Fruit Rots of Pumpkin

A Serious Threat to the Pumpkin Industry

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The word “pumpkin” is derived from the Latin word *pepo*, which is a botanical term for fruit with a hard rind and fleshy interior (51). The commonly used scientific name for pumpkin is *Cucurbita pepo* L. But some other cucurbits, such as *Cucurbita moschata* (Duchesne) Duchesne ex Poir (e.g., ‘Dickenson’ and ‘Libby Select’, known as processing pumpkin) (Fig. 1B) and *Cucurbita maxima* Duchesne (e.g., ‘NK 580’), which are winter squash, are also known as pumpkins. Tropical pumpkin or calabaza (*Cucurbita moschata*) is a popular type of hard squash in Latin America (37). Commonly, any *Cucurbita* fruit with an orange color is considered a pumpkin (Fig. 1A to C); however, *Cucurbita* cultivars with white fruit, such as ‘Lumina’ and ‘Cotton Candy’ are also considered pumpkins (Fig. 1D). In the United States, any cultivar of *C. pepo* used for baking, food stock, or Halloween jack-o-lanterns is considered a pumpkin (Fig. 1A and C). In this article, we will use the term “pumpkin” instead of “pumpkin fruit,” unless otherwise mentioned.

Pumpkins vary in shape, size, weight, and color (Fig. 1) (48). Most pumpkins are round, but some are oblong to pear-shaped. The diameters of pumpkins range from 5 cm to more than 50 cm (Fig. 1C). Pumpkins may weigh from less than 0.5 kg to

more than 500 kg. A common grouping of pumpkins is based on their sizes (48), as follows: giant (>22.5 kg), extra large standard (13.5 to 22.5 kg), large standard (7 to 13.5 kg), medium standard (3 to 7 kg), small standard (0.5 to 3 kg), and miniature (<0.5 kg). Giant pumpkins are *C. maxima*, and jack-o-lantern pumpkins are *C. pepo*.

Most pumpkins are produced by direct seeding in the field; however, due to increased seed prices more growers are using transplants (38). Depending on the fruit size, the number of pumpkin plants per hectare may range from 1,500 to 9,000. The number of fruits per plant ranges from

1 to 20, depending on cultivar and fruit size. Generally, pumpkins set about 30 days after seeding (49). A healthy fruit has green color and a solid stem at the beginning of fruit set (Fig. 2). Fruits reach full size by about 20 days after fruit set, often start to show color by 30 days after fruit set, and are usually fully colored by 45 days after fruit set (Fig. 2). In most cultivars, mature fruit have hard skin that cannot be cut with a fingernail. Even though pumpkins are ready for use as jack-o-lanterns by 50 days after fruit set, seed fill may not be complete until about 70 days after fruit set.

Fig. 1. Pumpkins: A, jack-o-lantern pumpkins (cv. Howden); B, processing pumpkins (cv. Dickinson); C, different pumpkin cultivars; D, white pumpkins (cv. Cotton Candy).

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Jack-o-lantern pumpkins are grown worldwide (70). Farm-gate value of jack-o-lantern pumpkins in the United States ranges from \$2,500 to more than \$12,000 per ha. Jack-o-lantern pumpkins are produced for food, animal feed, oil extraction from seed (in Europe), and entertainment (e.g., Halloween celebration in the United States). Seed of some pumpkin cultivars are used as a nut. Other jack-o-lantern pumpkin products are pumpkin ice cream, pumpkin wine, pumpkin butter, and pumpkin honey in North America.

The processing pumpkin (canned pumpkin) industry is limited to North America (1,2). More than 90% of processing pumpkins produced in North America are grown and processed in Illinois. The processing pumpkin industry was started by the Dickinson family in central Illinois in the late 1920s. Production of processing pumpkins grown in Illinois increased from less than 1,000 ha in the 1930s to more than 4,800 ha in 2009. The major processing pumpkin cultivar is 'Dickinson'. The city of Morton in Tazewell County in Illinois is known as the "Pumpkin Capital of the World" (Laatsch Nestle Food, *personal communication*). The gross values of processing pumpkin products exceed \$24,000 per ha.

Pumpkins can be infected by fungal, bacterial, and viral pathogens from the time of fruit set until harvest (2,6, 20,31,32,64,70). Also, some diseases may develop in pumpkins during transit and in storage. A variety of fruit rots may be seen on pumpkins during the growing season (Figs. 3 and 4). Major fruit rots of pumpkins are Phytophthora rot (*Phytophthora capsici*), Fusarium rot (*Fusarium* spp.), bacterial spot (*Xanthomonas campestris* pv. *cucurbitae*), black rot (*Didymella bryoniae*), and Sclerotinia rot (*Sclerotinia sclerotiorum*), ranked from the most to the least important, respectively (2,70). Recently, the incidence of Phytophthora blight on cucurbits has dramatically increased in the world (2,20,23,33). Pumpkin production and industry is seriously threatened by heavy crop losses resulting from Phytophthora fruit rot. For example, outbreaks of Phytophthora blight have threatened the processing pumpkin industry in Illinois (2,4). Similarly, the pickling cucumber industry in Michigan is jeopardized by the increased occurrence of

Phytophthora fruit rot (20). Because of heavy crop losses, growers often stop producing cucurbits on their own farms and move into different areas, sometimes traveling more than 80 km, to find fields that are not infested with *P. capsici*.

The importance of pumpkin fruit rots differs from one pumpkin growing area to another. Phytophthora and Fusarium fruit rots occur in almost all pumpkin growing areas. Black rot commonly occurs in the southeastern, mid-Atlantic, and northeast-

Fig. 2. Pumpkins from fruit set to maturity (cv. Howden).

Fig. 3. Infected pumpkins: A, *Choanephora* fruit rot (*Choanephora cucurbitarum*); B, scab (*Cladosporium cucumerinum*); C, anthracnose (*Colletotrichum orbiculare*); D, black rot (*Didymella bryoniae*); E, Fusarium rot (*Fusarium* sp.); F, Phytophthora rot (*Phytophthora capsici*); G, Plectosporium blight (*Plectosporium tabacinum*); H, Pythium rot (*Pythium* spp.); and I, southern blight (*Sclerotium rolfsii*) (courtesy G. J. Holmes).

Fig. 4. Infected pumpkins: A, *Sclerotinia* rot (*Sclerotinia sclerotiorum*) (courtesy M. T. McGrath); B, *Septoria* spot (*Septoria cucurbitacearum*); C, Bacterial wilt (*Erwinia tracheiphila*); D, angular leaf spot (*Pseudomonas syringae* pv. *lachrymans*); E, bacterial spot (*Xanthomonas campestris* pv. *cucurbitae*); F, Cucumber mosaic virus (CMV); G, Papaya ringspot virus (PRSV); H, Watermelon mosaic virus (WMV); and I, Zucchini yellow mosaic virus (ZYMV).

ern United States, whereas it is not a significant disease in the Midwest. In contrast, bacterial spot occurs widely in the northern regions, particularly in Illinois. Sclerotinia pumpkin rot is more common in northern regions of the United States.

Phytophthora Rot

Phytophthora rot, caused by the oomycete *Phytophthora capsici* Leonian, is one of the most serious threats to production of pumpkins and other cucurbits throughout the world (1,16,18,20,23–25,33). *P. capsici* was first described by Leonian on pepper in New Mexico in 1922 (35). The pathogen was subsequently described in more than 50 plant species, including all cucurbit crops (16,19,20,56).

Phytophthora infection of cucurbits is known as Phytophthora blight (5,20). *P. capsici* was first reported on cucurbits in 1937 in Colorado and California (57). Since then, Phytophthora blight has been observed in cucurbit growing areas throughout the world (15–18,20,55). *P. capsici* infection occurs in temperate, subtropical, and tropical environments in the world.

In 1996, Erwin and Ribeiro (16) reported that 49 plant species can be infected by *P. capsici*. Among the major hosts are red and green peppers (*Capsicum annuum*), watermelon (*Citrullus lanatus*), cantaloupe and honeydew melon (*Cucumis melo*), cucumber (*Cucumis sativus*), blue Hubbard squash (*Cucurbita maxima*),

Cucurbita pepo varieties including acorn squash, gourd, pumpkin, yellow squash, and zucchini squash, processing pumpkin (*C. moschata*), tomato (*Solanum lycopersicum*), black pepper (*Piper nigrum*), and eggplant (*Solanum melongena*). In 2004, Tian and Babadoost (56) reported six additional hosts: beet (*Beta vulgaris*), Swiss chard (*Beta vulgaris* var. *cicla*), lima bean (*Phaseolus lunatus*), turnip (*Brassica rapa*), spinach (*Spinacia oleracea*), and one weed species, velvetleaf (*Abutilon theophrasti*). At present, the reports indicate that more than 50 species in 15 plant families are hosts to *P. capsici*.

P. capsici can infect pumpkin fruit at any stage of development, during transit, and in storage (1,33,70). In fields, infections usually appear first in low areas where the soil remains wet for longer periods of time. Fruit rot generally starts on the area of the fruit that is in contact with the soil (Fig. 5A to D); however, when an infected leaf or vine comes in contact with a fruit, fruit rot will start at the point of contact. Symptoms on the upper surface of the fruit develop following rain or overhead irrigation, which provides splashing water for pathogen dispersal (Fig. 5C). Fruit rot typically begins as a water-soaked lesion (Fig. 5C) and expands, eventually covering the fruit with white mold (Fig. 5D) (1,33). Fruit infection progresses rapidly, resulting in complete collapse of the fruit. The pathogen produces numerous sporangia on infected fruit (Fig. 5A to D). The pathogen also infects seedlings (Fig. 6A), leaves (Fig. 6B), vines (Fig. 6C), and crown (Fig. 6D). Phytophthora foliar blight and fruit rot can result in total loss of the crop (1).

As an oomycete, the pathogen produces coenocytic hyphae, asexual sporangia and zoospores (Fig. 7B and C), and sexual oospores (Fig. 7D and E) (16). Colonies of *P. capsici* isolated from pumpkins grow from 10 to 36°C, with optimum temperatures of 24 to 33°C (25). Growth patterns of colonies are cottony, petaloid, rosaceous, and stellate (Fig. 7A). Sporangia of *P. capsici* are variable in shape and are papillate with a long pedicel (Fig. 7B and C). Sporangial shapes are influenced by light and other cultural conditions, and are subspherical, ovoid, obovoid, ellipsoid, fusiform, and pyriform. Length × width of sporangia varies from 32.8 to 65.8 × 17.4 to 38.7 µm (16). Length/breadth ratios of sporangia range from 1.3:1 to 2.1:1. Pedicel lengths are highly variable among the isolates, ranging from 35 to 138 µm. Zoospores are produced inside sporangia (Fig. 7C) and are biflagellate. Oospore (Fig. 7D) diameter of *P. capsici* ranges from 22 to 34 µm, and antheridia are amphigynous. *P. capsici* is heterothallic, and both A₁ and A₂ types may exist in the same field (16,25).

P. capsici is a soilborne pathogen and survives between crops as oospores in soil or mycelium in plant debris (16,18,20,

Fig. 5. Pumpkins infected by *Phytophthora capsici*: A, infected young fruit; B, fruit infection at maturity; C, initial infection of fruit; D, advanced infection of fruit.

Fig. 6. Pumpkin foliage infected by *Phytophthora capsici*: A, seedling damping-off; B, leaf spot; C, vine infection; D, crown infection.

43,44). Oospores are resistant to desiccation, cold temperatures, and other extreme environmental conditions, and can survive in the soil, in the absence of a host plant, for 3 years or longer (18,20,44). Oospores germinate and produce sporangia and zoospores. Zoospores are released in water and dispersed by irrigation or surface water (20). Zoospores are able to swim for several hours and infect plant tissues. Abundant sporangia are produced on infected tissues, particularly on affected fruit (Fig. 5), and dispersed by water or through the air. Sporangia can either germinate and infect host tissues directly, or they can release zoospores, which can infect the plant. If environmental conditions are conducive, the disease develops rapidly. Soil moisture conditions are important for disease development (18,20). Sporangia form when soil moisture is at field capacity, and they release zoospores when soil is saturated. The disease is usually associated with heavy rainfall, excessive irrigation, or poorly drained soil. Frequent irrigation increases incidence of the disease.

Isolates of *P. capsici* differ in genetics and virulence. The amplified fragment length polymorphism (AFLP) technique has been used to investigate genetic differences among isolates of *P. capsici* (20,25,50). The AFLP technique results in the selective amplification of restriction fragments from a digest of total genomic DNA with the polymerase chain reaction (PCR). Random amplified polymorphic DNA (RAPD) markers were employed to assess genetic variation among 24 isolates of *P. capsici* from 10 individual fields at six locations in Illinois (25). Unweighted mean pair group analysis clustered isolates into six groups (Fig. 8). The genetic distances ranged from 0.03 to 0.45. Inoculation of pumpkin seedlings in the greenhouse revealed that the isolates belonged to six distinct genetic groups differing significantly ($P = 0.05$) in virulence (Fig. 8).

No single method is available to provide adequate control of fruit rot or foliar blight of pumpkin caused by *P. capsici*. A combination of measures should be practiced to reduce the damage caused by *P. capsici* on pumpkin (4,18,20,24). The most effective strategy in controlling *P. capsici* is preventing movement of the pathogen to a new field. The following practices can help to manage Phytophthora blight in pumpkin fields: select fields with no history of Phytophthora blight; select fields that did not have cucurbit, eggplant, pepper, or tomato for at least 3 years; select fields that are well isolated from fields infested with *P. capsici*; select well-drained fields, or do not plant the crop in the areas of the field that do not drain well; clean farm equipment of soil between fields; avoid excessive irrigation; do not irrigate from a pond that contains water drained from an infested field; do not work in wet fields;

Fig. 7. *Phytophthora capsici*: A, growth patterns (cottony, petaloid, rosaceous, stellate); B, sporangia and zoospores; C, sporangium releasing zoospores; D, a single oospore; E, oospores produced in a culture plate.

Fig. 8. Clustering of 24 isolates of *Phytophthora capsici* from 10 different processing pumpkin fields at six locations in central Illinois with phylogenetic analysis using parsimony (PAUP) program (25). Genetic distance was calculated according to the equation developed by Nei and Li (41). Pathogenicity groups are based on disease severity of inoculated pumpkin seedlings in the greenhouse.

scout the field for *Phytophthora* symptoms, especially after major rainfall, and particularly in low areas; when symptoms are localized in a small area of the field, disk the area; discard infected fruit, but not in the field; do not save seed from a field where *Phytophthora* infection occurred; remove healthy fruit from the infested area as soon as possible and check them routinely; do not display fruit for sale in an area that is infested with *P. capsici*; and apply fungicides, when recommended. Seed treatment with either mefenoxam or

metalaxyl can protect pumpkin seedlings for 5 weeks after sowing (4). Several fungicides have been labeled for control/suppression of *P. capsici* in pumpkin fields (3,22). However, more than 10 applications of fungicides may be needed to prevent crop losses.

Fusarium Rot

Fusarium crown and fruit rot of cucurbits was first described in detail in South Africa in 1932 (36). *Fusarium* fruit rot is one of the most common pre- and posthar-

vest diseases of pumpkins, as well as of other cucurbit crops (10,14). A number of *Fusarium* species, including *F. graminearum*, *F. acuminatum*, *F. culmorum*, *F. moniliforme*, *F. semitectum*, *F. equiseti*, *F. scirpi*, and *F. solani*, have been reported as causal agents of cucurbit fruit rot (10). In a recent investigation, however, Elmer et al. (14) determined *Fusarium solani* f. sp. *cucurbitae* (Mart.) Appel & Wollenweb. emend. W.C. Snyder & H.N. Hans as the main causal agent of fruit rot of pumpkins in the United States.

Symptomatology of fruit rot of pumpkins can vary considerably, dependent on the stage of lesion development (Fig. 9). Symptoms range from small, pitted, corky spots to large, sunken areas covered with a white or gray mold (Fig. 9A and B) (10,14). Most infections occur in the areas that are in contact with the soil. The pathogen also causes a crown rot (Fig. 9C) (14,36). The initial symptom of crown rot in the field is usually wilting of leaves. Within several days, the entire plant wilts and dies. If the soil is removed from around the base of the plant, a very distinct necrotic rot of the crown and the upper portion of the taproot is evident. The entire affected plant usually wilts, collapses, and dies within 2 to 4 weeks.

F. solani f. sp. *cucurbitae* produces white to cream mycelium (Fig. 10A) (11). The pathogen forms all three asexual spore types (microconidia, macroconidia, and chlamydospores) (Fig. 10B to D) and a sexual stage when proper mating types are present (7,36). The teleomorph of *F. solani* is *Nectria haematococca* Berk. & Broome (syn. *Hypomyces solani* (Rke. & Berth.) W.C. Snyder & H.N. Hans) (58,59). Microconidia form abundantly in the aerial mycelium from elongated lateral phialides. They are hyaline, cylindrical wedge-shaped, or allantoid (Fig. 10B), 9 to 16 × 2 to 4 μm, and are usually single-celled but occasionally develop a septum and become 2-celled. Macroconidia are abundant in cream sporodochia. The macroconidia are long (40 to 100 × 5 to 7 μm), slightly curved, relatively wide, usually 3 to 4 septate, thick-walled with a short, rounded, and sometimes hooked apical cell and a notched base to the basal cell (Fig. 10C). They are often referred to as "sausage-shaped" because they are rounded at each end. Conidiophores are branched or unbranched monophialides. Chlamydospores are formed abundantly in most cultures within 2 to 3 weeks (Fig. 10D), and are usually most obvious in hyphae on the agar surface. They are usually globose to oval, smooth to rough walled, 10 to 11 × 8 to 9 μm, either intercalary or terminal. Chlamydospore formation is more frequent in darkness than in light (7). *N. haematococca* produces reddish orange or white perithecia with eight 2-celled ascospores in each ascus. Pathogenic isolates of *F. solani* are generally heterothallic, having two or

Fig. 9. Pumpkins infected by *Fusarium* spp.: A and B, *Fusarium* fruit rot; C, *Fusarium* crown rot.

Fig. 10. *Fusarium solani*: A, colony on potato dextrose agar; B, microconidia; C, macroconidia; D, chlamydospores.

more mating types. The pathogen has two races (14,36). Race 1 causes root, stem, and fruit rot and occurs worldwide. Race 2 causes fruit rot in the United States. The optimum temperature for growth and conidia formation of *F. solani* in culture plates is 28°C (7).

F. solani survives in soil (10,11). It is more abundant in higher rainfall areas or in irrigated soils. *F. solani* is also a seedborne pathogen. This pathogen may directly penetrate fruit under moist or wet conditions (10). Wounds facilitate fungal entry to fruit. Most infections by *F. solani* occur in the field (preharvest) and, to a lesser extent, during harvesting and handling.

Management of fruit rot of pumpkin, as well as fruit rots of other cucurbits caused by *Fusarium* spp., is difficult (10). Preharvest fungicide applications have been ineffective, because of difficulty in obtaining sufficient coverage of the fruit. Crop rotations of 3 years or longer with noncucurbit crops will help to reduce the incidence of pumpkin rot. Avoidance of wounding during harvest and packing, storage in dry conditions, and proper handling during transit and marketing reduce postharvest decay (10).

Bacterial Spot

Bacterial spot, caused by *Xanthomonas campestris* (Pammel) Dawson pv. *cucurbitae* (Bryan Dye.), is a serious disease in some pumpkin growing areas (6,32,39,42). The pathogen also infects summer and winter squashes, cucumbers, and gourds (5,6,65). The disease was first described on Hubbard squash in New York in 1926 (12). Subsequently, the disease was reported from cucurbit growing areas in Asia, Australia, Europe, and North America (65). The disease caused by *X. campestris* pv. *cucurbitae* is commonly known as bacterial leaf spot. Fruit rot due to infection with this pathogen exceeds 50% in some commercial fields in Illinois (M. Baba-doust, unpublished data).

The bacterium is a seedborne pathogen (65). It also survives in plant debris. Attempts to isolate the bacterium from soil have been unsuccessful. Very little is known about the biology and epidemiology of this bacterium. Leaf and fruit infection takes place during the summer months when temperatures are high and plant tissues are moist (most commonly after heavy rain or overhead irrigation).

X. campestris pv. *cucurbitae* infects leaves and fruit of pumpkin any time during the growing season (Fig. 11). Lesions on leaves are small (2- to 4-mm-diameter), angular, yellow to beige spots (Fig. 11C). Leaf infections can easily be overlooked because of their size. As lesions coalesce, they may appear more angular and similar to the spots caused by *Pseudomonas syringae* pv. *lachrymans* (65). The appearance and size of fruit lesions can vary, depending on rind maturity and the presence of

moisture (Fig. 11A and B). Initial lesions are small, slightly sunken, circular spots (1- to 3-mm-diameter), with a beige center and dark brown halo (Fig. 11B). Later, the cuticle and epidermis crack, and the lesions enlarge, reaching 10 to 15 mm in diameter, and become sunken. Penetration of bacteria into the flesh can lead to significant fruit rot in the field or later in storage.

X. campestris pv. *cucurbitae* is a yellow (Fig. 12), aerobic, gram-negative, non-spore-forming rod, measuring 0.5 to 1.3 × 0.4 to 0.6 µm, bacterium. It usually produces only one polar flagellum. On solid medium containing a utilizable carbohydrate, the colonies are yellow, convex, shiny, and of a slimy, mucoid consistency (21,47). Acid without gas is produced oxidatively from glucose, sucrose, fruc-

Fig. 11. Pumpkin infection with *Xanthomonas campestris* pv. *cucurbitae*: A, bacterial spots on an immature fruit; B, bacterial spots on a mature fruit; C, infected leaves.

Fig. 12. A comparison of colonies of *Xanthomonas campestris* pv. *cucurbitae* (causal organism of bacterial spot) with *Pseudomonas syringae* pv. *lachrymans* (causal organism of angular leaf spot) of pumpkin on three different culture media. *X. campestris* pv. *cucurbitae* is differentiated from *P. syringae* pv. *lachrymans* by its bright yellow color on yeast extract-dextrose-CaCO₃ (YDC) and on a selective medium for *X. campestris* (XCS) (52,66).

tose, galactose, arabinose, maltose, and cellobiose, and from mannitol, raffinose, and glycerol on prolonged incubation (6 to 20 days). Acid is not produced from inulin, dulcitol, *meso*-inositol, sorbitol, or salicin. Nitrite is not produced from nitrate by *X. campestris* pv. *cucurbitae*. Also, acetone and indole are not produced by this bacterium. Temperatures for multiplication of this bacterium are 30 to 32°C (optimum), 38 to 39°C (maximum), and 5°C (minimum). *X. campestris* pv. *cucurbitae* can be distinguished from *Pseudomonas syringae* pv. *lachrymans* by production of fluorescent pigment on King's B agar culture

medium (Fig. 12) (29). Also, *X. campestris* pv. *cucurbitae* is recognized from *P. syringae* pv. *lachrymans* with its bright yellow color on yeast extract-dextrose- CaCO_3 and on a selective medium for *Xanthomonas campestris* (XCS) (52,66,67).

Bacterial leaf spot and fruit rot, caused by *X. campestris* pv. *cucurbitae*, can be managed by the use of pathogen-free seed (39,42,65). The production of seed in arid regions under furrow irrigation is the best way to minimize seedborne pathogen populations, provided the seed crop was grown from disease-free seed on land where cucurbits had not been grown re-

cently. Minimizing moisture on plants by avoiding overhead irrigation or other means could help to reduce disease incidence. Crops for both seed and fruit production should be grown in fields that have had no cucurbits for at least 2 years. Where possible, only furrow irrigation should be used. Irrigation from surface drainage water near cucurbit fields should be avoided, as should entering fields for cultivation or harvesting when the foliage is wet from dew, rain, or irrigation. In warm, humid conditions, repeated application of copper sprays as foliar protectant can be helpful. Weekly copper application from the beginning of fruit set until harvest may be necessary to reduce disease incidence. Spray should be forced into the canopy to cover the foliage and fruit thoroughly.

Black Rot

Black rot (Fig. 13) was first reported in Europe and the United States in 1891. The disease is caused by the fungus *Didymella bryoniae* (Auersw.) Rehm (anamorph *Phoma cucurbitacearum* (Fr.:Fr.) Sacc.) (13,28,54). Black rot is an important pre- and postharvest fruit rot of pumpkins and winter squashes (54). Symptoms of black rot can appear in the field before harvest and continue to develop during transit and in storage. Small, water-soaked, dark-brown lesions develop and enlarge on fruit (69). As the lesions expand, they become black and sunken (Fig. 13A). Fungal fruiting bodies, pycnidia, develop on infected areas (Fig. 13B). The disease gets its name from the rot, which includes blackened fungal tissue on the rind (Fig. 13C). Affected fruit may collapse during or after harvest. Injury to the rind accelerates development of black rot on pumpkin.

When vegetative portions of pumpkins, and other cucurbit plants, are affected by *D. bryoniae*, the disease is known as "gummy stem blight" (Fig. 14) (27,28,54). Circular, tan to dark brown spots appear on the leaves, often first at or near the margins (Fig. 14B), and enlarge rapidly until the entire leaf is blighted (54). Circular, black or tan spots appear on the cotyledons and stems of young plants (Fig. 14C). Stem cankers develop in the cortical tissue, and a brown, gummy exudate is commonly produced on the surface (Fig. 14A).

Black rot and gummy stem blight occur commonly in the United States and Europe (54). Also, *D. bryoniae* and its anamorph, *P. cucurbitacearum*, have been isolated from most of the cucurbit production areas in Africa, the Americas, Asia, and Europe (27). *D. bryoniae* produces dark pseudothecia, which are globose, immersed, becoming erumpent, 125 to 213 μm diameter, opening by apical papillate ostiole (Fig. 15A) (45). The pseudothecia produce numerous bitunicate asci, which are cylindrical to subclavate, short stipitate or sessile, with eight ascospores per ascus, 60 to 90 $\times$ 10 to 15 μm (Fig. 15B). Ascospores are

Fig. 13. Black rot of pumpkin caused by *Didymella bryoniae*: A, pumpkin with black rot lesions; B, initiation of a black rot lesion; C, advanced black rot lesion.

Fig. 14. Gummy stem blight of pumpkin caused by *Didymella bryoniae* (anamorph *Phoma cucurbitacearum*): A, infected stem with gummy exudates (courtesy B. D. Bruton); B, gummy stem blight lesions on a leaf; C, infected stem with pycnidia of *P. cucurbitacearum*.

hyaline, ellipsoid, with mostly rounded ends, monoseptate, and slightly constricted at the septum, 14 to 18 × 4 to 7 µm. The upper cell of the ascospore is usually wider than the lower cell (Fig. 15B). *P. cucurbitacearum* produces dark pycnidia on the surface of infected tissues, which are solitary or aggregating, immersed, becoming erumpent, dark, 120 to 180 µm diameter (Fig. 14C and Fig. 15A and C). The conidia are hyaline, cylindrical with rounded ends, mostly 1-septate but a small percent unicellular, and 6 to 13 × 3 to 4 µm (Fig. 15D). No physiological specialization of the pathogen has been reported.

D. bryoniae survives between the growing seasons on diseased vines, crop debris, and seed of cucurbit crops (8,26,27,30,34,54). In most of the studies, *D. bryoniae* survived for fewer than 9 months in infested cucurbit debris (13,26,61). However, Keinath (27) studied survival of *D. bryoniae* in crowns of muskmelon plants with gummy stem blight cankers in South Carolina and reported that the pathogen was recovered from 2.5% of crowns buried 12.5 cm deep for 66 weeks. In contrast, he recovered the pathogen from 10% of the crowns placed on the soil surface for 103 weeks. Pathogen survival on seed has been studied in several locations. *D. bryoniae* was isolated from the seed coat, perisperm, and cotyledons of cucumber and pumpkin seed produced in several countries in Asia, Africa, and Europe (34). Also, it has been reported that gummy stem blight developed on cucurbit seedlings grown in the greenhouse, which suggests that the pathogen can be seedborne (8,30). Airborne ascospores are considered to be another source of inoculum (53,61).

Information on the effects of environmental conditions specifically on development of black rot on pumpkins is not available. However, several reports with valuable information on the epidemiology of gummy stem blight cucurbits and cucumber fruit rot caused by *D. bryoniae* are available (13,54,60,62,63). The optimum temperature for plant infection with *D. bryoniae* is 20 to 25°C in cucurbits. Moisture is more important for disease development than temperature. Peak ascospore dispersal occurs after rain and during dew periods at night. Free moisture on leaves for at least 1 h is necessary for infection, and further continuous leaf wetness is required for lesion expansion. Fruit is penetrated either through wounds or through flower scars at the time of pollination. Fruit rot begins to develop about 3 days after infection.

Black rot management should begin with effective control of gummy stem blight in the field (54,60,63). This will reduce the number of fruits lost during the season and provide better-quality fruit for transit and for storage. Seed should be treated with effective fungicides, and a 2-year crop rotation with noncucurbits is

essential for control of gummy stem blight and black rot. Control of gummy stem blight with protectant fungicides can be achieved. Several fungicides have been registered for control of gummy stem blight. Reducing moisture on the plant surface reduces the incidence of gummy stem blight and black rot infection. Care should be exercised to avoid rind injuries to fruit, as wounds provide entry for the black rot organism in storage. Under proper curing conditions, wounded areas heal themselves by producing corky tissue. Curing at 20 to 25°C or higher temperatures for 1 to 2 weeks hardens the rind.

Both pumpkins and winter squashes respond to this treatment. Storage of fruit should be at 11 to 16°C (optimum 13°C) with relative humidity of 55 to 75% (optimum 60%) (69).

Sclerotinia Rot

Sclerotinia rot of pumpkin and other cucurbits, caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, has been reported from the United States and many other countries throughout the world (5,9,32,46,68). *S. sclerotiorum* has a wide host range of horticultural crops. The disease commonly occurs on pumpkins later in the growing

Fig. 15. *Didymella bryoniae* (anamorph *Phoma cucurbitacearum*): A, pycnidia (left), pseudothecia (right); B, asci and ascospores; C, pycnidia; D, conidia.

Fig. 16. Pumpkin infection with *Sclerotinia sclerotiorum*: A, infected pumpkin (courtesy M. T. McGrath); B, sclerotia of the pathogen; C, colony and sclerotia of *S. sclerotiorum* on potato dextrose agar; D, apothecia (courtesy C. A. Bradley); E, asci and ascospores (courtesy H. F. Schwartz).

season. Very little research has been done on the etiology, epidemiology, and management of this disease on cucurbits.

The most obvious symptoms of Sclerotinia rot occur on the pumpkin fruit and fruit stem. The fungus may cause fruit rot of pumpkin in the field or postharvest decay. Fruit may be infected in areas contacting soil (Fig. 16A) or on the stem end, where water stands in the depressed area. The pathogen tends to infect dead tendrils and withered flowers still attached to developing fruit. White, cottony mycelium growth develops around the water-soaked infection site (Fig. 16A). Hard black sclerotia resembling small raisins are produced among the moldy growth (Fig. 16A and B).

S. sclerotiorum grows readily on culture media. Colony color on PDA is white (Fig. 16C) (40). Black sclerotia develop on the colony surface mainly near the edges of petri dishes after rapid hyphal extension has ceased (Fig. 16C). Sclerotia are round or elongated, up to 1 cm across, broadly reniform in vertical section, with a smooth or shallowly pitted surface (Fig. 16B). The fungus produces apothecia (Fig. 16D) that contain asci and ascospores (Fig. 16E).

The fungus can infect more than 300 different plant species. It survives in soil as sclerotia and as mycelium in plant debris. Sclerotia survive in soil for several years (9). A sclerotium either germinates to produce infective mycelium or produces apothecia and ascospores. Ascospores are released and dispersed by wind. Disease development is favored by cool, wet conditions. Once an outbreak occurs in a field, the potential for future outbreaks will remain high. Sclerotinia rot is a cool-season disease; hence it appears on pumpkins in wet years when summer temperatures are below normal (32).

No resistance to Sclerotinia rot is known in pumpkin. Crop rotation with nonhost crops (e.g., cereal grains) will limit the potential for damage to subsequent vegetable crops. Deep plowing immediately after harvest can help reduce disease incidence. Fields with good drainage should be considered for pumpkin production. Overhead irrigation should be used during the day when leaves will dry before dew forms. Fungicides may be effective if applied to young plants that could be threatened during cool, wet summers.

Interactions of the Fruit Rot Pathogens

Bacterial and Fusarium rots. Considerable interactions between *X. campestris* pv. *cucurbitae* and *Fusarium* spp. have been observed on infected pumpkins. Although fruit rots caused by either *X. campestris* pv. *cucurbitae* or *Fusarium* spp. can occur independently, widespread Fusarium rot following fruit infection with *X. campestris* pv. *cucurbitae* has been observed every year in Illinois (M. Babadoost, unpublished data).

Bacterial spots usually develop from midseason through harvest, while Fusarium rot commonly occurs at the end of the season. The lesions caused by the bacterium may provide an avenue for *Fusarium* spp. to rapidly colonize the fruit and cause its decay.

Phytophthora and Fusarium rots. Phytophthora fruit rot occurs any time from fruit set until harvest and in storage. Fruit infected with *P. capsici* is often colonized by *Fusarium* spp., such as *F. solani* and *F. oxysporum*, any time during the growing season. The Fusarium colony develops rapidly and masks Phytophthora growth. Hence, it is quite common to observe abundant Fusarium colonies and spores on fruit originally infected with *P. capsici*.

Phytophthora and bacterial rots. Stinking fruit rot is a common characteristic of fruit infected with *P. capsici*, but the odor is a result of fruit colonization by the soft-rot bacterium *Erwinia carotovora* subsp. *carotovora*. During warm, moist conditions, fruit infected with *P. capsici* is rapidly colonized by the bacterium. This secondary colonization and rapid breakdown of fruit tissues by *E. carotovora* subsp. *carotovora* results in the obvious odor. Infected fruit usually "melt down" within a few days, leaving only rind and seeds in the fruit growing site. Fruit infected with *P. capsici* and invaded by the soft-rot bacterium may also be colonized by *Fusarium* spp.

Fruit Rot Management

Management of pumpkin fruit rots is an overwhelming challenge for a number of reasons (20): cultivars resistant to fruit rot are not available (2,32); areas in contact with the soil are primary sites of infection and fungicide coverage of this site is not possible (2,20); because of the thick and dense canopy, management is usually difficult to achieve thorough chemical coverage of fruit (2); most of the fruit-rot-causing pathogens survive in soil for a long time (2,16,20); the pathogens causing fruit rots also cause foliar diseases (5); and the pathogens have wide host ranges (2,5,20). In general, no single method provides adequate control of all or any of the fruit rots of pumpkin (2). Effective management of fruit rots should include strategies with integration of all effective methods in the area. Practicing the following recommendations would reduce the incidence of fruit rots of pumpkin: introduction of the pathogen to the field should be avoided (exclusion); cropping rotations of 3 years or longer with nonhost crops are necessary; pathogen-free seed should be planted; to reduce fruit rot, control of foliage infection is necessary; and fungicide use for disease control should be considered as preventive rather than curative and fungicides with different modes of action should be alternated.

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