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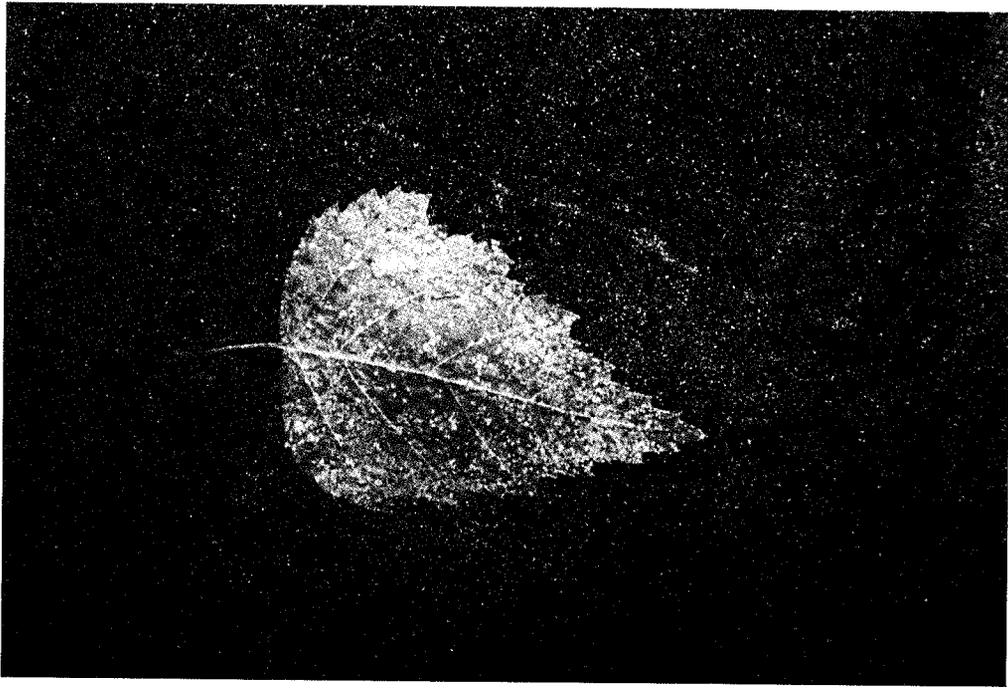


Figure 14.—Rust on birch (*Betula pendula*) caused by *Melampsorium betulae*. Uredinia occur on the under surface of the leaf.

Control

Observation

- Survey for disease appearance and dispersion in nurseries and young plantations in early July (period of uredinia pustule formation).

Cultural

- Use plant material selected for resistance to leaf rusts.
- Eradicate possible alternate hosts near nurseries or plantations.
- Collect or burn fallen leaves.

Chemical

- Before leaves emerge in spring, apply fungicide (DNOC or Thiram) to fallen leaves and the lower parts of plants.
- Apply fungicide (Zineb, Metiram and copper hydroxide) to foliage in summer.

Diseases Of Roots, Stems, And Branches

Diseases in Tree Nurseries and Young Forests

Damping - off

Class/Order: Deuteromycetes/Hyphomycetales

Pathogens

Primarily species from *Fusarium*, *Alternaria*, *Botrytis*, and *Verticillium* genera, but also by *Rhizoctonia*, spp., *Mycelia sterilia*, and *Pythium* spp.

Hosts

Species of pine (*Pinus*), including Scots (*P. sylvestris*); spruce (*Picea*), including Norway (*P. abies*), Siberian black (*P. obovata*), and blue (*P. pungens*); and larch (*Larix*), including European (*L. decidua*), Siberian (*L. siberica*), Sukachev (*L. sukaczewii*), and Kurilian (*L. kurilensis*); also species of beech (*Fagus*), elm (*Ulmus*) and birch (*Betula*)



Figure 15.—Current-year pine seedlings (*Pinus sylvestris*) severely affected by Fusarium root rot. Localized areas in beds of pine affected by Fusarium root rot.

Diagnosis

There are two forms of disease: pre- and post-emergence damping-off. Pre-emergence symptoms include lack of germination or emergence in seedling beds and rotten seeds and seedlings in the soil. Post-emergence symptoms include small areas of diseased seedlings (**Fig. 15**) that under favorable conditions for the fungus quickly merge and affect the entire planting area. A dark-brown constriction forms at the stem base, and seedlings fall to the soil and wither. Occasionally, fungal mycelium develops near the soil line in the zone of constriction, particularly during wet weather. Some diseased seedlings do not shed their seed cover. Lateral rootlets often die and rot. Diseased plants are easily pulled from the soil; the main root has no bark. The constriction at the stem base does not form in seedlings more than 4 weeks old. Another symptom is top withering caused by hyphal-induced embolisms in xylem vessels. Infected plants lose their turgor and wither but their root systems do not rot; they die 3 to 4 weeks after emergence.

Biology

Sources of inoculum are previously diseased seeds, seedlings, and soil. Pathogens, including *Fusarium* species, can exist as saprophytes on decaying plant tissues for long periods. *Fusarium* species can produce chlamydospores that remain in the soil for long periods and are highly resistant to unfavorable environmental factors and pesticides. The disease spreads by conidia carried or brought into the soil on seeds. The rate of mycelium spread in the soil is about 2-5 cm/day.

Disease development is most active in heavy, acidic, and waterlogged soils, though conditions for development can be favorable on dry and sandy soils. Weather conditions considerably influence disease development and distribution; mortality increases with low temperature and high humidity as well as high temperature and moisture deficit.

Damage

Kills seedlings; when conditions for disease development are favorable (high humidity and soil moisture), can affect up to 100 percent of the seedlings.

Distribution

Throughout Russia

Control

Cultural

- Select flat areas with light soils when establishing nurseries.
- Avoid sites formerly used to grow agricultural plants, e.g., potatoes, corn, or melons, due to the potential for previous *Fusarium* infection.
- Sow seeds at optimal time, observe rules of sowing, and use only certified seeds.
- Care for seeding beds after seedlings emerge and prevent weed growth and drying of soils.

Chemical

- Use chemical seed dressing where necessary. Dust seeds with fungicides (Bavistin, Benomyl, BAYMEB, Topsin-M, Daconil, Vitavax, Thiram, and Tersan). Fungicide treatment combined with micronutrient fertilizer (ZnSO₄, CoSO₄, CuSO₄, KMnO₄) is the most effective treatment.
- Apply fungicides (Thiram, Tersan, Karbation, or Vapam) to the soil where infection is heavy.
- Use fungicides in irrigation water in disease loci in nursery beds.

Biological

- Apply antagonistic antibiotics (Trichothecin, Phytobacteriomycin, or Phytolavin).

Diseases of Forest Stands

Wilts

Verticillium Wilt

Class/Order: Deuteromycetes/Hyphomycetales

Pathogen

Verticillium dahliae Kleb. and *V. albo-atrum* Rke. et Berth.

Hosts

Species of maple (*Acer*), particularly Norway (*Acer platanoides*), and birch (*Betula*), poplar (*Populus*), lime (*Tilia*), and elm (*Ulmus*)

Diagnosis

Affected trees fail to foliate in spring or die suddenly in midsummer (**Fig. 16**) in the acute form of disease development. In the chronic form, several branches die and then later the entire crown dies. Numerous epicormic shoots form on the stem but they soon die. Bark sloughs from dead trees. The wood of infected roots and stem initially has green stripes but later turns dark-green. An important diagnostic feature in the conidial stage is the conidiophores, which typically are branched vertically with colorless, ellipsoid conidia, 2-5 μ, on them. Microsclerotia and chlamydospores develop later. In natural conditions, the fungus sporulates rarely, however, spores develop rapidly in the laboratory (in moisture chambers).

Biology

Infection is initiated by conidia at the base of the stem or branch. Occasionally, trees are infected through damaged stems and branches. The mycelium penetrates the tree and spreads in the vascular system. From the lower part of the stem, mycelium grows upward and penetrates branches. When the fungus attacks, most of the roots (or the entire root system) are infected. The tree usually dies before mycelium reaches the stem. Sources of inoculum are infected wood, bark residues, and dead roots. The pathogen survives by forming microsclerotia and chlamydospores. These structures survive in the soil on dead debris for several years. Inoculum potential increases in soils where agricultural species such as potato (*Solanum tuberosum*), tomato (*Lycopersicum esculentum*), and sunflower (*Helianthus annuus*) have grown.

Damage

Kills seedlings in nurseries and artificially and naturally regenerated maple stands 1 to 2 years old. Dieback of trees in forest plantations and natural stands is possible during first 4 years of growth.

Distribution

European part of Russia, particularly the southwestern region



Figure 16.—Dieback of naturally regenerated maple (*Acer platanoides*) caused by *Verticillium dahliae*.

Control

Cultural

- Do not establish forest nurseries and plantations in areas where *S. tuberosum*, *L. esculentum*, and *H. annuus* have grown.
- In nurseries, remove diseased plants and their entire root system.
- Selectively remove infected trees or clearcut in disease loci in affected natural stands.
- Remove and destroy all felling residue.

Dutch Elm Disease

Class/Order: Ascomycetes, Microsporales

Pathogen

Ophiostoma ulmi (Buism.) Nannf. (Syn. *Ceratocystis ulmi* (Buism.) Mor. (anamorph: *Graphium ulmi* Schwarz.)

Hosts

Species of elm (*Ulmus*), including table top (*U. glabra*), Ohio (*U. laciniata*), European white (*U. laevis*), smooth leaf (*U. carpinifolia*), Japanese (*U. japonica*), Siberian (*U. pumila*), and Androssowi (*U. androssowii*)



Figure 17.—Elm (*Ulmus laevis*) affected by *Ceratocystis ulmi* (= *Ophiostoma ulmi*).

Diagnosis

Wilting foliage is the major symptom (Fig. 17). The most typical and distinctive feature of the disease is leaf roll along the primary rib. However, other symptoms are evident e.g., pigmentation and dropping of leaves. Unlike the asexual reproduction stage (conidia), the sexual ascus stage (black perithecia) occurs rarely. Most Russian strains belong to an aggressive pathogen race.

Biology

The pathogen can be distributed by air, water, and animals as a result of root grafting or by shoot infestation. Inoculation results from mechanical injury to trunks and branches by elm bark beetles, which play the major role in the disease distribution. *Scolytus scolytus* Fabr., *S. multistriatis* Marsch., and *S. pygmaeus* Fabr. are common in infected areas in Russia. The fungus is present on eggs and in larval galleries of the bark beetles. Spores were observed on 83.3 percent of beetles captured from infected trees. Beetles damage 1- and 2-year-old shoots in spring when they feed in twig crotches. Initially, necrotic zones develop in the current-year annual ring of any thin twig. In the chronic form of the disease, usually in trees more than 50 years old, the fungus remains in the upper part of the tree in the older sapwood elements. Symptoms usually arise annually with wilting of individual shoots in the crown; the disease continues to develop over several years until the tree dies. In the acute form, the pathogen not only spreads in the crown but also reaches the stem base and roots. Tree death is sudden and occurs in one growing season.

Damage

Causes massive dieback of elm species in forest and urban ecosystems. During epiphytotics, mortality of mature trees can approach 100 percent.

Distribution

Ukraine, Moldova, Volga River, Baltic Sea, central regions and European and Middle Asia parts of Russia (in the former U.S.S.R., the disease was first observed in the city of Odessa in 1935)

Control

Control of Dutch elm disease entails the suppressing both the pathogen and vector population.

Observation

- Survey elm stands in June and July for tree condition, presence of symptoms, and abundance of elm bark beetles.

Cultural

- Create mixed stands with a variety of deciduous species.
- Use disease-resistant planting material.
- Conduct selective and clear sanitation cutting and remove trees with disease symptoms (October through April) to reduce the abundance of overwintering bark beetles.
- Destroy infected wood and debark healthy portions of cut trees and branches.

Oak Wilt

Class/Order: Ascofymcetes, Microascales

Pathogen

Species of *Ceratocystis* (*C. roboris* Georg. et Teod., *C. valachicum* Georg. et Teod.) and species of *Fusarium* and *Verticillium*

Hosts

Species of oak (*Quercus*)

Diagnosis

The disease has both rapid and chronic development forms. The rapid form is characterized by leaves wilting at the top of the tree and branches. The disease quickly spreads to the entire crown. In this case, leaves curl, turn yellow or bronze, and fall 3 to 6 weeks after the first symptoms appear. If infection occurs at the end of summer, leaves turn tan and some fall while others remain on branches throughout the winter. Mycelial masses form under the bark of dead trees and arise from bark cracks. In the chronic form, wilting begins on several separate branches. Leaves are smaller than normal, turn yellow or tan, and drop. Often, buds on diseased branches fail to grow in spring, and/or leaves do not reach normal size and fall prematurely. Affected trees are characterized by sparse foliage and top dieback. Heavily weakened and dying trees produce adventitious sprouts. Morphological changes in the crown are not specific for the chronic form. Similar symptoms can be caused by other diseases as well as chemical, weather, and other adverse environmental factors.

The pathogen develops within the water-conducting xylem vessels, and causes dieback of adjacent parenchyma and extensive tyloses formation. Characteristic symptoms of this disease are evident when branches and trunks are cut; tan or dark-olive spots and streaks and dots form in the sapwood or the entire sapwood turns brown.

Biology

Infection usually occurs by spores, though infection by mycelium can occur through grafted roots. Spores are distributed by water, wind, and on seeds and tools, but most commonly by insects. The most active vectors are insects such as *Scolitus intricatus*, *Plagionotus detritis*, *P. arcuatus*, and *Agrilus angustulus*. Oak wilt usually develops in previously weakened stands. The percentage of diseased trees increases with tree age. The disease is prevalent in low-density stands. Oak dieback occurs in both mountain and floodplain stands but is more severe in the latter.

Damage

Weakens and kills oak stands (**Fig. 18**). Depending on age, nearly 75 percent of the stands can be affected. The death rate can reach 70 percent.

Distribution

European part of Russia

Control

Cultural

- Conduct sanitation cuts to remove and destroy infected trees, including trees infested by oak bark beetles; spray stumps with Fundasol or Nitraphen.
- Establish seed plantations in healthy and disease-resistant stands.
- Examine acorns for infection before seeding.



Figure 18.—Dead and dying trees of oak (*Quercus robur*) stands that were attacked by oak wilt – (*Ceratomyces roboris*).

Chemical

- Treat tree with contact insecticides during the feeding period of bark beetles.
- Use systemic fungicides to suppress the pathogen (injection into the trunk is the most effective method).
- Treat acorns with fungicide (Fundusol and TMTD).

Dieback and Canker Diseases

Cenangium Dieback of Pine

Class/Order: Ascomycetes, Helotiales

Pathogen

Cenangium abietis (Pers.) Rehm. (anamorph: *Dothichiza ferruginosa* Sacc.)

Hosts

Species of pine (*Pinus*), particularly Scots (*Pinus sylvestris*), and fir (*Abies*) and spruce (*Picea*)

Diagnosis

Symptoms first appear in the leader of current-year or the previous-year shoots. The bark turns red but necrotic areas do not have clear boundaries. Infected needles die and remain on the branches through

the growing season (**Fig. 19**). The conidial stage develops on infected branches and needles during the entire growing season. Small, black pycnidia-like tubercles develop in bark splits. Apothecia develop on dead branches and stems 1 or 2 years after infection. In wet conditions, apothecia are saucer-like with a green-yellow hymenial layer. In dry weather, apothecia resemble dark-brown, nearly black rough tubercles, 1-3 mm in diameter, raised from the bark splits. Conidia are colorless, oval or ovoid, 8-12 x 4 μ . Asci are clavate, 60-80 x 10-12 μ . Ascospores are colorless, ellipsoid or ovoid, with 1 or 2 oil drops, 10-12 x 5-7 μ .

Biology

The sources of inoculum are diseased plants and fallen needles. New infections on healthy trees are initiated by conidia and ascospores. High humidity promotes sporulation and infection. The fungus penetrates tissues of needles and bark injured by various agents, including insects. Many factors that result in poor growing conditions and weakened plants promote disease development. These include unfavorable climate conditions (drought and others), changes in water regime, insect injury (from *Aradus cinnamomeus* Panz. and *Ellopija fasciaria* L.), and industrial pollution.

Damage

The fungus usually occurs as a saprotroph on pine trees killed by various agents. More rarely, the disease develops as an epiphytotic in nurseries and pine plantations, causing dieback. Plant damage can range from 50-60 percent. In nurseries, seedlings can die if the epiphytotic continues for several years.

Distribution

European part of Russia, Siberia, Far East

Control

Cultural

- Remove infected trees to prevent disease spread.
- Remove and burn dead trees and branches as sources of inoculum.
- Create mixed hardwood/pine plantations as more biologically sustainable; hardwood species reduce the possibility of pine infection.



Figure 19.—Cenangium dieback of pine (*Pinus sylvestris*) caused by *Cenangium abietis*.



Figure 20.—Canker on poplar (*Populus nigra*) caused by *Nectria cinnabarina*. Sporodochia of the fungus are visible.

Nectria Canker and Dieback

Class/Order: Ascomycetes, Hypocreales

Pathogen

Nectria cinnabarina (Tode.) Fr. (anamorph: *Tubercularia vulgaris* Tode.)

Hosts

Numerous deciduous tree genera are affected, including species of maple (*Acer*), birch (*Betula*), beech (*Fagus*), ash (*Fraxinus*), alder (*Alnus*), poplar (*Populus*), lime (*Tilia*), elm (*Ulmus*), oak (*Quercus*), dogwood (*Cornus*), and mountain-ash (*Sorbus*), as well as barberry (*Berberis*) and currant (*Ribes*). Bosnian maple (*Acer platanoides*) is the most severely affected species.

Diagnosis

Bright-pink cushions of conidial stromata develop as longitudinal lines in bark cracks on the infected branches and trunks. Later, they darken and turn tan, and perithecia form on the same stromata (Fig. 20). Conidiophores are slightly branched. Conidia are elongate-ellipsoid, slightly curved, colorless, 5.5-8 x 3 μ . Asci are cylindrical or clavate, 60-90 x 9-12 μ . Ascospores are colorless, widely fusiform, 2-celled, 12-20 x 4-7 μ . Infected wood is blue gray.

Biology

In spring, infection occurs from ascospores that overwinter. During the growing season, infection occurs from conidia. Spores penetrate tree tissues at various bark injuries, including broken branches.

The mycelium spreads in the bark, cambium, and sapwood, and penetrates the trachids where it plugs vessels and causes rapid dieback.

Damage

The fungus often is reported as a saprophyte but it can be pathogenic in nurseries and young forest plantations. Infected seedlings in nurseries can reach 20 percent. Severe disease reduces vigor and causes dieback in nurseries. The canker significantly reduces productivity of mature trees.

Distribution

European part of Russia, Urals, Siberia, Far East

Control

Observation

- Survey stands during the summer when conidial sporodochia are visible.

Cultural

- Protect trees from environmental factors that can weaken them.
- Remove infected, dying, and dead trees in forest nurseries and urban stands; prune infected branches.
- Prevent or reduce mechanical injuries that serve as entry points for the pathogen.

Cytospora Canker

Class/Order: Deuteromycetes, Sphaeropsidales

Pathogen

Cytospora chrysosperma (Pers.) Fr. (teliomorph: *Valsa sordida* Nits.)

Hosts

Species of poplar (*Populus*) and willow (*Salix*)

Diagnosis

The pathogen develops in bark tissue and kills it. Thin, smooth bark that is affected is red-brown; the color of thick bark does not change. Dark-gray or brown conidial stromata form in the bark. Numerous pycnidia develop in the stromata and raise the bark surface as small tubercles. Mass sporulation occurs in May and early June, as well as in late August and September. Spore masses exude from pycnidia and congeal as golden-yellow or orange horns (cirris). Pycnidia can be 1.8 mm in diameter, chambered with a common opening. Conidia are colorless, allantoid, 3-5 x 1 μ .

Biology

Sources of inoculum are infected cuttings, seedlings, and infected trees in stands. Conidia are spread by rainwater or insects and infect healthy trees. The pathogen penetrates tree tissues through mechanical injuries and cracks at the base of branches. As the fungus grows, it secretes toxins that kills the bark. Mycelium then develops in dead bark. Pockets (loci) of the disease develop usually in stands predisposed to infection by unfavorable environmental conditions, e.g., droughts, flooding, winter damage, and unfavorable soil. Disease develops just after foliation and later in midsummer. Death may occur during one growing season or over several years. In the case of rapid disease development, affected trees do not foliate in the spring or die shortly after foliating. In chronic disease development, single branches die gradually in the crown; epicormic shoots develop on the stems but also are infected and die.

Damage

Kills weakened trees. Damage is most severe in nurseries, stool-beds, and forest plantations.

Distribution

Throughout Russia

Control

Cultural

- Cull diseased plant material in nurseries, forest plantations, and stands.
- Select resistant species and clones.



Figure 21.—Dothichiza canker of poplar (*Populus balsamifera*). Pycnidia with emerging mass of spores are visible.

Chemical

- Apply fungicides (Zineb and TMTD) as a drench before planting.

Dothichiza Canker of Poplar

Class/Order: Deuteromycetes, Sphaerosidales

Pathogen

Dothichiza populea Sacc. et Briard.

Hosts

Species of poplar (*Populus*) and their hybrids, particularly from the groups of black (*Aegeiros*) and balsam (*Tacamahaca*) poplar

Diagnosis

Initially, sunken, necrotic, oval areas several centimeters in diameter form on the bark of trunks and branches. Cankers form along the entire length of the main stem and branches, but more often where branches attach to the main stem and shoots attach to branches. Affected areas on the living stem and branches appear dark, but the bark yellows after death. Callus ridges several millimeters thick develop at the margins of the necrotic area. Gradually, cankers coalesce and girdle the stem or branch. In a large infected stem, mycelium spreads in the tissues over a 2- to 3-year-period, causing a perennial canker. Infected trees are sparsely leafed and there are numerous epicormic shoots on the stems. In spring, pycnidia up to 2 mm in diameter develop on dying and dead areas of the bark. Pycnidia usually form as longitudinal rows but can be distributed randomly (**Fig. 21**). Spores are black

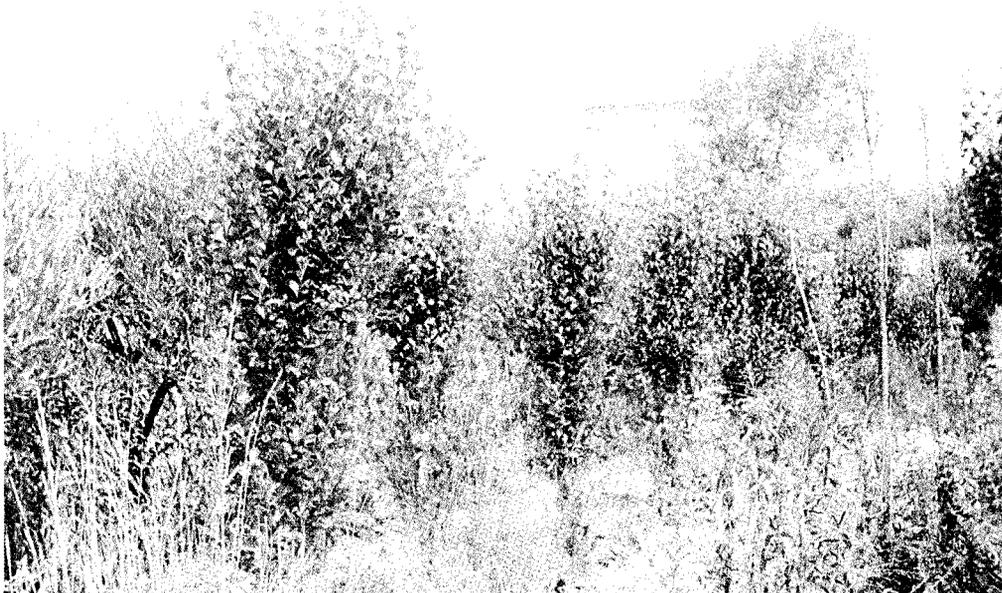


Figure 22.—Poplar (*Populus nigra*) dieback in ornamental nursery caused by *Dothichiza populea*.

or light-olive; when exuded they resemble small horns or strips, 2-4 mm in length. Conidia are colorless, ovoid, narrowed on the ends, rarely globose or ellipsoid, 1-celled, 8-13 x 6-12 μ .

Biology

Sources of inoculum are infected cuttings, transplants and diseased trees in forest plantations and stands. Infection occurs by conidia which mature early in the growing season. Mature spores can survive in pycnidia for nearly a year and are dispersed by rainwater and insects. Infection occurs from April until September. Trees are infected through mechanical injuries but also through natural openings such as lenticels, base of buds, shoot junctions, and cracks at the base of branches. Disease development occurs when conditions are unfavorable for poplar growth (poor aeration, soil compaction, waterlogging, overstocked stands).

Damage

Causes weakening and often mass dieback of poplar in nurseries, plantations, and stands of various types (**Fig. 22**).

Distribution

European part of Russia, south Urals, Siberia, Far East

Control

Observation

- Survey stands in early summer for disease symptoms.

Cultural

- Establish stool-bed plantations, nurseries, and forest plantations on sites where conditions are optimal for tree growth and development.
- Plant only healthy plant material.
- Minimize mechanical injuries when planting and cultivating.
- Select resistant species and clones that are adapted to the site conditions.

Chemical

- Apply fungicide (TMTD) to seedling roots as a drench before planting.

Clithris Canker and Dieback of Oaks

Class/Order: Ascomycetes, Phacidiales

Pathogen

Clithris quercina (Pers.) Rehm. (anamorph: *Cytospora quercella* Sacc.)

Hosts

Species of oak (*Quercus*), particularly peduncate (*Q. robur*)

Diagnosis

The bark of infected stems and branches turns red and later yellow-white upon death. Necrotic areas are sharply delimited from healthy bark. Necrosis can be elongate or circumferential. Pycnidia develop initially on the dead bark as small gray-white tubercles (hillocks). Later, numerous apothecia 5 mm long form in the dead bark. They are gray-white or dark-gray and curved. In humid conditions, apothecia open as relatively wide longitudinal splits uncovering a gray, jelly-like hymenial layer. Asci are clavate, with a stalk, 125-150 x 9-10 μ . The ascospores are thread-like, colorless, initially 1-celled but later multicelled, 90 x 1-1.5 μ .

Biology

Healthy branches and stems are infected from ascospores in early summer through injuries to bark. Trees weakened by poor growing conditions, e.g., repeated droughts, changing water regimes, or frost injury, are highly susceptible.

Damage

In the forest the fungus acts as a saprophyte, occurring on the lower dying branches and promoting the process of branch shedding. However, in the steppe zone it acts as a pathogen affecting plantations under 25 years, reducing vigor, and sometimes killing trees. Damaged plants range from 10-40 percent. The fungus causes sapwood decay as well as bark necrosis.

Distribution

European part of Russia

Control

Cultural

- Create optimal conditions for growth (soil moisture, fertilization, spacing) in nurseries and plantations.
- Remove damaged trees and felling debris after thinning.
- Minimize mechanical injury during tending operations.

Nummularia Canker

Class/Order: Ascomycetes, Sphaeriales

Pathogen

Nummularia bulliardii Tul.

Hosts

Species of oak (*Quercus*) and beech (*Fagus*)

Diagnosis

Symptoms are visible several years after infection. Thick, up to 3 mm, oval, cushion-like stromata form in infected bark and over entire length of the stem. Stromata are 1-6 cm wide and 2-40 cm long. Often, stromata are coalesced, girdling and extending up stems to a length of 1 m. Initially, stromata are brown but later turn black and coal-like. Numerous perithecia develop within the stroma. Perithecia openings appear as pointed hillocks on the entire stroma surface. Perithecia in stromata occur as a single layer; they are ovoid or sack-like, 0.5 mm high. Asci are elongate, cylindrical, on a short stalk, 100-210 x 10 μ . Ascospores are dark-brown, ellipsoid or globose, 1-celled, 12-14 x 6-10 μ . Decayed sapwood appears light-yellow in the main stem and branches.

Biology

The source of inoculum is diseased trees and felling residues with well-developed stromata. Tree infection occurs from ascospores that penetrate into tree tissues through bark cracks or mechanical



Figure 23.—Canker of oak (*Quercus robur*) caused by *Naemospora croceola*. Sporodochia with red drops of spore masses are visible.

injuries. Disease outbreaks occur most frequently in weakened stands. Predisposing stresses are severe droughts, frosts, water deficits, open-growing trees or overstocking, and bark injury from tending activities.

Damage

Affects 20- to 30-year-old trees in natural stands and forest plantations. Reduces tree vigor and causes some crown dieback. Some trees are killed.

Distribution

Steppe (southeastern) zone of European part of Russia

Control

Observation

- Survey stands for symptoms in summer and after leaf drop in autumn.

Cultural

- Create optimal conditions for the growth and development of plantations.
- Maintain proper spacing in closed plantations.
- Minimize mechanical injury of stems during tending activities.

Black Naemospora Canker

Class/Order: Deuteromycetes, Melanconiales

Pathogen

Naemospora croceola Sacc. (teliorph: *Diatrype stigma* (Hoffm.) Wint.)

Hosts

Species of oak (*Quercus* spp.), particularly English (*Q. robur*), and birch (*Betula*).

Diagnosis

Initially, infected bark turns red, but symptoms are not conspicuous (**Fig. 23**). Later, sporodochia form on the infected bark. They are round, yellow spots with dark margins, up to 3 mm in diameter. Red

drops or horns of congealed conidial masses usually are visible at the center of the spots. In 2 or 3 years, infections often girdle small stems and branches, and a stroma develops in dead bark. Stromata are flat, up to 2 mm thick, dark-brown, and covered with numerous longitudinal and transverse cracks. Numerous pointed hillocks that develop on the stroma surface can be seen with a hand lens. These are the openings of the perithecia, which are closely aggregated, bottle-shaped, with short nipple-like openings. Asci are clavate, cylindrical, on a lengthened stalk, 30-60 x 4-3 μ . Ascospores are light-brown, cylindrical, straight or curved, with 2 oil drops, 4-12 x 0.6-1.5 μ .

Biology

The sources of inoculum are diseased and dead trees and felling residues. Trees are infected by conidia and ascospores. Spores are dispersed by rainwater, insects, and other vectors. Weakened trees in forest plantations are affected.

Damage

Affects fine branches in the lower part of the crown, as well as the larger branches of severely weakened trees. During droughts in the steppe and forest-steppe zones, the disease affects forest plantations under 25 years of age and can cause tree mortality. Damage to oak plantations in the steppe zones can exceed 70 percent.

Distribution

Middle and southern areas of European part of Russia

Control

Observation

- Survey for symptoms and spread of disease in spring and summer.

Cultural

- Create optimal conditions for plantation growth and development.
- Timely remove diseased trees and their felling residues as sources of inoculum.

Thyrostroma Canker and Dieback

Class/Order: Deuteromycetes, Melanconiales

Pathogen

Thyrostroma compactum (Sacc.) Hoehn (syn.: *Stigmina compacta* (Sacc.) M.B. Ellis)

Hosts

Species of lime (*Tilia*) and elm (*Ulmus*)

Diagnosis

The first symptom of infection is dieback of the previous-year growth (**Fig. 24**). In spring, buds on these branches do not open; by the end of summer or during the next growing season, fungal stromata form on them. Dead branches often drop off, but the fungus grows into larger branches of the previous-year growth. These branches have a thin and smooth bark. Oval, pitching, necrotic depressions are formed along branches. Infected bark is darker than healthy bark. During the dormant season, necrosis spreads longitudinally and tangentially, killing bark and outer sapwood tissues and causing the rapid death of branches. Stromata form on dead branches, initially in the zone of original necrosis, and then on the entire branch (**Fig. 25**). Necrotic spots form on stems and branches with thin bark and around the base of small, infected twigs. During wound development, bark splits longitudinally and opens gradually. Later, bark sloughs and wood is exposed. Open cankers are elongate, often spindle-like, without changes in wood color, and have one ridge of callus (**Fig. 25**). Stromata are black, velvety, and cushion-like, and break through the bark surface. Spores have 1-3 vertical and 1-5 horizontal septa, usually 46-63 x 15.3-17.2 μ . Clusters of adventitious shoots with large leaves often develop at the base of dead branches and on limbs and trunks. These shoots are infected and killed, causing more intensive formation of clusters of adventitious shoots.

Biology

Infections by conidia occur in midsummer. Spores are disseminated by wind up to 10 m, though most fall within a radius of 3 m from their source, infecting new branches and twigs of the same tree as well as neighboring trees. Infection occurs through the buds. Rain and high humidity favor infection. The



Figure 24.—Elm (*Ulmus pumila*) dieback caused by *Thyrostroma compactum* (= *Stigmina compacta*).



Figure 25.—Lesion on branches of lime (*Tilia cordata*) caused by *Thyrostroma compactum*.

pathogen cannot colonize actively growing tissues in the tree; colonization occurs only during the dormant season. The pathogen tolerates low temperatures as growth occurs even at -2°C . Spores deposited on vulnerable twigs and branches remain viable for up to 7 months.

Damage

Causes cankers, resulting in branch dieback (**Figs. 24-25**).

Distribution

European part of Russia

Control

Cultural

- Use canker-free, healthy plant material to establish plantations.
- Prune infected branches at early stages of disease development to reduce inoculum.
- Establish quarantine areas to prevent the spread of diseased plant material to regions where the disease does not occur.

Ascocalyx Scleroderris Shoot Canker

Class/Order: Ascomycetes, Helotiales

Pathogen

Ascocalyx abietina (Lagerb.) Schläpfer. (*Gremmeniella abietina* (Lagerb.) Moretel., *Crumenula abietina* Lagerbe., *Scleroderris lagerbergii* Gremm.) (anamorph: *Brunchorstia pinea* (Karst.) Hohnk.)

Hosts

Species of pine (*Pinus*), spruce, (*Picea*), fir (*Abies*) and larch (*Larix*)

Diagnosis

Pinus species are affected more often than other species. Symptoms of the disease vary with plant age. Initial symptoms of pine infection in nurseries occur several days after snowmelt. The fungus penetrates into the needles and upper buds and affects the cambium of young branches and stems. Initially, needles are green but turn red at the base where they attach to the stem. Paired needles of the 2 to 3-year-old seedlings hang like an umbrella and fall at the slightest touch. In the nurseries this disease is called the "umbrella disease." Following snowmelt, needles of affected seedling in nurseries are flattened along the stem. They turn brown but remain attached to the stem for a long time. The upper bud and stem cambium die and bark sloughs away easily if the seedling is pulled from the soil. In disease centers in young pine stands more than 5 years old, needles hang flaglike. Upper shoots are short and thick, with shortened or thickened needles. Necrotic and sometimes girdling lesions develop on the stem and branches and kill upper shoots on plants with a stem diameter larger than 1 cm. Canker wounds sometimes form.

During summer, mature pycnidia form on needle bases and in the bark of dying and dead seedlings. Pycnidia resemble rough black warts, about 2 mm in diameter. Spores are colorless, curved, usually 3- or 4-celled, but can be 4- or 6-celled, 16-32 x 2-3.5 μ m. The sexual stage occurs rarely. Apothecia are black-brown, 1-1.8 mm in diameter (Fig. 26). Asci are colorless, clavate, 95-110 x 7.5-10 μ m. The ascospores are oval, with 2 or 3 septa, 15-24 x 4-4.8 μ m. In pine stands 15 to 20 years old, the tips of upper shoots usually die with characteristic symptoms of needles thinning and falling off. Pycnidia often form on these shoots. Similar symptoms can occur in middle-age, mature, and overmature stands.

Biology

Infection occurs by conidia and ascospores that are spread by wind or insects. Conidia dispersal occurs throughout summer; ascospore dispersal occurs from mid-July to September. Various factors cause tree weakening and reduced resistance to this disease, resulting in canker development. Unfavorable climate and soil conditions, hard frosts, and cold and rainy vegetative periods delay shoot development and disturb nutrient balance. Severe disease development occurs in low-lying areas where frequent fogs and autumn and spring frosts occur. Resistance to the disease is reduced in seedling beds and high-density plantings. Pine species planted offsite are highly susceptible to this disease.

Damage

Primarily kills seedlings in nurseries; mortality ranges from 10-60 percent. Mature trees are noticeably weakened and many are killed.

Distribution

European part of Russia, and Siberia

Control

Observation

- Survey nurseries for 10-14 days after snowmelt.
- Survey forest plantations during the summer.

Cultural

- Use indigenous seeds sources to increase seedling resistance to the disease.
- Remove infected plants from bordering forest plantations or stands.
- Select sites with flat topography for forest plantations; avoid cold, wet, shady areas.
- Use healthy indigenous planting material.
- Control the condition and density of plantings during the first 2 or 3 years after planting.



Figure 26.—Fruiting bodies of *Ascocalyx abietina* on a stem of fir (*Abies sibirica*).

Chemical

- In nurseries apply systemic fungicides such as BAYMEB, Benomyl (Benalate), and Daconil.

European Larch Canker

Class/Order: Ascomycetes, Helotiales

Pathogen

Dasyscyphus willkommii (Hart.) Rehm. (syn. *Lachnellula willkommii* (Hart.) Dennis).

Hosts

Species of larch (*Larix*), including European (*L. decidua*) and Siberian (*L. sibirica*); also fir (*Abies*) and Siberian stone pine (*Pinus sibirica*)

Diagnosis

Annual radial growth ceases in affected twigs and stem areas. Oval, pitching depressions form in the bark. Dead bark sloughs and cankers become open. Several cankers may form on the same stem. Cup-shaped apothecia on short stalks, 2-6 mm in diameter, form on the margins of cankers and on the dead branches. The outside surface of the apothecia is white and hairy and the hymenium is orange. Asci are oblong, 90-120 x 8-12 μ . Ascospores are colorless, oval or spindle-like, 1-celled, 16-25 x 6-9 μ . In young larch plantations, twigs and small stems die before the characteristic canker wound is formed. Resin exudes and apothecia form on the dead branches and stems.

Biology

Trees are infected by ascospores that mature in spring and autumn. The pathogen initially affects lower branches in areas of frost pockets or other bark injuries. It then penetrates into the main stem and develops for several decades. Mycelium develops in the tree tissues when the air temperature range is 3°-5°C. These conditions occur in spring and autumn, or in winter in regions with a mild climate. Epiphytotic may develop in milder winter regions. Where winters are severe, the fungus occurs usually as a saprophyte on dead branches. Larch plantations are affected severely under unfavorable conditions, e.g., waterlogged soils, overstocked stands, and unsatisfactory tending.

Damage

Causes gradual weakening of mature trees. Trees are killed in young stands (3-5 years old). Damage in larch plantations can reach 25 percent.

Distribution

Northwestern area of European part of Russia, Urals, Siberia, Far East

Control

Cultural

- Create mixed larch plantations with optimal density on optimal sites.
- Select and use resistant species of larch.
- Conduct timely sanitation cuttings and thinnings and remove and destroy slash.

Lachnellula Canker of Siberian Pine Understory

Class/Order: Ascomycetes, Helotiales

Pathogen

Lachnellula pini (Brunch.) Dennis

Hosts

Species of pine (*Pinus*), including Siberian stone (*P. sibirica*), mountain (*P. pumila*), and Scots (*P. sylvestris*)

Diagnosis

Disease development is perennial. The first symptom is significant pitching on stems and branches. Beneath the pitch are small shallow splits in the bark. The wounds deepen but remain covered by pitch (**Fig. 27a**). Dead bark and pitch sloughs, forming open target-like cankers. Cankers occur along the entire length of main stem, but are more common in the middle and lower portion. Cankers also occur on branches. Apothecia, which are cup-like, on short stalks, form aggregately during all stages of canker development. They are covered with short brown hairs. The disks are bright-orange, 2-5 mm in diameter (**Fig. 27b**). Asci are cylindrical, 105 x 20 µ. Ascospores are colorless and ellipsoidal or egg-shaped. Young ascospores are 1-celled; mature ascospores are 2-celled, 10-13 x 5-6 µ. In dry conditions, apothecia are closed and appear as small, brown triangles on the bark of the same color. In moist conditions, apothecia open; they are bright-orange and highly visible.

Biology

Infection occurs by ascospores penetrating woody tissues through natural openings in the bark as well as mechanical injuries, including attacks by insects. The disease occurs over a range of site conditions, but understory plants are affected less than fully insolated trees. The level of disease ranges from 11-56 percent on shaded trees and from 76-85 percent on fully exposed (to the sun) trees. Needles weakened by this canker fungus often are infected by *Lophodermium* needle cast (*Lophodermium pinastri* Chev.), which affects both cedar and mountain pine.

Damage

Causes decline and death of stone pine undergrowth and mountain pine. Affected mountain pine range from 15-85 percent. In undergrowth, about 50 percent of the plants are weakened and up to 40 percent are killed. Infected stone and mountain pine are susceptible to bark beetle and borer attacks, which accelerate decline and mortality and increases the susceptibility of branches to snow breakage.

Distribution

Northwestern part of Europe, Southern area of Siberia, Kamchatka



Figure 27a,b.—Canker on *Pinus pumila* caused by *Lachnellula pini*:
 a) initial stage of development; b) fruiting bodies on dead tree.

Control

- No control methods have been developed.

Stem Rust of Pine

Class/Order: Basidiomycetes, Uredinales

Pathogen

Cronartium ribicola Ditr.

Hosts

Species of pine (*Pinus*) including Siberian stone (*Pinus sibirica*), white (*P. strobus*); occasionally, Austrian (*P. nigra*), Swiss stone (*P. cembra*), and Korean (*P. koraiensis*)

Diagnosis

Initially, the pathogen develops on needles and causes yellow spots to develop. Later, the base of the needle swells and turns orange-yellow. Brown spermogonia form at the base of needles; aecia later develop at the same site. Distinct blister-like aecia are yellow-orange, about 10 mm long and 0.5-0.7 mm high. They release smooth, round or angular aeciospores with colorless walls and orange contents, 22-29 x 18-22 μ . Hypertrophied areas form on the stems and branches (Fig. 28) and gradually become open cankers.



Figure 28.—Blister rust on pine (*P. sibirica*) caused by *Cronartium ribicola*. Blister-like asci on the stem are visible.

Biology

Aecia develop on pine in spring or early summer. Mature aeciospores are released and infect species of *Ribes*. Numerous orange, cushion-like uredinia form on the undersides of *Ribes* leaves and produce urediniospores that reinfect *Ribes* leaves during the summer. In late summer, telia form on the same leaves. Telia have a columnar structure, about 3-4 mm high, and cover the entire lower surface of the leaf. After a short resting period, teliospores germinate and produce basidia with basidiospores from the end of summer to late autumn. Basidiospores infect the pine species.

Damage

Causes weakening and dieback of seedlings in nurseries, young forest plantations, understory trees, and trees in parks, squares, and botanical gardens. Lower branches die back rapidly. Damage of seedlings in nurseries can reach 60 percent.

Distribution

European part of Russia and Siberia

Control

Observation

- Survey in early summer for aecia on stems and twigs.

Cultural

- Create mixed-species pine plantations.
- Choose sites for new nurseries and pine plantations in areas without *Ribes* species and eliminate *Ribes* species near pine nurseries and plantations.
- Prune diseased branches (twigs) on plants when rust cankers first appear.

Broom Rust of Fir

Class/Order: Basidiomycetes, Uredinales

Pathogen

Melampsorella cerastii Wint.



Figure 29.—Broom rust of fir (*Abies alba*), caused by *Melampsorella cerastii*. Stem swelling is visible.

Hosts

Species of fir (*Abies*) including silver (*A. alba*), Siberian (*A. sibirica*), balsam (*A. balsamea*), concolor (*A. concolor*), Nordmann (*A. nordmanniana*) and Sachalin (*A. sachalinensis*).

Diagnosis

The pathogen causes galls or spindle-shaped swellings to form on branches (Fig. 29). Longitudinal cracks develop on these swellings and gradually widen and deepen. Damaged bark later sloughs off. Several galls may form along the stem. Numerous “witches’-brooms” are produced (Fig. 30). Spermogonia and aecia develop along the primary rib on needles of the broom. Aecia are round or cylindrical, orange, small, cushion-like. Aeciospores are globose or elliptical, orange, with colorless walls, 16-30 x 14-20 μ . Damaged needles of witches’-brooms usually drop in winter.

Biology

In spring, needles are infected by basidiospores formed from telia produced on the previous-year leaves of chickweed species *Stellaria* and *Cerastium*. Initially, the fungus damages young shoots and twigs and causes slight spindle-shaped swellings. The next-year buds on the diseased twig produce numerous short shoots called witches’-brooms. From the infected twigs, mycelium penetrates into the stele, develops for several years, and causes galls to develop. The galls grow slowly, can become large, and encircle the stem. Depending on the region, in mid- to late summer, aecia develop on the needles or on short shoots of witches’-brooms. The orange aeciospores are wind dispersed to the leaves of the alternate host plant, where uredinia and telia develop. Telia overwinter on the dead leaves of the alternate host and teliospores germinate in spring, producing basidia and basidiospores.



Figure 30.—Broom, caused by *Melampsorella cerastii* on a young fir (*Abies nordmanniana*).

Most rust infection occurs in clean, low-density fir stands, in stands where the alternate host predominates as ground vegetation, in small woodlots near housing settlements, and in gardens near the forest edge.

Damage

Infected understory trees have stunted growth and branch breakage is significant. Infected mature trees do not show symptoms for several decades. Diseased trees can have a dry crown or dry top. Cankers can be colonized by decay fungi such as *Phellinus hartigii* (Allesch. et Schnabl.) Pat., *Pholiota adiposa* (Fr.) P. Kumm., and *Hericium corolloides* (Scop. Fr.) Gray. Wounds from cankers and decay development can cause considerable losses of merchantable wood. Infection also promotes colonization of the stem by insects (bark beetles, borers, and weevils). The most serious insect pest is the bark weevil *Pissodes piceae* Tll. Insects attack living tissue near dead margins of cankers and increases the susceptibility of branches to wind breakage.

Distribution

European part of Russia, Urals, Siberia, Far East

Control

- Conduct salvage cuttings in disease centers and remove dead, suppressed, and infected trees.

Perennial Nectria Canker

Class/Order: Ascomycetes, Hypocreales



Figure 31.—Cankers caused by *Nectria galligena* on the trunk of a maple (*Acer platanoides*).

Pathogen

Nectria species, including *N. galligena* Bres., *N. ditissima* Tul., and *N. coccinea* (Pers.: Fr) Fr.

Hosts

Species of maple (*Acer*), beech (*Fagus*), oak (*Quercus*), apple (*Malus*), pear (*Pyrus*), hornbeam (*Carpinus*), poplar (*Populus*), and plum (*Prunus*)

Diagnosis

Cankers form on stems and branches; several may occur on the same tree. Initially, cankers are closed but later the bark cracks and sloughs off, exposing the wood. Infection occurs annually, giving the wood a “target-like” appearance (Fig. 31). White, cushion-like sporodochia of the conidial stage form between the bark cracks. Dark-red perithecia develop singly or in groups at the same site. Conidia are colorless, cylindrical, straight, with several septa, 54-62 x 5-6 μ . Asci are clavate. Ascospores are colorless, ellipsoid, 2-celled, 15-21 x 6-8 μ .

Biology

Conidia develop in spring and autumn during moist periods. Maturation and dispersion of ascospores may occur throughout the year. Ascospores overwinter and do not lose germinative power and viability. Ascospore discharge occurs after rain and fog. Spores penetrate into tree tissues at the juncture of dead branches with the stem or in cracks in dead bark. Sunscald, frost, mechanical damage, and insect injury can kill the bark. Long summers and mild winters with high levels of precipitation favor disease development.

Damage

Weakens but rarely kills trees. Fruit orchards suffer extensive damage. In beech understory, weakness and stem breakage occur near the canker. In addition, the cankers are colonized by wood-decay fungi.

Distribution

European part of Russia, Urals, Siberia, Far East

ControlCultural

- Conduct timely sanitation cuttings and thinnings and remove and destroy felling residues.
- Create mixed stands and maintain optimal stand density.
- Minimize mechanical injuries during tending activities.

Black Hypoxylon Canker**Class/Order: Ascomycetes, Sphaeropsidales****Pathogen**

Hypoxylon pruinaum (Kl.) Cke.

Hosts

European aspen (*Populus tremula*), white poplar (*P. alba*), and poplar hybrids from the Aegeiros and Tacamahaca selections

Diagnosis

Affects the bark, cambium, and wood of stems and branches. Initially, bark in lesion areas is slightly red. Later, it becomes blistered and cracked, and white liquid flows from the cracks. Cankers form gradually. They are irregularly elongate and extend along trunks and branches a length of 1-1.5 m. The conidial structures develop in bark in 1-1.5 years. They are dark-brown, blister-like pillars, up to 1 mm tall. Conidiophores with conidia form on them. In about 3 years after infection, black, smeared stromata develop inside the bark. Stromata, which are gray-black, multiangular, and up to 1 cm in diameter, contain groups of perithecia (**Fig. 32**). Perithecia are globose or bottle-like, completely immersed in the stromata. Asci are oblong, with stalks, 50-62 x 4-6 μ . Ascospores are elliptic, black, 1-celled, 10 x 6 μ .

Biology

Infection is initiated by both conidia and ascospores. Conidia form from May to August. Ascospores mature and actively discharge in high humidity during late autumn and early spring. The fungus infects tree tissues through injuries to the bark, including those from insects. Poplar species and hybrids differ in degree of resistance, which depends on bark moisture, chemical composition of cell sap, and physical properties of the bark. Disease outbreaks occur in a range of forest site conditions.

Damage

Weakens trees and causes dieback in natural stands, plantations, and urban street plantings. Diseased trees in stands can reach 40 percent. The disease also promotes extensive wind breakage of branches.

Distribution

European part of Russia, Urals, Siberia

ControlCultural

- Conduct timely sanitation cuttings and remove and destroy felling residue.

Cytophoma Canker of Ash**Class/Order: Deuteromycetes, Sphaeropsidales****Pathogen**

Cytophoma pulchella (Sacc.) Guthn.



Figure 32.—Hypoxylon canker of trembling aspen (*Populus tremula*) caused by *Hypoxylon pruinaum*.

Host

European ash (*Fraxinus excelsior*)

Diagnosis

Mycelium develops in trunks in the bark tissues, including cambium, but does not colonize the wood. Initially, oval, sunken, dark areas of bark form on the trunks. Later, the margins of the infected area split and boundaries are sharply defined. Dead bark in the center of the wound remains attached to the wood and does not slough off. As many as 8 cankers can form on one trunk. Stromata with numerous pycnidia develop in the dead bark and appear as dark tubercles. Spore masses that exude from the pycnidia resemble dirty-green ribbons. Spores are allantoid, colorless, 4-6 x 1-2 μ .

Biology

The disease is most severe in weakened stands. Conidia are dispersed by wind, rainwater, and insects. Infection occurs through old dead branches, leaf scars, and bark injuries.

Damage

Weakens and occasionally causes dieback of trees 7-15 years old. Coppice stands are damaged severely; up to 80 percent of the trees may be affected.

Distribution

Southeastern area of the European part of Russia