The word “pumpkin” is derived from the Latin word *pepo*, which is a botanical term for fruit with a hard rind and fleshy interior. The commonly used scientific name for pumpkins is *Cucurbita pepo*. But some other cucurbits, such as *Cucurbita moschata* (e.g., ‘Dickenson’ and ‘Libby Select’, known as processing pumpkin) (Figure 1, B) and *Cucurbita maxima*, which are winter squash, are also known as pumpkins. Most commonly, any Cucurbita fruit with an orange color is considered pumpkin (Figure 1, A-C). However, *Cucurbita* cultivars with white fruit, such as ‘Lumina’ and ‘Cotton Candy’ are also considered pumpkin (Figure 1, D). Here, term “pumpkin” will be used instead of pumpkin fruit.

Pumpkins can be infected by fungal, bacterial, and viral pathogens from the time of fruit set until harvest. Also, some diseases may develop in pumpkins during transit and in storage. Pumpkins can be infected by the following pathogens: (i) *Choanephora cucurbitarum* (Choanephora fruit rot, Figure 2, A); (ii) *Cladosporium cucumericum* (scab, Figure 2, B); (iii) *Colletotrichum orbiculare* (anthracnose, Figure 2, C); (iv) *Didymella bryoniae* (black rot, Figure 2, D); (v) *Fusarium* spp. (Fusarium rot, Figure 2, E); (vi) *Phytophthora capsici* (Phytophthora fruit rot, Figure 2, F); (vii) *Plectosporium tabacinum* (Plectosporium blight, Figure 2, G); (viii) *Pythium* spp. (Pythium fruit rot, Figure 2, H); (ix) *Sclerotium rolfsii* (southern blight, Figure 2, J); (x) *Sclerotinia sclerotiorum*.

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(Sclerotinia rot, Figure 3, A); (xi) *Septoria cucurbitacearum* (Septoria spot, Figure 3, B); (xii) *Erwinia tracheiphila* (bacterial wilt, Figure 3, C); (xiii) *Pseudomonas syringae* pv. *lachrymans* (angular leaf spot Figure 3, D); (xiv) *Xanthomonas cucurbitae* (bacterial spot, Figure 3, E); (xv) *Cucumber mosaic virus* (cucumber mosaic, Figure 3, F); (xvi) *Papaya ringspot virus* (papaya ringspot, Figure 3, G); (xvii) *Watermelon mosaic virus* (watermelon mosaic, Figure 3, H); and (xviii) *Zucchini yellow mosaic virus* (zucchini yellow mosaic, Figure 3, J).

**Major fruit rots of pumpkins are black rot, *Fusarium* rot, *Phytophthora* rot, *Sclerotinia* rot, and bacterial spot.**

**Black rot**

Black rot was first reported in Europe and US in 1891. The disease is caused by the fungus *Didymella bryoniae*. Black rot is an important pre- and post-harvest fruit rot of pumpkins and winter squashes. Symptoms of black rot can appear in the field before harvest and continue to develop during transit and in the storage. Small, water-soaked, dark-brown lesions develop and enlarge on fruit. As the lesions expand, they become blackish and sunken (Figure 4).

Fungal fruiting bodies (pycnidia), develop on infected areas. The disease gets its name from the rot, which includes blackened fungal tissue on the rind. Affected fruit may collapse during or after harvest. Large jack-o-lantern pumpkins are more susceptible to black rot than smaller pie types. When vegetative portions of pumpkins, and other cucurbit plants, are affected by *D. bryoniae*, the disease is known as “gummy stem blight. Black rot and gummy stem blight commonly occur in the United States (US). The disease, however, is not one of the most common diseases of pumpkin in Illinois.

*Didymella bryoniae* survives between the growing seasons on diseased vines, crop debris, and seed. In most of the studies, *D. bryoniae* survived for less than 9 months in infested cucurbit debris. Another source of inoculum of *D. bryoniae* is considered to be the air-borne sexual spores (ascospores) of the
pathogen. The optimum temperature for plant infection with *D. bryoniae* is 68-77°F (20-25°C) in cucurbits. Moisture is more important for disease development than temperature. Free moisture on leaves for at least 1 hour 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 control should begin with effective control of gummy stem blight in the field. Seed should be treated with effective fungicides, and a 2-year crop rotation with non-cucurbits is essential for control of gummy stem blight and black rot. Control of gummy stem blight by using protectant fungicides can be achieved. Several fungicides have been registered for control of gummy stem blight. Reducing moisture on plant surface reduces the incidence of gummy stem blight and black rot infection. Care should be exercised to avoid rind injuries to all 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 68-77°F (20-25°C) or higher temperatures for 1-2 weeks hardens the rind. Pumpkins and winter squashes respond to this treatment. Storage of fruit should be at 52-61°F (11-16°C) [the optimum is 55°F (13°C) with relative humidity of 55-75% (the optimum is 60%)].

### Fusarium rot

Fusarium crown and fruit rot of cucurbits was first described in detail in South Africa in 1932. Fusarium fruit rot is one of the most common pre- and post-harvest diseases of pumpkins, as well as other cucurbit crops. Several *Fusarium* species have been reported as causal agents of cucurbit fruit rot. But recently, *Fusarium solani* f.sp. *cucurbitae* was reported as the main causal of fruit rot of pumpkins in US.

Symptomatology of fruit rot of pumpkins can vary considerably, dependent on the stage of lesion development. Symptoms range from small, pitted, corky spots to large, sunken areas covered with a white or gray mold (Figure 5, A-B). Most infections occur on the sites that contact the soil. The pathogen also causes crown rot of pumpkin (Figure 5, C).

![Figure 5. Pumpkins infected with *Fusarium* spp. A and B, Fusarium fruit rot; C, *Fusarium* crown rot. (courtesy T.A. Zitter).](image)

![Figure 6. *Fusarium solani*: A, a colony on PDA; B, microconidia; C, macroconidia; D, chlamydospores.](image)
Fusarium solani. f.sp. cucurbitae survives in soil. It is more abundant in higher rainfall areas or in irrigated soils. Fusarium solani. f.sp. cucurbitae also is a seed-borne pathogen. Wounds facilitate fungal entry to fruit. Most of infections by F. solani f.sp. cucurbitae occur in the field (pre-harvest). Fusarium solani. f.sp. cucurbitae produces white to cream mycelium (Figure 6, A). The pathogen forms all three asexual spore types (microconidia, macroconidia, and chlamydospores) (Figure 6, B-D) and a sexual stage when proper mating types are present. The pathogen has two races. Race 1 causes root, stem, and fruit rot and occurs worldwide. Race 2 causes fruit rot in the US.

Management of fruit rot of pumpkin, as well as fruit rots of other cucurbits, caused by Fusarium spp., is difficult. Pre-harvest fungicide applications have been ineffective. Crop rotations of 3 year or longer with non-cucurbit crops will help to reduce the incidence of pumpkin rot. Growing pumpkin in the fields with cover crops (e.g., rye or crucifer crops) may help reduce incidence of pumpkin rot caused by F. solani f.sp. cucurbitae. Avoidance of wounding during harvest and packing, storage in dry conditions, and proper handling during transit and marketing reduce post-harvest decay.

**Phytophthora rot**

Phytophthora fruit rot, caused by the oomycete Phytophthora capsici, is one of the most serious threats to production of pumpkins and other cucurbits throughout the world. Phytophthora capsici was first described in New Mexico in 1922. The pathogen was subsequently described in more than 50 plant species, including all cucurbit crops from all over the world. Phytophthora infection on cucurbits is known as “Phytophthora blight.”

Phytophthora capsici can infect pumpkin fruit at any stage of development, during transit, and in storage. The infection usually appears first in low areas of the fields where the soil remains wet for longer periods of time. Fruit rot generally starts on the site of the fruit that is in contact with the soil (Figure 7). Fruit rot typically begins as a water-soaked lesion (Figure 7, C), expands, eventually covering the fruit with white mold (Figure 7, B and D). Fruit infection progresses rapidly, resulting in complete collapse of the fruit. The pathogen also infects seedlings, leaves, vines, and crown. Phytophthora foliar blight and fruit rot can result in total loss of the crop.

As an Oomycete species, the pathogen produces asexual sporangia and zoospores, and sexual oospores. Phytophthora capsici is a soil-borne pathogen and survives between crops as oospores in soil or mycelium in plant debris. 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 three years or even longer. Oospores germinate and produce sporangia and zoospores. Sporangia can either germinate and infect host tissues directly, or they can release zoospores, which can infect plant. Zoospores are released in water and dispersed by irrigation or surface water. Zoospores are able to
swim for several hours and infect plant tissues. If the environmental conditions are conducive, the disease develops rapidly. Soil moisture conditions are important for disease development. Sporangia form when the 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.

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. The most effective practice in controlling *P. capsici* is preventing the pathogen from being moved into a new field. The following practices can help to manage Phytophthora blight in pumpkin fields: (i) select fields with no history of Phytophthora blight; (ii) select fields that did not have cucurbit, eggplant, pepper, or tomato for at least 3 years; (iii) select fields that are well isolated from fields infested with *P. capsici*; (iv) select well-drained fields, or do not plant the crop in the areas of the field which do not drain well; (v) clean farm equipment of soil between fields; (vi) avoid excessive irrigation; (vii) do not irrigate from a pond that contains water drained from an infested field; (viii) do not work in wet fields; (ix) scout the field for the Phytophthora symptoms, especially after major rainfall, and particularly in low areas; (x) when symptoms are localized in a small area of the field, disk the area; (xi) discard infected fruit, but not in the field; (xii) do not save seed from a field where Phytophthora infection occurred; (xiii) remove healthy fruit from the infested area as soon as possible and check them routinely; (xiv) do not display fruit for sale in an area that is infested with *P. capsici*; and (xv) apply effective fungicides, when recommended. Seed treatment with either mefenoxam (0.42 ml Apron XL LS/kg seed) or metalaxyl (0.98 ml Allegiance FL/kg seed) can protect pumpkin seedlings until 5 weeks after sowing seed. Several fungicides have been labeled for control/suppression of *P. capsici* in pumpkin fields. For the update chemical control of Phytophthora blight of cucurbit, refer to the Midwest Vegetable Production Guide for Commercial Growers, publication C1373 (http://www.btny.purdue.edu/pubs/id/id-56/).

**Bacterial spot**

Bacterial spot (bacterial leaf spot), caused by *Xanthomonas cucurbitae*, has become a serious disease of pumpkin. The pathogen also infects summer and winter squashes, cucumbers, and gourds. The disease was first described on Hubbard squash in New York in 1926. Subsequently, the disease was reported from other cucurbit growing areas. Fruit rot due to infection with this pathogen exceeds 50% in some commercial fields in Illinois.

*Xanthomonas cucurbitae* infects leaves and fruit of pumpkin any time during the growing season (Figure 8). Lesion on leaves are small (2-4 mm), angular, yellow to beige spots (Figure 8, C). The appearance and size of fruit lesions can vary, depending on rind maturity and the presence of moisture. Initial lesions are small, slightly sunken, circular spots (1-3 mm in diameter), with a beige center and
dark brown halo (Figure 8, B). Later the cuticle and epidermis crack, and the lesions enlarge, reaching diameter of up to 10-15 mm and become sunken. Penetration of bacteria into the flesh can lead to significant fruit rot in the field or later in storage. The bacterium is known as a seed-borne pathogen. It also survives in plant debris. 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).

Bacterial leaf spot and fruit rot, caused by *X. cucurbitae*, can be managed by the use of pathogen-free seed. 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. 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 (e.g., in Illinois climate) when the bacterium is endemic, repeated application of copper sprays as foliar protectant can be helpful. Weekly copper application beginning fruit set until harvest may be necessary to reduce disease incidence. Spray should be forced into canopy to cover the foliage and fruit thoroughly.

**Sclerotinia rot**

Sclerotinia rot of pumpkin and other cucurbits, caused by *Sclerotinia sclerotiorum*, has been reported from the US and many other countries throughout the world. In the recent year, Sclerotinia fruit rot has been commonly observed in commercial pumpkin and winter squash field in Illinois. *Sclerotinia sclerotiorum* has a wide host range of horticultural crops. The disease commonly occurs on pumpkins later in the growing 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 occurs on pumpkin fruit and fruit stem. The fungus may cause fruit rot of pumpkin in the field or postharvest decay. Fruit may be infected on the site that contacts the soil (Figure 8, B) or stem end (Figure 8, A), where water stands in the depressed area. White, cottony fungal growth (mycelium) develops around water-soaked infection site (Figure 8, B). Hard black sclerotia resembling small raisins are produced among the moldy growth (Figure 9, A-D).

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. Disease development is favored by cool and wet conditions. Once an outbreak occurs in a field, the potential for future outbreaks will remain high. Sclerotinia rot is a cool season disease.
No resistant pumpkin cultivar to Sclerotinia rot is known. Crop rotation with non-host 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. Fungicide may be used to control Sclerotinia rot.

**Fruit rot management**

The most destructive fruit rot of pumpkin is Phytophthora rot. Importance of other fruit rots differs from one pumpkin growing area to another. For example, black rot commonly occurs in the Southeastern and the Northeast of US, while it is not a significant disease in the Midwest. In contrast, bacterial spot occurs widely in the North Central and Northeast regions, particularly in Illinois. Fusarium pumpkin rot has been reported from all pumpkin growing areas. Sclerotinia pumpkin rot is more common in northern regions of the US. Management of pumpkin fruit rots is an overwhelming challenge because of the following reasons: (i) resistant cultivars against the fruit rots are not available; (ii) site of the fruit contacting soil is primary site of infection and fungicide coverage of this site is not possible; (iii) because of thick and dense canopy, it is usually difficult to achieve through chemical coverage of fruit; (iv) most of the fruit-rot causing pathogens survive in soil for long time; (v) the pathogens causing fruit rot also cause foliar infection; and (vi) the pathogens have wide host-ranges. In general, no single method provides adequate control of all or any of the fruit rots of pumpkin. Effective management of the 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: (i) introduction of the pathogen to the field should be avoided; (ii) crop rotations of 3 years or longer with non-host crops are necessary; (iii) pathogen-free seed should be planted; (iv) to reduce fruit rot, control of foliage infection is necessary; and (v) fungicide use for disease control should be considered as preventive rather than curative and fungicides with different modes of action should be alternated.