Data Sheets on Quarantine Pests

Anisogramma anomala

IDENTITY

Name: *Anisogramma anomala* (Peck) E. Müller
Synonyms: *Apioporthe anomala* (Peck) Höhn
Cryptosporella anomala (Peck) Saccardo
Taxonomic position: Fungi: Ascomycetes: Diaporthales
Common names: Eastern filbert blight (English)
Bayer computer code: CRYPAN
EPPO A1 list: No. 201

HOSTS

Anisogramma anomala is a biotrophic parasite of *Corylus* spp., which is grown in culture only with difficulty (Stone *et al.*, 1994). It is indigenous on *Corylus americana*, a common understorey shrub of forests in eastern USA. It causes the severe disease eastern filbert blight on cultivated hazelnut, *Corylus avellana*. It has also been reported on other *Corylus* spp.

GEOGRAPHICAL DISTRIBUTION

Occurring naturally on wild *Corylus* spp. in eastern USA (Barss, 1930), *A. anomala* spread to Washington in 1973 (Cameron & Gottwald, 1978), to Oregon in 1986, and from those states to British Columbia (Canada).
EPPO region: Absent.
North America: Canada (British Columbia, Nova Scotia). USA (Connecticut, Delaware, Illinois, Iowa, Maine, Maryland, Massachusetts, New Jersey, New York, North Carolina, Oregon, Washington, Wisconsin).
EU: Absent.

BIOLOGY

Ascospores are the only known spore type; they are discharged from perithecia on diseased branches from autumn to late spring (Pinkerton *et al.*, 1990). Wetting of the stromata causes perithecial ostioles to open. Ascospores are then transported by rain water and splash droplets. Short periods of rainfall are sufficient to release spores and inoculate trees. The ascospores infect young vegetative tissue in spring (Gottwald & Cameron, 1980a), after budburst, through leaf emergence and shoot elongation (Stone *et al.*, 1992; Johnson *et al.*, 1994). Once established, *A. anomala* colonizes the cambial tissue. Stromata, containing perithecia, develop within cankers 12-16 months after initial infection (a cold period or a dormant period is required). Stromata form at the margins of cankers in successive years. Cankers expand at an average rate of 0.3 m per year (Gottwald & Cameron, 1980a). Cankers girdle branches, causing dieback of tree canopies and death of mature trees in 5-15 years. Younger trees may be killed within 4-7 years. New susceptible shoots may continue

to emerge from the roots. Perithecia mature in late summer or early autumn, erupting through the periderm within 2-4 mm of the stromata.

DETECTION AND IDENTIFICATION

Symptoms

The first symptoms appear 12-16 months after infection, as sunken cankers developing on branches and twigs. Cankers expand in all directions. They may coalesce when growing. Stromata then become visible and some branches may die.

Morphology

About 200 stromata are formed per canker, with 40-60 perithecia per stroma (Gottwald & Cameron, 1980b). The perithecia are dispersed and immersed in the stroma, which is outlined by a blackened zone. Ostiolar necks erupt through the outer tissues of the host. Ascomal wall two-layered. The asci are unitunicate, 8-spored with deliquescent base so that mature asci lie free in the perithecium at maturity. Ascospores are hyaline, bicellular, with two subequal cells (description of the genus *Diaporthe*) (Hanlin, 1990). The morphology of the fungus is fully described by Gottwald & Cameron (1979).

MEANS OF MOVEMENT AND DISPERSAL

Natural spread by rainsplash is only over short distances. The fungus is only liable to be carried long distances on infected planting material of *Corylus* spp.

PEST SIGNIFICANCE

Economic impact

In the USA, this fungus had no economic importance as long as it remained confined to the east of the country, on wild *Corylus* spp. Since 1986, it has spread to Oregon in the Willamette Valley, where 98% of American commercial hazelnuts are produced (Mehlenbacher *et al.*, 1994). Nearly all orchards within a 10 km radius of the initial site of disease have been destroyed, and the disease has continued to spread to the southwest. All cultivars grown commercially in this area are susceptible.

Control

Eradication was attempted but was not feasible as wild or volunteer *Corylus* plants in adjacent woodland provided an unmanageable reservoir of inoculum. Sanitation measures include destruction of infested wood, but are of little value because of the length of the latent period in the life cycle of the fungus. Chemical control has been applied, but was not very successful as the biology of the fungus was not well known (Pinkerton *et al.*, 1992). Experiments have only recently been carried out to determine efficient chemical treatments. Johnson *et al.* (1993) found that chlorothalonil, flusilazole and fenarimol are potentially valuable components of a programme for control of *A. anomala*. Four or five applications during the period of potential infection can completely control the disease. However such a programme may not be economically feasible for all growers. No known cultivar is fully resistant to the disease, but less susceptible clones have been identified and recommended to American growers. Breeding programmes are also being conducted. Some European cultivars showed high susceptibility to the disease (Pinkerton *et al.*, 1993).

Phytosanitary risk

A. anomala is an EPPO A1 quarantine pest and is also on the A2 list of Canada. The fungus is confined in North America at the moment but, given its biology, could be

expected to survive, find suitable conditions for infection and be very damaging to commercial plantations of *Corylus avellana* in the EPPO region.

PHYTOSANITARY MEASURES

A. anomala can be introduced with planting material of hazel. In the case of the USA, spread of the disease to the West is thought to have been due to the importation of infected *Corylus avellana* nursery stock or of wild *C. americana* seedlings from the Eastern regions. Planting material of *Corylus* spp. should be imported only from areas free from *A. anomala*.

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