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Data Sheets on Quarantine Pests

Didymella ligulicola

IDENTITY

Name:*Didymella ligulicola* (K.F. Baker, Dimock & L.H. Davis) von Arx
Synonyms: *Mycosphaerella ligulicola* K.F. Baker, Dimock & L.H. Davis
Anamorph: *Ascochyta chrysanthemi* F.L. Stevens
Taxonomic position: Fungi: Ascomycetes: Dothideales
Common names: Ray (flower) blight of chrysanthemum (English)
Ascochyta du chrysanthème (French)
Ascochyta-Krankheit der Chrysantheme (German)
Ascoquita del crisantemo (Spanish)
Notes on taxonomy and nomenclature: The name *Didymella chrysanthemi* (Tassi) Garibaldi & Gullino has been widely used, based on the supposed identity between the organism described in the USA and a little-known fungus described in Italy (*Sphaerella chrysanthemi* Tassi). Walker & Baker (1983) have shown that the American species is distinct and did not reach Europe until the 1960s.
Bayer computer code: MYCOLG
EPPO A2 list: No. 66
EU Annex designation: II/A2

HOSTS

Principal hosts of *D. ligulicola* are florists' chrysanthemums (*Dendranthema* spp.), mainly *D. morifolium*. Endives, *Dahlia pinnata*, globe artichokes, lettuces, *Rudbeckia hirta*, sunflowers and *Zinnia elegans* can be infected by artificial inoculation (Punithalingam, 1980).

Within the EPPO region the potential hosts of *D. ligulicola* would be chrysanthemums grown under glass and outdoors.

GEOGRAPHICAL DISTRIBUTION

Though Boerema & Van Kesteren (1974) have suggested that *D. ligulicola* was present in Italy before introduction from the USA, Walker & Baker (1983) consider the fungi involved to be distinct (see Notes on taxonomy and nomenclature). On this basis, the ray blight fungus originated in North America and the disease is indeed American ray blight.
EPPO region: Belgium, Denmark (found in the past but eradicated), Finland (found in the past but not established), France, Germany, Ireland, Israel, Italy, Luxembourg, Moldova, Netherlands, Norway, Romania, Tunisia, UK (including Northern Ireland, Guernsey and Jersey), Yugoslavia. Intercepted in Bulgaria and the Czech Republic.
Asia: Israel, Japan (Honshu).
Africa: Kenya, Malawi, Tanzania, Tunisia, Zimbabwe.
North America: Canada (Ontario), Mexico, USA (California, Florida, Mississippi, North Carolina, New York, Ohio, Pennsylvania, South Dakota; first observed in North Carolina in 1904).

Oceania: Australia (New South Wales, Queensland), New Zealand, Papua New Guinea.
EU: Present.
Distribution map: See IMI (1993, No. 406).

BIOLOGY

The fungus overwinters as mycelium or, more importantly, as spores; once established, it can survive long periods of drying and low temperatures (-29°C). The principal source of primary inoculum is ascospores, which mature during the winter andearly spring in pseudothecia on diseased tissue. Ascospores, which are discharged throughout the season and carried in air currents, cause scattered infections. Between 10 and 30°C, under experimental conditions, immature pseudothecia developed in 13 days from actual infection (in 3 days at 26°C); thus, maturation and expulsion may also be rapid. In some American isolates, release of ascospores is inhibited by light. Pseudothecia are reported to be rarely found in France.

Pycnidia form readily and abundantly on infected flower buds and peduncles and less so on stems and leaves. Pycnidia have been observed to develop under extremely dry conditions (in 18 weeks at 6% RH), although conidia are only dispersed in humid conditions. Conidia exude in gelatinous drops and are spread by rainsplash, misting, on black cloth covers or by workers, and cause characteristic streaks of infection in the crop. Given sufficient moisture, these spores can infect petals within 6 h and over a wide temperature range, 6-30°C. Conidia penetrate directly through or between epidermal cells, and a characteristic, much branched, short-celled mycelium quickly grows through the tissue, both intra- and intercellularly, causing a moist, brown decay. A phytotoxin is produced. For more information see Stevens (1907), McCoy (1971), Grouet (1974).

DETECTION AND IDENTIFICATION

Symptoms

All plant parts, including roots, may be attacked, but flowers and cuttings are particularly susceptible.

On cuttings

Cuttings are usually attacked at the terminal bud, whence infection spreads downwards to the whole plant. Unopened buds, bracts and stem tissue become darkened. On leaves, the fungus causes irregular brownish-black blotches, 2-3 cm across. Under favourable conditions, these rapidly coalesce and the leafrots. On stems, symptoms are associated with positions where the diseased leaves adjoin, with wounds, or at the cutting base. During rooting, symptom development may be arrested, but diseased tissues remain on the plant and constitute a dangerous source of inoculum.

On adult plants

Stem lesions, which may girdle the stem and are often localized at the base or nodes, are associated with an abnormalappearance in the corresponding shoots, without the latter being contaminated by the fungus. This is due to production of a phytotoxin which induces drooping of terminal growth, makes leaves smaller, chlorotic and more or less brittle, and causes slight dwarfing.

On flowers

Following infection, spots develop, initially on one side ofthe blossom only. The spots appear reddish on light-coloured cultivars and brownish on darker ones. Infection subsequently spreads rapidly and complete rotting of the flower head may occur, the infected florets sticking together. The fungus then grows down the peduncle, blackening and weakening the tissue, so that the head eventually droops and wilts.

Flower and leaf symptoms may be confused with those due to *Botrytis cinerea*, while rotting of cuttings resembles that due to *B. cinerea* or *Pythium* infection. In case of doubt, reproductive structures should be carefully examined. *B. cinerea* is distinguishable by the copious grey spores it produces. Septoria leaf spots have more definite lesion margins and the central areas have a characteristic sheen. For more information, see Stevens (1907), Nillsson (1963), Sauthoff (1963), Garibaldi & Gullino (1971), Grouet (1974).

Morphology

Pycnidia are visible with a x15 hand lens as depressed, thin-walled, globose bodies of two sizes: small (72 x 180 µm) and aggregated on the petals, and large (111 x 325 µm) and scattered on the stems and leaves.

Pycnidiospores exude in short columns, are hyaline, continuous (10-40%) and septate (60-90%, usually with one septum, occasionally with more), ovoid to cylindrical with a pronounced tendency to irregularity and an extreme variability in dimensions; continuous spores 6-22 x 2.5-8 µm, mostly 8.5-13 x 3.5-5.5 µm; septate spores 9-23 x 3-6.5 µm, mostly 13-15.5 x 4-5 µm. For more information, see Sauthoff (1963), Blakeman & Hadley (1968), Boerema & Bollen (1975).

D. ligulicola shows phialidic ontogeny. Septation of the spores is a secondary process, related to temperature, and is probably a function of spore size. In culture, on oatmeal agar at 20-22°C, with a variable common light-dark cycle, the majority of the pycnidiospores remain one-celled, 3.5-15 x 1.5-3.5 µm, mostly 4-8.5 x 2-3 µm.

Pseudothecia are less commonly found, are round and more erumpent than pycnidia, have dark-brown, thick-walled outer cells and are 96-224 µm in diameter.

Ascospores are hyaline to greyish, fusiform to elliptical, uniseptate, 12-16 x 4-6 µm.

Detection and inspection methods

D. ligulicola can be easily isolated on malt or potato dextrose agar. It develops a greyish-white aerial mycelium and reaches a colony diameter of 65 mm after 7 days of incubation at 24°C (Hahn & Schmatz, 1980).

MEANS OF MOVEMENT AND DISPERSAL

D. ligulicola has a relatively low dispersal potential on its own, but can be transmitted by infected cuttings, plants and flowers of chrysanthemums. Earth attached to roots can also be a source of inoculum.

PEST SIGNIFICANCE

Economic impact

The disease was recorded in North Carolina (USA) in 1904, and remained localized and of little importance until the late1940s when, concurrent with the intensification of chrysanthemum flower and pot plant production, it began to cause serious losses throughout the range. It is now considered the most serious fungal disease of chrysanthemums in Florida.

In 1975, in Connecticut, the disease was reported to be particularly important on chrysanthemum cuttings in propagating benches under mist; 50% losses occurred.

The increasing intensification of chrysanthemum production, with all-the-year-round cultivars, mist benches, use of dark covers, etc., favours spread and development of the disease. In addition, the fungus can develop under a wide range of conditions and, once established, is both difficult and costly to eradicate. The fact that the disease is recorded in California shows that it will persist even in areas with apparently unfavourable climatic conditions.

Control

In Europe, the disease has been controlled successfully with benomyl. However, the repeated and excessive usage of this fungicide over a number of years has led to resistance build-up and a consequent increase in ray blight importance. More recently dicarboximide derivates have been successfully used for control (Engelhard, 1984).

Currently there is no biological control method available. However, certain cultivation and phytosanitary requirements can reduce infection by *D. ligulicola*, especially during the rooting of cuttings (Hahn & Schmatz, 1980).

Phytosanitary risk

D. ligulicola is an A2 quarantine pest for EPPO (OEPP/EPPO, 1982). *D. ligulicola* also has A2 quarantine status for IAPSC. In the EPPO region at present, ray blight is of significant economic importance in countries where it occurs. Widespread establishment of the disease within the EPPO region could cause considerable economic losses to propagators and growers of chrysanthemums. Some countries (e.g. Finland) maintain an eradication programme.

PHYTOSANITARY MEASURES

Only chrysanthemum plants for planting are concerned. In countries where the disease occurs, growing-season inspections should be carried out, especially during rooting of cuttings, but also on mother plants and at flowering. Rooted or unrooted cuttings should come from rooting beds or plants, respectively, which were found free from *D. ligulicula* during the last growing season (OEPP/EPPO, 1990). Symptoms may develop in transit on blooms which are apparently healthy when cut.

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