Distribution of *Potebniamyces pyri* in the U.S. Pacific Northwest and Its Association with a Canker and Twig Dieback Disease of Pear Trees

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ABSTRACT

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Winter pears (*Pyrus communis*) in the United States are produced primarily in the Pacific Northwest. *Potebniamyces pyri* (anamorph *Phacidiopycnis piri*) is the causal agent of Phacidiopycnis rot, a recently reported postharvest disease on pears in the United States. Infection of fruit by *P. pyri* occurs in the orchard, and symptoms develop during storage. *P. pyri* was observed to be associated with cankers, dead bark, and twig dieback of pear trees. *P. pyri* was isolated from 40 to 50% of the twig samples exhibiting dieback symptoms from three commercial d'Anjou pear orchards, and 35% of dying bark samples from one orchard. However, little information is available regarding pathogenicity of *P. pyri* on pear trees. To determine the distribution of *P. pyri*, dying and dead bark samples were collected from pear orchards in various pear-producing areas in Oregon and Washington, and examined for presence of fruiting bodies (pycnidia or apothecia) of *P. pyri*. In the orchard, 2-year-old twigs were wounded using a sterile cork borer with or without spraying with a commercial aerosol tissue-freezing product at the wound sites. Wounds were then inoculated with either mycelial plugs from an agar medium or conidial suspensions of *P. pyri*. In a separate experiment, freshly made pruning wounds were inoculated with conidial suspensions of *P. pyri*. Canker development was monitored approximately monthly for up to 6 months after inoculation, at which time reisolation of *P. pyri* was attempted. *P. pyri* was found to be widespread in the Pacific Northwest. Incidence of trees infected by *P. pyri* based on presence of viable pycnidia in pear orchards ranged from 0 to 100%. Monthly tree inoculations in the orchard indicated that *P. pyri* in general did not cause cankers on non-cold-injured, wound-inoculated twigs, but apparently became established on cold-injured, wound-inoculated twigs and caused small cankers. Minor dieback developed on twigs inoculated at pruning wounds. At 6 months after inoculation, *P. pyri* was recovered from the majority of inoculated twigs. Thus, *P. pyri* appears to be a weak canker-causing pathogen on pear trees.

Additional keywords: postharvest decay

The discomycete *Potebniamyces pyri* (Berkeley & Broome) Dennis, anamorph *Phacidiopycnis piri* (Fuckel) Weindlmayr (5), is the causal agent of Phacidiopycnis rot, a recently recognized postharvest disease of pears in the United States (20). The disease had previously been reported only in Europe and India (14,15). A recent 2-year survey of postharvest diseases of pears indicated that Phacidiopycnis rot is an important component of storage rots of d'Anjou pears in Washington State, accounting for approximately 30% of the decay on packed fruit (20). Phacidiopycnis

rot causes three types of symptoms on d'Anjou pears: stem-end rot, calyx-end rot, and wound-associated rot, which originate from infections at the stem, calyx, and wounds on the surface of the fruit, respectively (20). Infection of pear fruit by *P. pyri* occurs in the orchard, and symptoms develop during storage (20).

P. pyri was originally described under the name *Phacidiella discolor* by Potebnia (10), and earlier reports indicated that this fungus is associated with a canker and bark necrosis of apple trees in Europe (1,3,6,16). However, pathogenicity of the fungus on apple trees has not been fully established (6,16). There is no information available regarding pathogenicity of *P. pyri* on pear trees. In the United States, winter pears are produced primarily in the Pacific Northwest (PNW). d'Anjou (*Pyrus communis* L.) is the major winter pear variety grown in the region. In order to develop relevant measures for control of Pha-

ciidiopycnis rot, sources of inoculum of *P. pyri* in pear orchards and the association of the fungus with pear trees must be determined. The objectives of this study were to determine the distribution of *P. pyri* in the pear-producing areas in the Pacific Northwest and to assess pathogenicity of the fungus on pear trees.

MATERIALS AND METHODS

Symptoms, occurrence, and isolation of the fungus. d'Anjou pear orchards with a history of Phacidiopycnis rot were identified in a previous study (20). Most of these orchards are located in the Wenatchee River Valley, the primary production area for d'Anjou pears in Washington State. To identify sources of *P. pyri* inoculum in pear orchards, branches with cankers, dying or dead bark, and twigs with dieback symptoms were collected from commercial pear orchards. Photos were taken and symptoms were described. Samples were examined under a stereomicroscope for the presence of fruiting bodies (pycnidia or apothecia) of *P. pyri*.

In May 2003, twigs with dieback symptoms were sampled from three commercial orchards, designated JW, FS, and MT, located in Leavenworth, Peshastin, and Monitor, WA, respectively. In each orchard, 20 twigs with dieback symptoms were collected. In addition, 20 samples of dead bark also were collected from branches of 10 trees (2 bark samples per tree) in FS. To isolate fungi associated with the symptoms of twig dieback and cankers, outer bark tissues of the twigs were scraped and small segments were cut from the marginal area between diseased and healthy tissues of diseased twigs. The segments were washed, surface-disinfested for 5 min in freshly prepared 0.5% sodium hypochlorite solution, rinsed three times with sterile water, cut into small pieces, and placed on acidified potato dextrose agar (APDA [Difco Laboratories, Franklin Lakes, NJ]: 4.0 ml of a 25% solution of lactic acid per liter of medium). Isolation plates were incubated at 20°C in the dark and examined for growth of *P. pyri* after 2 and 5 days. When necessary, fungi that grew on or in the agar were transferred onto fresh APDA. Pure cultures were then transferred onto oatmeal agar (OMA: 60 g of iron- and zinc-fortified single-grain

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oatmeal [Gerber, Fremont, MI] with 15 g of agar in 1,000 ml of deionized water, sterilized for 90 min). Plates were incubated at 20°C under 12-h alternating cycles of dark and fluorescent light to induce formation of pycnidia of *P. pyri* (21). Identification of *Potrebniomyces pyri*/Phacidio-pyrus piri was based upon the descriptions of the fungus by Potebnia (10) and DiCosmo et al. (5).

Distribution of the fungus. To determine the distribution of *P. pyri* on trees in the PNW, a region-wide survey for the presence of the fungus in pear orchards was conducted in 2002 and 2003. Dying or dead bark samples were collected from commercial pear orchards throughout the major pear-producing areas in the region (Table 1). In each orchard, 10 trees were arbitrarily selected, with trees at least two rows apart between rows and five trees apart within a row. Ten dying or dead bark samples each at a size of 4 to 16 cm² were collected from branches or twigs of each tree. Bark samples were examined under a stereomicroscope for the presence of fruiting bodies (pycnidia or apothecia) of *P. pyri*. If pycnidia or apothecia of the fungus were present, three pycnidia or apothecia were selected and crushed in sterile water. Conidia or ascospores were streaked on APDA to test viability. Plates were incubated for 2 to 5 days in the dark at 20°C and examined for the presence of colonies of *P. pyri*.

Pathogenicity tests. Pathogenicity of *P. pyri* on pear twigs was tested using isolate 210, which was originally recovered from a decayed d'Anjou fruit. Mycelium plugs (5 mm in diameter) from 7- to 10-day-old cultures grown on apple juice agar (200 ml apple juice [TreeTop, Selam, WA], 3.0 g CaCO₃, 15 g agar, and 800 ml deionized water) at 20°C in the dark were used as inoculum. For inoculation with conidia, conidial suspensions were made from pycnidia that formed on 10- to 12-week-old OMA cultures grown at 20°C under 12-h alternating cycles of dark and fluorescent light (21). Pycnidia were collected

by scraping them off the surfaces of cultures with a sterile scalpel and were gently crushed with a pestle in a mortar. Approximately 25 ml of sterile water was added to the mortar to make conidial suspensions, which were then filtered through four layers of cheesecloth. Concentrations of the conidial suspensions were adjusted to 1 × 10⁵ conidia per ml.

Inoculations of twigs were conducted in a research block of d'Anjou pear trees planted in 1986 at the Washington State University Tree Fruit Research and Extension Center (WSU TFREC) in Wenatchee, WA. Trees were irrigated using under-tree sprinklers, and no fungicides were applied during the course of study. Prior to inoculation, selected 2-year-old twigs (average 8 mm in diameter) were sprayed with 70% ethanol and air-dried. For inoculation with mycelial plugs, there were three treatments: (i) Twigs were wounded to a depth of 1 to 2 mm before reaching the cambium with a sterile 5-mm-diameter cork borer, and then a 5-mm mycelium plug of the fungus was placed at each wound. (ii) After wounding as in treatment one, an approximately 4-cm twig section with the wound at its center was cold-injured by spraying with a commercial aerosol tissue-freezing product, Freeze'IT (Curtin Matheson Scientific, Houston, TX) (13). The spray was applied for 10 s from a distance of 2 to 3 cm with the twig held as close to horizontal as possible in order to avoid or minimize runoff of the aerosol product (the instant temperature induced by the product was -51°C). Twigs were then inoculated with the fungus 10 to 15 min after the aerosol was applied. (iii) Twigs were wounded as in treatment one but treated with sterile APDA plugs as a control. Inoculation sites for all treatments were covered with moist cheesecloth and sealed with Parafilm (Pechiney Plastic Packaging, Chicago, IL), which was removed 3 weeks after inoculation. An inoculation was performed 26 May 2002, then starting 28 October 2002, was repeated approximately monthly until 24

March 2004. On each inoculation date, three twigs were inoculated per treatment on each of four trees for the study.

For inoculation with conidial suspensions in a separate experiment, five different treatments were applied. The first three were like the mycelium-plug treatments, except inoculum type was different. For the two additional treatments, twigs were pruned, and pruning wounds were either inoculated with conidial suspensions or treated with sterile water as a control. Pieces of sterile cheesecloth (2 × 2 cm² each) were dipped in the conidial suspension and placed over the wound; the control consisted of wounds treated with cheesecloth moistened with sterile water. Inoculation sites were then wrapped with Parafilm, which was removed 3 weeks after inoculation. Three twigs were inoculated per treatment on each of four trees at each inoculation time.

Canker development was monitored approximately monthly for up to 6 months after inoculation. For the wound control and non-cold-injured, inoculated treatments, the diameter of bark necrosis (canker) that included the wound on each twig was measured. For the cold-injured twigs, the length between the center of the wound and the edge of bark necrosis toward the main trunk of the tree was measured. For the pruning-wound inoculations and controls, the length of dieback on each twig was measured. Twigs were removed from trees 6 months after inoculation. Reisolation of *P. pyri* from inoculated twigs was attempted as described. For treatments with no cankers, wound areas (inoculation sites) were selected for isolation.

Weather data were obtained from a weather station near the pear orchard, maintained by the WSU TFREC.

Data analysis. For the final measurements of canker size or dieback, a *t* test was performed with SAS PROC TTEST (version 8.2, SAS Institute, Cary, NC) to evaluate differences in canker size or dieback between the non-cold-injured, wound-inoculated treatments and the non-

Table 1. Recovery of *Potrebniomyces pyri* from dying or dead bark sampled from pear orchards in the U.S. Pacific Northwest

Location ^a	No. of orchards	Cultivars	Sampling dates	% Trees with viable pycnidia ^b	% Bark samples with viable pycnidia ^c	% Bark samples with viable apothecia
Omak	6	d'Anjou	7 Nov 2002	0–90 (33) ^d	0–32 (10)	0
Wenatchee	8	d'Anjou	5 Sep 2002 19 Nov 2002	20–100 (85)	2–78 (49)	0–9 (3)
Quincy	2	d'Anjou	22 Aug 2002	40–50 (45)	7–11 (9)	0
Yakima	3	d'Anjou	22 Aug 2002	0–90 (30)	0–20 (7)	0
	3	Bartlett	22 Aug 2002	20–90 (60)	2–25 (16)	0
Hood River	5	d'Anjou	19 May 2003	20–90 (56)	2–37 (14)	0–2 (1)
	1	Bartlett	19 May 2003	10	1	0
Medford	1	d'Anjou	21 May 2003	60	25	3
	5	Bosc	21 May 2003	0–60 (22)	0–13 (4)	0
	2	Comice	21 May 2003	20–50 (35)	2–9 (6)	0

^a Hood River and Medford are located in Oregon; other areas are located in Washington State.

^b In each orchard, 10 trees were arbitrarily selected, and the selected trees were at least two rows apart between rows and five trees apart within a row.

^c In each orchard, 10 samples of dying or dead bark were collected from each of 10 selected trees.

^d Data are presented as ranges when more than one orchard of the same cultivar was sampled from each location; values in parentheses are means.

inoculated wound controls. The analyses were performed separately for different inoculation dates.

RESULTS

Symptoms, occurrence, and isolation of the fungus. *P. pyri* was associated with cankers on branches (Fig. 1A), bark necro-

sis (Fig. 1B), and dieback of twigs and fruit spurs (Fig. 1C and D). The canker area was slightly sunken. Dieback of twigs usually originated from infections at pruning wounds. Pycnidia of the fungus often formed on dead or dying bark tissues. Pycnidia of the fungus were either immersed in the bark tissues or partially

erumpent from the dead bark tissues of twigs or branches (Fig. 1E). Apothecia of the fungus were less frequently observed (Table 1). A voucher specimen is deposited at the Washington State University Mycological Herbarium (accession no. WSP 70479). Macroconidia were commonly observed inside pycnidia and were hyaline,

Fig. 1. Symptoms and signs of *Potebniamyces pyri* on d'Anjou pear trees. **A**, cankers on a branch with pycnidia on the bark; **B**, bark necrosis of a branch with pycnidia in dead bark tissues (woody tissues of the branch were apparently healthy); **C**, dieback of a twig (arrow indicates margin between healthy and dieback or canker tissues); **D**, dieback of a fruit spur (arrow indicates margin between healthy and dieback tissues); **E**, pycnidia partially erumpent from the bark of an infected twig; **F**, macroconidia. Scale bar in **E** = 1.3 mm; **F** = 12 μ m.

subglobose to obovoid, and guttulates (Fig. 1F). Microconidia, which are considered spermatia in the life cycle of *P. pyri* (19), also were observed in pycnidia collected from pear orchards but occurred much less frequently than macroconidia.

P. pyri was commonly isolated from twigs with dieback symptoms and dead or dying bark tissues. In May 2003, *P. pyri* was isolated from 50, 40, and 45% of the twigs with dieback symptoms sampled from FS, JW, and MT orchards, respectively, and from 35% of the dead bark samples collected from FS orchard. Other fungi commonly recovered from these samples were *Aureobasidium pullulans*, *Alternaria* spp., and *Cladosporium herbarum*.

Distribution. *P. pyri* was observed on dead or dying bark tissues collected from all major pear-producing areas in the PNW (Table 1). Incidence of trees infected by the fungus in the orchards ranged from 0 to 100%. Pear trees in the Wenatchee area were heavily infected by the fungus. In addition to d'Anjou pear trees, *P. pyri* was also observed on trees of other pear varieties including Bartlett, Bosc, and Comice (Table 1). Among the orchards in which *P. pyri* was detected, 10 to 90% of the sampled trees had viable pycnidia of the fungus, whereas 2 to 78% of the bark samples had viable pycnidia of the fungus. Apothecia of *P. pyri* were observed on samples from the Wenatchee area, WA, as well as the Hood River and Medford areas, OR, but at a low frequency (Table 1).

Pathogenicity tests. The vast majority of the non-cold-injured, wounded twigs inoculated with mycelial plugs did not develop cankers by 6 months after inoculation, but small cankers developed on those inoculated on 20 December 2002, 30 October 2003, and 25 November 2003 (Fig. 2). The fungus established on cold-injured, inoculated twigs and was able to advance a limited distance into healthy tissues during the 6-month period following most inoculations. At 6 months after inoculation, *P. pyri* was reisolated from the majority of non-cold-injured wounds that had been inoculated with the fungus; the fungus also was reisolated from the margins between healthy and diseased tissues on cold-injured, wound-inoculated twigs. No *P. pyri* was recovered from noninoculated controls except one noninoculated twig from the inoculation experiment on 20 December 2002.

On twigs inoculated with conidial suspensions of *P. pyri* in February and March of 2002 and 2003, generally no distinct cankers developed by 6 months after inoculation (Table 2). Small dieback occurred on pruning wound-inoculated twigs 6 months after inoculation, and the lengths of dieback on pruning wound-inoculated twigs were greater ($P < 0.15$) than those on pruning wound controls. *P. pyri* was recovered from the vast majority of inoculated

twigs and was not recovered from the non-inoculated wound and pruning wound controls.

DISCUSSION

In this study, we found that *P. pyri* was widespread in major pear-producing areas of the U.S. Pacific Northwest and that the fungus was commonly associated with dying or dead bark tissues, cankers, and twig dieback of pear trees in the region. *P. pyri* is the causal agent of Phacidiopycnis rot, a recently reported postharvest fruit rot disease on pears in the United States (20). The information presented in this study is of value to the pear industry in the region in assessing the economic importance of *P. pyri* as a decay-causing pathogen on fruit and a canker-causing pathogen on trees in different pear-producing areas, as well as in implementing relevant measures for disease control.

Many canker-causing fungi are weak pathogens. They cause cankers when trees are under stress caused by various factors such as winter injury, drought, or nutrient deficiencies (7,12,17). Stresses caused by freezing temperatures are particularly injurious and often associated with damage to woody plants (18). Host tissues injured by cold temperatures may become predis-

posed to invasion by weak canker-causing pathogens (12). For example, cankers caused by *Botryosphaeria* spp., *Cytospora* spp., and *Leucostoma* spp. are often associated with cold injury to host plants (4,8,11,13). In our study, we found that *P. pyri* was able to colonize wounded twigs and in general did not cause cankers, whereas the fungus established on cold-injured, inoculated twigs and caused small cankers, indicating that *P. pyri* was a weak canker-causing pathogen. Our finding is in general agreement with previous reports, although complete pathogenicity tests were not established in these reports (6,16). Southee and Brooks (16) did a large number of inoculation experiments with pure cultures of *P. pyri* on wounded branches of cultivar Lord Derby apple trees. They found that the fungus does not cause cankers and stops advancing soon after penetrating tissues. We did not perform histological evaluations of the fungus at inoculation sites, but recovery of *P. pyri* from inoculated wounds not exhibiting canker symptoms indicates that the fungus likely penetrated the tissues to some extent and survived at wound sites during the course of our experiments.

In most dicotyledonous trees, as trees age, sequent periderms may arise at suc-

Fig. 2. Canker development on twigs of d'Anjou pears trees 6 months after inoculation with mycelium of *Potentialmyces pyri*. Two-year-old twigs were wounded with a sterile 5-mm-diameter cork borer. The cold-injured treatment was achieved by spraying with a commercial aerosol tissue-freezing product. Twigs were inoculated by placing a 5-mm-diameter mycelium plug at each wound. Wounds treated with sterile agar plugs were used as noninoculated controls (wound only). Canker development was monitored approximately monthly for up to 6 months after inoculation. For the control and non-cold-injured inoculated twigs, diameters of cankers at wounds were measured, and measurements include the wound size; for the cold-injured inoculated twigs, the length between the center of the wound and the edge of canker toward the main trunk of the tree was measured. Canker sizes at 6 months are presented except that the 5-month data from the inoculation on 24 Feb 2003 are presented.

cessively greater depths and cause an accumulation of dead tissues on the surface of the stems or branches. This contributes to the formation of rhytidome (also called scale bark or shell bark) on rough-barked species (2). The dead bark tissues provide substrates for many fungi (2). *P. pyri* is a caulicolous and corticolous fungus (5). d'Anjou pear trees have rough bark. In this study, most of the orchards sampled were old blocks (>15 years). The naturally occurring dead tissues on the bark of pear trees may provide a suitable avenue for *P. pyri* to colonize trees. This may partially explain the wide distribution of the fungus in the region.

Recovery of *P. pyri* from wound-inoculated twigs 6 months after inoculation indicates that the fungus was able to colonize wounded twigs at different temperatures and survived in the inoculated twigs during the 6-month period of each experiment. However, low recovery rates of *P. pyri* from twigs inoculated on 24 July 2003 were observed. The average temperatures during the first 10 days and 3 weeks after the inoculation on 24 July 2003 were 28 and 26°C, respectively. These high temperatures may affect the establishment

of the fungus on inoculated twigs because *P. pyri* is a low-temperature fungus; mycelium grows at temperatures from -3 to 25°C and does not grow at 30°C (21). Conidia of *P. pyri* germinate at 0 to 30°C (9). The average temperatures during the fall, winter, and spring in Wenatchee are within the temperature ranges of mycelial growth and conidial germination of *P. pyri*. The germination of conidia and the growth of mycelia at freezing temperatures indicate that *P. pyri* may be active to either colonize the bark tissues or develop in infected twigs during the dormant period of pear trees from late fall to early spring. This may explain the expansion of bark necrosis or cankers on twigs inoculated in late fall and early winter.

d'Anjou pear trees are commonly pruned in late winter and early spring in north-central Washington. Fresh pruning wounds may provide an avenue for *P. pyri* to invade trees. Observations of minor dieback which developed from pruning wounds inoculated with conidial suspensions of the fungus indicate that conidia of the fungus have the ability to germinate and colonize pruning wounds at low temperatures following early spring pruning.

This also suggests that pruning during wet weather may increase the likelihood of twig dieback originating from pruning-wound infections.

Teleomorphs of Phacidiaceae have a distinct seasonality (5). Ascomata are initiated in the autumn, mature during the winter, and release mature ascospores in the spring and early summer, and apothecia of *P. pyri* may occur intermixed with pycnidia of the fungus on the trees (5). In the orchard survey, apothecia were observed in the Wenatchee, Hood River, and Medford areas, but much less frequently in comparison with observations of pycnidia. Because the sample sizes were relatively small and samples were collected at different times of the year in the various areas, it is unclear whether the teleomorph state of the fungus occurs in other pear-producing areas in the PNW. We speculated that if the teleomorph is present in those areas from which apothecia were not observed during the orchard survey, it may be at the same low frequency as was observed in the Wenatchee, Hood River, and Medford areas. The dominance of pycnidia on dead and dying bark tissues suggests that the conidial state of the fungus is the main type of inoculum in pear orchards in the region.

Table 2. Pathogenicity of *Potrebniomyces pyri* on twigs of d'Anjou pear trees inoculated with conidial suspensions of the fungus

Inoculation date	Treatment ^a	Canker size (mm) at 2 or 3 mo ^b	Canker size (mm) at 6 mo ^c	Recovery of <i>P. pyri</i> at 6 mo (twigs yielding <i>P. pyri</i> /total twigs) ^d
28 Feb 2002	Wound only	5	5	0/4
	Wound + inoculum (ino)	5	5.5	11/12
	Wound + cold injury (CI) + ino	40.6	45	10/12
	Pruning wound only (PW)	3	3.6	0/4
	PW + ino	5	5.8 G	11/12
25 Mar 2002	Wound only	5	5	0/4
	Wound + ino	5	5.1	12/12
	Wound + CI + ino	53.5	57.6	10/12
	PW	0	4.8	0/4
	PW + ino	5.4	8.4 G	10/12
27 Feb 2003	Wound only	5	6	0/12
	Wound + ino	6	6.1	6/12
	Wound + CI + ino	39.7	40.3	11/12
	PW	2.8	3.7	0/12
	PW + ino	4.6	6.9 G	12/12
31 Mar 2003	Wound only	5	5.4	0/12
	Wound + ino	6	7.3 *	9/11
	Wound + CI + ino	28.4	30.7	12/12
	PW	2.8	3.4	0/12
	PW + ino	4.8	5.1 G	10/12

^a 2-year-old twigs were wounded using a sterile 5-mm-diameter cork borer. The "cold injury" treatment was achieved by spraying with a commercial aerosol tissue-freezing product. Pruning wounds were made just prior to inoculation.

^b For non-cold-injured, inoculated twigs and wounded-only twigs, diameters of cankers at wounds were measured, and measurements include the wound size; for the cold-injured, inoculated twigs, the length between the center of the wound and the edge of canker toward the main trunk of the tree was measured. The lengths of dieback on pruning wound treatments were measured. Twigs inoculated in February and March were measured at 3 and 2 months after inoculation, respectively.

^c * Indicates that the sizes of cankers or dieback on inoculated twigs were significantly ($P < 0.05$) greater than those on noninoculated controls; G indicates that the sizes of cankers or dieback on inoculated twigs were greater ($P < 0.15$) than those on noninoculated controls from the same inoculation dates.

^d A total of 12 twigs on four replicate trees was used for each treatment, except that four replicate twigs were used for wound and pruning wound controls in 2002.

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LITERATURE CITED

- Barthelet, J. 1943. Recherches sur quelques parasites des arbres fruitiers. Ann. Epiphyties 9:27-45.
- Biggs, A. R. 1993. Anatomy and histochemistry of wound responses in bark tissues and implications for management of canker diseases. Pages 13-33 in: Cytology, Histology, and Histochemistry of Fruit Tree Diseases. A. R. Biggs, ed. CRC Press, Boca Raton, FL.
- Brooks, F. T. 1928. On the occurrence of *Phacidiella discolor* (Mout. & Sacc.) *Potrebni* in England. Trans. Br. Mycol. Soc. 13:75-81.
- Brown-Rytlewski, D. E., and McManus, P. S. 2000. Virulence of *Botryosphaeria dothidea* and *Botryosphaeria obtusa* on apple and management of stem cankers with fungicides. Plant Dis. 84:1031-1037.
- DiCosmo, F., Nag Raj, T. R., and Kendrick, W. B. 1984. A revision of the Phacidiaceae and related anamorphs. Mycotaxon 21:1-234.
- Ficke, W., and Peter, E. 1982. Occurrence of apple bark necrosis pathogens so far not identified in the German Rep. Dem. Arch. Phytopathol. U. Pflanzenschutz 18:31-38.
- Guyon, J. C., Jacobi, W. R., and McIntyre, G. A. 1996. Effects of environmental stress on the development of Cytospora canker of aspen. Plant Dis. 80:1320-1326.
- Latorre, B. A., and Toledo, M. V. 1984. Occurrence and relative susceptibility of apple culti-

- vars to *Botryosphaeria* canker in Chile. *Plant Dis.* 68:36-39.
9. Liu, Q., and Xiao, C. L. 2005. Influence of nutrient and environmental factors on conidial germination of *Potebniamyces pyri*. *Phytopathology* 95:572-580.
 10. Potebnia, A. 1912. Ein neuer Kresbserreger des Apfelbaumes *Phacidiella discolor* (Mout. Et Sacc.) A. Pot., seine Morphologie und Entwicklungsgeschichte. *Z. Pflanzenkrank.* 22:129-153.
 11. Proffer, T. J., and Jones, A. L. 1989. A new canker disease of apple caused by *Leucostoma cincta* and other fungi associated with cankers on apple in Michigan. *Plant Dis.* 73:508-514.
 12. Schoeneweiss, D. F. 1975. Predisposition, stress, and plant diseases. *Annu. Rev. Phytopathol.* 13:193-211.
 13. Scorza, R., and Pusey, P. L. 1984. A wound-freezing inoculation technique for evaluating resistance to *Cytospora leucostoma* in young peach trees. *Phytopathology* 74:569-572.
 14. Sharma, R. L. 1991. Prevalence of storage rots of China pear in Himachal Pradesh. *Plant Dis. Res.* 5:109-111.
 15. Snowdon, A. L. 1992. Post-harvest diseases and disorders of fruits and vegetables. Vol. 1. General Introduction and Fruits. CRC Press, Boca Raton, FL.
 16. Southee, E. A., and Brooks, F. T. 1926. Notes on a pycnidial fungus associated with a dying-back of apple branches. *Trans. Br. Mycol. Soc.* 11:213-219.
 17. Stanosz, G. R., Blodgett, J. T., Smith, D. R., and Kruger, E. L. 2001. Water stress and *Sphaeropsis sapinea* as a latent pathogen of red pine seedlings. *New Phytol.* 149:531-538.
 18. Wene, E. G., and Schoeneweiss, D. F. 1980. Localized freezing predisposition to *Botryosphaeria* canker in differentially frozen woody stems. *Can. J. Bot.* 58:1455-1458.
 19. Wollenweber, H. W. 1937. Der schwarze Rindenbrand der Quitte. *Angewandte Bot.* 19:131-140.
 20. Xiao, C. L., and Boal, R. J. 2004. Prevalence and incidence of *Phacidiopycnis* rot in d'Anjou pears in Washington State. *Plant Dis.* 88:413-418.
 21. Xiao, C. L., and Sitton, J. W. 2004. Effects of culture media and environmental factors on mycelial growth and pycnidial production of *Potebniamyces pyri*. *Mycol. Res.* 108:926-932.