EPPO Datasheet: *Phialophora cinerescens*

Last updated: 2023-10-10

IDENTITY

Preferred name: Phialophora cinerescens
Authority: (Wollenweber) van Beyma
Taxonomic position: Fungi: Ascomycota: Pezizomycotina:
Eurotiomycetes: Chaetothyriomycetidae: Chaetothyriales:
Herpotrichiellaceae
Other scientific names: Verticillium cinerescens Wollenweber
Common names: phialophora wilt of carnation, wilt of carnation
view more common names online...
EPPO Categorization: A2 list
view more categorizations online...
EPPO Code: PHIACI



more photos...

HOSTS

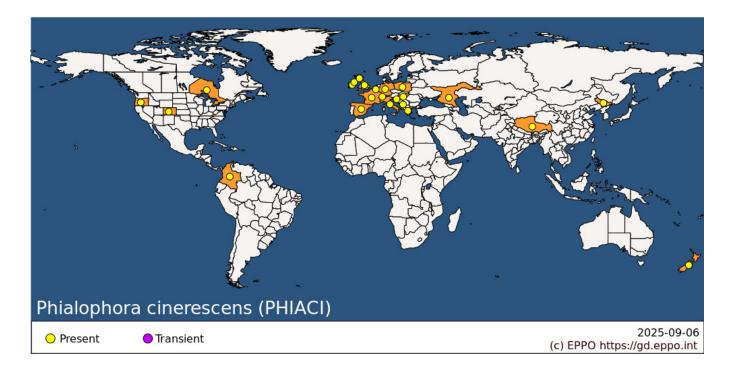
Carnations (*Dianthus* spp.) are the main hosts of *Phialophora cinerescens*. There are more than 45 species in the genus *Dianthus*, and studies have shown that *Dianthus* species and their cultivars presented different levels of susceptibility to *P. cinerescens*. Many *D. caryophyllus* cultivars, as well as several garden species of *Dianthus*, were tested for their response to *P. cinerescens* and could be rated as resistant, moderately resistant, moderately susceptible, or very susceptible (EFSA, 2013; Garibaldi & Pergola, 1975; de Granada *et al.*, 1997; Zenkteler, 1992). As some isolates demonstrated different virulence on carnation cultivars, different races of *P. cinerescens* were proposed (de Granada *et al.*, 1997). Hosts from other plant genera include *Saponaria officinalis*, *Silene compacta, Silene latifolia alba, Petrocoptis grandiflora, Viscaria vulgaris* (Bankina, 1995; USDA, 2010).

However, it should be noted that there is a lack of recent studies on the host–pathogen relationship between *P. cinerescens* and carnations.

Host list: Dianthus anatolicus, Dianthus andronakii, Dianthus arboreus, Dianthus arenarius, Dianthus armeria, Dianthus balbisii, Dianthus barbatus, Dianthus bicolor, Dianthus biflorus, Dianthus capitatus, Dianthus carthusianorum subsp. atrorubens, Dianthus carthusianorum subsp. carthusianorum, Dianthus carthusianorum, Dianthus caryophyllus, Dianthus chinensis, Dianthus collinus subsp. glabriusculus, Dianthus cyri, Dianthus deltoides, Dianthus furcatus, Dianthus gallicus, Dianthus giganteiformis, Dianthus giganteus, Dianthus gratianopolitanus, Dianthus hyssopifolius, Dianthus integer, Dianthus knappii, Dianthus lusitanus, Dianthus nardiformis, Dianthus orientalis, Dianthus pavonius, Dianthus petraeus subsp. noeanus, Dianthus petraeus subsp. petraeus, Dianthus petraeus, Dianthus plumarius, Dianthus rupicola, Dianthus seguieri, Dianthus serotinus, Dianthus spiculifolius, Dianthus sternbergii, Dianthus superbus, Dianthus sylvestris, Dianthus tatrae, Dianthus trifasciculatus, Petrocoptis grandiflora, Saponaria officinalis, Silene compacta, Silene latifolia subsp. alba, Viscaria vulgaris

GEOGRAPHICAL DISTRIBUTION

P. cinerescens is an indigenous European species, although its presence has not been reported in several European countries. The fungus has been documented in certain areas of Asia, New Zealand, and North America, particularly in the United States, and Canada. Numbers of reports of *P. cinerescens* from South America are limited, but the fungus has been identified in some regions of Colombia, especially in tropical and subtropical areas where it may be found in soil and organic matter.



EPPO Region: Belgium, Croatia, France (mainland), Germany, Greece (mainland), Hungary, Ireland, Italy (mainland), Montenegro, Poland, Russian Federation (the) (Southern Russia), Spain (mainland), Switzerland, United Kingdom (England, Northern Ireland, Scotland)
Asia: China (Jilin, Xizhang)
North America: Canada (Ontario), United States of America (Colorado, Oregon)
South America: Colombia
Oceania: New Zealand

BIOLOGY

P. cinerescens is a soil-borne pathogen, with very limited natural spread through infested soil (Akhmed & Sidorova, 1990). This is a slow developing fungus, requiring a relatively low temperature for growth and sporulation *in vitro* (Bankina, 1995). Infection occurs via conidia which penetrate the roots directly through small wounds in the hair roots (Bankina, 1995). When hyphae come into contact with a carnation plant, they penetrate the root tips or enter through wounds and then colonize the vascular tissue. The pathogen invades the vessels and causes changes in the structure of living cells, simultaneously blocking the diseased vessels with polysaccharides and phenolic compounds produced by the host (Wolkan *et al.*, 2016).

P. cinerescens produces abundant, small spores on the ends of phialides grouped in penicillium-like clusters. Sporulation is possible at temperatures of $10-28^{\circ}$ C and reaches its maximum at $18-23^{\circ}$ C (Akhmed & Sidorova, 1990; Bankina, 1995). At low temperatures (e.g. 11° C is indicated as optimal temperature in vitro; Bankina, 1995), more spores are produced but in a longer time, and these spores tend to be larger than those produced at higher temperatures (Akhmed & Sidorova, 1990; Bankina, 1995). The optimum temperatures for the development of the pathogen are $15-20^{\circ}$ C, but the fungus is able to attack plants successfully even at temperatures of $11-15^{\circ}$ C (Garibaldi, 1969; Wolkan *et al.*, 2016).

The incubation period is comparatively long, varying between 17 days (in susceptible cultivars) and 120 days (in more resistant ones) (Bankina, 1995; Wolkan *et al.*, 2016). The fungus is able to survive saprophytically for many years in soil and it is very persistent even in deeper soil layers (Arbelaez, 1988). A few studies indicated that plants became infected by *P. cinerescens* even 16 years after the last case of vascular wilt in the same area (EFSA, 2013). For more information, see Moreau (1970), Hawksworth & Gibson (1998), EFSA (2013).

DETECTION AND IDENTIFICATION

Symptoms

Disease symptoms may vary, but usually the first symptom caused by *P. cinerescens* is a discoloration of leaves and stems which become bluish grey (Hawksworth & Gibson, 1998). Initial symptoms also include chlorosis of lower leaves which may have reddish or brownish pigmentation. Generally, symptoms are similar to those typical to the fusarium wilts and include systemic vascular occlusion progressively resulting in wilt, starting at the base and extending upward (Bankina, 1995; Wolkan et al., 2016). Moreover, another vascular wilt pathogen, Fusarium oxysporum f. sp. dianthi, may be simultaneously present in the infected plant. To differentiate these two pathogens, the morphological examination of pure cultures using light microscopy is needed. P. cinerescens causes a brown discoloration of the vascular system of the stem and plant base (Voronenko et al., 1978; Wolkan et al., 2016). Subsequently, there is a rapid wilt of the whole plant without rotting. The root system remains intact and apparently unaffected (Garibaldi, 1969). Peeling-off the cortex or taking longitudinal or transverse sections of the stem will reveal a browning of the vascular zone (Peresse, 1971). This discoloration tends to be localized to a number of small groups of vessels and tracheids, so appears to be a typical wilt in that longitudinal brown stripes can be seen when the cortex is removed (Garibaldi, 1969). There is no extensive rotting of the pith or cortex (Garibaldi, 1969). The stems may develop sharp twists (kinks) at the nodes and the internodes may be somewhat shortened (Bolton, 1978; USDA, 2010). The incubation period varies significantly between 17 and 120 days and depends on susceptibility of cultivars (Hellmers, 1958; Bolton, 1978; Bankina, 1995). The host susceptibility depends on level of phenolic compounds which is a crucial factor in the resistance of carnations to the fungus (Niemann et al., 1994). For more information, see Hawksworth & Gibson (1998), Arbelaez (1988), and EFSA (2013).

Morphology

Sporulation is mainly nematogenous but plectonematogenous in parts, abundant, conidiophores are septate, clearly differentiated from the vegetative mycelium, strongly branched with the slender phialides forming compact clusters around the hyphae, hyaline becoming pale-brown with age, smooth-walled, mainly 8–20 x 2–3 µm (Hawksworth & Gibson, 1998). Conidiogenous cells (phialides) are flask-shaped and arranged in densely verticillate bunches on the conidiophores, crowded together on the conidiophores giving a brush-like appearance or, in old cultures, arising directly from large conidia, ampulliform, often slightly curved towards the axis of the conidiophore, somewhat inflated below, with a distinct apical collarette with an almost flared appearance, generally hyaline but becoming brownish with age, smooth-walled, variable in size, mainly 8-12 x 2.5-3.5 µm, ending in a darker, very short but distinct collarette with a minute flaring margin (Hawksworth & Gibson, 1998). Conidia formed singly at the apices of the conidiogenous cells, aggregating together with those from adjacent phialides to form dense slimy heads, simple, hyaline to pale brown, smooth-walled, subcylindrical to almost allantoid, generally with one end rounded, not markedly constricted centrally, usually distinctly 2-guttulate but with many very small guttules when young, in young cultures 3–6 (...7) \times 1.5–2.5 (...3) µm, in older cultures thicker walled and darker in colour, variable in size, mainly 4–8 (...12) \times 2.5–3.5 (...4.5) µm; the largest sometimes with ornamented walls and giving rise directly to phialides or acting as pleurophialides (Hawksworth & Gibson, 1998). Hyphae septate, hyaline to pale-brown, 1-3 µm wide; on ageing, frequently develop irregularly swollen cells covered with flat warts and up to 6 µm wide (Hawksworth & Gibson, 1998). These cells have been mistakenly identified as chlamydospores although both chlamydospores and sclerotia absent (Hawksworth & Gibson, 1998).

Colonies of the fungus have pure grey tinge, sometimes have an orange tinge, and become almost black with age, growing slowly on 2% malt extract agar, reaching 1.5–2.0 cm in diameter in 3 weeks at 20°C, tending to be zonate, floccose; characteristically woolly, light mineral-grey in the centre (Hawksworth & Gibson, 1998).

Detection and inspection methods

A rapid and early screening can be done via fluorescence microscopy which shows infected areas of *P. cinerescens* as bright fluorescent spots at the level of the woody vessels (Bonifacio & Rumine, 1984).

P. cinerescens is most often diagnosed by morphological examination of fungal structures using light microscopy (Hawksworth & Gibson, 1988) or through DNA-based identification by sequencing of the ITS region followed by a blast search at GenBank (Wolkan *et al.*, 2016). Identification of *P. cinerescens* is also based on isolation of the fungus from small stem sections of the cuttings (EFSA, 2013).

PATHWAYS FOR MOVEMENT

P. cinerescens is an indigenous European species, which is present in many parts of the EPPO region and in many climates of Europe, Asia, America, and New Zealand although its presence has not been reported in several European countries (EFSA, 2013; EPPO, 2023). The environmental conditions are suitable in most parts of Europe and for most of the host growing season (EFSA, 2013). The fungus spreads through the soil, by splash-dispersed conidia, persistence of infected plant debris in the soil or mycelial growth (Hawksworth & Gibson, 1998), so as a soil-borne pathogen, *P. cinerescens* has an extremely limited potential for natural spread. The main pathway for spread is international trade in carnation cuttings. However, current cultural practices and control measures strongly reduce the probability of infection of carnations, therefore, spread to new areas is considered very unlikely (EFSA, 2013).

PEST SIGNIFICANCE

Economic impact

Nowadays, the damage caused by *P. cinerescens* to carnations is very low and unlikely to increase in the future because the existing control measures provide a high efficiency to stop the spread of the pathogen. The disease at this moment is either absent, or eradicated, or present with limited distribution in the majority of the European countries (EFSA, 2013; EPPO, 2023). No negative environmental consequences are foreseen. Due to the declining importance of phialophora wilt of carnation worldwide, there have been very limited studies on the disease in recent decades. However, the disease was once of great economic importance in many carnation-cultivating areas of the EPPO region. In Colombia, this fungus was formerly regarded as one of the two most important pathogens of carnations (Arbelaez, 1988).

Control

Control of this pathogen can be achieved by soil disinfestation by physical (heat) or chemical (fumigants) means, by fungicide drenching and root dipping and by application of systemic fungicides to soil. Moreover, there are several highly effective and highly feasible cultural practices as growing the crop in raised benches, use of artificial substrates and individual containers, soil/substrate disinfestation and use of pest-free irrigation water (Wolkan *et al.*, 2016). Soil solarization using clear plastic mulch is a common pre-plant practice in current agricultural systems in conducive climates to destroy soil pathogens, including *P. cinerescens* (Wolkan *et al.*, 2016).

Clones of carnation cultivars resistant to *P. cinerescens* and screening of the resistant seedling were used (Leus, 2018). In practice, the disease is mainly controlled by use of disease-free planting material from mother plants derived from nuclear stock subjected to meristem tip culture. A protocol for certification of carnation cuttings is described in detail in PM 4/2(2) (EPPO, 2002).

Phytosanitary risk

The risk of spread to new area (for the EU) was assessed by the EFSA Panel (EFSA, 2013) as very low: the pathogen is present but not widely distributed and it is under official control on carnation. Moreover, the pest has a low impact nowadays because certified plant material (i.e., cuttings) is used in the propagation of carnation and the crop is grown on disinfested substrates/soil on raised benches (Suffert, 2012; EFSA, 2013). *P. cinerescens* is no longer included in the EU plant health law (Regulation 2016/2031). It remains, however, in quarantine lists of several countries in Africa, America, Asia, and Europe.

PHYTOSANITARY MEASURES

In the countries where *P. cinerescens* occurs, growing season inspections should be carried out and cuttings should be taken from separately grown mother plants which have been tested by an EPPO-approved method (EPPO, 2002, 2023).

REFERENCES

Arbelaez G (1988) Control of *Fusarium oxysporum* and *Phialophora cinerescens* on carnation by combined soil treatment and application of antagonists. *Acta Horticulturae* **216**, 77–84.

Akhmed D & Sidorova S (1990) Morphological and cultural variability and virulence causative agent of clove phyalophorosis. *Mikologiya i Fitopatologiya* **24**(**3**), 248–251 (in Russian).

Bankina B (1995) Distribution and biological characteristics of *Phialophora cinerescens*, the causative agent of carnation in Latvian. Abstract of a PhD dissertation. Riga: Latvian University, 84 pp. (In Latvian).

Bolton AT (1978) Fan-mould of carnation caused by *Phialophora cinerescens*. *Canadian Plant Disease Survey* **58** (4), 83–85.

Bonifacio A & Rumine P (1984) [Use of the fluorescence microscope in the diagnosis of vascular diseases of carnation]. *Informatore Fitopatologico* **34**, 43–44.

de Granada EG, de Amézquita MO, Calderón OL & Arbeláez G (1997) Response of some standard carnation varieties to *Phialophora cinerescens* (Wollenw.) van Beyma. *Agronomía Colombiana* **14**(2), 149–153.

EFSA Panel on Plant Health (PLH) (2013) Scientific Opinion on the risk to plant health posed by *Phialophora cinerescens* (Wollenweber) van Beyma for the EU territory, with the identification and evaluation of risk reduction options. *EFSA Journal* **11**(1), 3070.

EPPO (2002) EPPO Standards PM 4/2(2). Certification scheme for carnation. EPPO Bulletin 32, 49–53.

EPPO (2023) EPPO Global Database page on *Phialophora cinerescens*. https://gd.eppo.int/taxon/PHIACI (last accessed 22 June 2023).

Garibaldi A (1969) [Influence of several factors on symptom expression in phialophora wilt of carnation]. *Phytopathologia Mediterranea* **8**, 19–27 (In Italian).

Hawksworth DL & Gibson IAS (1998) *Phialophora cinerescens*. *CMI Descriptions of Pathogenic Fungi and Bacteria* No. 503. CAB International, Wallingford, UK.

Hellmers E (1958) Four wilt diseases of perpetual-flowering carnations in Denmark. *Dansk Botanisk Arkiv* 18, 1–200.

Jenkins JEE (1958) Methods for the detection of vascular wilt pathogens in carnation cuttings. Plant Pathology 8, 23.

Leus L (2018) Breeding for disease resistance in ornamentals. Ornamental Crops 97(11), 97–125.

Mabbett T (2011) Reigning queen of carnations. Practical Hydroponics and Greenhouses 117, 13-18.

Niemann GJ, Geibel M, Treutter D & Feucht W (1994) A crucial role of phenolic metabolism in resistance of carnations to wilt diseases. *Acta Horticulturae* **381**, 565–571.

Moreau M (1970) La verticilliose de l'oeillet: un modèle intéressant pour aborder les rapports hôte-parasite dans les maladies vasculaires. *Horticulture Française* **1**, 19–28.

Péresse M (1971) Pénétration directe et transport passif de spores de *Phialophora cinerescens* (Wr.) van Beyma dans le xylème de l'oeillet. *Phytopathologia Mediterranea* **10**(3), 260–268.

Pergola G & Garibaldi A (1975) Observations on the resistance of carnation cultivars to *Phialophora cinerescens* and *Fusarium oxysporum* f. sp. *dianthi. Annali dell'Istituto Sperimentale per la Floricoltura* **5**(2), 59–66.

Rumine P & Parrini C (1982) [The results of experiments on the control of *Phialophora cinerescens* with benzimidazoles in the Pescia region]. *Annali dell'Istituto Sperimentale per la Floricoltura* **13**, 79–91 (in Italian).

Suffert M (2012) Re?evaluation of EPPO?listed pests. EPPO Bulletin 42(2), 181-184.

Sparnaaij LD & Demmink JF (1976) Breeding for resistance to *Phialophora cinerescens* (Wr.) van Beyma in glasshouse carnations (*Dianthus caryophyllus* L.). *Euphytica* **25**, 329–338.

Schol-Schwarz MB (1970) Revision of the genus Phialophora (Moniliales). Persoonia 6, 59–94.

Tramier R (1967) Les principales maladies de l'oeillet. Bulletin Technique d'Information No. 217.

USDA (2010) *Post-entry Quarantine Manual*. U.S. Department of Agriculture. Available online: <u>https://www.aphis.usda.gov/import_export/plants/manuals/domestic/downloads/postentry.pdf</u> (last accessed July 22, 2023)

Voronenko EN, Primakovskaya MA & Savelyeva AV (1978) Phialophora wilt of carnation. *Plant Protection* (Moscow) **7**, 43 (in Russian).

Wolcan SM, Malbrán I, Mourelos CA, Sisterna MN, González MDP, Alippi AM & Lori GA (2016) Diseases of Carnation. *Handbook of Florist's Crops Diseases*, Centro de Investigaciones de Fitopatología, Comisión de Investigaciones Científicas. Buenos Aires, Argentina. online access <u>https://digital.cic.gba.gob.ar/handle/11746/8206</u> (last accessed July 22, 2023)

Zenkteler E (1992) Rootstocks resistant to *Fusarium oxysporum* f. sp. *dianthi* and *Phialophora cinerescens* for grafting glasshouse carnations. *Phytopathologia Polonica* **16**, 17–23.

ACKNOWLEDGEMENTS

This datasheet was extensively revised in 2023 by Kateryna Davydenko, Ukrainian Research Institute of Forestry and Forest Melioration and Swedish University of Agricultural Science. Her valuable contribution is gratefully acknowledged.

How to cite this datasheet?

EPPO (2025) *Phialophora cinerescens*. EPPO datasheets on pests recommended for regulation. Available online. https://gd.eppo.int

Datasheet history

This datasheet was first published in the EPPO Bulletin in 1982 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 (1st and 2nd edition). CABI, Wallingford (GB).

EPPO (1982) Data sheets on quarantine organisms, *Phialophora cinesrescens*. *EPPO Bulletin* **12**(1), 89-83. https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1365-2338.1982.tb01961.x



Co-funded by the European Union