

**Data sheets on quarantine pests**  
**Fiches informatives sur les organismes de quarantaine**

## ***Stegophora ulmea***

### **Identity**

**Name:** *Stegophora ulmea* (Fries) Sydow & Sydow

**Synonyms:** *Gnomonia ulmea* (Fries) Thümen, *Sphaeria ulmea* Fries, *Dothidella ulmea* (Fries) Ellis & Everhart, *Lambro ulmea* (Fries) E. Müller

**Taxonomic position:** Fungi: Ascomycetes: Diaporthales

**Notes on taxonomy and nomenclature:** the anamorph is of acervular type, containing both macroconidia, of 'Gloeosporium' type, and microconidia, of 'Cylindrosporella' type. Various anamorph names in different form-genera have been used ('Gloeosporium' *ulmeum* 'Gloeosporium' *ulmicolum*, *Cylindrosporella ulmea*, *Asteroma ulmeum*), but it is not clear which is appropriately to be preferred. Besides, other 'Gloeosporium' spp. have been described from elm (Stipes & Campana, 1981)

**Common names:** black spot of elm, twig blight, elm leaf scab, elm leaf spot, anthracnose (English); anthracnose de l'orme (French); Ulme-Blattfleckenkrankheit (German); antracnosis del olmo (Spanish)

**EPPO code:** GNOMUL

**Phytosanitary categorization:** EPPO A1 list no. 315

### **Hosts**

The fungus causes a serious foliar disease on many elm (*Ulmus*) species (Stipes & Campana, 1981; McGranahan & Smalley, 1981, 1984a). In North America, *S. ulmea* is most virulent and conspicuous on *Ulmus americana* (American white elm). The following are also known to be hosts of *S. ulmea*: *Ulmus alata* (winged elm), *Ulmus carpinifolia* (European field elm), *Ulmus crassifolia* (cedar elm), *Ulmus glabra* (Wych elm), *Ulmus hollandica* (Dutch elm), *Ulmus japonica* (Japanese elm), *Ulmus laciniata*, *Ulmus laevis* (European white elm), *Ulmus parvifolia* (Chinese elm), *Ulmus procera* (English elm), *Ulmus pumila* (Siberian elm), *Ulmus serotina*, *Ulmus thomasi* (rock elm), *Ulmus rubra* (slippery elm). In addition, *Zelkova serrata*, an ornamental Ulmaceous tree, which is also grown for timber, is susceptible (Sinclair *et al.*, 1987).

### **Geographical distribution**

Sinclair *et al.* (1987) gives the main account of distribution. The fungus is considered to be native in North America and

widespread from the Great Plains to the Atlantic Ocean. Sydow (1936) reported a foliar disease of *Ulmus davidiana* caused by *Stegophora aemula* in China stating that the pathogen differs from 'the closely related *Gnomonia ulmea*' by the 'mode of growth' on elm. Since, 1999, *S. ulmea* has repeatedly been detected in consignments of bonsais from China, in UK and the Netherlands, suggesting that the pathogen probably occurs in China. In Europe, there is a doubtful record of '*G. ulmicolum*' on leaves and fruits of elm in Romania (Georgescu & Petrescu, cited by Peace (1962)), which has not been confirmed since. In the Netherlands, *S. ulmea* was introduced into a glasshouse in 2000, on ornamental bonsais, but was successfully eradicated (OEPP/EPPO, 2001)

**EPPO region:** Netherlands (found in one glasshouse in 2000 and successfully eradicated), Romania (doubtful)

**North America:** Canada (at least Quebec, Nova Scotia, but likely to occur in other provinces), USA (widespread from the Great Plains to the Atlantic Ocean)

**Asia:** China (no published record but possibly present, since repeatedly detected on exported bonsais)

**EU:** absent

### **Biology**

Sinclair *et al.* (1987) give many details on the biology of *S. ulmea*. The disease cycle is initiated in the spring when new leaves are infected with ascospores from overwintered perithecia in leaf debris. An additional, unidentified, source of inoculum, thought to originate from overwintered buds, can result in severe infection (McGranahan & Smalley, 1984b). Ascospores are released into the air following a period of alternating wetness and drying after several days with temperatures of at least 7°C. Ascospore release is synchronized with foliar development of elms. The ascospores germinate optimally at temperatures of approximately 8°C (experimental data shows a reduction in germination above 16°C with total inhibition at 24°C). Ascospores infect the lower leaves and twigs. Primary infection can occur from spring onwards and early infections can be damaging in wet conditions. There are conflicting opinions on the resistance of elms to primary infection and subsequent disease development. For example, under laboratory conditions, McGranahan & Smalley (1984b) found that young expanding leaves near the bud were most susceptible to infection, and that frequency of lesions declined

with leaf maturity. However, Pomerleau (in McGranahan & Smalley, 1984b) gives a contrasting account where frequency of leaf spots increased with maturity. As Pomerleau's observations were of elms in the field, they are of greater significance to forest pathology. Following infection by ascospores, yellow lesions (1 mm diameter) develop on the upper surfaces of the leaves (Sinclair *et al.*, 1987). Pomerleau also observed an increase in lesion frequency 2–3 weeks after a wet period. Acervuli develop at the centre of disease lesions and black stromata (0.5 mm diameter) form beneath the acervuli. Several acervuli and stromata can coalesce to form irregular black masses of 5 mm diameter surrounded by a narrow band of whitish dead tissue.

During spring and early summer, secondary inoculum is produced when acervuli split to liberate a white mass of macroconidia (*Gloeosporium ulmicolum*). The macroconidia mature 10–20 days after primary infection. Macroconidia germinate well over a wider temperature range than ascospores (4–28°C) with no particular optimum. Moisture is important in secondary infections. Rain splash is presumed necessary for dispersal of macroconidia from lower to upper leaves. The disease can be inhibited during dry summers. In addition to leaves, petioles, stems and green fruits of elm can be invaded by the fungus, which can result in leaf and twig blight and crumpling of the fruit.

Microconidia (*Cylindrosporella ulmea*) are produced in lesions during midsummer, and act as spermatia in the sexual cycle of the fungus. Perithecia begin to develop in lesions during late summer and autumn and development continues in senesced leaves, where the perithecia overwinter. In parts of America where evergreen elms are grown, for example Chinese elm (*U. parvifolia*), symptoms of the disease are present all year.

## Detection and identification

### Symptoms

Initial diagnostic symptoms on elms are small yellow spots (1 mm diameter) which turn black as stromata develop, hence the common name 'black spot'. The black spots measure up to 5 mm in diameter. Other symptoms are premature shedding of leaves and, in a severe infection, blight of young leaves and shoots and complete defoliation by early August. Green fruits can become infected and crumpled. Disease symptoms can be observed throughout the year in species of evergreen elm.

### Morphology

Macroconidia are hyaline, unicellular spores 8–10 × 3–3.5 µm and microconidia measure 4–6 × 1–2 µm. Measurements of the two-celled, hyaline ascospores are not recorded. The flask-shaped perithecial bodies are 200–385 µm wide and 150–230 µm deep with beaks measuring 80–100 µm in length (Sinclair *et al.*, 1987).

## Detection and inspection methods

Inspection of leaves in the spring would be the most effective method of detecting the disease. Yellow lesions and black stromata on leaves are typical diagnostic symptoms and can be seen throughout the season. Leaf and twig blight and premature defoliation are also indications of infection. Microscopic inspection of leaves can be used to confirm the presence of the fungus.

McGranahan & Smalley (1984a) developed techniques for isolation of the fungus which had previously been considered to be an obligate biotroph. Initial isolation from infected leaves was by plating surface-sterilized leaf discs onto solid media. Mycelial growth on solid media was slow but optimum on potato dextrose or oatmeal agar. Mycelial growth was optimum at 16–24°C. Macroconidia formed in culture. *In vitro* germination of ascospores was optimum at 8°C and macroconidia germinated between 8 and 28°C. Microconidia did not germinate under *in vitro* conditions.

## Pathways for movement

Overwintered ascospores in perithecia within leaf debris and an unidentified inoculum source in dormant buds are the primary sources of inoculum. Ascospores are forcibly expelled from perithecia and are airborne. They are presumed to have the potential to spread over a longer distance than the secondary inoculum, the macroconidia, which are presumed to be locally splash-dispersed throughout an infected tree from lower to upper leaves. As the fungus can survive in leaves, twigs, fruits and dormant buds, movement over long distances could occur via plants for planting, cut foliage and bonsai plants.

The recent detections of *S. ulmea* on imported bonsais (*U. parvifolia*) show that the pathogen has a pathway to enter the EPPO region. It is conceivable it could be introduced to nursery elm stocks if diseased bonsai elms or Japanese *Zelkova* were stored in close proximity. Transplanted, infected trees could be a source of inoculum each season if senesced leaves are not removed and inoculum was allowed to build-up. This would introduce the fungus to the environment and would eventually result in infection of elms in amenity or forest situations where these exist.

## Pest significance

### Economic impact

Black spot was the best known disease of elms in North America prior to the advent of Dutch elm disease, but the effects of infection are rarely fatal on mature elms and recovery has been observed even where repeated defoliation has occurred (Sinclair *et al.*, 1987). In the 1930s, nursery stocks of young *U. americana* in northern Illinois were severely damaged by infection by the fungus in combination with '*Gloeosporium inconspicuum*'. The worst damage was

attributed to '*G. inconspicuum*' (Trumbower, 1933). *S. ulmea* can cause significant defoliation and twig dieback on susceptible elm cultivars in nurseries.

### Control

There is no recent information on fungicidal control of this disease. Cultural control involves reduction of inoculum by removal of all leaf debris, which might be achieved in nurseries, or managed parks and gardens, but not in other situations. If leaf infection has already occurred, the pathogen can in any case survive in dormant buds. On nurseries where overhead watering is practised, cessation of such watering would reduce inoculum spread.

Resistance to *S. ulmea* varies greatly between species and is genetically determined. Most elm species grown in North America are susceptible to *S. ulmea*. McGranahan & Smalley (1981) ranked nine elm species in the Arlington arboretum in order of increasing susceptibility to *S. ulmea* thus: *U. thomasi*, *U. laciniata*, *U. parvifolia*, *U. pumila*, *U. carpinifolia*, *U. japonica*, *U. americana*, *U. glabra*, *U. laevis*. In Wisconsin a similar study ranked nine species in order of increasing susceptibility thus: *U. thomasi*, *U. laciniata*, *U. parvifolia*, *U. pumila*, *U. carpinifolia*, *U. japonica*, *U. americana*, *U. glabra* and *U. laevis* (Sinclair *et al.*, 1987). The European species (*U. laevis* and *U. glabra*) were the most susceptible to the disease in both studies.

### Phytosanitary risk

*S. ulmea* is not present on European elms. The European species *U. laevis* and *U. glabra* are highly susceptible to *S. ulmea*. Both these species are already severely depleted in many European countries by the epidemic of Dutch elm disease (*Ophiostoma novo-ulmi*). Though an elm-breeding programme, initiated by British, Dutch and Americans scientists, has produced several cultivars which are thought to be resistant to *O. novo-ulmi*, several of these are crosses of species also named as being particularly susceptible to black spot (*S. ulmea*). For example, F1 hybrids of *U. pumila* × *U. japonica* ('Sapporo Autumn Gold') and *U. pumila* × *U. carpinifolia* ('Regal') have been developed with good resistance to *O. novo-ulmi* and were on sale in the UK from 1988. The Canadians have also released selections of *U. japonica* for replacing elms lost to Dutch elm disease (Burdekin & Rushworth, 1988). All these *Ulmus* species were identified as being susceptible to *S. ulmea* (McGranahan & Smalley, 1981).

This suggests that *S. ulmea* has the potential, in Europe, to be damaging to bonsais, to elm nursery stocks, and to amenity and

forest elm populations. Damage would principally be a reduction in quality since the symptoms on leaves, fruits and twigs are unsightly rather than fatal. However, for ornamental plants, quality losses could reach 100%.

It may be noted that uncertainty remains about the distribution of *S. ulmea* in China, and possibly elsewhere in the Far East (even falling in the EPPO region). Since the import of bonsai *Ulmus* from the Far East probably represents a greater volume of trade, under less controlled conditions, than that of breeding material of *Ulmus* from North America, it is important to resolve this uncertainty.

### Phytosanitary measures

*S. ulmea* was added in 2003 to the EPPO A1 action list, and endangered EPPO member countries are thus recommended to regulate it as a quarantine pest. It is suggested that plants for planting of *Ulmus* should be dormant and free from leaves, and either originate from a pest-free area, or else from a pest-free place of production where the plants were fungicide-treated.

### Acknowledgements

This data sheet was originally drafted by C. Sansford, Central Science Laboratory, York (GB).

### References

- Burdekin DA & Rushworth KD (1988) *Breeding Elms Resistant to Dutch Elm Disease*. Arboriculture Research Note 2/88/Path. Arboricultural Advisory and Information Service Department of the Environment, London (GB).
- McGranahan G & Smalley EB (1981) Relative susceptibility of elm species to black spot. *Phytopathology* **71**, 241 (Abstract).
- McGranahan G & Smalley EB (1984a) Conidial morphology, axenic growth and sporulation of *Stegophora ulmea*. *Phytopathology* **74**, 1300–1303.
- McGranahan G & Smalley EB (1984b) Influence of moisture, temperature, leaf maturity and host genotype on infection of elms by *Stegophora ulmea*. *Phytopathology* **74**, 296–300.
- OEPP/EPPO (2001) Phytosanitary incidents reported on bonsais and pot plants in the Netherlands. *EPPO Reporting Service*, 2001/045. EPPO, Paris (FR).
- Peace TR (1962) *Pathology of Trees and Shrubs*, p. 427. Clarendon Press, Oxford (GB).
- Sinclair WA, Lyon HH & Johnson WT (1987) *Diseases of Trees and Shrubs*. Comstock Publishing Associates, London (GB).
- Stipes RJ & Campana RJ (1981) *Compendium of Elm Diseases*. American Phytopathological Society, St Paul (US).
- Trumbower JA (1933) Control of elm leaf spots in nurseries. *Phytopathology* **24**, 62–73.