Oak Decline in Central Europe: A Synopsis of Hypotheses

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ABSTRACT A brief overview is provided about the occurrence of oak decline in Central Europe during the past three decades, its development in space and time, the tree species affected, and the symptoms that have been recorded in different countries. Emphasis is placed on the critical discussion of several concepts and hypotheses about ‘forest decline’ in general and on ‘oak decline’ in particular, and on the importance of insects and fungi in the decline process. Excessive impairment of the water balance and metabolic disorders in oaks, attributed to climatic extremes, anthropogenic site deterioration, insect defoliation, and fungal infection, is frequently overcome by the trees, or results in mortality. The latter occurs either due to the impact of xylophagous insects and root pathogens, or by the physiological collapse of the tree. Despite the simplicity of this common scheme, the pathological processes involve numerous variations due to local site conditions and the locally involved stressors. Thus, ‘oak decline’ is considered a complex of different diseases, their ecosystem complexity requires better understanding.

A few oak species (Quercus robur, Q. petraea, and Q. cerris), being significant components of natural hardwood forests in Central European lowlands, represent an economically important basis of forestry in certain parts of Europe. In the past two decades, oak forests have been affected seriously by decline phenomena over wide areas of Central Europe. Foresters are extremely worried about the situation and seek predictive explanations from scientists who, in turn, are making efforts to understand the decline phenomena and their causes. However, the so-called oak decline turned out to be rich of facets. Therefore, scientific investigations resulted in distinct and diverging causal hypotheses, resulting in a confusing picture about the origin of pathologies observed in oak forests.

The general discussion of oak decline cannot be separated from the broader issue of forest decline and environmental perturbations such as air pollution and climate change. Additionally, pests and diseases appear to be involved in the dieback of oaks, though their importance in the causal chain is not thoroughly understood. However, as members of the ecosystem their impact on the health of oaks would also be influenced by the impact of external physical and chemical factors.

From the beginning, some questions stood in the forefront: Is the oak decline observed a novel phenomenon, or does it, at least, contain new pathogenic components? Can the oak decline syndrome in Central Europe be defined as a causally homogenous pathology? Can forest management be used to overcome the impact of oak decline and, if so, which techniques are most promising? Answers to these questions can be provided only after the pathological processes, which lead to disease symptoms and dieback, are sufficiently understood. A synoptic view of oak decline in Europe may be found in publications by Führer (1987), Hämmerli and Stadler (1989), Luisi et al. (1993), and Wulf and Kehr (1996).
addition, Schlag (1994) discusses oak decline from a phytopathological point of view. In this paper I have tried to summarize the essential facts and theories about oak decline and to elucidate the contributions of insects to this phenomena.

**Oak Decline in Space and Time**

Oak forests in eastern Europe were affected several times during the 20th century by regional episodes of dieback (Marcu 1966). In the most recent period of the phenomenon, the first reports originated from the same region: 1967 in western Russia, 1971 in Romania. The affected area spread to central and western Europe during the second half of the seventies and early eighties, when first observations were reported from most adjacent countries (Hämmerli and Stadler 1989). The temporal sequence of reports about oak decline suggests a geographic extension from the continental over the sub-Mediterranean zone to the zone of maritime climate. On the British Isles, oak decline was first reported in 1989 (Fig. 1). Igmandy (1987) reported that the decline of *Q. petraea* in Hungary began in 1978 in the colline northeast and extended within three years to the west of the country, finally reaching the eastern regions of Austria in 1984. This pattern of spread somehow resembled that of epidemic diseases and suggested that pathogenic agents could be involved in the phenomenon of oak decline.

![Figure 1. Years when recent oak decline phenomena were first recorded in individual European countries (Hämmerli and Stadler 1989).](image)

It is noteworthy that since the beginning of the recent period of oak decline (Fig. 1), oak forests in most countries have not fully recovered. Although extensive tree mortality was limited to rather small areas, the condition of oak forests has not yet stabilized. In the countries of Central Europe, where oak forests play a major role in forestry, the impact of
decline is considered even worse than that which occurs in coniferous forests (Rösel and Reuther 1995, Wulf and Kehr 1996). This conclusion is drawn from the results of crown transparency assessments, which revealed a continuous deterioration in oak, in contrast to conifer species, which demonstrated a recovery at least regionally (Huber 1992, 1994). It must be mentioned, however, that conifer forests (Picea abies, Pinus silvestris) in certain parts of Central Europe have been fatally affected by ‘conventional’ agents, i.e., windthrow and epidemics of well-known pest species such as bark beetles and defoliators (Führer 1996, Majunke et al. 1996, Wulf and Berendes 1996). Unfortunately, it appears that such facts are not clearly reflected by the forest health assessment procedures that are commonly utilized.

**Tree Species Affected**

Due to their dominant representation in Central European hardwood forests, *Quercus robur* and *Q. petraea* are the species most affected by the decline syndrome. These same species are reported to have suffered from dieback during former periods of oak decline in southeastern Europe (Marcu 1966). Leontovyc and Capek (1987) listed nine oak species indigenous to Slovakia, based on their relative susceptibility to the syndrome of decline. *Quercus robur*, *Q. petraea* along with *Q. polycarpa* and *Q. dalechampii* were ranked among the susceptible species. However, one must question the specificity of the indicators of susceptibility and the definition of the stress impact such that a comparison could be made among the different oak species.

When analyzing the causative mechanisms involving in the oak decline syndrome, the tree species concerned must be considered individually. Because of their ecophysiological specificity, each species is subjected to specific ecological limitations. This is expressed in the diverging distributional patterns and eco-geographical ranges of the individual oak species. *Quercus robur*, for example, is more susceptible to drought but less susceptible to winter cold than is *Q. petraea*. Therefore, *Q. robur* prefers humid soils in warm lowland sites and is widely distributed to the continental East, while *Q. petraea* prefers drier colline sites, but is not common in regions that experience severe winters (Mayer 1977). Thus, it is not surprising that, when both species are present in a region, ‘oak decline’ often affects only one or the other species (Landmann 1992, Ackermann and Hartmann 1992). Careful consideration of the ecophysiological specificity of individual tree species is a first important step towards gaining a better understanding of the oak decline phenomenon.

In contrast to the decline phenomenon in conifers, where the severity of symptoms usually is related to the advanced age of the trees, the syndrome in oaks has been recorded from all age classes, thus de-emphasizing the age-related trend of disease. This characteristic of oak decline is especially discouraging to foresters because it negates a potential solution to the problem, which would be to reduce the rotation period. From the pathologist’s perspective, the age-independent sensitivity of oaks could indicate either that conifers differ significantly in their pathophysiological responses, or that different causal factors are involved in oak and conifer decline phenomena. Innes (1993), after considering this and other peculiarities of the patterns of the oak decline syndrome suggests that basic differences exist between conifer and oak decline in Europe.
Symptomatology in Oak Decline

The list of symptoms reported in the literature in connection with oak decline is long and complex. Macrosymptoms include: crown transparency, yellowing, excessive twig abscission, dieback of branches and the whole crown, epicormic sprouts on branches and trunk. Less obvious are the following symptoms: undersized leaves, reduced shoot length, necrotic or dead foliage, dead twigs and buds, anomalous ramification, bark fissures and necroses, slime flux, reduced radial increment, sapwood discoloration, root necroses, damaged mychorrhiza, fungus infections in different organs, several kinds of insect infestation, nutrient imbalances, biochemical stress indications, etc.

Despite this long list, only a few selected symptoms are usually visible in individual cases, and these may differ from one site to another or from time to time, suggesting that different types of diseases may be involved. Most of the individual symptoms can be interpreted as being inter-connected, and may reflect sequential phases of physiological decline or recovery. They are, in part, unspecific manifestations of physiological stress acting upon the trees, or they may be directly or indirectly related to the impact of specific pest organisms. But uncertainty still remains about the origin of some of the symptoms. This is particularly the case concerning the allocation of individual symptoms to distinct positions in the causative chain of stressing influences: what is a primary effect, and what is secondary? This question also is relevant to our understanding of the causes of tree mortality.

Tree mortality is not a common feature of oak decline in Central Europe and information about mortality is incomplete because foresters usually cut the diseased trees before they die in order to prevent deterioration in timber quality. Hence, actual impacts on mortality may be greater than those recorded from monitoring sites. In controlled sites that were significantly affected by oak decline, tree mortality in peak years varied considerably: in Bavaria < 1% (Huber 1992, 1994), in Lower-Austria < 2% (Schopf and Mitterböck 1991), in Saxony ca 10% (Kontzog 1996), in Hungary > 22% (Igmandy 1987). These data are in contrast to the high rates of trees affected by oak decline, which frequently ranges from 70 to 90%. Thus, an apparently high proportion of affected trees recovers or at least survives in a weakened condition. The mortality of trees associated with the oak decline syndrome obviously depends on site specific conditions.

Hypotheses for the Causes of Oak Decline

Manion and Lachance (1992) edited a collection of essays that addressed concepts of forest decline. Although they did not directly focus on oak decline, their principles can be examined relative to their applicability to the oak decline syndrome in Central Europe. It can be demonstrated readily that most of the suggested concepts are partially appropriate to the phenomenon of oak decline, but none fully fit the diverse overall pattern of symptoms.

As a general assumption, the syndrome, although diverse in many details, is the result of synergies between sequentially or simultaneously acting influences on the trees. These influences can be classified as ‘predisposing’, ‘contributing’, and ‘inciting’ factors, respectively (Manion 1981). Careful examination is required to identify the real position of the individual influences in the causative constellation, however, and some factors could prove to be functionally unimportant.
A hypothesis for explanation of a *Q. petraea* dieback in Slovakia and Hungary was based on an epidemic concept analogous to the North American oak wilt disease (Leontovyc and Capek 1987, Igmandy 1987). A ‘tracheomycosis’, caused by fungi of the genus *Ceratocystis* and transmitted by insects (*Scolytus intricatus* Ratz., *Agrilus* spp. etc.), was considered to be the main causative factor responsible for tree mortality. This mechanism could neither be observed with European species of *Ceratocystis* in oak in other countries, nor did careful searches for a possible introduction of *C. fagacearum* reveal positive results. So far this ‘epidemic concept’ remained unconfirmed.

There is no clear evidence that air pollution is involved as the causative agent in oak decline. The role of air pollutants in the oak decline syndrome is frequently discussed. Excess nitrogen (agriculture-born ammonium) input and ozone impact is assumed to represent a risk potential causing predisposition for or synergism with natural stressors (Krapfenbauer 1987, Rösel and Reuther 1995, Hartmann 1996). Their relative significance in comparison with other stressors, though rated differently by different authors, is considered generally to be rather low.

Referring to the book of Manion and Lachance (1992), the ‘cohort senescence theory’ of Mueller-Dombois deserves consideration as a possible model to explain oak decline.

Several authors report that disease was more severe and mortality higher in trees that were over 60 years of age, however, they also emphasize that age classes below 60 years appear also to be affected (Wulf and Kehr 1969). This trend for predisposition of trees to be age-related can be regarded as a manifestation of senescence. Similarly, it has been suggested that coppice-derived trees suffer more from the syndrome than seedling-derived trees (Marcu 1987). This may correspond to the demographic aspect of predisposition and susceptibility as stated in the Mueller-Dombois theory, but it does not explain the anomaly of the present condition of oak forests.

Auclair et al. (1992) emphasize the importance of climatic perturbations in triggering forest dieback. Frost damage, which leads to xylem embolism, in combination with periods of drought, is regarded as the driving force leading to crown dieback in hardwoods. There is consensus among most European researchers that climatic factors are strongly involved in causing the oak decline syndrome. The occurrence of drought periods preceding the start of or enhancing the progress of the decline process is mentioned in many reports (Donaubauer 1987, Marcu 1987, Leontovyc and Capek 1987, Eisenhauer 1990, Gonschorrek 1995, Innes 1993). Extreme frost events that occurred after relatively warm winter periods were repeatedly recorded in the years preceding the manifestation of the oak decline syndrome, suggesting that cold damage has an inciting effect on the syndrome (Marcu and Tomiczek 1989, Rösel and Reuther 1995, Hartmann 1996). Even if the ‘climatic perturbation hypothesis’ seems to provide a plausible explanation of oak decline in Central Europe, many questions remain unanswered. Hartmann (1996) concludes that site condition and defoliation by insects are more important than climatic stresses, and, indeed, the role of organisms and of site is not considered sufficiently in the discussed hypothesis.

The ‘host-stress-saprogen model’ proposed by Houston (1992) requires that host trees are affected by stress factors, such as climatic perturbations (Auclair et al. 1992) or biotically caused defoliation in order for pathogens to successfully infect the root system. This model is based on the generally accepted theory of stress-induced predisposition of trees to pathogen/pest attack. The organisms considered as being mainly involved in the decline
process are ‘secondary’ agents that are situated near the end of the causative chain. In Houston’s examples, oaks seriously affected by the decline syndrome were often infected by species of *Armillaria*. However, the pathological role of these infections often remains dubious because the taxonomic identity of the species of *Armillaria*, which possess different pathogenicities, usually was not ascertained. In his study in Austria, Halmschlager (1997) mainly isolated the saprophytic *A. gallica*, but in certain cases only the pathogenic *A. ostoyae*, from the roots of diseased oaks. He concluded that in sites where *A. ostoyae* is present, this fungus might play a significant role in the physical decline of oaks. In Germany secondary root infections caused by certain species of *Phytophthora* are regarded to be more important than those caused by *Armillaria* (Hartmann 1996). In view of the fact that the potential of pathogenicity in the observed fungi is not completely clear, it seems justified to consider even the pathogenic species as ‘secondary’ rather than as among the ‘primary’ pathogenic agents, thus requiring a stress-born predisposition of the host tree. Under this assumption, the host-stress-saprogen model from the pathologist’s view could be partially, i.e., in certain cases, applicable to oak decline in Central Europe.

In connection with the host-stress-saprogen model the xylophagous insects also must be considered. In the literature, importance is attached to only a few species, which are well known to be associated with the stressed condition of the host tree and that cause lethal impairments of the tree’s conductive system. In Slovakia and Hungary, *Scolytus intricatus* Ratz. (Coleoptera, Scolytidae) was the dominant species (Patocka 1985, Szontagh 1987). Much attention was paid to this insect in connection with the ‘tracheomycosis model’, in which a vector-function was ascribed to the bark beetle. However, in other countries *S. intricatus* seemed to be irregularly associated with oak mortality, and occurred only occasionally. Large-scale inventories of symptoms in oaks affected by the decline syndrome in Austria revealed that *S. intricatus* was found infrequently, except in specific sites (Schopf and Mitterböck 1991, Schopf 1992). Representatives of the genus *Agrilus* (Coleoptera, Buprestidae) are mentioned in reports from most Central European countries. *Agrilus angustulus* Ill. was found to play a dominant role in the mortality of younger age classes of *Q. robur* that occurred locally in Hungary and Slovakia (Szontagh 1987). In Austria it was the dominant species among the Buprestidae that were found in diseased oaks, but without obvious significance in the causative chain of the decline syndrome (Schopf 1992). A completely different situation is described from Germany (Hartmann 1996), where *A. biguttatus* F. is considered to play a key role in oak dieback and mortality. Among the cambiophage insect groups, the Cerambycidae are inconspicuous. *Xylotrechus antilope* Schonh. is mentioned by Szontagh (1987) as being economically important in Hungary because it damages timber by excavating sites for pupation. In Austria, where this insect proved to be the dominant Cerambycid species found in dying oaks, the chronology of attack suggested that *X. antilope* was actively involved in tree mortality (Schopf 1992).

Wood boring insects usually occurred not earlier than in advanced phases of the dieback of whole trees or portions of them (Schopf 1992, Hartmann 1996). While these insects were mainly recorded from dead wood, their initial attacks oftentimes occurred when the trees were still alive. *Xyleborus monographus* F. (Col., Scolytidae) and *Xiphydria longicollis* Geoffr. (Hym., Xiphydriidae) are representatives of this group. Although their contribution to the physiological decline is rather improbable, the loss of value in wood caused by these insects is significant.
In summary, xylophagous insects proved to participate frequently in the final phase of the causative chain that leads to mortality of oaks. The involved species varied regionally or locally, probably according to the eco-geographic situation or individual site and stand condition. The incidence of their infestation may also depend on additional circumstances that contribute to the actual level of population density, such as the frequency of forest hygiene and control measures. Due to the ecological characteristics of the relevant species, it seems correct to classify them principally as ‘secondary’ pests, thus fitting in the host-stress-saprogen model, that is characteristic for certain species of fungi. Within limitations they seem to be able to substitute for one another, however, this does not mean that they are ecophysically and pathologically equivalent in their host tree - xylophage/parasite associations. Furthermore, there is no evidence that xylophages or phytopathogenic fungi of the types considered would be indispensable components that exhibit a killing function in the dieback phenomenon. The dominance of truly saprophytic/saprophagous species and the complete absence of potentially pathogenic species, can be interpreted as an indication that the dead tree had succumbed physiologically from abiotic influences. The ‘host-stress-saprogen’ model would not be applicable in such cases where biotic incitement of the pathological mechanism is lacking.

**Further Involvement of Biotic Agents**

When using this rough classification to describe the causative chain of oak dieback, many pests and pathogens behave as “primary” disease agents rather than as secondary agents. Defoliators and mildew fungi apparently act independently of the physiological condition of the host trees. By destroying ‘only’ foliage and buds, which are renewable organs of the tree, they are considered to be incapable of killing trees outright. Nevertheless, their outbreaks, which lead to a loss of photosynthetic capability, and induce an energy-consuming second flush of foliage in oaks, represent an important stressor, which severely predisposes trees to attack by other detrimental influences. This is the simplified, essential conclusion drawn from the reports in which defoliator outbreaks are thought to contribute to the oak decline phenomenon. Springfeeding species belonging to several families of Lepidoptera (Lymantriidae: *Lymantria dispar* L., *Euproctis chrysorrhoea* L.; Geometridae: *Operophthera brumata* L., *Erannis defoliaria* Cl., *Colotois pennaria* L.; Tortricidae: *Tortrix viridana* L., *Archips xylosteana* L., etc.) cause episodes of defoliation in all countries considered here (Marcu 1987, Szontagh 1987, Donaubauer 1987, Hartmann 1996). Sometimes their outbreaks take place temporally in alternate years or even simultaneously, this way causing more or less continuous periods of defoliation over consecutive years. Trees affected by repeated defoliation are highly susceptible to abiotic stressors such as drought and strong frost. Coincidence of spring defoliation with epidemic mildew (*Microsphaera alphioides* Griff. and Maubl.) infection of the regenerated foliage in late summer may be fatal for the trees (Lobinger and Skatulla 1996).

The statements concerning the role of defoliators need some qualifications. Although defoliation by insects is considered to be one of the most important components among predisposing stresses in Germany (Hartmann 1996), there is evidence that oak decline phenomena frequently occur in absence of severe insect defoliation (Donaubauer 1987, Schopf and Mitterböck 1991, Schopf 1992a). Marcu (1987), and Varga (1987) list insect
defoliation as only one of the possible factors responsible for the initiation of decline phenomena. Therefore, defoliation by insects should not be regarded generally as the initiating factor in oak decline. Another point deserves critical examination that is the chronology of insect outbreaks and other stress episodes in relation to when decline symptoms appear. Coincidence in time may provoke confusion in the interpretation of symptoms on the one hand; on the other, a site-related or climatically caused delay of recovery after defoliation may conceal a causative connection. Finally, there is the question, concerning how far insect defoliation and subsequent decline phenomena coincided locally over time. Small local outbreaks of defoliators are frequently overlooked. In the past, delineation of defoliated areas usually was not sufficient to reconstruct the differential pattern of infestation intensity in a region of interest. Similarly, the decline phenomena show locally differentiated patterns of intensity. Unfortunately, therefore, the availability of reliable data has not been adequate for us to reconstruct the pattern of these events on past epidemics.

Besides the xylophagous insects and the defoliators, a third group of rather inconspicuous pest insects is frequently found at high densities in oak stands associated with the decline syndrome: sapsuckers, gallmakers and shootminers. Varga (1987) mentions that serious infestation of *Kermes quercus* L. (Homoptera, Kermesidae) occurred in chronically devitalized stands of *Q. robur* in Hungary. From Austria, Schopf and Mitterböck (1991), Schopf (1992a), Gotsmy and Schopf (1992) mention *Asterolecanium variolosum* Ratz. (Homoptera, Asterolecaniidae) and *Mytilococcus ulmi* L. (Homoptera, Diaspididae), but in much higher densities, *Iassus lanio* L. (Homoptera, Cicadellidae) and *Harpocera thoracica* Fall. (Heteroptera, Miridae) are associated with oak stands that exhibit decline symptoms. In contrast to the Coccoidea, which affect the host tree only by their sap sucking habits, all of *I. lanio* and *H. thoracica* developmental stages feed on young tissues of buds, flowers and growing shoots and, additionally, injure the host plant by ovipositing into the base of young buds. In heavily infested trees, the density of egg-pockets of Hemiptera exceeded 2.2 per young shoot on average; the intensity of feeding damage on shoots and leaves was correspondingly high. These insects attained outbreak levels only in a portion of the sites investigated, and they appeared to prefer healthy oaks rather than obviously weakened trees. Another insect that attacks oaks and is easily overlooked is *Andricus quercusradicis* F. (Hymenoptera, Cynipidae), which builds its tiny galls inside the young shoots and in the bases of leaf stalks, thus causing the death of infested shoots Schopf and Mitterböck (1991). In contrast to the Hemipterans, *A. quercusradicis* was at outbreak densities in nearly all investigated sites, showing a slight preference for trees affected by the decline syndrome. In extreme cases, over 80% of the young shoots per tree were infested with galls. In the same study, authors report the occurrence of outbreaks of the oak shoot moth, *Stenolechia gemmella* L. (Lepidoptera, Gelechiidae) in certain sites in Austria. The larva, which mines the young shoots, causes considerable damage to the terminals of branches.

It is difficult to evaluate the importance of this complex of insects to the syndrome of oak decline. Our knowledge about the geographical distribution and dynamics of these species is poor because they are rather inconspicuous animals and are frequently overlooked. Very scarce records, with the exception of Austria, come from Hungary (Varga 1987 concerning *K. quercus*; Szontagh pers. comm. concerning *A. quercusradicis* after reinspection of diseased trees) and Slovakia (Patocka 1980 concerning *S. gemmella*). Possibly, the importance of these species was limited to the pannonian climatic region. Very little is known about the host-plant
relationships, population dynamics and epidemiology of these insects. Thus, it is difficult to judge whether they are involved in the initiation of tree decline or whether they benefit from it. The contemporaneous occurrence of outbreaks in numerous isolated sites suggests that the different species had directly or indirectly responded to a region-wide factor, probably weather. In their pathological function they clearly contribute to the disturbance of photosynthetic efficiency, and to the expression of visually recognizable symptoms, which include bushy shape of branches, necroses and malformation of leaves, and possibly pathological twig abscission (Schopf et al. 1991). It would be speculative to suggest that these species might be transmitters of hitherto undiscovered or functionally unidentified pathogens, such as viruses or MLOs (Nienhaus 1987, Ahrens and Seemüller 1994).

**Synopsis**

Recalling the fact that decline phenomena are the manifestation of ecosystemic processes, and that oak forest ecosystems in Central Europe are very diverse, we should not expect that the manifestation of oak decline should be uniform. The circumstances under which oak trees show symptoms of decline or dieback, or eventually die are likewise diverse. The genesis and course of pathological processes differ from case to case, regardless of whether the final result is recovery or death of trees. Although many different factors are considered to be involved in the causality, their negative effects seem to be based on a few ecophysiological disfunctions present in the trees. These problems can be triggered by abiotic influences or by organisms, or by both factors in combination. In summation, sets of impairing influences are acting on the trees, which are mainly interconnected to causative chains according to the predisposition-incitement principle. The diversity of stressor sets is derived from the variable combinations of static and dynamic environmental traits to which trees are exposed.

Static traits are represented by the properties that are characteristic of individual sites, which consist of mosaics of different edaphic, hydrologic, and nutritional conditions on both a small and large scale. Hartmann (1996) emphasized the importance of site conditions with respect to the effects of drought or extreme wetness. The type of climate can also be regarded as a static condition, characterized by the average amplitudes of climatic extremes, and determining the adaptedness of the oak population, the type and outbreak behavior of potential pest populations, etc. This pattern of static traits is understood to be a basic pattern of qualitatively and gradually different predisposing conditions that are being acted upon by a variety of dynamic stress factors. Under the influence of a differentiated pattern of dynamic stressors, this may produce a heterogenous pattern of tree stress and, consequently, a heterogenous pattern of tree response. The local configuration of predisposing conditions and the likelihood of interactions with stress-enhancing dynamic influences will collectively determine the local risk of damage. Dynamic variables include the episodic meteorological extremes, populations of potentially harmful organisms, and, even if not environmental, the changing vulnerability of the target, the oaks. Crucial to the pathological process is the spatial and temporal occurrence of ecophysiologically risky static and dynamic influences.

The basic physiological properties of the trees that seem to be affected adversely in every variant of oak decline syndromes, are the carbon budget and the water balance. There are numerous ways and mechanisms by which these functions can be reversibly or irreversibly
disturbed. Disturbance that results from water stress (alone) is usually reversible, whereas that caused by cambio-xylophagous insects is generally irreversible. Different factors and agents can cause uniform effects, and can operate mutually or self-enhancing on the progressively stressed tree.

The impact of stressors can directly or indirectly affect adversely the photosynthetic apparatus in the leaves and the water and nutrient absorbing apparatus in the roots. The impact on one (e.g., leaves) can cause negative effects on the other (e.g., fine roots), and is mediated by the intact conductive system in the trunk and branches. Critical conditions in the foliage and/or roots also can be provoked by functional disturbances of the conductive system. A simplified diagrammatic representation of their interactions is provided in Figure 2.

Figure 2. Oak decline: eco-physiological decline cycle, indicating essential effects of environmental factors on the tree, and showing the positions and contributions of defoliating and xylophagous insects, respectively, in the decline process.

As demonstrated in Figure 3, the carbon budget can be negatively affected by defoliation and, alternately, by water and/or nutrient-stress, which causes a reduction of photosynthesis, and which, in itself, can be induced by physical and chemical influences of the environment. Water and/or nutrient-stress also can arise due to the insufficient function of the root-mychorrhizae system, which is, in turn, a consequence of the affected carbon budget; therefore, this results in a self-enhancing, destructive cycle. Another feedback of water and/or nutrient-stress caused by disturbance of the carbon budget is a reduction of cold hardiness. Thus, under episodes of extreme cold, embolisms occur in the xylem, which adversely affect the ability of vessels to transport water (Tyree and Sperry 1989). The severity and duration of impact of the individual or combined influences determine the fate of the affected tree.
Survival and recovery are possible if the stress does not exceed a critical intensity and duration and, additionally, if the tree is spared the successful attack by aggressive xylophagous insects or root pathogens. Excessive stress impact results in the physiological collapse of the tree and finally in its demise. In this case the associations of xylophages and fungi will be characterized by the non-aggressive, saprophilic life habits of the participating species.

**STRESS FACTORS IN OAK DECLINE**

**EXOGENIC**

- drought
- cold
- defoliation
- mildew infection
- ozone
- nitrogen

- cold
- cambio-xylophagous organisms

- drought
- water-logging
- soil acidification
- nutrient imbalance
- pathogenic fungi

**SYSTEMIC**

- water stress
- nutrient imbalance
- reduced photosynthesis

- reduced frost hardiness
- embolism
- reversibly reduced conductivity
- reduced resistance to 'secondary' pests
- irreversible block of conductivity

- assimilate deficiency
- oxygen deficiency
- reduced pathogen resistance
- loss of functionality

**Figure 3.** List of exogenic and systemic stress factors relevant for the genesis of oak decline phenomena, arranged by their main points of attack in the tree.

Figure 3 indicates that very different exogenic and endogenic mechanisms that are functionally interchangeable can adversely affect the sensitive target ‘carbon budget’ of the tree. But, there is an additional point where environmental conditions influence these functional cycles, the root-mychorrhizal system, where vulnerability is very high. The normal function of the root-mychorrhizal system can be disrupted by deprivation of assimilates, or by the direct impact of abiotic stressors such as extreme drought or anaerobic conditions caused by excessive soil moisture. It has been suggested that advanced soil acidification causes damage to fine roots of oaks (Hartmann 1996). Root mortality under the influence of
continuous oxygen deficiency in heavy, water-saturated soils is reported frequently as a dieback of oaks (Varga 1987, Prpic and Raus 1987, Hartmann 1996). The coincidence of these soil conditions with defoliation prevents the rapid transpiration of surplus water, thus prolonging adverse conditions for the root systems. Weakened or damaged roots are more susceptible to aggressive pathogens that may be present (Halmschlager 1997). Infections by pathogens that are initiated in such situations, then cause persistent disruption in the function of the root system, increases the susceptibility of the tree to other stress agents.

The roles of pest organisms in the pathological pathway and the nature of their pathophysiological effects on the tree are relatively clear. However, the circumstances under which these organisms reach outbreak status are not well documented. The action of defoliators as well as that of the relevant ‘secondary’ organisms can be crucial at either the beginning or the end of the disease process. In contrast to stresses caused by meteorological factors, the origin of which is external to the ecosystem, pest organisms are basic components of the system itself. Their biological organization is different from that of green plants, and the temporal patterns of their life cycles differ considerably from that of their hosts, the oak trees. Due to their obvious and significant role in the oak decline syndrome, pest organisms and their dynamics deserve particular consideration within the context of ecosystemic disturbances (Führer 1997).

In order to clarify the role of defoliators in the context of oak decline, the following facts should be considered: The geographical distribution of the relevant defoliator species indicates that they are omnipresent in all regions where oak decline occurs in Central Europe. Nevertheless, and considering the large number of individual oak stands in their region, outbreaks of the different defoliator species and local populations show neither strong synchrony nor local or regional homogeneity. This cannot be solely because of differences in the coincidence of bud burst. The opinion that outbreaks of oak defoliators are only dependent on the synchrony between bud burst and egg hatch is a misjudgment of the situation with insects (Innes 1993). The highly diverse pattern of fluctuation caused by the occurrence, delay, extension or suppression of mass outbreaks, is the result of the successful regulation of insect abundance in multitrophic systems. In addition to the influence of environmental factors on the food-chain members, the host plant interaction among herbivores, the interspecific competition between herbivores, and the herbivore - natural enemy interactions, and the complicated structure of the natural enemy complexes themselves, are relevant variables in this regulation process (Führer 1997). Better knowledge of significant details would improve our ability to predict the spatial pattern of defoliator outbreaks, and in this way predict better the onset of oak decline.

As with the defoliators, the ‘secondary’ pest organisms, i.e., cambio-xylaphagous insects and root pathogens, demonstrate a high diversity of participation and local representation in oak stands affected by the decline syndrome (Szontagh 1987, Schopf 1992, Hartmann 1996). Although no basic limitations of geographical distribution seem to exclude them from individual oak forest sites in Central Europe, their outbreak behavior may be influenced by site conditions, which directly affect the pest species or indirectly provide favorable or unfavorable habitat for the pest population. Distribution and abundance of specific organisms depends also on the availability of suitable food substrate in the form of weakened of dying branches, trunks and roots. Thus, forest management practices on individual sites may strongly influence the populations of these ‘secondary’ pest species.
Another important consideration is the physiological potential of the individual cambioxylophage and pathogen species to successfully attack the host tree. The species/populations observed in connection with oak dieback no doubt exhibit considerable variation in their relative aggressiveness. This is a critical factor related to their significance in the causative chain of dieback because progressively stressed trees are considered to have gradually and qualitatively different susceptibility to attack by these organisms. In this context it appears noteworthy that the host tree selection by cambio- and xylophagous insects does not necessarily correspond with the categories of crown transparency. Schopf (1992) reports cases where trees that appeared to be healthy during summer did not survive until next spring because of severe infestation by ‘secondary’ pests, while other trees on the same site, which looked to be mortally ill in summer, remained uninfested and survived to the following year. We lack knowledge on the host tree relationships and population dynamics of cambioxylophagous insects. Therefore, we depend mainly on assumptions when we try to interpret the epidemic patterns that are observed and to evaluate the significance of individual species to the syndrome of oak decline. Because of their potentially crucial role in oak decline, it would be highly desirable if we could predict their patterns of abundance.

As previously stated, site conditions in many respects play a decisive role in the behavior of pest organisms. Hence, alterations of site conditions can influence the risk of injurious effects caused by abiotic and biotic agents. Characteristics of sites that are frequently exposed to anthropogenic alteration are hydrology, availability of nutrients, and soil acidity. Due to the outstanding significance of drought stress among the factors involved in oak decline, site hydrology was the subject of attention in relevant investigations. The prevailing opinion was that gradual manifestation of the oak decline syndrome often was related to site hydrology, which, under the influence of region-wide weather extremes, causes periods of excessive drought or excessive wetness. Such effects, which happen even in natural situations (Hartmann 1996), can be expected to occur even more frequently when manmade changes in the hydrology of oak forest sites may exceed the adaptive capacity of the existing oak stands. Changes in site hydrology, which increase the probability of both excessive drought and wetness, usually occur when human interventions into the landscape water regime cause either a drop of groundwater level (Hager et al. 1992), or the increased likelihood of long-term flooding of the sites (Prpic and Raus 1987). Increasing demands for water, attributed to changes in land use (river regulations, drainage of wet lands, irrigation of agricultural land, settlement of industrial centers, extensive urbanization, etc.) has taken place in all Central European countries during the past decades. Since forested land in Central Europe, particularly where oaks are dominant, is entangled spatially with human settlements, agricultural land and industries, significant adverse ecological effects may result from human-related activities. This is especially true in the case of water regimes, which may sustain alterations that are irreversible. Many authors such as Donaubauer (1987), Marcu (1987), Varga (1987), and Schume (1992) suggest that manmade changes to site hydrology contribute significantly to the oak decline phenomenon by increasing the predisposition of trees on these sites to climatically induced stresses.

Persistent deterioration of site hydrology (similar to permanent latent loads of photooxidants, nitrogen, etc.) probably exposes oaks to a permanent latent stress, even during periods of moderate climate. Considering the self-intensifying mechanisms of the stress response, and the additive or synergistic effects of accompanying stress factors, one should...
expect that such affected forests will become extremely sensitive to an episodic impact of an additional stressor. Because of this high predisposition to stress, the incitement of visible disease symptoms may occur in response to a very minor environmental event. Hartmann (1996), for example, regards the aggravating influence of episodes of severe late-winter frost during the period 1985-1987, on oak forests that had previously been stressed by defoliation, for the synchronized initiation of oak dieback in Germany. Initiation of the visible decline process caused by a similar mechanism may have occurred elsewhere and earlier, without preceding defoliation (Donaubauer 1987, Marcu and Tomiczek 1989). The effect of climatic perturbations, which doubtlessly are essentially involved in the past/present period of oak decline, are more significant now than in the past because oak forests are more predisposed to stress. This increased susceptibility has anthropogenic origin, which lies mainly, but not exclusively, outside the responsibility of forestry.

Concerning the causative climatic effects, oak decline phenomena cannot be explained by global climate change, nor can they be used as possible indicators of global climate change. In past centuries, the climate in Central Europe has changed periodically between more continental and more maritime phases. These changes were also reflected by changes in plant health and pest problems in both agriculture and forestry (Auer et al. 1994, Pschorn-Walcher 1954), which may provoke more easily the coincidence of different stress impacts during the continental phases. Hence, the oak decline syndrome seems to be related less to the hypothetical global change of climate, but much more to the declining health of oak forest ecosystems combined with the well-documented fluctuation of climate in Europe.

Conclusion

‘Oak decline’ is not a disease, but rather a process driven by several ‘diseases’ involving site factors, environmental factors, pests and pathogens. Due to differences in local conditions, the constellations of participating stressors, their individual weights and the duration of their impact can vary between sites, resulting in qualitatively and quantitatively different loads and patterns of stress. The local response of oak stands, i.e., the manifestation of disease, or so-called ‘oak decline syndrome’ is correspondingly variable and depends on which tree species are involved. In this respect ‘oak decline’ does not differ fundamentally from the general term ‘forest decline’ (Führer 1991). Its separation in time of occurrence from other recent ‘tree declines’ may be due to different spatial patterns and regional dynamics of climatic stress in Central Europe during the past three decades. After all, oaks are growing in regions that are climatically different to those where Norway spruce and white fir are found. Therefore one should not expect that the impact of climatic stress must occur simultaneously in both areas. In addition, differences in the susceptibility of tree species (conifers, hardwoods), and of their speed of response and recovery, also may have contributed to the chronological separation of ‘oak decline’.

In spite of the basic similarity with other ‘tree declines’, there seem to be striking differences in the dominant causative factors that are involved. While nutrient deficiencies and imbalances, combined with soil degradation and air pollution, play a major role in the initiation of decline phenomena of conifers, water stress and outbreaks of defoliators, combined with frost damage, seem to be the primary initiators of oak decline. In view of this divergent array of causative factors, the different pathological characteristics of tree diseases, and the different
nature of the hosts, we must first distinguish between diseases of conifers on one hand, and of oaks on the other. Focusing on oak decline phenomena only, the lack of uniformity in the array of factors and their pathological performance, does not justify that we consider oak decline as one ‘complex disease’ but as a ‘complex of diseases’ in the sense of Kandler (1992). The term ‘disease’ in this context implies the involvement of more than one causative agent, i.e., the combination of predisposing and inciting factors, thus describes better a ‘complex’ phenomenon.

Each variant of oak disease is based on impairment of the water balance and a metabolic disorder of the tree, particularly of the carbon budget. The ways in which this occurs differ from place to place, depending, in part, on the local site conditions. The nature, intensity and duration of the participating stress factors, together with the degree of predisposition, determines the dimension of the pathological response of the tree. The constellation of ‘primary’ stressors (climate, defoliators, pollutants etc.) and their stress-enhancing coincidence in time may be accidental, as is the occurrence of aggressive ‘secondary’ pest organisms. Here again the diversifying effects of site and stand properties must be emphasized. It should be noted that every involved causative complex follows its special laws: climatic extremes and man-made flooding of sites are neither inter-related nor related to the impact of pollutants; ecophysiology of trees exhibits endogenously induced patterns and dynamics that differ in part among tree species; outbreaks of defoliating insects are determined by specific rules of population dynamics, which differ from those of cambico-xylophagous insects and of root pathogens; additionally, dynamics of site conditions and oak stand quality are highly dependent on human actions. Hopefully, this enumeration has demonstrated that in order to interpret oak decline phenomena and to explain their genesis, many disciplines must be studied. Furthermore, in order to understand what is common and what is specific to the individual cases of oak disease, every group of causative factors must be given special consideration. This applies, in particular, to all further attempts to conduct research and apply forest practices to counteract the progression of oak dieback in Central Europe. The need for such attempts appears to be very urgent, because the state of the art is based much more on assumptions than on well founded scientific knowledge.

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