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Vascular Wilt Diseases

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Introduction

The vascular wilts include some of the most destructive of all tree diseases, in terms of both the scale of the damage and the speed of attack. Vascular wilt diseases mainly occur in angiosperms and only one example in a gymnosperm has been documented. True vascular wilts are caused by fungi, although there are some similar diseases caused by bacteria. Pine wilt disease caused by the pine wood nematode *Bursaphalenchus xylophilus* is covered elsewhere (*see* **Pathology**: Pine Wilt and the Pine Wood Nematode).

The Characteristics of Vascular Wilt Diseases

The fungi that cause vascular wilt diseases are initially restricted to the xylem elements. They can often achieve rapid dissemination within these elements through the passive movement of spores in the transpiration stream. Xylem anatomy can influence this process, with ring-porous genera, such as oaks and elms, that have large, long earlywood vessels which are particularly vulnerable to invasion. Vascular wilt pathogens often grow out from the xylem into the surrounding tissues after the tree has died. This enables them to establish contact with the outside world and, in particular, with insects that can act as vectors for transport to new hosts. Typically, vascular wilt pathogens do not have the capacity to survive long in the tissues of a dead host.

Generally, all vascular wilt diseases display similar symptoms. Leaves and young shoots on one or more branches suddenly wilt and die. If such a branch is cut, a marked discoloration can often be observed in the xylem of the current year. In a severe attack, symptoms can rapidly develop to kill the whole tree. Much still remains to be learnt about the process of pathogenesis. Vascular wilt fungi have the capacity to produce conidia in water-filled xylem vessels without causing cavitation (i.e., the breaking of the column of water). This can lead to rapid dissemination of the fungus throughout the tree in the transpiration stream. However, at some stage cavitation and an interruption to the water flow will occur. Gums, gels, and toxins may also be produced. In some cases the tree responds by producing balloon-like tyloses within the vessels by extrusion from adjacent parenchyma cells.

Ascomycetous fungi (and related mitosporic fungi) are responsible for most true vascular wilt diseases. Important examples are Dutch elm disease (*Ophiostoma ulmi* and *O. novo-ulmi*), oak wilt (*Ceratocystis fagacearum*), black stain root disease of conifers (*Leptographium wageneri*), and verticillium wilt (principally *Verticillium dahliae*). These diseases are discussed separately below.

Fungal Wilt Diseases

Dutch Elm Disease

Dutch elm disease has caused devastation to elms in Europe and North America. It is caused by two different, although closely related, species of fungi: *O. ulmi* and *O. novo-ulmi*. *Ophiostoma ulmi* was responsible for the first epidemic, which began in the

Netherlands (hence the name) in the late 1910s and quickly spread through much of Europe. In the late 1920s it was introduced to North America, where it proved to be particularly damaging on *Ulmus americana*. The second epidemic, caused by *O. novo-ulmi*, and proving to be very destructive in Europe, began in the late 1960s. It is now known to have developed from two centers, one in Eastern Europe and the other in the mid-west of North America. Two subspecies of *O. novo-ulmi* are associated with these two centers. Hundreds of millions of elms have been killed and huge sums spent in attempts at control and in the removal of dead trees. Most elm species are susceptible to the disease, with the American elm being especially vulnerable to both species of the fungus (**Figure 1**). Where there is some resistance, as in the Siberian elm, it is often found in trees that are lacking in aesthetic and silvicultural qualities.

The first symptoms of Dutch elm disease on a previously healthy tree typically take the form of the wilting or yellowing of the leaves on a vigorously growing branch in midsummer. Such a branch will show pronounced streaking in the vessels of the outermost xylem ring. The speed of subsequent symptom development down the branch and into the rest of the tree will depend, *inter alia*, on the species of host and pathogen involved. In some cases symptoms may remain relatively restricted in the first season, only to flare up throughout the crown early in the second season. Rapid general symptom development occurs if a tree becomes infected as a result of the transmission of the pathogen via grafted roots from an adjacent tree.

Various bark beetles act as vectors of the pathogen. The most important of these are *Hylurgopinus rufipes* and *Scolytus multistriatus* in North America and *S. scolytus* in Europe. (*see* **Entomology**: Bark Beetles). From the infection viewpoint, the key event is the establishment of the fungus within water-filled xylem vessels to which it has gained access by the feeding activities of the beetles. Early stages of colonization may be quite slow. However, once in the large, long, springwood vessels, spores of the fungus can be disseminated very rapidly under the influence of transpiration. They can also be drawn from a diseased tree to a neighboring healthy one if the trees have developed on a common root system or have grafted together (**Figure 2**).

Deleterious viruses may be present within the mycelium of *O. ulmi* and *O. novo-ulmi* and can lead to a reduction in pathogenicity. These viruses, known

Figure 1 English elm (*Ulmus procera*) killed by Dutch elm disease during the early 1970s. Photograph courtesy of J. Gibbs.

Figure 2 A hedgerow of English elm which has originated as sucker growth. Dutch elm disease is spreading rapidly through the common root system that links the trees. Photograph courtesy of J. Gibbs.

as d-factors, can spread from one unit of mycelium to another, provided that the latter is vegetatively compatible (i.e., belong to the same vc-group). Within *O. novo-ulmi,* one vc-group predominates, making it vulnerable to infection. However, hybridization between *O. novo-ulmi* and *O. ulmi,* once the former has moved into a new area, can lead to an exchange of vc-genes and hence to the formation of new vc-groups.

Management of Dutch elm disease requires effective removal and destruction of diseased or dead trees to reduce breeding material for the bark beetles. Disruption of root grafts between infected and healthy trees can restrict movement of the pathogen. In the case of trees of high value, fungicidal injections can be used. Breeding programs have resulted in the release of a number of resistant trees.

Oak Wilt

Oak wilt, caused by *C. fagacearum,* is a wilt disease known only in North America, where it mainly affects oaks (*Quercus* spp.) belonging to the red oak group. Currently the disease occurs in 21 states of the USA and has been recorded on 20 species of oak. *Ceratocystis fagacearum* is considered to be potentially as destructive as the fungi causing Dutch elm disease, but while they have effective vectors for dispersal, the oak wilt fungus currently lacks such vectors.

In red oaks such as *Q. rubra* and the northern pin oak (*Q. ellipsoidalis*), disease symptoms spread rapidly throughout the crown and trees can die within a few weeks. In contrast, white oaks such as *Q. alba* and bur oak (*Q. macrocarpa*) are substantially less susceptible. The disease progresses slowly with only a few branches being affected each year. In some cases trees may recover. In the live oaks of Texas (*Q. fusiformis* and *Q. virginiana*) the leaves become chlorotic or bronze, often with a yellow or brown color along the veins. Trees may defoliate and die quickly but commonly survive for several years (**Figure 3**).

Through experiments conducted in the USA, it has recently been discovered that the European white oaks *Q. robur* and *Q. petraea* are highly susceptible to *C. fagacearum*. This discovery, combined with the recognition that the European oak bark beetle, *S. intricatus*, has the characteristics to make it an effective vector for this fungus, has reinforced

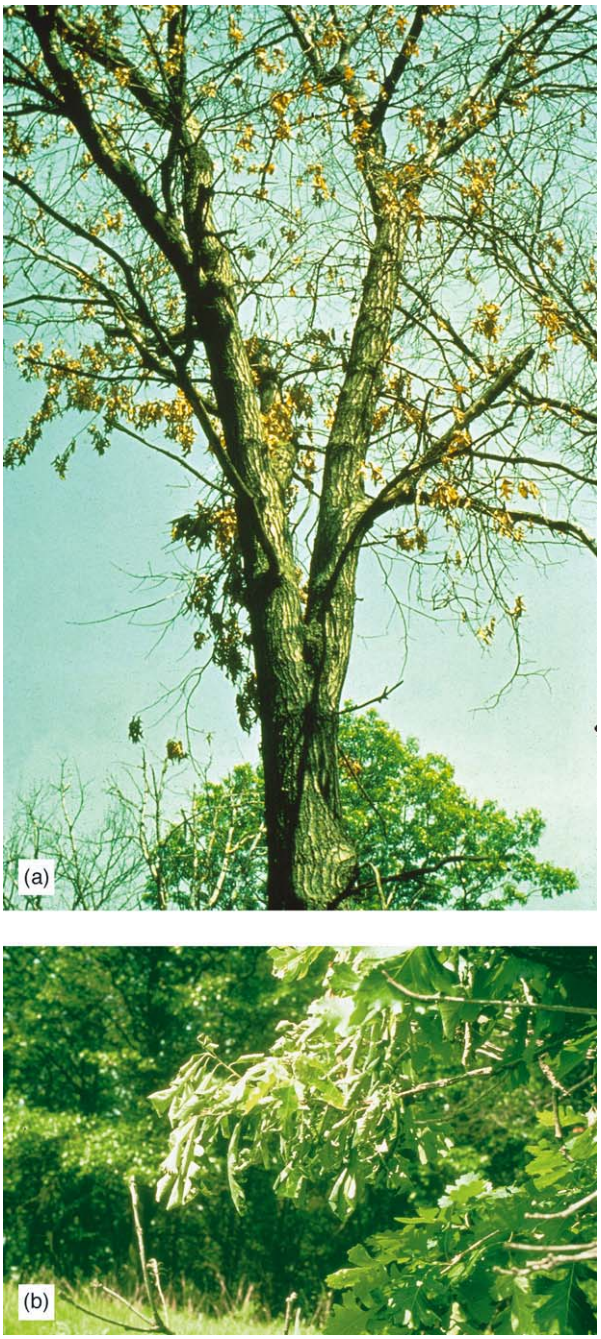

Figure 3 (a) A red oak (*Quercus ellipsoidalis*) killed by oak wilt. A healthy tree can be seen in the background. (Courtesy of JN Gibbs; in Forestry Commission Collection.) (b) A white oak showing symptoms of oak wilt on one branch while adjacent branches remain healthy. Courtesy of JN Gibbs; now in Forestry Commission Collection.

concerns that the fungus could be extremely destructive were it to cross the Atlantic. Strict quarantine regulations are in force.

Ceratocystis fagacearum moves rapidly through the xylem, as spores are drawn along in the transpiration stream. This process is aided by the fact that the fungus commonly enters a tree via root grafts with a neighboring tree that is already diseased. After tree death, *C. fagacearum* grows out of the xylem and forms mats of mycelium just below the bark. The bark splits open as a result of pressure exercised by a special structure, the pressure cushion, and volatiles produced by the fungus act as attractants for insects such as sap-feeding beetles (Coleoptera: Nitidulidae). The insects become contaminated with conidia and ascospores from the fungus mats and, if they move to fresh xylem wounds on healthy trees, are capable of transmitting the pathogen. Trees are most vulnerable in late spring and early summer shortly after the new earlywood (springwood) vessels have become functional. There are several weak points in this association between insect and fungus. For infection to take place, wounds on susceptible trees have to be less than 3 days old. Moreover, relatively few of the insects visiting the wounds are carrying spores of the fungus.

Oak wilt is best managed by preventing infection. This is achieved through removing diseased stems on which sporulating mats might form (or by treating them to stop mat production) and by taking steps to minimize the creation of wounds on valued trees in late spring/early summer. Root grafts can be disrupted to prevent tree-to-tree spread. As with Dutch elm disease, high-value trees can be injected with fungicides, although this is very costly and is restricted to very specific situations.

Black Stain Root Disease of Conifers

Black stain root disease is caused by *L. wageneri*. The disease is restricted to the western USA and parts of western Canada where it has caused significant losses, particularly to Douglas-fir (*Pseudotsuga menziesii*) and ponderosa pine (*Pinus ponderosa*). It is recognized to be of high quarantine significance as it is seen to pose a serious threat to both conifer plantations and indigenous forests elsewhere in the northern hemisphere.

External symptoms of the disease may develop gradually over several years. These include reduced height growth, reduced needle size, and premature needle fall. In the xylem of the roots and lower stems, a black stain can be found, typically in bands that follow the annual rings. Resinosis may be observed and the presence of the disease predisposes the tree to attack by bark beetles and other pathogens.

As in the case of other wilt pathogens, *L. wageneri* colonizes only the xylem elements of the host. Members of the Pinaceae do not have long vessels but only short tracheids, and hence rapid passive transport of spores is not a feature of this disease.

Rather, the fungus grows from tracheid to tracheid via the bordered pits. The dark mycelium of the fungus, together with a discoloration of the tracheids themselves, is principally responsible for the visible stain. In pines, the stained wood may be impregnated with resin and many tracheids may become occluded with tyloses.

Leptographium wageneri represents a species-complex including three different host specific variants, *L. wageneri* var. *wageneri* (occurring on pinyon pine), *L. wageneri* var. *pseudotsugae* (occurring on Douglas-fir), and *L. wageneri* var. *ponderosum* (occurring on jeffrey, lodgepole, and Ponderosa pines). Although all three varieties of *L. wageneri* can infect tree species other than those from which they were isolated, this rarely occurs in nature.

Black stain root disease spreads over relatively small distances through root grafts. Also, the fungus can grow for short distances (up to *c.* 15 cm) through the soil and infect healthy rootlets. For dissemination over longer distances, *L. wageneri* requires insect vectors, in particular bark beetles (Coleoptera: Scolytidae). It has been associated with two weevils *Pissodes fasciatus, Steremnius carinatus*, and the root-feeding bark beetles, *Hylastes nigrinus* and *H. macer*. These last species are thought to be the primary vectors on Douglas-fir and ponderosa pine respectively. These insects colonize declining roots of diseased trees and introduce the pathogen to the roots of healthy trees if attracted to them for maturation feeding or by the presence of preexisting wounds. Infection centers enlarge at the rate of about $1\,\mathrm{m\ year^{-1}}$ but in Douglas-fir stands they tend to slow down markedly when the trees reach 30–35 years of age.

The incidence of black stain root disease is increased by environmental factors. The disease is prevalent near roads or railroad tracks, where logging has occurred or trees have been thinned. Management treatments for disease-prone areas should be those that cause least site disturbance and tree injury. In affected stands, resistant species should be favored.

Verticillium Wilt

Verticillium wilt caused by *Verticillium albo-atrum* and *V. dahliae* is widespread in both temperate and tropical regions of the world. In the northern hemisphere, many woody plants are affected, all in the class of Dicotyledons. Gymnosperms and woody monocotyledons are not susceptible. The two fungi differ in a number of characters and, where proper attribution to species has been made, almost all the cases of damage to woody plants involved *V. dahliae*. Verticillium wilt is principally a problem in nurseries and ornamental plantings, such as those in gardens, parks, and streets, and it rarely occurs in forests and natural stands. It affects several important shade tree genera, including maple (*Acer*), lime (*Tilia*), and ash (*Fraxinus*), as well as some orchard trees such as cherry and apricot (*Prunus* spp.).

Symptoms of this disease can vary depending on the host and the environmental conditions. Most species display wilting and browning of the foliage first, while others may exhibit rapid chlorosis and necrosis of the foliage. In some cases, symptoms develop rapidly to tree death, while in others they are chronic and include slow growth, sparse and distorted foliage, stunted twigs, and dieback. In some species, examination reveals marked xylem staining that varies in color with the host, e.g., in maples it is typically green (**Figure 4**). In others, such as ash, no discoloration can be seen. Most fatalities occur in nursery stock and small trees. In mature trees, chronic infection is more usual, with symptoms

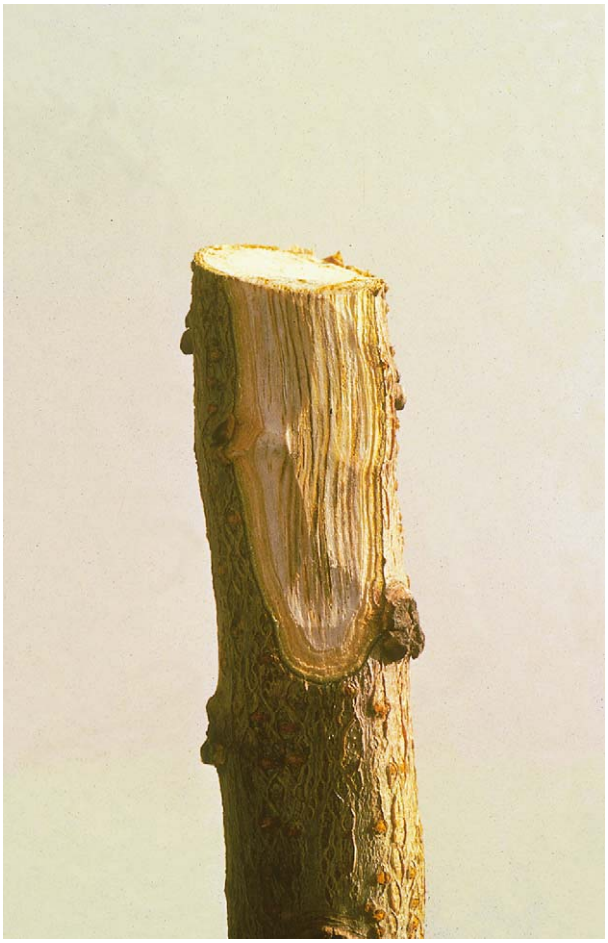

Figure 4 Staining caused by *Verticillium dahliae* in the xylem of a small stem of the Indian rain tree (*Koelreuteria paniculata*). Photograph courtesy of J. Gibbs.

affecting just part of the crown, and recovery the following year being quite common.

Verticillium dahliae differs from the vascular wilt fungi described above in having considerable capacity to survive in the absence of its host. This survival is in the form of dark microsclerotia, which can persist in the soil for up to 10 years. Most information on the infection process comes from studies of herbaceous hosts. Hyphae from germinating microsclerotia invade via root tips and root wounds. In plants not immune to vascular infection, the fungus grows into the xylem where conidia are produced and disseminated into the stem. In the past, fungal toxins affecting leaf function were thought to be key to the development of symptoms but current views emphasize the importance of vascular disruption. Effective host resistance mechanisms are thought to depend on processes such as the rapid production of gels in the vessels to trap and immobilize the conidia, followed by the production of toxic secondary metabolites.

There is little evidence for host specialization in *V. dahliae* and some of the most dramatic disease outbreaks have occurred when tree nurseries have been established on old fields of susceptible species like potato or cotton. Such sites should not be used unless there is the option of carrying out soil fumigation. Once the disease is present in a tree, there may be some scope for promoting natural recovery by improving the growing conditions, although heavy irrigation and nitrogen fertilization should be avoided.

Other Fungal Vascular Wilt Diseases

Strains of *Fusarium oxysporum* cause important vascular wilt diseases on herbaceous and woody plants, particularly in warm temperate and tropical regions. A tree example is provided by *F. oxysporum* f. sp. *pernicosum* that has become a limiting factor in the use of mimosa (*Albizzia procera*) as an ornamental in parts of the USA. The fungus is soilborne, germinating from chlamydospores in the presence of host plants to penetrate both wounded and unwounded rootlets and then to colonize the xylem. Another race of the same fungus is known from *A. procera* in Puerto Rico. *Fusarium oxysporum* has also been implicated in the death of *Acacia koa* in Hawaii. *Ceratocystis albofundus* is the causal agent of Ceratocystis wilt on black wattle in South Africa and other parts of Africa. In the Seychelles, takamaka disease, caused by *Leptographium calophylli*, is a serious problem on takamaka (*Calophyllum inophyllum* var. *takamaka*), a broad-leaved evergreen tree that plays an important role in littoral habitats around the Indian Ocean. The bark beetle *Cryphalus trypanus* has been shown to act as a vector.

Bacterial Wilts

There are a number of wilt diseases of trees caused by bacteria in which the causal organism is restricted to the tissues of the xylem during pathogenesis, if not to the xylem elements. Most notable are bacterial wilt of willow and bacterial wilt of *Eucalyptus*.

Watermark Disease of Willow

This disease was first recorded in eastern England during the 1920s on *Salix alba* var. *caerulea* grown for the manufacture of cricket bats. In the 1930s it was recorded in the Netherlands but it is not known from any other European country. In 1993 it was found affecting several species of willow (*S. bakko*, *S. sachalensis*, and *S. kinuyanagi*) in natural forests in a mountainous part of Hokkaido, Japan. The symptoms involve the wilting of young leaves and shoots in early summer. The foliage turns a reddish-brown and the dead leaves are typically retained on the tree. Cross-sections of the stems of affected shoots show a marked brownish 'watermarking' of the xylem. In large branches and trunks this normally follows the position of one or more annual rings. On exposure to air, the discolored tissues darken and a brownish-black liquid may be exuded (**Figure 5**).

The causal bacterium is a Gram-negative nonsporing rod with peritrichous flagella. It falls within the *Erwinia amylovora* group and has been named *E. salicis*. Microscopic examination of the stained tissues of a diseased tree reveals masses of bacteria in some of the vessels, often in association with tyloses. Ray parenchyma cells show plasmolysis and become necrotic, and bacteria can also be seen in these.

Large numbers of bacteria can ooze from affected branches and it used to be thought that infection resulted from the dissemination of these bacteria by wind and rain to injured tissues on healthy trees. However, serological studies in the UK have shown that the bacterium can be detected in the wood of many healthy trees, and also in the coppice stools that provide cuttings for willow propagation. Isolates from adjacent diseased trees often fall into different groups on the basis of electrotyping and this supports the idea that the bacterium is disseminated within the cutting material in a latent or endophytic state, only developing to cause disease under the influence of some as yet unknown environmental factors.

In the UK a sanitation felling program for symptomatic willows is conducted in commercial

Figure 5 Cricket-bat willow (*Salix alba*) felled because of watermark disease. Staining due to the disease can be seen in the xylem of both trunk and stump. Photograph courtesy of J. Gibbs.

cricket-bat willow plantations. Although there was an initial fall in disease incidence following the introduction of this program in the 1930s, there has been little subsequent evidence for a further reduction in disease. In the Netherlands, serious losses in the 1960s can be attributed to the widespread amenity planting of a number of very susceptible clones of *S. alba*.

Bacterial Wilt of *Eucalyptus*

The bacterium *Ralstonia solanacearum* has been reported to cause bacterial wilt in commercial *Eucalyptus* plantations. This disease was first described in Brazil and later in China, Taiwan, Australia, Venezuela, and South Africa. *Ralstonia solanacearum* is divided in different biovars based on their nutritional requirements and in different races based on their host ranges. Biovars 1 and 3 are able to infect *Eucalyptus* and race 1 has been recorded from all the areas where the disease occurs on *Eucalyptus*.

Symptoms of bacterial wilt on *Eucalyptus* include wilting, leaf drop, reduced growth, discoloration of the vascular system, and death of stems. Infected trees may die within 6 months of showing the first signs of infection. Bacterial exudation can also be seen from the cut surfaces of the stems. Fortunately, only some *Eucalyptus* species are susceptible to this disease and it can be managed by planting resistant species or hybrid close.

See also: **Pathology**: Diseases of Forest Trees; Insect Associated Tree Diseases; Pine Wilt and the Pine Wood Nematode. **Tree Breeding, Practices**: Breeding for Disease and Insect Resistance.

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