

# EPPO Datasheet: *Stagonosporopsis chrysanthemi*

Last updated: 2023-12-05

## IDENTITY

**Preferred name:** *Stagonosporopsis chrysanthemi*

**Authority:** (F. Stevens) P.W. Crous, N. Vaghefi & P.W.J. Taylor

**Taxonomic position:** Fungi: Ascomycota: Pezizomycotina:  
Dothideomycetes: Pleosporomycetidae: Pleosporales: Didymellaceae

**Other scientific names:** *Ascochyta chrysanthemi* F. Stevens,  
*Didymella ligulicola* (Baker, Dimock & Davis) von Arx,  
*Mycosphaerella ligulicola* Baker, Dimock & Davis, *Phoma*  
*ligulicola* var. *ligulicola* Boerema, *Stagonosporopsis ligulicola* var.  
*ligulicola* (Baker, Dimock & Davis) Aveskamp, Gruyter & Verkley

**Common names:** flower blight of chrysanthemum, ray blight of  
chrysanthemum, stem canker of chrysanthemum

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**EPPO Categorization:** A2 list

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**EPPO Code:** MYCOLG



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## Notes on taxonomy and nomenclature

Not to be confused with *Didymella chrysanthemi* (Tassi) Garibaldi & Gullino (syn. *Sphaerella chrysanthemi* Tassi = *Mycosphaerella chrysanthemi* (Tassi) Tomilin). The name *Didymella chrysanthemi* has been erroneously used as synonymous to *Didymella ligulicola* (for example, see Punithalingam, 1980). This was based on a study by Garibaldi & Gullino (1971) that reported morphological identity of the ray blight pathogen described in the USA (at the time known as *Didymella ligulicola*) and a little-known fungus described in Italy (at the time known as *Sphaerella chrysanthemi*). Later, Walker & Baker (1983) studied the type specimen of both species and demonstrated that the American species was morphologically distinct and rejected the synonymy of *Didymella ligulicola* and *Didymella chrysanthemi* suggested by Garibaldi & Gullino (1971). For more information see Walker & Baker (1983) and Vaghefi *et al.* (2016b).

Taxonomy of the ray blight pathogen was revised in 2012 based on multi-locus phylogenetic analyses (Vaghefi *et al.*, 2012). Currently, three *Stagonosporopsis* species are recognized as the cause of ray blight disease on Asteraceae. *S. chrysanthemi* is identified as the cause of ray blight on chrysanthemums while *S. inoxydabilis* and *S. tanacetii* are recognized as the cause of ray blight on other Asteraceae species in Europe and Australia, respectively (Vaghefi *et al.*, 2016b).

## HOSTS

The principal hosts of *S. chrysanthemi* are florists' chrysanthemums, *Chrysanthemum x morifolium* and *C. indicum*, the former being the original host on which this pathogen was described (Stevens, 1907). Later, annual chrysanthemum (*Chrysanthemum carinatum*), endives (*Cichorium endivia*), Dahlia (*Dahlia pinnata* and *D. variabilis*), globe artichokes (*Cynara scolymus*), lettuces (*Lactuca sativa* var. *crispa*), rudbeckia (*Rudbeckia hirta*), sunflowers (*Helianthus annuus*), and Zinnia (*Zinnia elegans*) were reported to be infected by artificial inoculation (Chesters & Blakeman, 1967). Pyrethrum (*Tanacetum cinerariifolium*) is also established as an experimental host for *S. chrysanthemi* after *in vitro* inoculation (Vaghefi *et al.*, 2016b). Records established prior to the molecular reclassification of the pathogen in 2012 (Vaghefi *et al.*, 2012) should be treated with caution as the identity of the isolates are not confirmed. For example, DAR 28714 previously identified as *Didymella ligulicola* (syn. *S. chrysanthemi*) on Zinnia (Walker & Baker, 1983) was later reclassified as *Stagonosporopsis caricae* (Marin-Felix *et al.*, 2019).

Within the EPPO region the main potential hosts of *S. chrysanthemi* would be chrysanthemums grown under

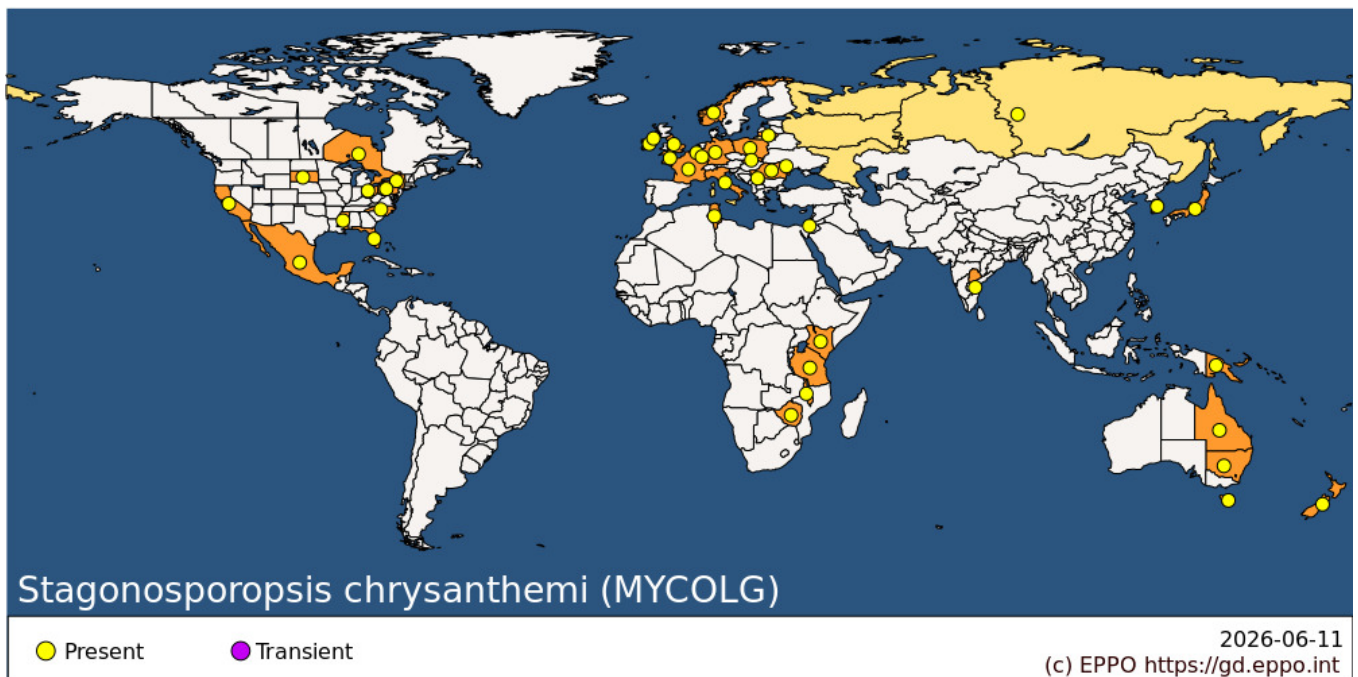
protected conditions and outdoors.

**Host list:** *Chrysanthemum indicum*, *Chrysanthemum lavandulifolium*, *Chrysanthemum x morifolium*, *Lactuca sativa*

## GEOGRAPHICAL DISTRIBUTION

Though Boerema & Van Kesteren (1974) have suggested that *S. chrysanthemi* (*D. ligulicola* at the time) 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 fungus causing ray blight of chrysanthemum originated in North America and is believed not to have reached Europe until the 1960s (Walker & Baker, 1983).

Records dated prior to the molecular characterization of the pathogen (Vaghefi *et al.*, 2012) should be treated with caution as the identity of the isolates cannot be confirmed and some reports of *S. chrysanthemi* were subsequently found to belong to other *Stagonosporopsis* species.



**EPPO Region:** Belgium, France (mainland), Germany, Ireland, Israel, Italy (mainland), Lithuania, Luxembourg, Moldova, Republic of, Norway, Poland, Romania, Russian Federation, Serbia, Slovakia, Tunisia, United Kingdom (Channel Islands, England, Northern Ireland)

**Africa:** Kenya, Malawi, Tanzania, United Republic of, Tunisia, Zimbabwe

**Asia:** India (Andhra Pradesh), Israel, Japan (Honshu), Korea, Republic of

**North America:** Canada (Ontario), Mexico, United States of America (California, Florida, Mississippi, New York, North Carolina, Ohio, Pennsylvania, South Dakota)

**Oceania:** Australia (New South Wales, Queensland, Tasmania), New Zealand, Papua New Guinea

## BIOLOGY

A principal source of primary inoculum contributing to ray blight epidemics is ascospores, which mature during the winter and early spring in pseudothecia on diseased host tissue (Baker *et al.*, 1949). Ascospores are discharged throughout the season and carried in air currents (McCoy, 1973) causing scattered infections through much of the flowering season (Baker *et al.*, 1949). Airborne ascospores are forcibly discharged (McCoy, 1973) and may disperse over longer distances and infect neighbouring fields (Blakeman & Hadley, 1968). Under experimental conditions on chrysanthemum stem and leaf tissue, pseudothecia developed in seven days at 20°C from the time of inoculation (McCoy *et al.*, 1972). Lower temperatures delayed pseudothecia formation (25 days at 10°C) while temperatures higher than 25°C inhibited development of pseudothecia (McCoy *et al.*, 1972). In naturally infected plants,

pseudothecia were reported to be less abundant, but once plant samples were dried and incubated in the laboratory, an increase in the number of pseudothecia was observed within six to eight weeks (Baker *et al.*, 1949).

Pycnidia, which form readily and abundantly on infected flower buds and peduncles and less so on stems and leaves, are also overwintering structures that can survive temperatures as low as -29°C (Baker *et al.*, 1949). Upon artificial inoculation of chrysanthemum leaves and stems in the laboratory, pycnidia developed in three days at 26°C (optimal growth temperature) while pycnidial formation was inhibited at 30°C (McCoy *et al.*, 1972). Pycnidia are produced under both dry and humid conditions (relative humidity of 2 to 85%) but splash-dispersal of pynidiospores occurs only in humid conditions (Baker *et al.*, 1961). Pynidiospores exude in gelatinous drops (cirri) and are spread by rain splash, misting, infested tools and clothing or hands of workers. Infection by waterborne pynidiospores result in localized streaks of infection in the crop (Baker *et al.*, 1949).

Given sufficient moisture, spores can infect petals within 6 hours and over a wide temperature range (6-30°C) but the optimal temperature for infection is 20-26°C (Baker *et al.*, 1949). pynidiospores 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 (Baker *et al.*, 1949). *S. chrysanthemi* produces a phytotoxin that is able to induce leaf symptoms similar to those caused by the pathogen itself (Schadler & Bateman, 1974). For more information see Stevens (1907), McCoy (1971), Vaghefi *et al.* (2016b).

The fungus can survive as epiphytic mycelium on chrysanthemum cuttings for 12 weeks (Chesters & Blakeman, 1966) and, more importantly, as pseudothecia and pycnidia, which are the overwintering organs and can survive over a broad range of temperatures on infected plants outside (-29 to 30°C) (Baker *et al.*, 1949). Sclerotia have been reported to survive in the soil for eight weeks (Blakeman and Hornby, 1966), however, the identity of the isolates is not confirmed. Therefore, this record should be treated with caution as the original descriptions of the ray blight pathogen (Stevens, 1907; Walker & Baker, 1983) did not mention production of sclerotia.

## DETECTION AND IDENTIFICATION

### Symptoms

Ray blight caused by *S. chrysanthemi* affects chrysanthemum under favorable conditions on potted plants, stock beds and cuttings grown in the field and protected areas (Engelhard 1984; Baker *et al.* 1949). Flowers and cuttings are particularly susceptible, but all plant parts are affected and present symptoms (Baker *et al.*, 1949, 1961).

#### *On cuttings*

Cuttings are usually attacked at the terminal bud, from which point the 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 (Baker *et al.*, 1949). Under favourable conditions, these rapidly coalesce and the leaf-rots. On stems, symptoms are associated with the points at which 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 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 abnormal appearance 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 of the 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 (Baker *et al.*, 1949).

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 (caused by *Septoria* spp.) have more definite lesion margins and the central areas have a characteristic sheen. For more information, see Stevens (1907), Nilsson (1963), Sauthoff (1963).

## Morphology

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

*S. chrysanthemi* 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 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.

*S. chrysanthemi* is homothallic and produces abundant pseudothecia in culture and on infected plants incubated in the laboratory for three to eight weeks (Baker *et al.*, 1949; Vaghefi *et al.*, 2016a,b). Pseudothecia are less commonly found on infected stems and flowers exposed outdoors during the winter, and 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

Visual examination in a place or site of production is not effective at detecting *S. chrysanthemi* as the disease may be latent and symptoms may be confused with other diseases (EFSA, 2013). If *S. chrysanthemi* is suspected, samples should be sent to the laboratory for identification.

Isolation can be performed from all plant parts showing symptoms, but also seeds can be plated and the obtained culture characterized and identified. *S. chrysanthemi* can be easily isolated on pea agar and cherry decoction agar (Stevens, 1907; Boerema *et al.*, 2004). For morphological studies, potato dextrose agar (Baker *et al.*, 1949), oat meal agar or malt agar can be used (de Gruyter *et al.*, 2002; Boerema *et al.*, 2004).

Colony diameter on OA approximately 7 cm after seven days, regular to slightly irregular, colourless/greenish olivaceous to dull green olivaceous, often in a zonate pattern; with sparse to abundant, felted to floccose, white to pale olivaceous grey aerial mycelium; reverse grey olivaceous to fawn/hazel or olivaceous grey, more or less discolouring due to a diffusible pigment. On MA, slower growth, 5-6 cm in diameter after seven days, sometimes with a pale luteous pigment production. Only the anamorph of the pathogen is produced in culture (Van der Aa *et al.*, 1990) with pycnidia developing on and in the agar medium (OA).

It should be noted that identification of *S. chrysanthemi* based on micro-morphological features requires expertise and is not always possible due to morphological similarity to other *Stagonosporopsis* spp. that cause ray blight on Asteraceae, thus, molecular sequencing is necessary for correct identification (Vaghefi *et al.*, 2016b). A multiplex PCR test capable of differentiating the three *Stagonosporopsis* species associated with ray blight symptoms on Asteraceae has been developed (Vaghefi *et al.*, 2016a).

An EPPO diagnostic protocol on *Stagonosporopsis chrysanthemi* is under development.

## PATHWAYS FOR MOVEMENT

*S. chrysanthemi* has a relatively low dispersal potential on its own. Pathways for movement include host plants for planting (including seed), cut flowers of host plants and soil and other growing media (EFSA, 2013). Infected cuttings of host plants can be a pathway for movement, as the pathogen is capable of surviving epiphytically on symptomless plant cuttings. Soil and other growing media attached to roots can also be a source of inoculum and therefore potted plants of hosts, and non-hosts with infected soil, can be a pathway for movement.

## **PEST SIGNIFICANCE**

### **Economic impact**

There are no recent reports of the effects of *S. chrysanthemi* on chrysanthemum crops (EFSA, 2013). Historically, the disease has caused economic impacts, and these are recorded for North America. The disease was recorded in North Carolina (USA) in 1904 and remained localized and of little importance until the late 1940s when, concurrent with the intensification of chrysanthemum flower and pot plant production, it began to cause serious losses throughout the distribution range. In North America from 1946-47, the disease was described as destructive (EFSA, 2013). In 1975, in Connecticut, the disease was reported to be particularly important on chrysanthemum cuttings in propagating benches under mist and 50% losses occurred.

*S. chrysanthemi* is present in the EU sporadically but there are no reports of the pathogen causing damage on chrysanthemum crops in the EU (EFSA, 2013). Additionally, EFSA (2013) conclude that the overall impact of *S. chrysanthemi* in the EU is minor.

### **Control**

Cultural practices in places of production can reduce the spread and infection by *S. chrysanthemi*. EFSA (2013) highlight that the use of pathogen-free propagation material, monitoring of the crop, avoiding overhead irrigation, proper management of waste plant material, soil disinfestation with steam, use of clean irrigation water and general hygiene measures can be used to avoid occurrence and spread.

Rapid removal of infected plants can help reduce disease spread.

In Europe, historically, the disease has been controlled successfully with benomyl. However, repeated and excessive usage of this fungicide (and thiophanate-methyl) over a number of years has led to the development of resistant isolates (Punithalingam, 1980). EFSA (2013) note that recent information on fungicides for *S. chrysanthemi* is poor.

Currently there is no biological control method available (CABI, 2012).

### **Phytosanitary risk**

There is the potential for further establishment and spread of *S. chrysanthemi* in the EPPO region (EFSA, 2013). Host plants of *S. chrysanthemi* are popular ornamentals in the EPPO region and are widely grown in public and private gardens. The crop is cultivated in open fields and greenhouses. The Netherlands and Italy are the main European producers of chrysanthemum cut flowers whereas potted plants are produced throughout the EPPO region. Maintaining the current import procedures, production practices and control measures can keep the phytosanitary risk to a minimal level.

## **PHYTOSANITARY MEASURES**

Certified host plant material should be used (see EPPO, 2002). 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 *S. chrysanthemi* during the last growing season. Symptoms may develop in transit on blooms which are apparently healthy when cut. For imported consignments and inspections of places of production of chrysanthemum plants, visual inspection should be conducted and if suspected symptoms are detected, a sample should be sent for

laboratory testing.

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## How to cite this datasheet?

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## Datasheet history

This datasheet was first published in the EPPO Bulletin in 1983 and revised in the two editions of 'Quarantine Pests for Europe' in 1992 and 1997, as well as in 2023. It is now maintained in an electronic format in the EPPO Global Database. The sections on 'Identity', 'Hosts', and 'Geographical distribution' are automatically updated from the database. For other sections, the date of last revision is indicated on the right.

CABI/EPPO (1992/1997) *Quarantine Pests for Europe (1<sup>st</sup> and 2<sup>nd</sup> edition)*. CABI, Wallingford (GB).

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