

**Themis J. Michailides**  
University of California, Berkeley  
Kearney Agricultural Center, Parlier, CA

**Robert A. Spotts**  
Oregon State University  
Mid-Columbia Agricultural Research and Extension Center, Hood River

# Postharvest Diseases of Pome and Stone Fruits Caused by *Mucor piriformis* in the Pacific Northwest and California

In the Pacific Northwest, pome fruits, i.e., pear (*Pyrus communis* L.) and apple (*Malus domestica* Borkh.), occupy approximately 87,000 ha, whereas stone fruits, including peach (*Prunus persica* (L.) Batsch), nectarine (*P. persica* var. *nectarina* (Aiton) Maxim.), plum and prune (*P. domestica* L.), cherry (*P. avium* L.), and apricot (*P. armeniaca* L.), occupy only 9,845 ha. In contrast, in California, stone fruits occupy 94,000 ha and pome fruits occupy only 13,000 ha, although since the 1980s this acreage has been increasing.

Losses of pome and stone fruit from decay that develops in cold storage often depend on conditions of storage. Temperatures below 5 C prevent brown rot and Rhizopus or Gilbertella rot on stone fruit and reduce Botrytis and Penicillium rot on pome fruit, but other rots caused by fungi that can tolerate low temperatures still result in losses in storage. Among these, *Mucor piriformis* Fischer has become a serious threat for fruit in cold storage in the Pacific Northwest and is sometimes a problem for the California stone-fruit industry.

Until recently, *Mucor* spp. were of relatively minor importance as postharvest pathogens. In the last two decades, however, they have caused serious decay of strawberries (*Fragaria* × *ananassa* Duchesne), pears, apples, guavas (*Psidium guajava* L.), peaches, nectarines, and vegetables, such as tomato (*Lycopersicon esculentum* Miller) and sweet potato (*Ipomoea batatas* (L.) Lam.). In Europe, *M. piriformis* and other *Mucor* spp., such as *M. mucedo* L.:Fr., *M. racemosus* Fres., and *M. strictus* Hagem, occasionally have been reported as causing rot

of pome fruits (3,8). During 1970–1980, *Mucor* rot caused major losses in pears and apples in the Pacific Northwest (2). Because chemical control of this disease is not possible, significant losses of pome fruit in cold storage occur each year. *Mucor* rot of pears and apples in the Pacific Northwest and Canada and of stone fruit in California is caused only by *M. piriformis*; therefore, this discussion refers exclusively to this species.

*M. piriformis*, initially described by A. Fischer in 1892, was first reported as a cause of fruit decay in 1895 (31). Subsequent reports that cover approximately a century on *Mucor* rot of pome and stone fruit are listed in Table 1. In 1975 (2), a *Mucor* rot outbreak on pome fruit occurred in the mid-Columbia area of Oregon and Washington. In 1977, *Mucor* rot caused significant losses of stone fruit in California, when an unusual amount

of decay developed during cold-temperature (approximately 5 C) transit of fresh-market peaches from California to markets in the eastern United States and of nectarines shipped from Chile to California.

From 1962 to 1990, several *Mucor* spp., including *M. circinelloides* van Tieghem, *M. racemosus*, *M. plumbeus* Bonorden, and *M. hiemalis* Wehmer, were isolated from stone fruit in the field and in cold storage in California, but only *M. piriformis* caused serious fruit losses in cold storage. During 1988–1989, *M. piriformis* was commonly isolated from infected Asian pears, Fuji apples, and plums, all grown in California.

## Symptomatology

Infection occurs through the stem end, the calyx, or puncture wounds. In 1988 we also observed *Mucor* rot initiating

**Table 1.** A century of reports on *Mucor* rot caused by *Mucor piriformis* in pome and stone fruit throughout the world

Year reported	Affected fruit	Location	Literature citation
1895	Pear	Pilze, Germany	31
1931	Apple	Washington, USA	10
1938	Apple	Northern Ireland	3
1940	Pear	Washington, USA	9
1972	Pear	British Columbia, Canada	12
1973	Stone fruit	Maryland, USA	23
1975	Pear	Oregon and Washington, USA	2
1977	Peach <sup>a</sup>	Pennsylvania, USA	1
1979	Peach, nectarine	Maryland, USA	24
1980	Peach, nectarine	California, USA	(TJM) <sup>b</sup>
1982	Pear, apple	Victoria, Australia	32
1987	Prune	California, USA	(TJM)
1989	Asian pear, apple, plum	California, USA	(TJM)
1990	Peach	British Columbia, Canada	22

<sup>a</sup> Pathogen was identified as *Mucor albo-ater* Naumov, a synonym of *M. piriformis*.

<sup>b</sup> Identified by the first author.

from the core area of Fuji apples. Infected tissues of pome fruit become soft, watery, and light brown, and usually sporangiophores protrude through cracks in the skin (Fig. 1) or emerge through lenticels. Infected fruit decay completely after about 2 months in cold storage (0 C), releasing large quantities of juice. Well-rotted apples or pears kept in an airtight chamber for several days emit a characteristic alcoholic odor.

On peaches or nectarines, *Mucor* rot commonly begins from puncture wounds or wounds caused by insects. Lesions are circular, light brown, soft, and watery and are soon covered with masses of shiny, erect sporangiophores that appear at breaks in the fruit skin or emerge through lenticels. Peaches and nectarines infected in cold storage sometimes show a narrow band of clear, water-soaked tissue at the lesion margin and usually give off a pleasant, aromatic odor. The diseased internal tissue is light brown, very soft, and watery and separates easily from the healthy tissue.

The advanced stage of *Mucor* rot is easily distinguished from that of *Rhizopus* or *Gilbertella* rot, although all look identical in the beginning stages. All three are tan soft rots, i.e., acid-tolerant polygalacturonases produced by the fungi cause infected tissues to become watery and soft. With advanced *Mucor* rot, erect, white or yellowish, shiny sporangiophores with gray to black sporangia (in the laboratory) or brown sporangia (in the field) densely cover the decay lesions (Fig. 2A). Under high relative humidity, the sporangia absorb water, the sporangial wall dissolves, and the whole sporangium becomes a "sporangial drop." At 0 C, *Mucor* rot develops very rapidly.

In contrast, with *Rhizopus* rot, caused by *Rhizopus stolonifer* (Ehrenb.:Fr.) Vuill., the mycelium of the fungus is

interwoven with stolons, dark sporangiophores, and black powdery sporangia, with a dirty, cottony appearance (Fig. 2B). At 20 C, *Rhizopus* rot covers the whole fruit soon after infection. The sporangial wall dries and falls apart, and the sporangiospores appear as a black powder. At 0 C, *Rhizopus* rot does not develop.

In the field, *Gilbertella* rot of stone fruit, caused by *Gilbertella persicaria* (E.D. Eddy) Hesseltine, resembles *Rhizopus* rot because of the dense black sporangia (Fig. 2C). However, the sporangiophores of *Gilbertella* are short and the sporangia remain wet ("sweaty"). The sporangial wall splits in half on top but remains attached at the tip of the sporangiophores. Rot develops rapidly at 20–25 C but does not develop at 0 C.

### The Pathogen

*Mucor* spp. belong in the family Mucoraceae of the order Mucorales in the class Zygomycetes and occur typically as saprophytes in soil and dung. Synthetic Mucor agar (SMA) is used to grow members of the Mucorales for identification and taxonomy. The following description of *M. piriformis* is based on two isolates from stone fruits grown at 21 C in darkness.

The fungus produces gray columellate

sporangia on single or branched (monopodially or sympodially) short and tall, shiny sporangiophores (Fig. 3). Young sporangia are milky-white or yellowish (turning to mouse-gray), globose, 264–283  $\mu\text{m}$  in diameter, with deliquescent spiny walls. Columellae are smooth, cylindrical-ellipsoidal, pyriform, subglobose, up to 182  $\times$  144  $\mu\text{m}$ . Sporangiospores are ellipsoidal (8.6–9.2  $\times$  7.8–8.1  $\mu\text{m}$ ) to subspherical (7.3–7.8  $\times$  4.6–5.3  $\mu\text{m}$ ) and smooth. Germinated sporangia and columellae are common in 10-day or older cultures, but chlamydospore-like structures (gemmae) are not very common. Isogametangia (Fig. 4A) produce zygospores that are spherical to subglobose, measuring 151–180  $\times$  127–156  $\mu\text{m}$  (Fig. 4B). To date, zygospores have been found on decayed pear fruit in several orchards in the Hood River Valley in Oregon and in a nectarine orchard in Parlier, California. Production of zygospores on media and fruits in vitro and their germination in the laboratory are relatively easy (20).

The fungus grows well and sporulates extensively at 0–24 C. Growth is optimal at 21 C (Fig. 5). Mycelium of the fungus grows slowly at 26 C (maximum temperature) but not at 27 C. Sporangiospores germinate between –1 and 24 C but not at 27 C or higher, but they can grow in a yeastlike fashion at 26–27 C. The thermal death points of the mycelia and sporangiospores are 43–46 and 52–55 C, respectively, depending on the isolate (17).

**Resistant structures.** Zygospores are considered resistant structures because the episporal wall is melanized. *M. piriformis* does not produce chlamydospores, but sometimes gemmae (Fig. 6) are produced in hyphae and sporangiophores that come in contact with soil (13). Gemmae germinate by producing a single sporangium or a branched sporangiophore, but their role in survival of the fungus in soil is unknown. Mycelia buried in soil fragment and show retraction of protoplast and frequent septation in the hyphae. Although the role of retracted protoplasts has not been determined, similar retraction of protoplasts in germ tubes of sporangia from *Pythium ultimum* Trow are believed to act as a mechanism to counter lysis in soil (29).

**Genetics of *M. piriformis*.** Like most of the Mucorales, *M. piriformis* is heterothallic with + and – mating types. Both mating types, with the + type predominating, were discovered in soils of pear orchards in Oregon (18), but most fruit decaying on the orchard floor in November and December were infected with a single mating type. Only 15% were infected by both types, and 1.6% of decaying fruit had zygospores. In California, although both mating types of the fungus are found, most isolates from stone fruit do not mate with either + or – mating types ("neutral" isolates).



Fig. 1. (A) A d'Anjou pear and (B) a Fuji apple infected by *Mucor piriformis*.

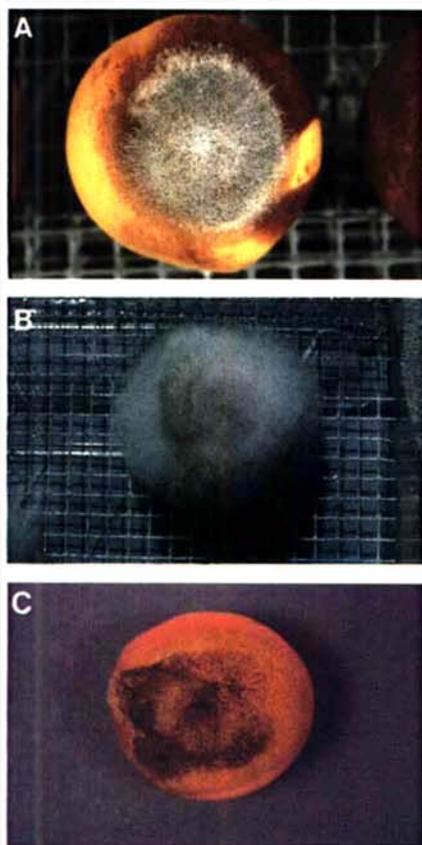


Fig. 2. (A) *Mucor* rot of nectarine caused by *Mucor piriformis*, (B) *Rhizopus* rot of peach caused by *Rhizopus stolonifer*, and (C) *Gilbertella* rot of peach caused by *Gilbertella persicaria*.

In the laboratory, zygospores of *M. piriformis* germinate in petri dishes at  $23 \pm 1$  C after drying and wetting periods and germination continues even after the dishes are transferred to 0 C. More than 95% of germinated zygospores developed one sporangiophore; 95.6% of the germinated zygospores yielded only the + mating type of germ-sporangiospores, 2.2% yielded only the - type, and 2.2% yielded both types. Zygospores formed on artificially inoculated peach and nectarine fruits germinated and produced 95.8% + and 4.2% - germ-sporangia (20).

## Techniques

**Isolation from decaying fruit and recovery from soil and debris.** When sporulating, the fungus can be recovered easily by placing a few sporangia on potato-dextrose agar (PDA) or acidified PDA (APDA) and incubating the dishes at 20–21 C. Contamination of cultures by *R. stolonifer* may be avoided by incubation below 5 C, at which only *M. piriformis* will grow. If sporulation does not occur, peeled pieces of fruit tissue from the active margins of decay lesions can be placed on PDA or APDA.

The serial dilution plating technique on APDA is very efficient for recovering propagules from infested soil or debris. A preliminary test is useful for estimating the numbers of propagules in soil. Dishes are incubated at 21 C for 22–24 hours, then at 0 C for 2–3 days. Colonies of *M. piriformis* are distinguished from those of *Pythium*, *Phytophthora*, and *Rhizopus* by their morphology. Under transmitted light, colonies of *M. piriformis* have sparse, dichotomously branched mycelium, the pattern of branching resembling that of snow crystals (Fig. 7). Colonies can be counted by observing the dishes through transmitted light. Numbers can be expressed as propagules (sporangiospores) per gram of dry soil or, if bulk soil density and moisture are known, per cubic centimeter of soil.

**Recovery of the mating types from soil.** Surface-sterilized pear slices are placed in the bottom of petri dishes and covered with two sheets of Whatman No. 1 filter paper. Then, 0.5 g of air-dried soil is distributed evenly over the surface of the filter papers, and the petri dishes are incubated at 5 C for 10–13 days. When propagules of both mating types occur in the same petri dish, zygospores develop on the filter paper. Sporangia can be picked from opposite sides of the line of zygospores and transferred to APDA to obtain pure cultures of + and - mating types (18).

**Induction and germination of zygospores of *M. piriformis*.** To induce zygospore development, + and - mating types are paired on APDA in 90-mm-diameter petri dishes. Cultures are incubated at 21 C for 2 days, then at 9–10 C for 10 days. Continuous incu-

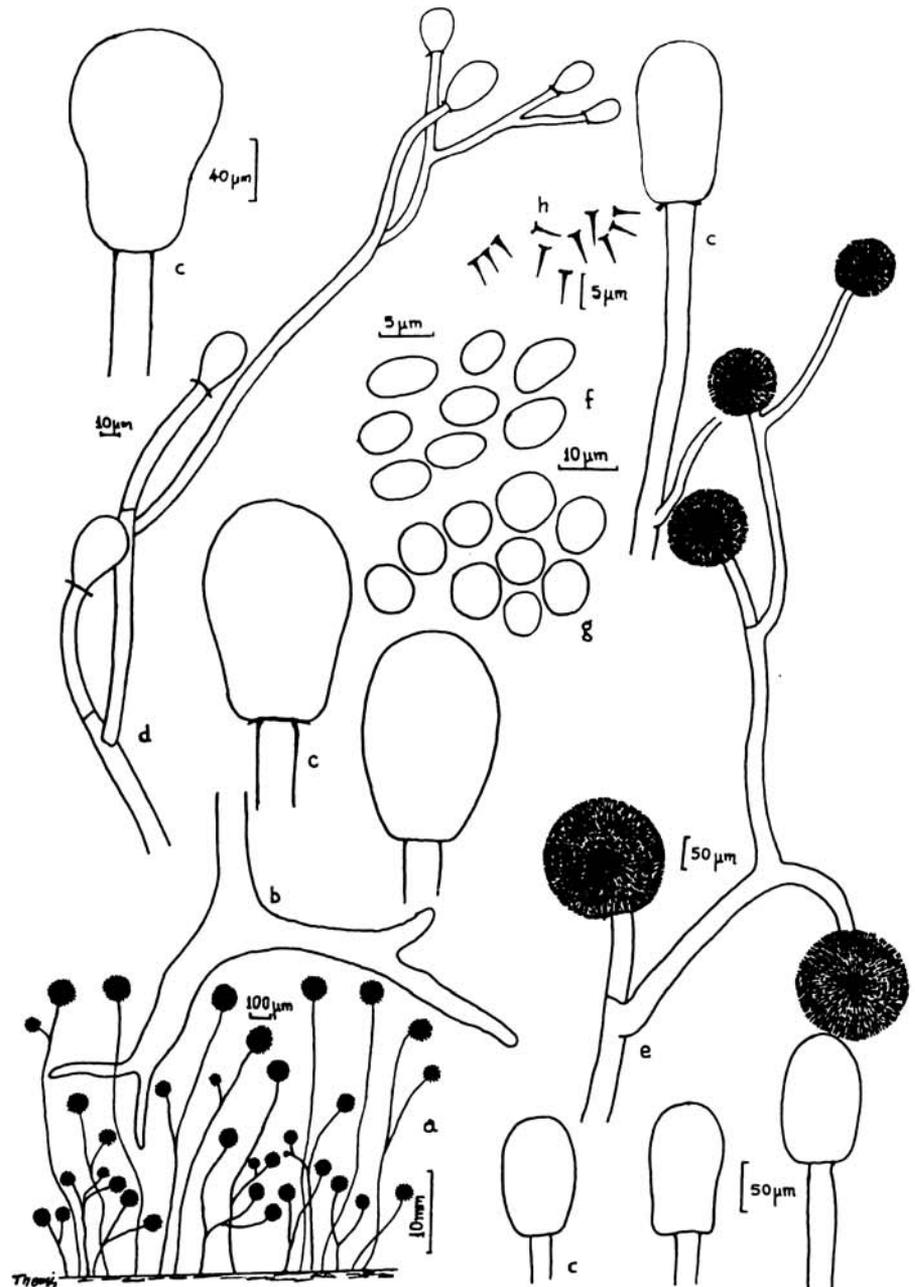


Fig. 3. *Mucor piriformis*, California isolate: (A) Turf, consisting of short and tall sporangiophores; (B) base of a tall sporangiophore; (C) columellae; (D) a sympodially branched short sporangiophore bearing (E) spiny sporangia; sporangiospores from sporangia on (F) tall and (G) short sporangiophores; and (H) spines on the sporangial wall.

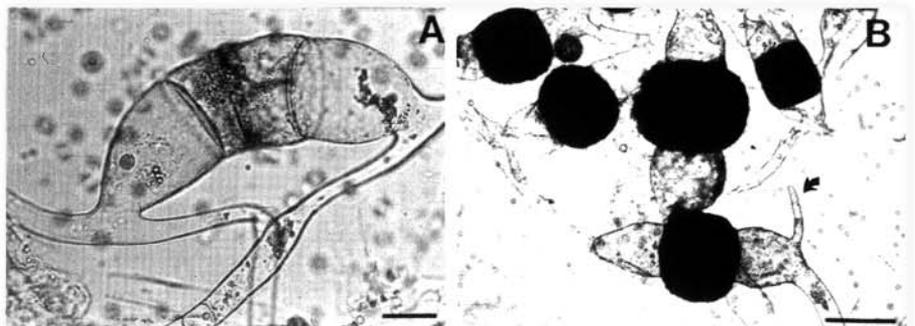


Fig. 4. Isogametangia of *Mucor piriformis* (A) in early stages of mating (B) produce mature zygospores; arrow indicates the characteristic fingerlike projection on one of the suspensors. Scale bar = 100  $\mu$ m.

bation of cultures at 21 C does not induce zygospor development. Strips of agar 10 mm wide with zygospor produced along the line of contact of the mating types are cut and removed aseptically, washed with sterile distilled water to eliminate sporangia and sporangiospores, then halved. The resulting rectangular pieces are placed in deep petri dishes (9 × 2.5 cm) on two Whatman No. 1 filter papers (20). The dishes are incubated at 23 ± 1 C, with lids offset 1 cm to allow slow drying of the agar. After 4–6 days at room temperature, zygospor are washed with sterile water, excess water is removed, and the lids of the dishes are left partially offset (about 1 cm) for 4–5 hours, then replaced over the dishes. Germinating zygospor can be observed 2 days after the dishes have been covered.

### Ecology, Epidemiology, and Disease Cycle

Sporangiospores of *M. piriformis* in soil of pear orchards are clustered in both vertical and horizontal planes (Fig. 8). More than 75% of the spores are in the top 2 cm of the soil, where *M. piriformis* colonizes organic matter such as fallen fruit (6). Fallen fruit are infected through contact with infested soil or by fruit-to-fruit spread involving rodents, birds, and insects. In addition, fall and winter rains wash spores from decayed fruit into the soil or splash them on other fruit.

*M. piriformis* cannot compete effectively with other soil microbes at 20 C

and above but is a strong competitor at temperatures below 15–20 C (7). The fungus survives best in cool, dry soil. In California, soil temperatures above 26 C in peach and nectarine orchards greatly reduce sporangiospor survival and development of *M. piriformis* (15). Propagules of *M. piriformis* increase in soil only when favorable nutrient status, cool temperature, and high moisture level prevail.

When temperature permits, the fungus can survive 19–20 months on infected fruit and up to 1 year on endocarps of stone fruit buried in soil. Nutrients on the endocarp contribute to germination, growth, and sporulation of the fungus (Fig. 9) (13). Sporangiospores can also germinate, grow, and sporulate on peach leaves fallen on the ground or on common weeds of pome and stone fruit orchards (Fig. 8), such as chickweed (*Stellaria media* L.) and ryegrass (*Lolium perenne* L.), when temperatures are low and antagonistic microflora are limited (16). Other species of *Mucor* reported to cause decay of stone fruit can usually survive as sporangiospores or chlamydo-spores in soil and can withstand higher soil temperatures.

In pome and stone fruit orchards, infected fruits can be buried intact or be crushed by tractor tires and mixed with and buried in soil during cultivation. If stone fruits remain intact on the soil surface, the fruit tissue disintegrates and the sporangiospores of the fungus persist on the endocarps until the following season (Fig. 8) (13).

Because *M. piriformis* is heterothallic, infection of orchard fruit from both mating types is necessary for the formation of zygospor. Insects or other animals can pick up propagules of the two mating types and transfer them to the same fruit, assuring zygospor development (21). Several vinegar flies collected in pear and nectarine orchards and plated on APDA developed colonies bearing zygospor of *M. piriformis*. In addition, in laboratory studies, dried-fruit beetles (*Carpophilus hemipterus* L. and *C. freemani* Dobson) and vineyard flies (*Drosophila melanogaster* Meig.) acquired propagules of + and – mating types of *M. piriformis* from decaying peaches and plums and successfully transferred them to healthy fruit, resulting in the development of zygospor.

**Sources of inoculum.** Infested soil and debris are the major sources of inoculum for the infection of fruit by *M. piriformis*. In Oregon, when soil attached to picking bins was collected and assayed for the fungus, 95% of the samples were infested with 1,042–8,333 propagules of *M. piriformis* per gram of dry soil. Inoculum builds up in the soil when fruit on the ground becomes infected and debris contaminates the surrounding soil. The fruit are inoculated as the soil and debris on picking bins are removed when the bins are immersed in dump tanks or are stacked in storage. Sporangiospores of *M. piriformis* did not survive on the surface of pear fruit (26), and we were unable to capture propagules from the air using a spore trap operated for 8 hours during commercial harvest (19). In addition, no propagules were found in washings from fruit and leaf samples taken from pear trees during harvest (19). However, propagules of *M. genevensis* Lendner and, occasionally, of *M. plumbeus* (an airborne fungus) were recovered from leaf surfaces of pear trees.

**Survival and population dynamics of propagules.** Although sporangiospores originating from sporangia are the primary long-term survival structures (15), sporangiospores produced from germinated zygospor or chlamydo-spore-like structures could be another source of inoculum. Sporangiospor levels of the fungus fluctuate in an annual cyclic pattern, with a sharp increase occurring about 1–3 months after harvest (Fig. 10) (5). Colonization of fruit dropped on the soil before and during harvest coincides with the abundant sporulation of the fungus during this period.

In California, propagules of *M. piriformis* decline exponentially when high (>27 C) temperatures occur in orchard soils (15,17). Depending on the location, isolates of *M. piriformis* from California survived on decayed peaches buried intact 5 cm deep in soil for more than 1.5 years. Mycelia of the fungus buried in soil survived for only 3 weeks in the field. In laboratory studies, temperatures

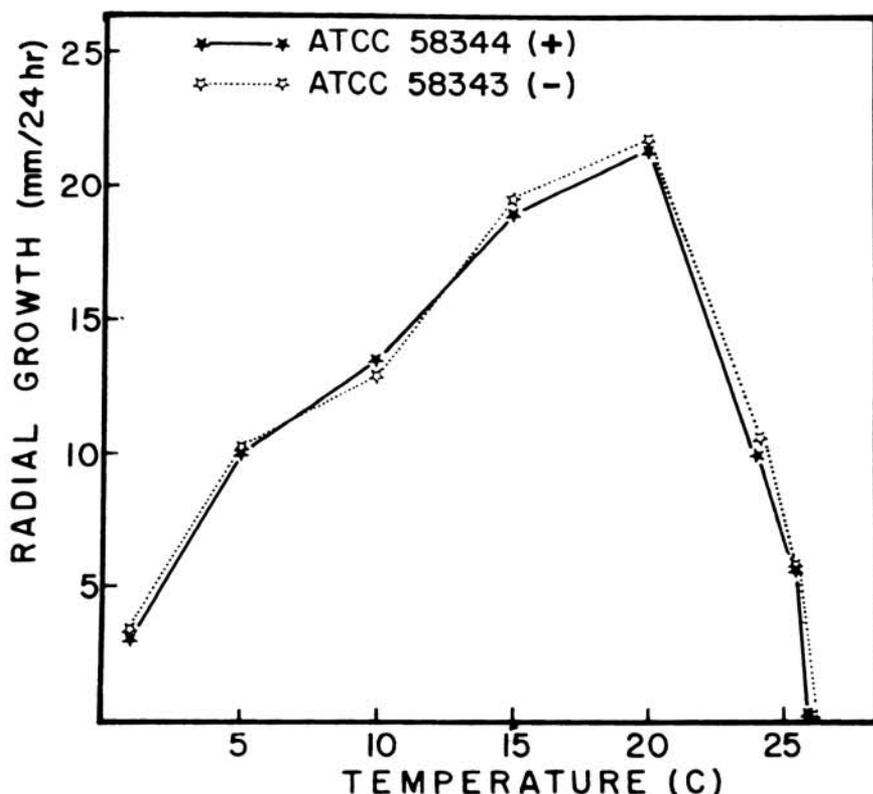


Fig. 5. Radial growth of mating types + and – of *Mucor piriformis* on acidified potato-dextrose agar at selected temperatures.

of 27–33 C result in a rapid decline of sporangiospores in soil, reduced and abnormal germination, and death. At 27 C, however, surviving sporangiospores show yeastlike growth in artificial media (17). Sporangiospores survived better in dry (–1,300 bars matric potential) than in wet (–0.3 bar matric potential) or wet-dry (–0.3 to –1,300 bars) soil.

**Dispersal of propagules.** *M. piriformis* is a wet-spored fungus (spores are embedded in a mucilaginous matrix) that relies primarily on rain splash and insects and birds for dispersal of its sporangiospores. The fungus is spread from fruit to fruit by nitidulid beetles (*C. hemipterus* and *C. fremani*) and vineyard flies (*D. melanogaster*) (21), both very common insects in stone fruits and the flies very common in pome fruit orchards. Of vineyard flies collected in three pear orchards in Hood River, Oregon, 68–94% produced colonies of *M. piriformis* when plated on APDA.

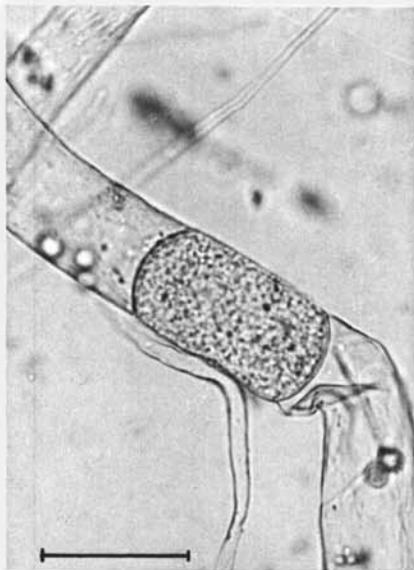


Fig. 6. Chlamydospore-like (gemmae) structure of *Mucor piriformis*. Scale bar = 50  $\mu$ m.

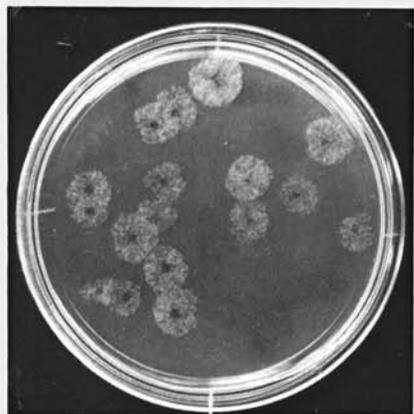


Fig. 7. Petri dish with colonies of *Mucor piriformis* developed from plated soil suspensions after 24 hours at 21 C and 48 hours at 0–1 C.

Pieces of infected fruit scattered by birds feeding on fruit on the ground provide another means for spreading the fungus in fruit tree orchards. *M. piriformis* can also be spread through soil by machinery passing over decaying fruit or by cultivation of the orchard floor.

**Infection courts and decay development.** Like *Rhizopus* spp., *M. piriformis* requires wounds for infection unless spore suspensions contain nutrient sources for the germination of spores. On pears and apples, the fungus is associated with stem punctures and animal wounds (rodents, birds, and insects). Edney (8) reported that *M. strictus* (a relative of *M. piriformis*) infected pear fruit that had small skin blemishes, whereas fruit without skin blemishes was not infected. In cold storage (–1 C), initial postharvest infections usually occur via wounds, whereas secondary spread of the fungus is by infected fruit contacting healthy fruit (Fig. 11) or by dissemination of sporangiospores in exuded juice dripping from infected fruit. Polygalacturonases produced by the fungus soften and liquefy the fruit tissues. All tested strains of *M. piriformis* from California and Chile produced significant amounts of polygalacturonases at 0, 6, and 21 C. Isolates of less virulence produce smaller amounts of polygalacturonases (14). Secondary spread in cold storage is com-

mon in pears but not in apples.

Sporangiospores of *M. piriformis* are so virulent that inoculations with single sporangiospores produced infections in every case. At 20 C, 4 days after inoculation, lesion diameter ranged from 40 to 44 mm. The fungus progresses faster along the suture line of stone fruit because tissue firmness in this area is less than on the sides (“cheeks”) of the fruit.

Pears and apples can be inoculated experimentally by first being wounded with a sharp instrument equivalent in dimensions to the fruit stem, since stems are the most common cause of wounding. Then, fruit is dipped in a suspension of sporangiospores, usually 4,000 spores per milliliter, or is first dipped in a fungicide suspension, dried, and then inoculated with a spore suspension.

Pear fruit becomes particularly vulnerable to infection during the last month before harvest, and overmature fruit is especially susceptible to decay (25). Similarly, green stone fruit is resistant to infections, but late-harvested, overmature fruit is susceptible to decay.

### Control Strategies and Prospects

Infection and decay by *M. piriformis* occur at temperatures as low as 0 C. Rapid cooling of fruit and storage at a low temperature reduce rotting but do not prevent infection, and the viability of sporangiospores is not reduced by ex-

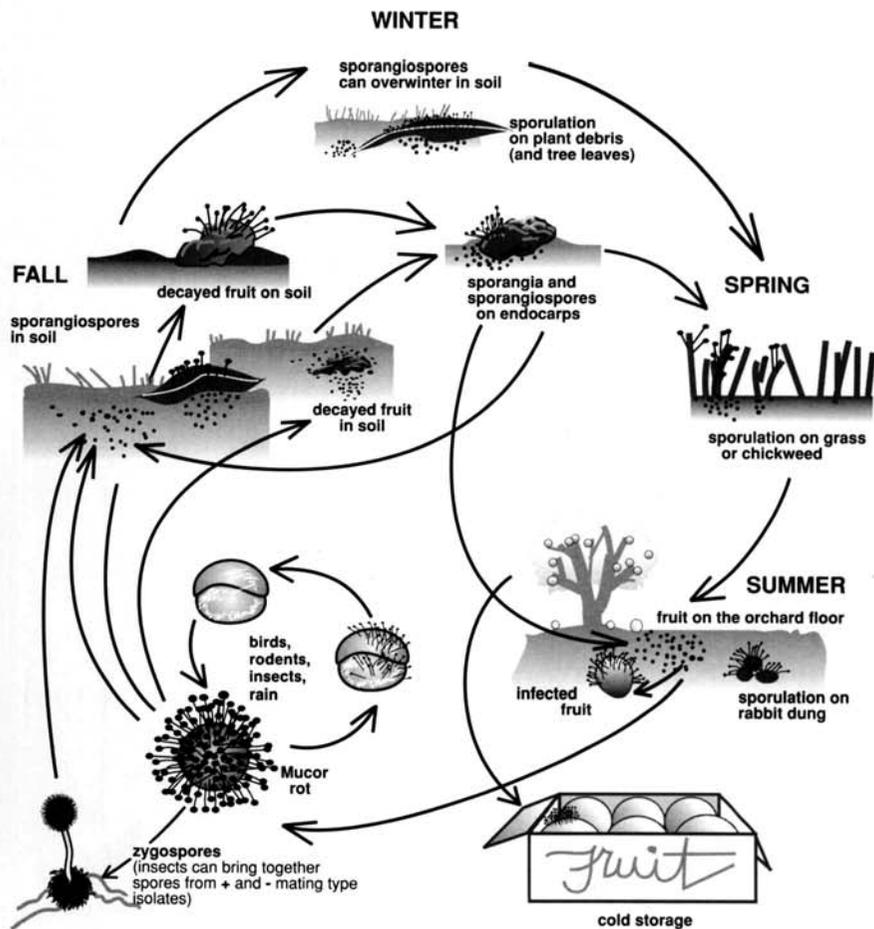


Fig. 8. The life cycle of *Mucor piriformis* in a fruit tree orchard.

posure to chill temperatures (4). Fungicides currently registered for postharvest treatment of pome and stone fruits are ineffective against *M. piriformis* and other Zygomycetes generally. The fungus is highly resistant to fungicides (benzimidazoles, dicarboxamides) commonly used on fruits to control other diseases, such as gray mold caused by *Botrytis cinerea* Pers.:Fr. Thus, Mucor rot can be reduced only by other control measures. Most fruit losses caused by Mucor rot are associated with unsanitary practices (24). Therefore, sanitation practices, such as removing fallen fruit from the ground, can reduce inoculum sources in soil and subsequently control rot in storage. In Oregon, growers of pear orchards remove fallen fruit from the ground to reduce buildup of sporangiospore inoculum of *M. piriformis*. In Hawaii, removal of fallen fruit from the ground reduced the percentage of guava fruits infected by *M. hiemalis*, which is spread by three species of fruit flies (11).

*M. piriformis* also survives on wooden fruit bins, so these should be thoroughly washed or steam-cleaned and covered



Fig. 9. Growth and sporulation of *Mucor piriformis* on peach endocarps in the absence of mesocarp tissues.

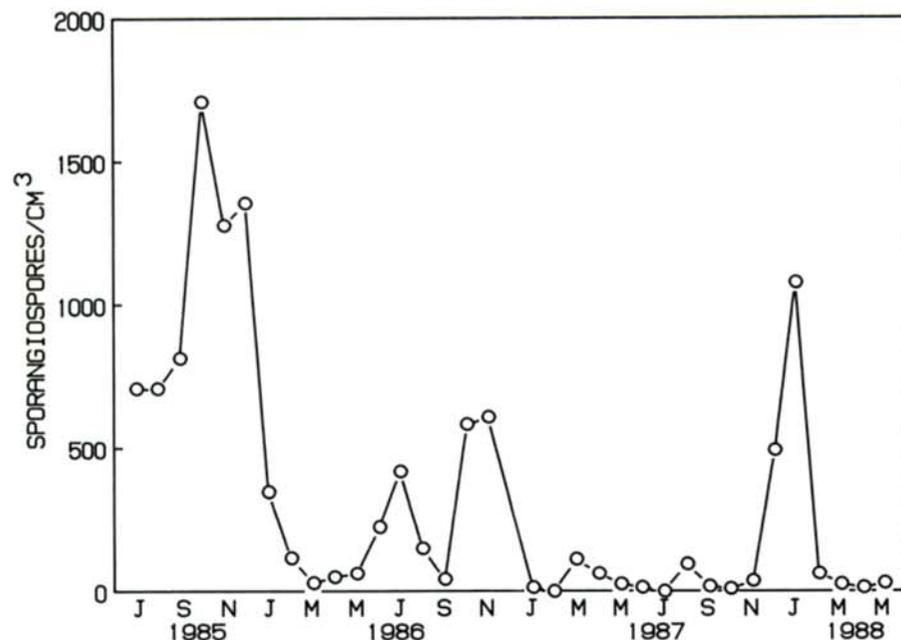


Fig. 10. Population densities of sporangiospores of *Mucor piriformis* in the upper 5 cm of soil in a pear orchard in the Hood River Valley of Oregon over a 36-month period, beginning July 1985. Each point is the average of three values.

internally with paper or plastic pads to protect fruit from bruising and wounding. Bins also should be designed and positioned to minimize collection of soil and debris on lower surfaces. Soil in loading areas where bins are stacked should be covered with wood chips or sawdust to avoid contact between bins and soil. In orchards with level terrain, bins may be placed on trailers to avoid contact with soil during harvest. If possible, fruit should be harvested in dry weather, when the incidence of infection is lower than in wet weather (2). Fruit that falls to the ground during harvest should never be placed in bins with fruit harvested from trees. During 1988–1989, for instance, excessive decay caused by *M. piriformis* developed on plums during export from California to the Orient after pickers had collected fruit from the ground. Fallen fruit that had been wounded during picking or by insects could have been the source of inoculum for infection of healthy fruit collected from the trees.

In packinghouses, dump tank and flume water should contain chlorine or sodium *o*-phenylphenate to keep spore levels as low as possible. The concentration of sporangiospores in dump-tank water increases as the packing season progresses but usually remains less than 500 spores per milliliter (27), enough to inoculate about 10–20% of wounded fruit floated in unchlorinated contaminated water (28). Pears require addition of a flotation salt to the water-handling system. Chlorine is more effective for reducing spore viability in sodium sulfate than in sodium silicate or sodium carbonate, and sodium *o*-phenylphenate is more effective in sodium ligninsulfonate than in other flotation salts (30).

A thorough, fresh-water rinse of fruit removes spores and reduces decay.

Prestorage heating of d'Anjou and Bosc pears at 27 C for 2 days reduces Mucor rot without adversely affecting the storage life or the quality of ripened fruit. In cold storage, Mucor stem-end rot is less with low-oxygen atmosphere (1% O<sub>2</sub>, 99% N<sub>2</sub>) than with regular air. Lowering the relative humidity in storage even slightly will reduce Mucor rot; therefore, fruit should be dry when placed in cold storage. Wrapping fruit individually in copperized paper also reduces secondary spread of Mucor rot.

The concept of thermotherapy to control postharvest decay is not new, but several limitations have prevented the application of this system in packinghouses. Tests showed that peaches and pears dipped in hot water (47 C) for 30 minutes had lower inoculum levels and fewer postharvest infections (Fig. 12) (17). It may become possible to maintain the water in the dump tanks at 47 C to kill the propagules of *M. piriformis* or to prevent decay from propagules that have been introduced into fruit wounds in the field.

Future attempts to control Mucor rot more effectively should emphasize effective sanitation to prevent contamination of fruit in the field, in the packinghouse, and in storage. More information is needed on the possibility of using heat to control Mucor decay in pome fruits, keeping in mind that such procedures may favor Rhizopus rot on stone fruits. Biological control of postharvest pathogens is now a reality. Its use in reducing Mucor rot should be one of the priorities,



Fig. 11. Secondary spread of *Mucor piriformis* in stored peaches.



Fig. 12. Effect of heat treatment on infection of d'Anjou pear fruit by *Mucor piriformis* (California isolate). Fruits were dipped for 30 minutes in water (left) at 21 C and (right) at 47 C.

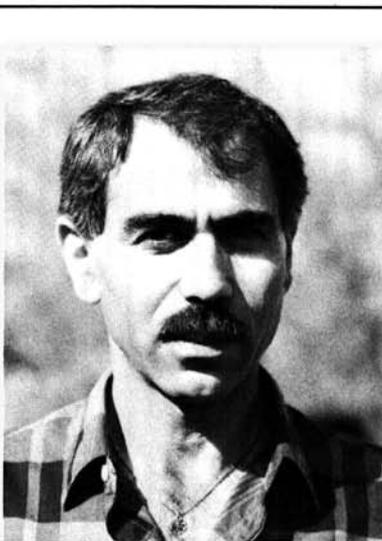
particularly since no chemical treatments for this disease are yet available.

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**Themis J. Michailides**

Dr. Michailides is an assistant research plant pathologist with the Department of Plant Pathology, University of California, Berkeley, at Kearney Agricultural Center, Parlier. He received his M.S. degree in irrigations and agricultural development from the Superior College of Agricultural Sciences, Athens, Greece, in 1973 and his M.S. and Ph.D. degrees in plant pathology from the University of California at Davis. His responsibilities since joining the University of California at Berkeley in 1989 have included research on fungal diseases of fruit and nut trees and other crops and postharvest diseases. He has a special interest in the study of ecology, epidemiology, and control of postharvest pathogens of fruit and nut trees.



**Robert A. Spotts**

Dr. Spotts is professor of plant pathology at Oregon State University's Mid-Columbia Agricultural Research and Extension Center, Hood River. He received his Ph.D. degree in plant pathology at Pennsylvania State University in 1974. He served on the faculty of Ohio State University for 4 years and spent a sabbatical at the Horticultural Research Institute in Victoria, Australia. He is responsible for research on diseases of pome and stone fruits, and he specializes in the biology, epidemiology, and control of preharvest and postharvest diseases of pear.