or rejected with existing evidence.

Waldsterben belongs to a group of diseases called forest diebacks or declines for want of better terms (13,21). Because these diseases are characterized by collaboration of several biotic and abiotic pathogenic factors acting simultaneously or one after another, deciding which are primary and which are predisposing or secondary is difficult (12). In almost every case, a broad spectrum of predisposing, inducing, and contributing stress factors is involved (21). Since these factors can differ in time, space, and intensity, the symptoms also can vary from location to location.

Nationwide surveys of Waldsterben symptoms were undertaken in 1982, 1983, and 1984 by the regional governments of West Germany. In 1984, 50% of the country's total forest area was showing Waldsterben symptoms (2). Intensity of damage differs widely among the major tree species and geographic regions of West Germany (Table 1, Fig. 1).

Symptoms on Various Species

White fir. White fir, a high-volume tree species of outstanding ecological value, is most abundant at moderate elevations in parts of the Alps and in several other middle-elevation mountain ranges in central Europe (Fig. 1). Many fir forests probably have been weakened by previous occurrences of Tannensterben (22,26). The 80- to 200-year-old fir forests are more seriously affected than younger ones. Usually, the time from first symptoms to death of the trees is more than 5 years.

Fig. 4. "Stork's nest crown" induced by Waldsterben in 40-year-old white fir. Although these structures often are observed in healthy firs of 100+ years, they are now frequent in 50- to 80-year-old trees and are even seen in some 25- to 30-year-old trees. This symptom is unique to white fir.

Fig. 5. Pathological wetwood in stem wood of white fir. Spread of this bacterial infection from the central core to the sapwood of the stems or roots inhibits water transport. In Norway spruce, this symptom is less conspicuously colored and less frequent.

The first symptom is usually loss of needles from the older (10- to 12-year-old) branches in the basal and middle parts of the crown (Fig. 2). The crown becomes progressively more transparent (Fig. 3), until only the uppermost 1–2 m remain unaffected and dark green. Diseased needles turn grayish green, not red or brown. A "stork's nest crown," formed when the leading shoot is stunted while the laterals continue to grow (Fig. 4), is often observed on older trees and sometimes on younger ones. The root wood and basal part of the stem wood often contain bacterial wetwood (Fig. 5), which inhibits water transport if the infection extends into the sapwood portion of the tree (5,25).

The fine root system of white fir is often seriously damaged (Fig. 6A), and root regeneration is poor. Frequently, mycorrhizae are almost totally absent and such root pathogens as *Phytophthora*

sp. or *Armillaria* invade (7,8). Surprisingly, the water status of the green needles remaining on affected trees often is about normal (18).

At present, white fir is the most seriously affected tree species in central Europe. In parts of eastern Bavaria and in Czechoslovakia, white fir appears to be no longer viable as a commercial forest tree species.

Norway spruce. Norway spruce is the most important tree species in West Germany (Table 1) and other parts of central Europe because of its straight stems and high volume. Norway spruce is usually shallow-rooted and thus is susceptible to windthrow and to drought. It is predominantly a tree of the Alps and of all elevations in the middle mountains of Germany (Fig. 1).

Symptoms usually occur on older trees first. Young trees are seriously affected only at high elevation; in the flat plains of southern Germany, the youngest symptomatic trees are about 40 years of age. Symptoms often develop irregularly, and the crown of a tree may carry both healthy and diseased branches in a random pattern. Needles of similar age on the same branch often vary in color and size, without a perceptible order.

Again, the most conspicuous symptom is transparency of tree crowns owing to loss of needles (Fig. 7); usually, but not always, older needles are lost first (Fig. 8). The majority of lost needles are grayish green and some are yellowish green. Yellowing is observed mainly on spruce at high elevation, appearing first on the upper (light-exposed) side and later on the lower (shaded) side of the needles and progressing from the lower to the upper branches and from the older needles near the stem to the younger ones farther out on the branches (Fig. 9). During certain years in some regions, a few trees in a stand show a variable pattern of needle chlorosis of varying intensity.

Some spruce trees that naturally have an almost vertical habit of branching show a so-called silver tinsel symptom. When needle loss is severe, the "hanging" bare branches resemble those of a Christmas tree decoráted with silver tinsel.

Occasionally, infection by secondary and weak needle parasites such as *Rhizosphaera kalkhoffii* or *Lophodermium* spp. cause needle browning before needle fall (17). In some areas, single-node shoots with intact and apparently healthy green needles are dropped to the forest floor after abscission (Fig. 10); to our knowledge, this active casting of intact green shoots has never been reported in any disease of spruce. Wetwood also occurs in the root and stem wood of Norway spruce, but its role, if any, in Waldsterben of spruce is still uncertain.

The fine root system of diseased spruce is often very weak, with few or absent

Fig. 6. Typical root symptoms of Waldsterben: (A) Fine roots of declining white fir with typical knots. (B) Deteriorated mycorrhizal short roots on declining Norway spruce (left) compared with active mycorrhizal root tips on spruce with few decline symptoms (right).

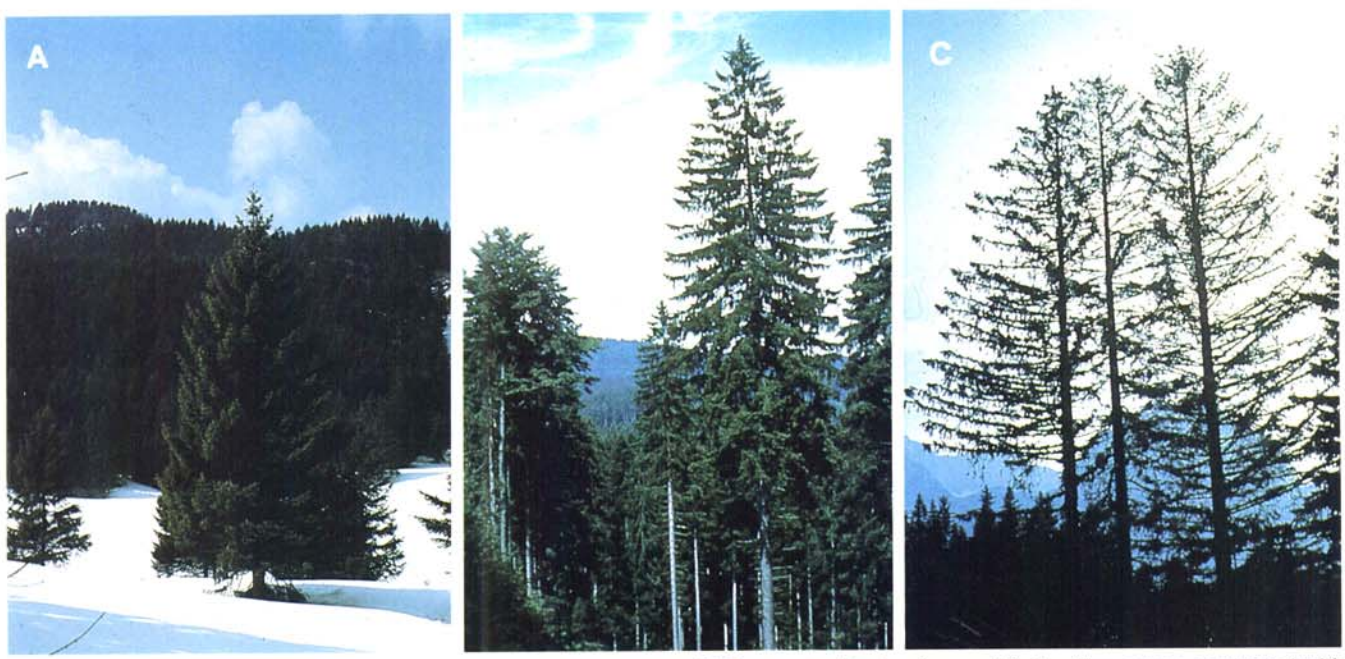

Fig. 7. Typical crown symptoms of Waldsterben in Norway spruce: (A) Dense needle development in healthy spruce compared with increasing transparency of the crown at (B) moderate and (C) advanced stages of the disease.

mycorrhizal short roots (Fig. 6B) and poor ability to form new feeder roots. *Cylindrocarpon* sp. and *Mycelium radicis atrovirens* frequently are abundant in roots of affected trees (20).

A very common and striking symptom in spruce is the abundance of adventitious shoots formed on the upper side of branches of first and second order (Fig. 11). These shoots give heavily diseased trees an "uncombed," larchlike appearance.

Exposed spruce usually show symptoms earlier and are more seriously affected than trees within a closed stand. The period of time between first symptoms and the stage of damage where the tree has to be cut varies from a few weeks to 3 years.

Scots pine. Scots pine is distributed in many parts of central Europe. It reaches modest size, is deep-rooted, and grows well in both pure and mixed stands. Waldsterben symptoms on Scots pine sometimes resemble those of chronic sulfur dioxide or ozone fumigation, especially with regard to discoloration and needle loss. Affected trees usually are in the middle (40- to 80-year) or older age class.

Healthy pines keep their needles for about 3–4 years. Waldsterben-affected pines lose their oldest needles first, beginning at the inner and basal parts and extending to the outer and upper parts of the crown. The crown becomes progressively thin and transparent (Fig. 12A) so that a heavily damaged tree often carries only a single small ring (umbrella) of dwarf shoots around the terminal leader. Needles turn from dark green to grayish green. Occasionally, yellow spots appear near the needle tips and turn into dark brown necrotic bands (Fig. 12B). Reduced needle length is often prominent and may begin with some short needles forming together with normal ones on the same shoot. Necrotic zones develop on young branches and resemble hail wounds. The period between first symptoms and the final stage of disease varies between 1 and 3 years.

European beech. Beech is a very important timber species in central Europe. This deep-rooted, mighty tree thrives in humid areas, growing in pure and mixed stands on either alkaline or acid soil. Beech is an important ecological component of the original "Bergmischwald" (mountain mixed forest).

Older beeches (80+ years) are most frequently affected by Waldsterben, although saplings in the upper elevations of the Bavarian Forest also show symptoms. No other tree species shows so many different and unique symptoms (32), but not all symptoms are evident on every tree and at all times of the year. The most striking are deviations in crown shape and decrease in crown density due to pathological changes in branching habit and in the number, size, shape, and retention of leaves (Figs. 13–15).

The natural balance in the ratio of short shoots to long shoots is disturbed. First-order branches almost exclusively form short shoots, so that the crown of an affected beech tree is characterized by a few dominant long shoots. Consequently,

Fig. 8. Typical loss of needles from Waldsterben-affected Norway spruce. Needles are often actively cast while still dark green but may turn grayish green or yellowish green before abscission. Needle loss typically progresses from the older ones near the stem to the younger ones farther out on the branch.

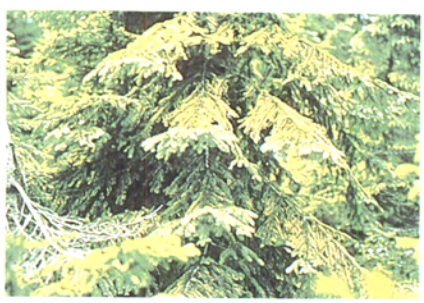

Fig. 9. Typical yellowing of needles of Waldsterben-affected Norway spruce. This symptom, observed mainly in high-elevation spruce forests, resembles typical symptoms of magnesium, iron, or manganese deficiency. Yellowing appears first on the upper (light-exposed) side and later on the lower (shaded) side of the needles and progresses from the lower to the upper branches and from the older needles near the stem to the younger ones farther out on the branch.

Fig. 10. Green shoots of Waldsterben-affected Norway spruce on the forest floor after active casting (abscission). To our knowledge, active casting of green shoots has never been reported in any other disease of conifers.

Fig. 11. Abundant adventitious shoots on the upper side of a branch of a Waldsterben-affected Norway spruce. These shoots are sometimes the only green foliage remaining in the crown of a severely affected tree. This symptom is also seen in white fir and European larch.

Fig. 12. Typical crown symptoms of Waldsterben in Scots pine: (A) Normal needle development in healthy pine (right) compared with transparent crown of affected pine (center). (B) Needles of affected pine are sparse with necrotic or yellow spots near the tips.

most leaves are concentrated close to these long shoots and large parts of the inner crown lack leaves (Fig. 13). Again, the crown of a diseased tree becomes transparent. This tendency is accentuated by leaves decreasing in number and size from the base to the top of an annual long shoot and by pronounced premature shedding of leaves (32). Many of these leaves are dropped while still *green* (Fig. 14)—a very unusual symptom indeed! Abnormal leaf fall often starts in June, soon after the leaves are fully formed, and increases in intensity during the summer months. Usually, the percentage of green leaves falling to the ground is higher than that of colored or dead ones.

Some trees show marked yellowing of light-exposed leaves, frequently starting at the leaf margin and ending in necrotic patches. Also, leaves on affected trees are occasionally abnormal in shape, with irregular (toothed) margins (Fig. 15) rather than the smooth margins of healthy beech leaves.

Old, diseased beeches sometimes contain dead branches; heavy branches at the crown base die first, followed by the upper ones. Bark necrosis is occasionally observed, typically in small but at times in large patches in the branches that are dying back from the top. In almost every Waldsterben-affected tree, the youngest branches are very brittle and break easily when bent. This brittleness is in distinct contrast to branches of healthy beech, which can be bent around very sharp curves before breaking.

Finally, the fine root system and mycorrhizal development are disturbed and *Armillaria* infections are common. Old beeches can die within a single growing season, but this is not generally the case. More study is needed before firm generalizations can be drawn about this aspect of Waldesterben in beech.

Other tree species. Since the summer of 1982 we have seen new disease symptoms on several other tree species of lesser economic importance (30). Sycamore maple (*Acer pseudoplatanus*) began to show similar symptoms at about the same time and in the same forest sites as beech. The same was true for mixed stands of Douglas-fir (*Pseudotsuga menziesii*) and Norway spruce. Some differences from the usual symptom pattern have been observed on birch (*Betula verrucosa*), alder (*Alnus glutinosa*), ash (*Fraxinus excelsior*), and oak (*Quercus* spp.). European larch (*Larix decidua*) looks and behaves differently. Like beech, larch shows a striking disorder of branching rhythm, combined with early leaf fall, again of green needles.

The Common Symptoms

The symptoms of Waldsterben are neither constant nor uniform. They vary from species to species, season to season, region to region, soil type to soil type, elevation to elevation, and stand to stand—and even among individual trees in the same stand. This is expected to a certain degree, considering our present knowledge of other tree diseases of either single or multiple etiology. But in spite of this variability, certain common symptoms and physiological traits are part of the syndrome in nearly every tree species (Table 2).

Growth-decreasing (hypoplastic) symptoms. *Loss of foliar biomass.* Increased transparency of crowns owing to loss of needles, leaves, and even shoots is very common (Figs. 2, 3, 7, 8, 10, 12–14), occurring in both gymnospermous and angiospermous species. Loss of foliar biomass inevitably leads to decreased photosynthate for allocation to feeding of roots and other vital functions, including height and diameter growth. We do not know if decreased foliar biomass leads to decreased root biomass, or vice versa, or whether each decreases independently of the other. Maintenance of a general balance between roots and shoots may be expected in all tree species.

Loss of feeder root biomass. Comparative investigations between healthy and Waldsterben-affected old fir, spruce, and beech trees (19,31) invariably show that affected trees have markedly fewer living feeder roots, less abundant or no mycorrhizae, reduced capacity to form new feeder roots, and increased frequency of infection by secondary root pathogens (Fig. 6). Fungi of minor pathogenic ability apparently can invade roots of diseased trees. Poor feeder-root development seems to be associated more with mineral soils than with soils high in organic matter.

Decreased annual increment. Measurements in old fir and spruce trees taken by different investigators in different regions of central Europe have led to almost identical conclusions. Long before foliar and branching symptoms become visible, the annual increment of stems is reduced (Fig. 16). Tree rings become narrower, and the amplitude of ring-width responses

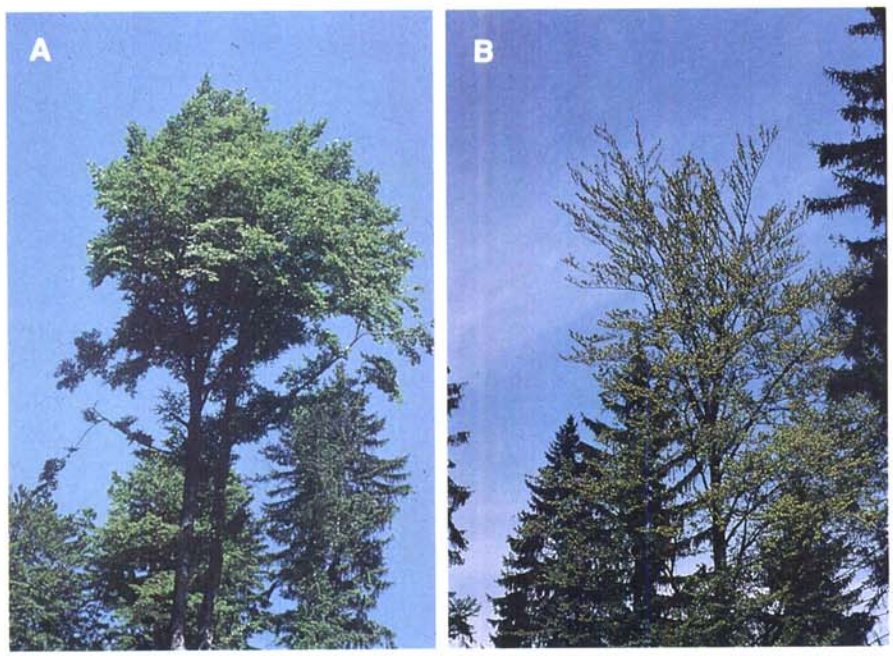

Fig. 13. Typical crown symptoms of Waldsterben in European beech: Comparison of (A) healthy beech with (B) diseased beech shows significant changes in pattern of long and short shoots and abnormal location of leaves (mainly on long shoots) within the crown of the diseased tree.

Fig. 14. Green leaves of Waldsterben-affected European beech on the forest floor after active casting (abscission). This symptom has rarely been reported in diseases of forest trees.

Fig. 15. Abnormal leaf shape occasionally seen in Waldsterben-affected European beech. Leaf margins are irregularly lobed (toothed) rather than smooth.

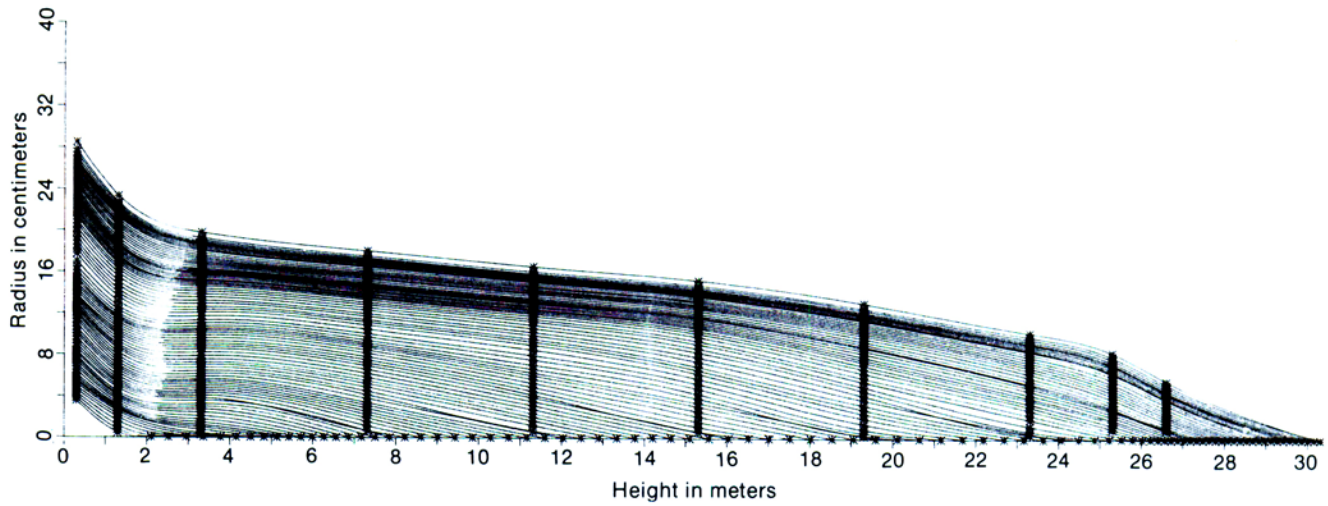

Fig. 16. Change in width of recently formed annual rings with increasing height along the stem of a Waldsterben-affected Norway spruce. In some cases, diameter growth near the base of a diseased tree is absent or so slight that several rings are missing, compared with the upper parts of the stem. (Courtesy Helmut Kenneweg, University of Göttingen)

to climatic variation is reduced. These tendencies are expressed more clearly in Waldsterben-damaged trees than in healthy ones. Diameter growth reduction began earlier in white fir (about 1960) than in Norway spruce (about 1970) and seems to be present also in beech (starting about 1978–1980). Photosynthate is allocated to wood formation differently in Waldsterben-affected trees, i.e., growth is depressed in the basal but not in the upper parts of stems (Fig. 16) (3,15). Changes in height growth of Waldsterben-affected trees have not been studied sufficiently, so determining if total biomass growth rather than just diameter growth is affected has not been possible.

Premature senescence of older needles. Foliage loss typically proceeds from the stem out to the branch and stem tips (Figs. 8 and 9). Thus, the oldest needles and leaves are lost first, suggesting that premature senescence may be a mechanism by which Waldsterben is induced. This is in distinct contrast to the pattern of dieback from the top common in other diseases of complex etiology, especially in angiosperms, in which the youngest branches die first (21).

Increased susceptibility to secondary root and foliar pathogens. Loss of vigor in trees is a very common consequence of many stresses and frequently results in increased abundance of secondary or weakly parasitic and/or pathogenic insects, fungi, bacteria, nematodes, viruses, and other pathogens.

Death of affected trees. Death appears to be the end result of all the growth-decreasing influences.

Death of herbaceous vegetation. Symptoms of tree decline are occasionally accompanied by damage to ground vegetation. This is observed with various species of ferns and grasses in the middle mountains of Germany (Fig. 17). Plants frequently die during the vegetation period but sometimes sprout again in the fall. This is true especially for *Lycopodium* spp. but also for *Rubus* spp. and *Vaccinium* spp.

A recent observation is death of vegetation directly under the crown projection (drip line) of diseased trees (Fig. 18). Since this phenomenon does not occur under the crowns of healthy beech or spruce, it appears that phytotoxic compounds build up in affected leaves or needles and are leached out or that deposits of toxic substances on the tree canopy are leached out onto the ground vegetation.

Summary. These various growth-decreasing symptoms could very well be the result of the following, alone or in various combinations: toxic air pollutants, nutrient deficiencies, drought, nutrient-induced inhibition of mycorrhizae, soil toxicity, and induction of self-limiting toxins or allelopathic chemicals, with or without secondary root or foliar pathogens.

Abnormal-growth (hyperplastic) symptoms. *Active casting (abscission) of green leaves and shoots.* This symptom is one of the most astonishing of the whole Waldsterben phenomenon because it is so completely out of harmony with the common symptoms of tree diseases. Fir, spruce (Figs. 8 and 10), beech (Fig. 14), maple, and oak are especially affected. The loss goes on continuously during the growing season, and even during the dormant season in some conifers. It is not induced by windstorms, snowstorms, or ice storms or restricted to special developmental stages of the trees.

Although squirrels and certain insects are known to cut green leaves and shoots, visual and microscopic observations confirm that such loss, at least in beech, results from a clearly discernible abscission layer at the base of the petiole of the fallen leaves. Active casting of undamaged green leaves is an extremely rare symptom in plant diseases caused by fungi, bacteria, viruses, nematodes, and air pollutants studied so far. The only known exceptions appear to be oak wilt in the early stages (13) and a recent air pollution case involving aniline (9,10). Diseased plants usually retain leaves; dropped leaves almost invariably turn brown or at least yellow and show clear signs of pathogenesis or senescence before dropping.

Stork's nest formation. This symptom is seen only in white fir. Although a normal occurrence in trees more than 120 years old (22), stork's nest formation is considered a pathogenic trait of Waldsterben because it affects even 25- to 30-year-old firs (Fig. 4). The formation may be a type of premature senescence.

Altered branching habit. The branching habit of Waldsterben-affected beech is obviously out of order (Fig. 13). Often, short shoots are formed instead of long shoots, changing the general shape and structure of the crown in both young and old trees and markedly decreasing the number of leaves (32).

Adventitious shoots are common in Norway spruce (Fig. 11) and white fir and occur to a lesser extent in larch. The shoots appear, singly or in low numbers, on the upper side of the first- and second-order branches, usually close to the branch nodes. The shoots carry sound needles with apparently normal photosynthetic activity. Proliferation of adventitious shoots markedly alters the appearance of heavily damaged trees, with first-order branches looking like bottle brushes. Many of these shoots stand in a row across the branches and frequently form the only green portions of the tree canopy during the final stage of Waldsterben. Adventitious shoots are known to occur in healthy trees; they occasionally appear after various stress situations, such as drought, but are more common in Waldsterben than has been seen with other stress factors.

Altered morphology of leaves. The leaf margins of Waldsterben-affected beech trees are often uneven and toothed (Fig. 15). There appears to be little or no correlation between intensity of disease and irregularity of leaf margins. In pine, short and long needles are common (Fig. 12B).

Altered allocation of photosynthate. Annual rings high on the stems of diseased trees often are much wider than those low on the stems, where rings may even be absent (Fig. 16). Also, root biomass is markedly decreased in relation to foliar biomass (Fig. 6). These observations suggest that the trees have lost the normal ability to allocate photosynthate to diameter growth along the stem and to balance root growth with shoot growth.

Excessive seed and cone production. All species of trees affected by Waldsterben have produced large crops of cones and seeds during the past 3 or 4 years. Norway spruce sometimes produces so many cones that those formed are less than half the usual size.

Summary. These abnormal-growth symptoms lead to the supposition that Waldsterben disturbs the balance of plant hormones. Indeed, recent experimental findings show a marked difference in the abscisic acid content of shoots of healthy and of Waldsterben-affected spruce trees (29).

Water-stress symptoms. *Altered water balance.* So-called pathological wetwood is part of the Waldsterben syndrome in white fir (Fig. 5). This abnormal wood is inhabited by bacteria that tend to spread from the central core of the stem into the sapwood, where they inhibit water conduction (22,25). Pathological wetwood originates in wounds of the root system and is one of several factors that increase water stress in affected trees. The phenomenon has not been investigated adequately in other species.

Freezing-point measurements show that green needles of Waldsterben-affected trees have practically the same water status as those of healthy trees. Despite reduced water supply to the tree as a whole, diseased trees apparently maintain the water status of their active needles. In white fir, the transpiration rate of these needles is lower than that of green needles of healthy trees (16,18).

Possible Causes

From the very first observations of Waldsterben in 1979 and 1980, scientists, foresters, politicians, and various public-interest groups have expressed opinions, ideas, and hypotheses about the causes of the syndrome (28). Many were attempts to explain some part of the total symptom picture or to blame some stress factor, e.g., acid rain, sulfur pollution, ozone, drought, or soil changes. Some were designed to serve a political, economic, or emotional purpose. In many cases, these

Fig. 17. Death of bracken fern in a Waldsterben-affected natural stand of white fir in the Bavarian Forest.

Fig. 18. Death of herbaceous vegetation directly beneath the canopy of Waldsterben-affected Norway spruce in the Bavarian Forest. Transition from lush green vegetation (*Calamagrostis villosa*) outside the "drip line" to dead vegetation inside the line is abrupt.

efforts have confused rather than advanced our understanding of the Waldsterben phenomenon.

General areas of agreement. Excluding the opinions and ideas characterized by political or economic interests or emotional background, a consensus has begun to emerge about the following facts or assumptions:

1. Waldsterben must be understood as a disease syndrome probably caused by a complex of several predisposing and stress-inducing factors that are followed by numerous secondary effects of abiotic and biological origin (21,28).

2. The primary causes are not any of the known forest pathogens or insects, although some of these agents are secondary or contributing stress factors.

3. Climatic extremes such as drought or frost may play a role but are probably predisposing or secondary factors instead of primary causes.

4. Some experimental findings, various field observations and survey results, and numerous debates about alternative explanations have led to a general agreement that atmospheric deposition of air pollutants or pollutant-related toxic, nutrient, acidifying, or growth-altering substances are among the primary causal factors.

Recent surveys, especially in the province of Baden-Württemberg (24), indicate that symptoms of Waldsterben are greatest at high elevation, in the direction of predominant winds, and in trees with wind-exposed canopies. These observations lend further credence to the idea that air pollutants are among the causal factors. This general conviction has led to some technical improvements in regional air quality in West Germany, the Netherlands, and some other countries in central and northern Europe. It has also led to almost total concentration of research activities on air pollutants, to the exclusion of other possible causes, such as radioactivity or microwaves.

Five hypotheses and one speculative idea. Five major hypotheses or "schools of thought" and a more speculative idea have been developed by various forest scientists in attempts to explain one or another part of the Waldsterben syndrome. The order in which they are presented here is arbitrary and not related to priority or weight of observational, experimental, or theoretical evidence.

The acidification-aluminum toxicity hypothesis. Bernard Ulrich and colleagues at the University of Göttingen developed this hypothesis and predicted a general decline of forest ecosystems in central Europe prior to the widespread development of Waldsterben (35). This hypothesis, based on Ulrich's long-term studies of nutrient cycling on the Solling plateau near Göttingen, holds that the natural acidification of forest soils (due to humus disintegration, nitrification, and greater uptake of cations than of anions from the soil) is accelerated as a direct or indirect result of deposition of acidic or acidifying substances from the atmosphere. Increased acidity in the soil leads to increased concentrations of soluble aluminum ions. Aluminum toxicity results in necrosis of fine roots, which leads to increased moisture and/or nutrient stress and eventually to "drying out" and death of the trees, particularly during drought periods. Ulrich believes that gradual acidification of soil is a predisposing factor and that periodic "acid pushes" are a primary inducing stress factor in the Waldsterben syndrome. We believe this hypothesis is relevant primarily to the growth-decreasing and water-stress symptoms listed in Table 2.

The ozone hypothesis. This hypothesis, advocated especially by Bernhard Prinz of the Landesanstalt für Immissionsschutz at Essen, is based mainly on field observations of foliar symptoms and measurements of ozone concentrations in various parts of West Germany as well as on controlled exposures of seedlings of various tree species. We believe this hypothesis is most relevant to the foliar loss and other growth-decreasing symptoms listed in Table 2.

The magnesium-deficiency hypothesis. This hypothesis is advocated especially by Karl Rehfuess of the Department of Soil Science at the University of Munich. According to this hypothesis, based

primarily on field observations of symptoms and both soil and foliar chemical analyses in high-elevation spruce stands, the yellowing of spruce foliage is a product of extreme magnesium deficiency in trees with plentiful supplies of nitrogen and calcium. Rehfuess (23) states that acid deposition "may contribute to these growth disturbances; it adds nitrogen to the ecosystem but may leach out magnesium and calcium from needles and soils. The leaching from foliage is presumably accelerated by episodic ozone or frost damage to cuticles and cell membranes." We believe this hypothesis is relevant almost exclusively to the yellowing of spruce foliage at high elevation.

The general-stress hypothesis. A group of botanists, plant pathologists, and physiologists in the Department of Forest Botany at the University of Munich base this hypothesis on field and laboratory observations of symptoms in various tree species, especially spruce and beech. The observations include changes in gas-exchange rates and formation of plant growth hormones and secondary metabolites during symptom development. According to this hypothesis, air pollution with associated atmospheric deposition of nutrient, growth-altering, or toxic substances has led in recent years to a decrease in net photosynthesis and associated diversion of photosynthate from mobile carbohydrates to less mobile and potentially toxic secondary metabolites. This in turn leads to a poorer energy status in roots and to accumulation of toxic substances in shoots, leading to poor development of fine roots and mycorrhizae and to foliar decline symptoms. Reduced energy status increases the susceptibility of the trees to other stress factors, such as drought, nutrient deficiency, and the usual secondary or contributing biotic pathogens. We believe this hypothesis is relevant to the growth-decreasing, abnormal-growth, and water-stress symptoms listed in Table 2.

The excess-nutrient or excess-nitrogen hypothesis. This hypothesis is based on a synthesis of widely scattered observations and theoretical considerations of how the nutrient status of forests can be related to recent changes in the chemical climate of industrial regions. Forest trees have always obtained an important part of their nutrients from the atmosphere. Sixteen elements are required; all are dispersed through the atmosphere and all can be taken up by the foliage as well as through the roots. During the preindustrial period, natural processes were the only sources of airborne nutrients. Since the mid-1800s, as the industrial revolution has gathered momentum, more and more substances have been added to those that circulate naturally among the air, water, and soil and to the organisms that inhabit the land and surface waters of the earth. Today, forests in many parts of central Europe receive much heavier loadings of airborne nutrients than were deposited during the preindustrial period. In certain locations, the atmosphere provides essentially all the nutrients needed to grow some forests.

Nitrogen is the element that most often limits the productivity of forests. Air emissions of nitrogen compounds have increased greatly in recent years, and we can reasonably postulate that a series of detrimental effects could be contributing to the present decline of forests in central Europe. Excess nitrogen is known or suggested to induce the following:

1. Increased growth and hence increased demand for all other essential nutrients, leading to deficiencies of these other elements, as suggested by Abrahamson (1);
2. Inhibition or necrosis of mycorrhizae, as demonstrated 36 years ago by Björkman (6) and in recent simulated rain experiments by Shafer et al (33);
3. Increased susceptibility to frost, as observed by Tisdale and Nelson (34) and suggested by Friedland et al (11), owing to delay in both cuticularization of epidermis and conversion of starch to sugars;
4. Increased susceptibility to root-disease fungi, such as *Pythium, Rhizoctonia*, and *Phytophthora*, as indicated by Huber (14);
5. Changes in root-shoot ratios, as shown by Aung (4); and
6. Altered patterns of nitrification, denitrification, and possibly nitrogen fixation, as suggested by Arthur Johnson (*personal communication*).

We believe this hypothesis is most relevant to the growth-decreasing and water-stress symptoms listed in Table 2.

Air transport of growth-altering organic substances. This is the most speculative of all current suggestions about possible causes of Waldsterben. During the German-U.S. Scientific Exchange on Forest Decline, Fritz Führ of the Institute for Radioagronomy at Julich encouraged our group to consider the possibility that among the thousands of synthetic organic compounds produced in central Europe, some might contribute to the symptoms of forest decline. We know of only a single documented case demonstrating the validity of Führ's speculation. In this case (9,10), loblolly pines in the vicinity of two chemical plants near Raleigh, North Carolina, were changed in growth habit and later killed. As with Waldsterben-affected spruce and beech (Figs. 10, 14, and 15), the affected pines showed changes in shape of leaves (twisted needles) and dropping of leaves by abscission while still green. Both observations suggest that the air pollutant(s) affecting the trees had altered the normal balance of growth regulators. Controlled exposures of susceptible seedlings to various volatile compounds produced by these two industrial plants confirmed that as little as 0.4 ppm of aniline in air for 1 hour could induce the twisted needle habit of growth and the dropping of green needles.

We know of no case in which regional (rather than local) air transport of growth-altering organic substances has been shown to affect plant growth. Nevertheless, we believe this general idea deserves further evaluation as a plausible explanation of the abnormal-growth symptoms listed in Table 2.

Our Concern and Our Hope

This paper and three German publications (28,30,31) describe the symptoms of Waldsterben in detail. Never before have so many different tree species growing under so many different soil, site, and climatic conditions shown so markedly similar and serious effects. Many of the observations we report here are scientifically unprecedented—the number of species affected, the geographic scale of the effects, the uncertainty about the probable cause or causes. Furthermore, the effects are of very great social and economic importance to the people of central Europe. Many scientists and the public at large are concerned that if the rate of intensification continues, the forest ecosystems of central Europe may not be sustainable. We share this concern and accept the idea that atmospheric deposition of nutrient, acidifying, toxic, or growth-altering chemicals may be involved. We hope this paper will attract to the Waldsterben problem some of the world's most able minds in plant pathology, plant physiology, soil science, forest ecology, and atmospheric chemistry. We hope you will add your intellectual resources to those already seeking improved understanding of one of the most remarkable forest disease problems of this century!

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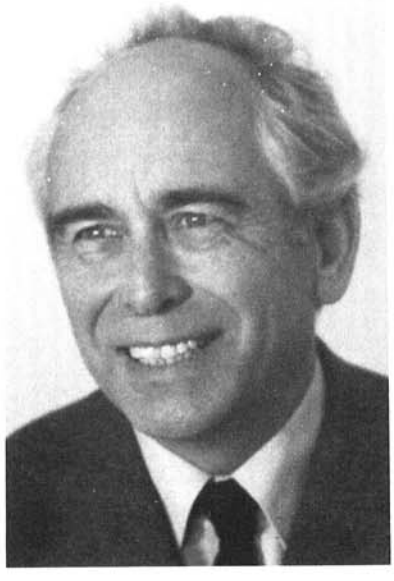

Peter Schütt

Dr. Schütt has served as professor in the forest faculty at the University of Munich and as editor of the *European Journal of Forest Pathology* since 1970. Waldsterben has been the principal object of his research since the inception of the disease in West Germany. He and his colleagues in the Department of Forest Botany and Pathology have published three books on the Waldsterben phenomenon: *So stirbt der Wald* (1983 and 1985) and *Der Wald stirbt an Stress* (1984).

Ellis B. Cowling

Dr. Cowling is associate dean for research in the School of Forest Resources and director of the Acid Deposition Program at North Carolina State University, Raleigh. He has been engaged in research on the ecological effects of airborne chemicals since 1971. He served as chairman of the National Atmospheric Deposition Program (NADP/NC-141/IR-7) from 1975 to 1983 and assisted in the development of the National Acid Precipitation Assessment Program (NAPAP) in the United States during 1978–1981.