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Data Sheets on Quarantine Pests

Apiosporina morbosa

IDENTITY

Name: *Apiosporina morbosa* (Schweinitz) von Arx
Synonyms: *Sphaeria morbosa* Schweinitz
Dibotryon morbosum (Schweinitz) Theissen & H. Sydow
Otthia morbosa (Schweinitz) Ellis & Everhart
Plowrightia morbosa (Schweinitz) Saccardo
Cucurbitaria morbosa (Schweinitz) Ellis
Anamorph: *Fusicladium* sp. (also referred to as *Cladosporium* or *Hormodendron*)
Taxonomic position: Fungi: Ascomycetes: Dothideales
Common names: Black knot (English) Schwarzer Rindenkrebs (German)
Bayer computer code: DIBOMO
EPPO A1 list: No. 10
EU Annex designation: II/A1

HOSTS

The principal host is plums (*Prunus domestica*); American, European and Japanese cultivars are attacked.

A. morbosa has also been reported on apricots (*P. armeniaca*), cherries (*P. cerasus*), damsons and peaches, and on various wild North American *Prunus* spp. (*P. americana*, *P. pennsylvanica*, *P. serotina*, *P. virginiana*). The potential host range extends to *Prunus* spp. generally.

Prunus spp. are cultivated as fruit crops throughout the EPPO region. Wild species occur widely and could be a potential reservoir of the pest.

GEOGRAPHICAL DISTRIBUTION

EPPO region: Absent.
Asia: A record of *A. morbosa* on pears in Taiwan (Chao & Wu, 1979) seems very dubious.
North America: Indigenous. Canada (throughout) and USA (throughout), especially in western regions; Mexico.
EU: Absent.
Distribution map: See CMI (1977, No. 48).

BIOLOGY

The spores germinate and enter through wounds or directly at the bases of small twigs of the current season's growth. Infection takes place in a short time, usually immediately after bud break. Where infections occur on older wood, it is always in conjunction with fruit or leaf buds. The minimum temperature at which infection will occur is considered to be ll°C. After infection, a swelling develops late that same year or the following spring. Olive-green

conidia are produced sparingly on the surface of the swelling. Although these spores are extremely cold-resistant (survived 192 days at -20°C) and could overwinter, they are not thought to be important in spread of the disease. Ascospores, the primary source of inoculum, are produced in multilocular ascostromata found on mature knots. Peak ascospore production, in Pennsylvania (USA), occurs after petal fall in May (Smith, 1970) but, in Michigan (USA), it occurs before petal fall (Ritchie *et al*., 1975). Following wetting in rain, the ascospores are forcibly ejected up to 45 mm (50% < 10 mm) and carried in air currents to new growth.

The life cycle cannot be completed in less than 2 years in Nova Scotia (Canada), but can be in 1 year in Michigan (USA) and Ontario (Canada). Prunings containing excised knots can continue to produce viable spores for several weeks. Knots, once formed, continue to produce ascospores each year, although their productivity decreases with age.

Other organisms which are often associated with the knots and which may affect the maturation of ascostromata include: *Trichothecium roseum*, *Coniothyrium* spp., *Cladosporium* spp. and *Fusarium* spp.

Occurrence of the disease is sporadic and it is not clear what are the conditions that favour severe epidemics. Rosenberger & Gerling (1984) discuss factors which may affect the amount of ascospore inoculum (use of benzimidazole fungicides and mycoparasites).

A. morbosa is not readily perpetuated on peaches, ascospores formed on this species re-infecting it only weakly. However, ascospores from plums can readily infect peach trees. Data on cross infection between wild and cultivated species are conflicting and mostly old (see Sutton & Waterston, 1970), so it is not clear whether host-specific forms exist within *A. morbosa*. Recent publications refer to wild species as potential sources of inoculum for orchards.

For more information on the biology of the pathogen, see Gourley (1962), Klos (1964), Smith (1970), Wainwright & Lewis (1970), Ritchie *et al*. (1975).

DETECTION AND IDENTIFICATION

Symptoms

In early spring, branches infected the previous year develop small, light-brown swellings which gradually enlarge (Klos, 1964). The intercellular fungal cells are elongate with thick walls. Knots first become visible just below the point of attachment of the leaf petioles to the stem. They are corky and covered by a velvety olive-green growth due to the conidial stage. Later in the summer, they turn black and become hard and brittle. Knots naturally range from 1 to 15-20 cm in length and from 0.5 to 4 cm in diameter; they very often coalesce to form larger ones and may even girdle the stem. The green coloration on young knots is far less pronounced on peaches than plums. Hyperparasitism by *Trichothecium roseum* results in a pink or white coloration of the knots. Attacked trees become dwarfed and stunted.

Morphology

Conidia occur singly or in chains and are 0- to 1-septate, smooth-walled, pale-brown, ovate, obovate or irregular in shape; 5-13 x 3-5 µm. Ascospores are clavate, apex obtuse, tapered towards the base, 1-septate near the base, smooth-walled, olivacious; 14-18 x 4.5-6 µm.

A full description is given by Sutton & Waterston (1970).

Detection and inspection methods

Plants for planting of Prunus should be inspected for the presence of knots or cankers. Infected material should be excised at least 10 cm below the visible swelling.

MEANS OF MOVEMENT AND DISPERSAL

Under natural conditions, *A. morbosa* spreads readily by ascospore dispersal within orchards. Movement by human agency (pruning, transport of plants) has not especially been noted. In international trade, *A. morbosa* is liable to be carried on infected plants for planting of *Prunus*. However, it is not known ever to have been intercepted.

PEST SIGNIFICANCE

Economic impact

In North America, *A. morbosa* can be serious in plums but is not of economic importance in peaches (Gourley, 1962). The losses caused on plums have been estimated to be 10% and on cherries 1% (Cramer, 1967). In plums (*Prunus domestica*) and *P. salicina*, resistance to *A. morbosa* is valued in new cultivars (e.g. Norton & Cosper, 1984).

Destructive outbreaks of *A. morbosa* usually occur in small home orchards where no control is practised. Within a few years of attack, trees lose vigour and become worthless. Girdling may result in death of individual branches, but stunting of the trees is the most serious effect. Secondary pathogens may gain access through knots. The disease is thought to be responsible for the disappearance of cherry trees from orchards in Ontario (Canada) in the 19th century. It is severe on both wild and cultivated plums. It has been reported as the limiting factor in plum culture in Nova Scotia (Canada) (Gourley, 1962). On *P. serotina*, the cankers formed render the tree worthless for timber.

Control

In some cases, commercial orchards have been successfully protected by chemical spraying coupled with routine inspections, pruning and removal and destruction of infected parts. Rosenberger & Gerling (1984) have recently tested a range of fungicides against black knot in plums, and estimated that a zineb + captan spray applied during a single epidemic could save 8535 USD per ha in subsequent losses. However, the economic benefit of fungicides applied on an annual basis would be diminished by the sporadic incidence of the disease.

A. morbosa has been envisaged as a biological control agent for *P. pennsylvanica* growing as a weed tree on cleared forest land in Canada (Wall, 1986).

Phytosanitary risk

A. morbosa is listed as an A1 quarantine pest by EPPO (OEPP/EPPO, 1979) and is also of quarantine concern for IAPSC. In the EPPO region, it is potentially dangerous to plums (and probably other *Prunus* spp.), wherever grown.

PHYTOSANITARY MEASURES

OEPP/EPPO (1990) recommends that plants for planting of *Prunus* from Canada and USA should come from a place of production intensively treated (by chemical treaments or pruning) against *A. morbosa*, and found free from *A. morbosa* during the last two growing seasons.

BIBLIOGRAPHY

Chao, G.P.; Wu, W.S. (1979) Mycological investigations on the cultivated plants at Highland farms. *Memoirs of the College of Agriculture, National Taiwan University* **19**, 87-95.

CMI (1977) *Distribution Maps of Plant Diseases* No. 48 (edition 2). CAB International, Wallingford, UK.

Cramer, H.H. (1967) Plant protection and world crop production. *Pflanzenschutz-Nachrichten Bayer* **22**, 274-275.

Gourley, C.O. (1962) A comparison of growth, life cycle and control of *Dibotryon morbosum* on peach and plum in Nova Scotia. *Canadian Journal of Plant Science* **42**, 122-129.

Klos, E.J. (1964) How to recognize and control black knot of plum and cherry. *Extension Bulletin, Michigan State University* E-469, 2 pp.

Norton, J.D.; Cosper, R.D. (1984) AU-Roadside - a high quality disease-resistant plum. *HortScience* **19**, 206.

OEPP/EPPO (1979) Data sheets on quarantine organisms No. 10, *Dibotryon morbosum*. *Bulletin OEPP/EPPO Bulletin* **9** (2).

OEPP/EPPO (1990) Specific quarantine requirements. *EPPO Technical Documents* No. 1008.

Ritchie, D.F.; Klos, E.J.; Yoder, K.S. (1975) Epidemiology of black knot of "Stanley" plums and its control with systemic fungicides. *Plant Disease Reporter* **59**, 499-503.

Rosenberger, D.A.; Gerling, W.D. (1984) Effect of black knot incidence on yield of Stanley prune trees and economic benefit of fungicide protection. *Plant Disease* **68**, 1060-1064.

Smith, D.H. (1970) Epidemiology of the black knot disease of plum. *Phytopathology* **60**, 1441-1444.

Sutton, B.C.; Waterston J.M. (1970) *Dibotryon morbosum*. *CMI Descriptions of Pathogenic Fungi and Bacteria* No. 224. CAB International, Wallingford, UK.

Wainwright, S.H.; Lewis F H. (1970) Developmenta1 morphology of the black knot pathogen on plum. *Phytopathology* **60**, 1238-1244.

Wall, R.E. (1986) Effects of black knot disease on pin cherry. *Canadian Journal of Plant Pathology* **8**, 71-77.