WILT DISEASES

To wilt means to lose freshness or to become flaccid. Wilting in plants may be temporary, due to too rapid transpiration; or it may be permanent, due to continued loss of water beyond the recovery point. Disease organisms, by reducing or inhibiting water conduction, may cause permanent wilting.
Because wilt diseases are systemic, and tied up with the entire vascular system of a plant, they are usually more important, and harder to control, than localized spots or cankers. In many cases the fungus enters the plant from the soil through wounds or root hairs and cannot be controlled by protective spraying. Often, although the fungus is present only near the base of a plant, the first symptom is a flagging or wilting or yellowing of a branch near the top. Many species of *Fusarium* are responsible for important wilts and "yellows." *Verticillium* is a common cause of wilt in maples, other trees, and shrubs, but most important among the wilt pathogens are two species of *Ceratocystis*, one causing oak wilt, the other Dutch elm disease.

Acremonium (Cephalosporium)

► Leaf Spots.

Acremonium diospyri (formerly *Cephalosporium diospyri*). **Persimmon Wilt**, a lethal disease of common persimmon. Wilt appears in scattered localities from North Carolina to Florida and west to Oklahoma and Texas, but most infection is in north central Florida and central Tennessee. Spread is rapid and death quick. First notice of the disease was in Tennessee in 1933. By 1938 only 5% of the persimmons in the infected stand were alive. Topmost branches wilt suddenly, then the rest of the tree, with defoliation and death. The fungus fruits in salmon-colored spore masses in cracks in dead bark of dying trees or under bark of dead rings. Fine, blackish streaks are present in five or six outer rings of trunk, branches, and roots. No control is known.

Cephalosporium diospyri (see *Acremonium diospyri*). **Persimmon Wilt**, a lethal disease of common persimmon.
Cephalosporium sp. **Sunflower wilt**.

Ophiostoma (Ceratocystis)

Ascomycetes, Ophiostomatales

Perithecia enlarged at base, with thin walls, and long slender neck, ascus wall evanescent, ascospores hyaline. Conidial stage may be *Chalara* with endogeneous spores or *Graphium* with external conidia or conidiophores united into a dark stalk (synnema).

Ceratocystis fagacearum (*Chalara quercina, Endoconidiophora fagacearum*). **Oak Wilt**, our most serious disease of oaks, now known in 20 states from Texas and Oklahoma east to Pennsylvania and South Carolina. It has also been reported in Florida. Although apparently present in the Upper Mississippi Valley for many years, the disease did not cause concern, and the fungus was not described until 1943, since when it has become a major threat to our forest economy and to trees in residential areas. All native oak species are susceptible, also chinquapin, chestnut, lithocarpus (and apples in experimental inoculation); but red oaks succumb most rapidly. Scouting for the disease has been done largely by airplane, the discolored foliage being visible up to a half mile.
First symptoms are a slight crinkling and paling of leaves, followed by progressive wilting, bronzing, and browning of leaf blades from margins toward midribs and defoliation progressively downward and inward throughout the tree. Red oaks almost never recover and may be killed within 4 to 8 weeks after symptoms appear. White and burr oaks may persist for some years, with affected branches dying in a staghead effect.
The first internal symptoms are the formation of gums and tyloses in the xylem. After wilting, mycelial mats are formed between the bark and wood, and the bark cracks from the pressure exerted. Perithecia are formed in these mats, which have a sour odor and attract insects. Nitulid beetles, fruit flies, brentids, springtails, bark beetles, and possibly other insects get conidia and ascospores on or in their bodies as they feed, and can inoculate other trees through wounds. We know that ascospores remain viable several months on insects and can be distributed through fecal pellets, but we do not yet know how great a role they play in the spread of oak wilt. Birds have been suspected as carriers but are not yet indicted. Local spread is largely by root grafts,

one tree infecting others within 50 feet and with grafts possible between red and white oaks, not limited to the same species.

Control. In residential areas infected trees should be removed. In forests, felling may wound other trees and spread the disease more than letting the dead tree remain but treated so that it is not infective. Different states handle the problem in different ways. In Pennsylvania, each infected tree is cut, with all other oaks within 50 feet, and ammate crystals are placed on each stump. In North Carolina stumps and felled trees are thoroughly sprayed. In West Virginia the trees are left standing, but a deep girdle into the heartwood dries out the tree so that mycelial mats and spores do not form.

Ceratocystis (Ceratostomella) ulmi (**Graphium ulmi**) (see *Ophiostoma ulmi* and *O. novo-ulmi*). **Dutch Elm Disease**, on American, Sibirian, Slippery and European elms in 31 states, Maine to North Carolina and west to Oklahoma, and on cedar.

Ophiostoma ulmi and **O. novo-ulmi** (formerly *Ceratocystis* (*Ceratostomella*) *ulmi* (*Graphium ulmi*)). **Dutch Elm Disease**, on American, Sibirian, Slippery and European elms in 31 states, Maine to North Carolina and west to Oklahoma, and on cedar. This fatal disease is not really of Dutch origin but is so named because it was first investigated in Holland. It was noticed in Europe about 1918, first in France, then in Belgium and Holland. It spread throughout central and southern Europe, then into England and Wales. In many places it virtually exterminated the elms, including those on the famous avenues at Versailles. It is suspected that the fungus came to Europe from Asia during World War I.

Dutch elm disease was discovered in Ohio in 1930 and in New Jersey in 1933. It has spread north through New England and has become very serious in the Midwest. In 1948, the disease was found in Denver, Colorado, and in 1976 in California. It is now fairly widespread in reports of its occurrence in the United States. The spread of the fungus is linked with the presence of the large and small European bark beetles, *Scolytus scolytus* and *S. multistriatus*. Only the latter is established in this country, having arrived in Boston about 1919. Patient detective work established the fact that the fungus came here in elm burl logs imported for furniture veneer. After one such infected elm burl was found in Baltimore in 1934, months of scouting went on in the vicinity of ports of entry, railroad distributing yards, and veneer plants. Such backtracking showed the infected material had come in at four ports of entry and had been carried by 16 railroads over 13,000 miles in 21 states. From this source the disease got its start in at least 13 areas in 7 states.

Elm nursery stock is, of course, quarantined, and elm burls are embargoed; but who who would have believed that dishes could have anything to do with killing our elms? Dishes have to be crated, however, and several times since 1933 English dishes crated with elm wood carrying bark beetles and *Ceratocystis* have been intercepted. All American and European elms are susceptible. Asiatic elms, *Ulmus parvifolia* and *U. pumila*, are resistant. A seedling elm, named Christine Buisman for its Netherlands' discoverer, is highly resistant, though not immune, and is now available. Other promising seedlings have been tested by the U.S. Department of Agriculture.

Symptoms are apparent from the latter part of May until late fall. The acute form of the disease is characterized by sudden and severe wilting. First the young leaves, then all leaves wilt and wither, sometimes so rapidly that they dry, curl, and fall while still green, before they can turn the usual brown of dead leaves. Sometimes terminal twigs are curled into a shepherd's crook. Chronic disease symptoms are gradual, often taking all summer for complete defoliation. In many cases individual branches or "flags" appear, the yellowed leaves conspicuous against the rest of the tree; but sometimes all leaves gradually turn yellow. In another type of chronic disease, trees leaf out late in spring, with sparse chlorotic foliage and a staghead appearance.

When an affected twig is cut across, the vessels or water-conducting tubes show dark brown or black, being clogged with bladderlike tyloses and brown gummy substances (see Fig. 3.62). The production of these substances is thought to be stimulated by a toxin secreted by the fungus and carried in the sapstream. Symptoms are not dependent on the physical presence of fungal hyphae in all parts of the tree. The fungus lives in the sapwood, fruiting in cracks between wood and loosened bark and in bark beetle galleries under the bark. This fruiting is of the anamorph state, spores being produced in structures called coremia. These are black stalks about 1 mm high with enlarged heads bearing vast numbers of minute, pear-shaped spores embedded in a translucent drop of sticky liquid. Spores in the vessels increase in a yeastlike manner. The perithecial stage, not found in nature, has been produced in culture by crossing plus and minus strains of the fungus.

Although the smaller European elm bark beetle is chiefly responsible for spread of the pathogen, at times the native elm bark beetle, *Hylurgopinus rufipes*, is the agent. When the adult beetles emerge from under the bark of dead or dying trees, they bring along sticky spores on their bodies and deposit them as they feed in the crotches of young twigs or leaf axils of near-

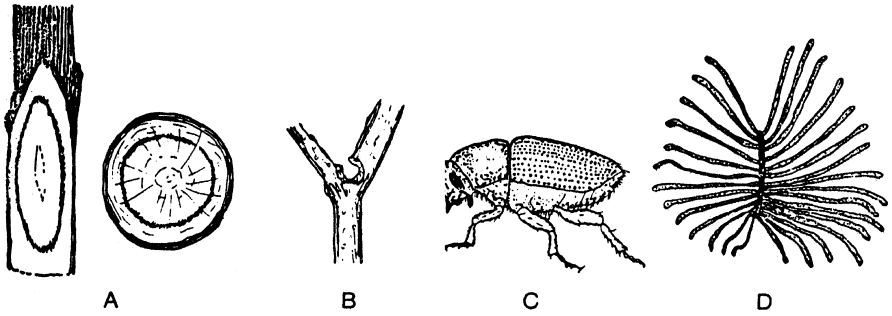

Figure 3.62 Dutch Elm Disease. **A** branch cut to show discoloration of wood; **B** wound in twig crotch due to beetle feeding; **C** bark-beetle carrier of the fungus; **D** egg and larval galleries of the beetle engraved on sapwood

by healthy trees. Although the beetles feed on healthy wood, usually within 200 feet of their original tree, they breed only on weakened or dying wood and may fly some distance for it. The European female tunnels out a brood gallery 1 or 2 inches long in the wood, and when the larvae hatch, they tunnel at right angles across the wood (Fig. 3.62). There is a second brood in August and September, but the overwintering one, emerging in May, is most to be feared. Because the disease often follows traffic routes, automobiles probably account for a good deal of long-distance spread. So far as we know, the only other natural means of infection is by root grafts, made when trees are planted so close together that their roots touch. This is another argument for diversified planting, rather than streets closely lined with but one type of tree.

Control. In the first few frantic years an enormous amount of money (more than $26 million) was spent on trying to eradicate the disease by removing and burning diseased trees; and while this was undoubtedly helpful, it did not stop the spread of wilt. The Federal government has now left the control of Dutch elm disease up to the communities and is restricting its efforts to research. Many towns have taken a laissez-faire attitude, thinking that our elms are doomed anyway, so why waste money? Other, more enlightened communities have proved that a sustained control program keeps the disease down to a negligible 1 or 2%, or less, and that the cost is far, far less than that of continuous removal of dead trees.

The Midwestern Chapter of the National Shade Tree Conference, in its *Guide for Community-Wide Control of Dutch Elm Disease*, suggests:

1. Survey of the total elm tree population to be protected.

2. Symptom scouting for detection of diseased trees and sanitation scouting for badly weakened elms and wood piles containing elm wood.
3. Destruction of known sources of elm wood actually or potentially hazardous for spread of disease. Elm wood piles should be destroyed completely, or each log stripped of bark and the bark destroyed. Diseased trees should be burned, on site if possible, or thoroughly sprayed. Wood chips from diseased elms may still carry the fungus; material should be burned, not used for mulches.
4. Spraying of healthy trees to prevent infection.
5. Maintenance of elms in healthy condition to prevent invasions of bark beetles. This includes proper watering and fertilizing, spraying to control summer foliage pests if necessary.

A single annual DORMANT SPRAY is now considered sufficient to protect healthy elms from bark beetles if enough material is used and complete coverage is obtained. This spray was originally a very heavy dosage of DDT, which caused some bird mortality and other environmental problems. Some communities, of which Greenwich, Connecticut is a good example, figure that they cannot afford not to spray, for it costs less to spray for control than to remove a dead tree. Where dormant spraying and sanitation have been combined consistently, the annual loss from Dutch elm disease has been kept to 1% or less.

Chemotherapy, injection of chemicals that will inactivate the fungus, has been a promising line of research for many years. A parasitic European wasp is now being bred at several laboratories for release against the bark beetles. To have elms in our future we must keep on planting them. Some forms, such as the Christine Buisman and Groeneveld elms, are quite resistant although not immune. Chinese and Siberian elms are resistant.

Dothiorella

►Cankers.

Dothiorella ulmi. **Dothiorella or Cephalosporium Wilt** of elms. **Dieback**, rather common on American elms, occasional on slippery and Siberian elms in central and eastern states. The names are confusing. In culture the fungus develops spores as in *Cephalosporium*, but in nature *Dothiorelia*-type pycnidia are developed on bark of killed twigs. The fungus has also

been classified as *Deuterophoma*. Spores are extruded in a sticky mass and are disseminated by wind, rain, possibly insects. Infection is through insect or other wounds on foliage. The mycelium proceeds from leaf petioles into wood, where it is confined to the vessels. The foliage wilts and yellows; there are gradual dying back of the crown and a brownish discoloration in outer rings of the wood. Without laboratory diagnosis the disease cannot be positively separated from Dutch elm disease, but the elliptical cankers on the stems, with small black specks of pycnidia, provide one diagnostic symptom. Older trees die 3 to several years after first symptoms; nursery trees, in 1 or 2 years. Some trees recover, and some remain infected for many years without showing much effect.

Control. Prune out infected branches a foot or more below the lowest point of discoloration. Promote vigor by feeding, watering, aerating soil. The inclusion of a fungicide in sprays for elm-leaf beetles or cankerworms might be helpful.

Fusarium

► Rots.

Fusarium annuum (*F. solani*). **Fusarium Wilt** of chili pepper. Underground stems are dry, brown, but the roots soft and water-soaked; plants wilt and die rapidly. Spores are spread in irrigation water and with wind-blown particles of soil. Avoid heavy, poorly drained soils.

Fusarium foetens. **Wilt** on begonia.

Fusarium oxysporum. **Wilt** on pyracantha and basil. **Blight and Wilt** on purple coneflower (*Echinacea*).

Fusarium oxysporum f. sp. **apii**. **Celery Wilt**, **Yellows**, general in northern celery districts. There are three strains of the fungus, all causing stunting, vascular discoloration, crown and root rot, but one form causes the entire plant to turn yellow at high temperatures, producing brittle stalks with a bitter taste. Another strain causes downward curling of young heart leaves, and the third produces no above-ground symptoms except stunting. The fungus persists indefinitely in soil. Golden, self-blanching varieties are more susceptible. Grow green petiole celery or somewhat resistant Michigan Golden, Cornell 19, Tall Golden Plume, Golden Pascal or Emerson Pascal.

Fusarium oxysporum f. sp. **asparagi**. **Fusarium Wilt** of asparagus, a major factor in asparagus decline in California, found in most plantings. The fungus lives in soil and may be distributed on seed.

Fusarium oxysporum f. sp. **barbati**. **Fusarium Wilt** of sweet william. New growth is yellowed; plants are stunted; leaves point downward and are tinged with tan as they die. Roots and lower stem are discolored brown. Plant in new or sterilized soil.

Fusarium oxysporum f. sp. **batatas**. ►Rots.

Fusarium oxysporum f. sp. **betae**. **Fusarium Yellows** on sugar beet.

Fusarium oxysporum f. sp. **callistephi**. **Aster Wilt**, one of the most serious diseases of China aster, unless resistant seed is used. Plants wilt, wither, and die at any age from seedlings to full bloom. Older plants are often stunted, with a one-sided development and a brown discoloration of the vascular system. Sometimes all lower leaves are wilted, with blackening at base of stem, often with a pink spore mass at ground level. Plants in full bloom may suddenly droop their heads. Such symptoms are in contrast to the mycoplasma-like disease, aster yellows, where the plant remains upright, although stunted and yellow. The fungus is seed-borne and persists in the soil many years.

Control. Sterilize soil for seedbeds. Some seedsmen provide seed of wilt-resistant varieties, but maintaining resistance means continuous selection from asters grown on heavily infested soil under conditions highly favorable for infection, and this is an expensive process.

Fusarium oxysporum f. sp. **cattleyae**. **Wilt** of cattleya orchids. The fungus was isolated from a private collection in Ohio. Leaves wilted, roots abscised and decayed; flowers fewer, smaller, short-lived.

Fusarium oxysporum f. sp. **chrysanthemi**. **Fusarium Wilt** on chrysanthemums.

Fusarium oxysporum f. sp. **conglutinans**. **Cabbage Yellows**, **Fusarium Wilt**, general on cabbage and other crucifiers, probably the most destructive disease of such hosts in the Midwest, perhaps other sections. It is serious on cabbage, kohlrabi, and collards. Brussels sprouts, cauliflower and broccoli are moderately susceptible in hot dry seasons. The fungus, which can live many years in the soil, enters through the roots, usually right after transplanting or at the first hot weather, with potassium deficiency as well as heat thought to favor infection. The fungus progresses upward in the xylem, not invading other elements until the plant dies.

The most striking symptom is the dull yellow to greenish color of the foliage, together with a warping or curling of basal leaves. Leaves are killed and

shed from the base up; the woody tissue in the stem is brown, with a water-soaked appearance. The fungus is spread by soil clinging to farm implements, drainage, water, wind, animals and infected seedlings. Once the disease is established, general sanitation and crop rotation are of little help against a fungus that can survive so long without a susceptible host.

Control. Once soil is infested resistant varieties offer the only hope. Many have been developed, including Jersey Queen, Marion Market, Wisconsin Golden Acre, Resistant Detroit, resistant strains of Early Jersey Wakefield, Charleston Wakefield, Globe, Wisconsin All Season and Wisconsin Hollander.

Fusarium oxysporum f. sp. **cucumerinum**. **Cucumber Wilt**. A newly recognized form of *Fusarium* highly pathogenic to cucumber and muskmelon in Florida, only slightly pathogenic to watermelon.

Fusarium oxysporum f. sp. **cyclaminis**. **Fusarium Wilt** on cyclamen.

Fusarium oxysporum f. sp. **cubense**. **Wilt** of banana.

Fusarium oxysporum f. sp. **dianthi**. **Carnation Fusarium Wilt**, **Yellows**, **Branch Rot**, general. The first symptom is a slow withering of shoots, often accompanied by change of color from normal deep green to lighter green to pale straw yellow. Plants appear wilted, especially during the warmer part of the day. Only one side of the plant may be affected, resulting in distortion and tendency to curl. If the stem is split, a brownish streak is seen in the vascular system. There may be a dry, shreddy rot of affected wood and cortex. Plants may be infected at any age, but succumb faster if attacked when young. This species of *Fusarium* does not rot roots; see *F. roseum* under Rots for the form causing stem and root rot on carnation.

Control. Sterilize greenhouse soil and benches; take cuttings from healthy mother block; avoid overwatering. Drenching newly flatted or benched plants has reduced the number of wilted plants but does not replace steaming or otherwise sterilizing soil.

Fusarium oxysporum f. sp. **erythroxyli**. **Wilt** of *Erythroxylum*.

Fusarium oxysporum f. sp. **gladioli**. ►Rots.

Fusarium oxysporum f. sp. **hebae**. **Fusarium Wilt** of *Hebe buxifolia*, and veronica. Reported as killing nursery plants in California.

Fusarium oxysporum f. sp. **lactucum**. **Wilt** of lettuce.

Fusarium oxysporum f. sp. **lycopersici**. **Fusarium Wilt** of tomato, general, in many sections the most damaging tomato disease in field and greenhouse. Chief losses are in states where air temperatures are rather high during most of the season, susceptible varieties dying or producing little fruit. Losses go

up to 30,000 tons of canning tomatoes, or 10 to 35% of the crop in many states.

In seedlings there is downward curvature of the oldest leaves followed by wilting and death. In older plants the disease is most evident as fruit begins to mature, lower leaves turning yellow, first on one side of the stem or leaflets on one side of the petiole. One shoot may be killed before the rest of the plant shows symptoms.

The fungus enters through roots and grows into the stem, where it produces the toxic substances causing wilting and eventual death. The vascular system in the stem shows a dark brown discoloration. In severe infections the fungus grows into fruit and seeds, but such fruits usually drop, and seed is not used. Almost all original infection comes from the soil, the *Fusarium* operating best in light sandy soils and at temperatures between 80° and 90° F, but the disease is spread widely in transplants. It is encouraged by low potassium and high nitrogen nutrients.

Control. Start seedlings in clean soil; do not grow in the same land more than once in 4 years. The use of resistant varieties is the chief means of control. Marglobe, Pritchard and Rutgers are moderately resistant, but infestation by nematodes may predispose even these to wilt. Pan America, Southland, Homestead and Jefferson are more highly resistant. Treating soil with nematicides may reduce incidence of wilt even though the wilt pathogen is not killed.

Fusarium oxysporum f. sp. **melonis**. **Muskmelon Fusarium Wilt**, similar to that of watermelon, important in Minnesota, New York, New Jersey and Maryland. Seeds rot in soil; seedlings damp-off; vines wilt. Fungus persists in soil and is carried internally in seed. Varieties Golden Gopher and Iroquois are quite resistant.

Fusarium oxysporum f. sp. **niveum**. **Watermelon Wilt**, general on watermelon, also on citron. The fungus is transported in and on seed and persists in soil 15 to 18 years. It rots seeds or seedlings, causes wilting of plant, sometimes with cottony mycelium on surface of dying vines. Resistant varieties include Improved Kleckley Sweet and Klondike.

Fusarium oxysporum f. sp. **perniciosum**. **Mimosa Wilt** on mimosa from New Jersey and Maryland to Florida. This extremely pernicious wilt started about 1930 at Tryon, North Carolina, and mimosas have wilted and died at a rapid rate ever since. The wilt appeared in one city block at Morgantown, North Carolina in 1943, and by 1947 trees were dead and dying on 232 blocks.

The first external symptom is a wilting and yellowing of leaves on some of the branches, causing foliage to hang down, then die and drop. Death of the tree follows from a month to a year after first infection. The trunk has a brown ring of discolored sapwood, usually in the current annual ring, and the color may extend out into the branches. The xylem is plugged with brown gummy substances. Small branches may have a one-sided wilting with the bark flattened over collapsing tissue. The disease has been spreading in Maryland since 1947, in Florida since 1952.

As with other *Fusaria*, this is a soil fungus entering through the roots, and eradication of diseased trees has no effect on spread of the wilt. Nematodes, by their wounds, may increase the incidence of wilt. Out of a great many seedlings grown from seed collected from Maryland to Louisiana, inoculated several times with the fungus and planted in infested soil, some have remained mostly disease-free. These have been propagated by the U.S. Department of Agriculture. Released for commercial sale are Charlotte and Tryon.

Fusarium oxysporum f. sp. **pisi**. **Pea Wilt**, caused by race 1 of this pathogen and **Near Wilt**, caused by race 2. Race 1, confined to pea, produces stunted plants, pale yellow green, with leaves curled downward, stem thickened and brittle near the ground. Plants wilt and die prematurely. The disease may cause more or less circular bare spots in the field, enlarging each year if peas are planted continuously, encouraged by high soil temperature. Some commercial pea varieties are resistant to race 1 but not to race 2. Delwiche Commando was the first variety introduced resistant to both races.

Fusarium oxysporum f. sp. **raphani**. **Radish Wilt**. Young plants turn yellow and die; others are stunted, with discoloration of roots.

Fusarium oxysporum f. sp. **spinaciae**. **Fusarium Wilt** of spinach. Plants are pale; leaves roll inward, gradually die. The wilt is serious in Texas and Virginia. One form of the mosaic-resistant Savoy spinach is also resistant to wilt.

Fusarium oxysporum f. sp. **tracheiphilum**. **Wilt** of cowpea.

Fusarium solani f. sp. **pisi**. **Wilt** of chick-pea.

Hendersonula

Deuteromycetes, Coelomycetes

Pycnidia dark, separate; spores dark with several cells.

Hendersonula toruloidea. **Branch Wilt** of walnut. **Canker**, destructive to Persian walnuts but associated with sunburn of affected branches. The fungus is a wound parasite.

Phialophora

► Rots.

Phialophora gregata. **Wilt** of chick-pea.

Phomopsis

► Blights.

Phomopsis sp. **Wilt** on ice plant.

Phytophthora

► Blights.

Phytophthora cactorum. **Wilt** of blue laceflower and baby's breath.
Phytophthora cinnamomi. **Rhododendron Wilt**. A wilt of young stock, grafted plants 2 to 3 years old, seldom on older shrubs, most severe on *Rhododendron ponticum*. The foliage is first dull yellow, then permanently wilted, roots are decayed; stems are brown at soil level and below. Remove infected stock from frames immediately; avoid excessive irrigation; keep soil acidity at pH 4.0 to 4.5; provide shade and mulch for young plants. This pathogen also causes wilt of Japanese umbrella tree.
See under Rots for this fungus at work on many other plants.

Pythium

► Rots.

Pythium myriotylum. **Wilt** on peanut.
Pythium tracheiphilum. **Wilt** on lettuce and also leaf blight.
Pythium aphanidermatum. **Wilt** of *Nicotiana*.

Rhizoctonia

► Blights.

Rhizoctonia solani. **Wilt** of watermelon.

Sclerotinia

► Blights.

Sclerotinia minor. **Wilt** of lupine and wild garlic.

Verticillium

Deuteromycetes, Coelomycetes

Conidia one-celled, hyaline, globose to ellipsoid, formed at tips of whorled branches and separating readily from tips.

Verticillium albo-atrum. **Verticillium Wilt**, **Maple Wilt** of many ornamental trees, shrubs, fruits, flowers and vegetables. The fungus was first isolated from potatoes in Germany in 1870 but apparently was present in California as early as 1850. It attacks nearly 300 cultivated plants of widely diverse types and may persist as a saprophyte in the soil 15 years or more.

Of the ornamental tree hosts silver maples are most susceptible, then sugar and red maples, elms, with occasional reports on ailanthus, alfalfa, aspen, ash, boxelder, beech, black locust, camphor-tree, carob, catalpa, Chinaberry, cucumber, deerbrush, dogwood, goldenrain, horse-chestnut, India hawthorn, redbud, linden, magnolia, oak, osage-orange, olive, pistachio, persimmon, periwinkle, Russian olive, sassafras, strawberry, smoke-tree, tulip-tree, walnut, mango, sunflower and hickory. Maples may wilt suddenly in midsummer, often a large branch or one side of the tree drying and dying while the other side stays fresh. The sapwood of the infected side has greenish streaks, and sometimes slime flux develops on the bark. The disease can be chronic, progressing slowly for several seasons, or acute, affecting the entire tree in a few weeks. In elms the leaves may be smaller than normal, with a drooping flaccidity in hot hours of the day. Later there is a slight yellowing, deepening until the foliage is a striking lemon yellow. Defoliation starts at time of first yellowing, and quite often branchlets drop as well as leaves. Sapwood

discoloration is brown, and the disease cannot be told positively from Dutch elm disease without laboratory cultures. Tyloses and gums are formed in the wood as with other toxin-producing fungi. The fungus always progresses upward through the xylem vessels so there is little danger of downward infection of the main trunk from pruning operations. Progress is slowed by adequate moisture and by high nitrogen fertilizers, ammonium sulfate being particularly helpful.

Verticillium wilt is also a problem on rose understock. Ragged Robin, Odorata, and Multiflora are very susceptible, Dr. Huey less susceptible, and Manetti resistant.

In fruit trees the wilt is often known as black heart or verticillosis. It is common in apricots, less so in almonds and peaches; branches may drop their leaves and die. Also susceptible are sweet and sour cherry, avocado, plum and prune.

On bush fruits – raspberry, blackberry, dewberry and youngberry – the disease is commonly known as blue stem. The symptoms appear late in the season, leaves turning pale, cane tips bending downward, canes taking on a bluish color, lower leaves wilting and drying. Death is often delayed until the season after first infection. Black raspberries are more susceptible than red. The disease is sometime serious on strawberries, especially in California, but cannot always be separated from root rots. Plants may collapse in large areas at the beginning of hot weather.

Verticillium wilt is very destructive to mint in Michigan and Indiana, also reported, though not so serious in Oregon and Washington. Infected plants

Figure 3.63 Verticillium Wilt on Tomato

are stunted, defoliated, and killed; yield of oil is greatly reduced. The fungus attacks all species of mint, but peppermint is most susceptible. There are some resistant hybrids. Deep plowing, inverting the soil, has reduced the amount of wilt.

Verticillium is especially damaging to tomatoes in Utah and California. First symptoms are yellowing of older leaves and wilting of tips during the day;

Figure 3.64 Verticillium Wilt on Potato

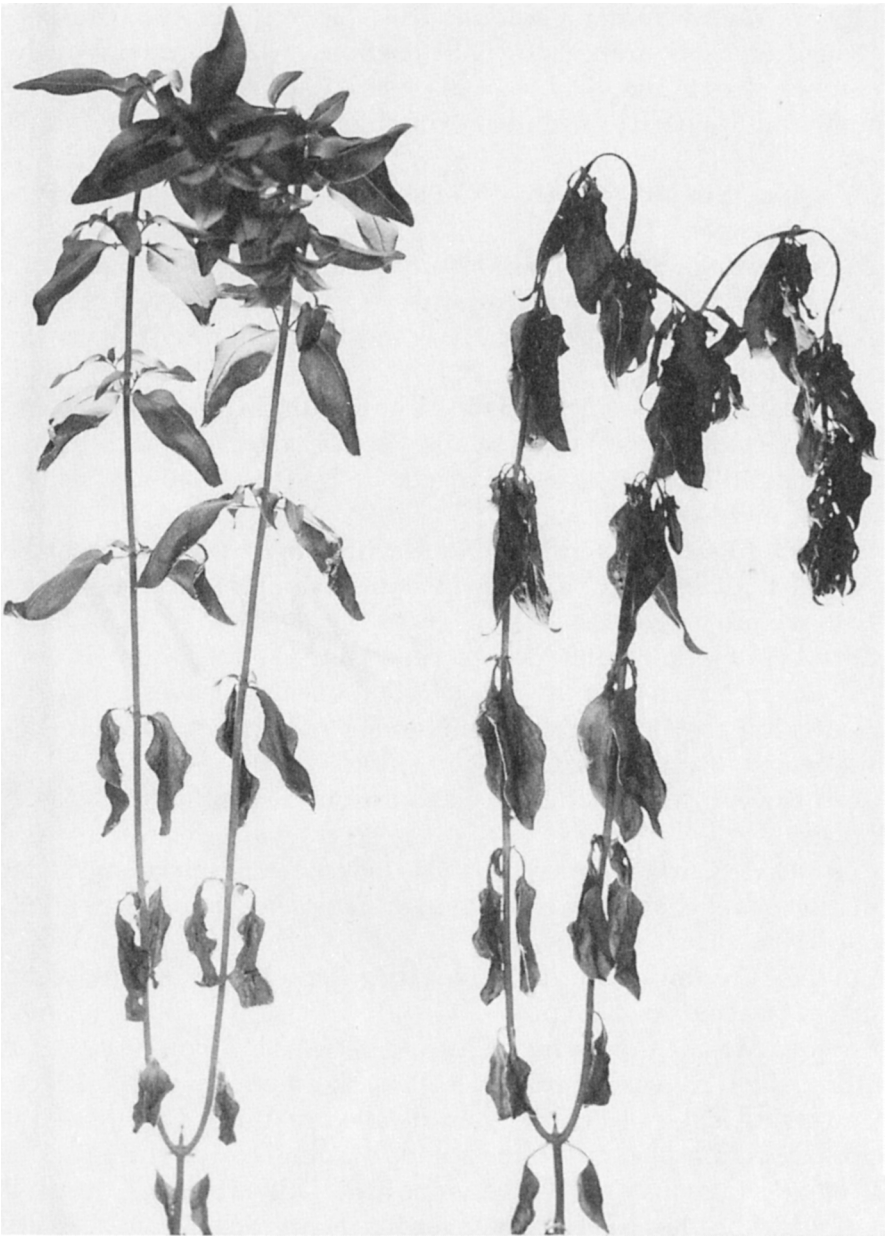

Figure 3.65 Verticillium Wilt on Snapdragon

later, margins of all leaves curl upward, then leaves drop (see Fig. 3.63). Plants are stunted; fruit is small. Moderately resistant varieties Riverside and Essar have been developed for California. Symptoms on potatoes are rather indefinite, but often there is yellowing of lower leaves, shortening of internodes, and rosetting of the top (see Fig. 3.64). Resistant varieties may be symptomless hosts. Verticillium wilt is common on eggplant and okra, rather rare on pepper. It occurs on Chinese yard-long bean, rhubarb and New Zealand spinach.

On herbaceous perennials in eastern gardens I find Verticillium wilt common on aconite and chrysanthemum, with leaves turning dark brown and hanging down along the stem. When the stem is cut across near the base, a circle of black dots indicates the fungus in the vessels. Such plants seldom die immediately but flower poorly and gradually peter out. Wilt was serious on greenhouse chrysanthemums until a wholesale commercial concern started to provide healthy propagating stock from cultured cuttings. Other ornamental hosts include abutilon, aralia, barberry, begonia, China aster, carnation, dahlia, fremontia, geranium, marguerite, peony, poppy, snapdragon (see Fig. 3.65), stock and viburnum.

Control. Sometimes it is possible to prune out an infected maple and still save the tree, but often the dying tree must be taken out. Neither maple nor elm should be replanted in the same spot. Do not transfer plants from areas where wilt has appeared. Do not set raspberries following potatoes or tomatoes; do not use tomatoes after eggplant or potatoes without a long rotation. Proper fertilization and adequate watering may help trees to recover from wilt.

Verticillum dahliae is considered by some a synonym of *V. albo-atrum* and by others as a distinct species; reported as causing wilt of dahlias, mint, marigold, ice plant, barley, wheat, oat, potato, Leucospermum, impatiens, giant hyssop, globe artichoke, ash, cabbage, Cineraria and Echinacea, and other plants. This form has microsclerotia and grows on agar at slightly higher temperatures.

Verticillium fungicola. **Dry Bubble** of oyster mushroom. Infection of sporophores at pin or button stage cause development of typical dry bubbles; mature sporophores show cracking and curling of tissues, and depressed, brown, necrotic areas.