Alternaria Diseases of Carrot

Carrot (*Daucus carota* L. subsp. *sativus* (Hoffm.) Arcang.) is one of the most popular and commonly consumed vegetables (52). The carotene in one medium-sized carrot supplies our daily requirement of Vitamin A (53). Commercial carrot production is an economically important industry worldwide. China, the United States, and Russia are the major producers of carrots (53). In the United States, nearly two million English tons of carrots are produced annually on over 40,000 ha at a value of \$500 million (53).

Wild carrot (*Daucus carota* L.), also known as Queen Anne's Lace, is probably the progenitor of the present-day carrot, and the two readily cross. The center of origin of the carrot is thought to be in Middle Asia in the region of Afghanistan and Turkistan (52,53). The first cultivated carrots were white, purple, red, yellow, green, and black, but never orange. The roots were thin and turnip-shaped, and early usage of the carrot focused on its medicinal properties (53).

Several types of carrot cultivars are grown today, and different cultures have particular preferences for the shapes and root colors of carrots (52). In Asia, reddish-orange, long, thick, conical-shaped roots are preferred. European consumers prefer yellowish-orange, relatively short, slender, cylindrical-shaped roots such as Nantes and Nantes-like cultivars; whereas preferences in North America are for deep orange, long, slightly tapered roots such as Imperator and similar cultivars (52,53).

The carrot plant is biennial and produces a rosette of leaves on a compressed stem and a fleshy edible storage root in the first year of growth. For commercial production, carrots are direct seeded, generally not thinned, and grown as an annual crop. Seeds are small (500 to 1,000 seeds per gram) in comparison to other vegetable seeds, and emergence is relatively slow. At optimum temperatures of 15 to 20°C, ger-

mination usually is completed in 7 to 12 days. Seed is sown at depths of 3 to 10 mm at rates of 500,000 to 3,000,000 seeds per hectare. Roots usually achieve marketable size within 70 to 120 days, but during the cool season some crops may require more than 150 days of growth to achieve marketable size. The taproot may achieve maximum length in as little as 25 to 30 days after emergence and ultimately reach a depth of 1 m. Although carrots are usually grown on sandy soils, they tolerate a wide range of soil types as long as the upper meter of soil is uniform and free of barriers to root growth (53).

Modern carrot production utilizes self-propelled multirow mechanical harvesters, which undercut and lift carrots from the ground by their tops using a system of gripper belts (Fig. 1). This is followed by removal of the foliage, which is discarded, while the roots are conveyed and collected in large containers or wagons. Obviously, strong and healthy foliage is an important feature for this type of mechanical harvesting. Other types of harvest machines first flail or cut off the foliage. The roots are then dug and lifted in a manner similar to the harvesting and handling of potatoes. For bunch carrots with attached leaves, a subsurface blade is drawn horizontally to undercut the roots for easy hand-lifting from the soil. A specific number or weight of uniform carrots with intact foliage is then tied together as a bunch (52,53).

Alternaria Leaf Blight

Alternaria leaf blight of carrot was first described in Germany in 1855, when it was reported causing sporadic disease losses in several Northern European countries (26). At the time, the causal agent was identified as *Sporidesmium exitiosum* Kühn v. *dauci* Kühn. Over the next 90 years, the disease apparently did not play a prominent role in carrot production in Europe (37). In the United States, *Alternaria* leaf blight was first reported in Louisiana in 1890, and the causal agent was identified as *Macrosporium carotae* Ellis & Langlois (15). As commercial carrot production expanded along the East Coast, leaf blight was reported from Massachusetts to Florida, and damage to carrot crops was significant.

Following these first reports of *Alternaria* leaf blight, the disease spread to all carrot production areas in the world and has become one of the most destructive diseases of carrots. The disease commonly occurs when carrots are cultivated during moderate temperatures and the leaves are exposed to prolonged periods of wetness due to rainfall, dew, or sprinkler irrigation. Statistics for annual losses due to *Alternaria* leaf blight are not available, but in Israel severe epidemics have been reported to reduce yields by 40 to 60% (6).

Causal agent. *Alternaria* leaf blight is caused by the fungus *Alternaria dauci* (Kühn) Groves & Skolko 1944 (syn. *Sporidesmium exitiosum* Kühn var. *dauci* Kühn 1955, *Macrosporium dauci* (Kühn) Rostrup 1888, *Macrosporium carotae* Ellis & Langlois 1890, *Alternaria brassicae* (Berk.) Sacc. var. *dauci* (Kühn) Bolle 1924, *Alternaria porri* (Ellis) Ciferri f. sp. *dauci* (Kühn) Neergaard 1945) (51,57). Reproduction occurs by means of asexual conidia produced in abundance on erect conidiophores. In culture, hyphae are subhyaline to olive-brown and septate. Conidiophores are olive-brown and straight to flexuous with a single terminal or one to two geniculate conidiogenous sites. Conidia are typically borne singly, but occasionally a sturdy terminal secondary conidiophore bearing a secondary spore is produced. Conidia are medium to dark olive-brown, long ellipsoid to obclavate, 60-100 × 15-25 µm (spore body), with 7 to 11 transepta and 1 to 3 longisepta in fewer than half of the transverse segments (Fig. 2A). Mature conidia are rostrate with a terminal filamentous beak 80-250 × 5 µm tapering distally to ca. 2 µm, occasionally with a single lateral branch up to 100 µm in length (12,20,37,57). Sexual reproduction of *A. dauci* is not known to occur.

A. dauci is a member of the *porri* species-group of *Alternaria* fungi, which are characterized by a large conidium body and the presence of a long, narrowly tapered to filiform beak (46,57). Included in this group of *Alternaria* species are a large number of other pathogens prominent on particular hosts, such as *A. carthami* on safflower, *A. cucumerina* on cucurbits, *A. porri* on onion, *A. sesami* on *Sesamum* spp., *A. solani* on tomatoes and potatoes,

Fig. 1. Example of a mechanical carrot harvester that: A, undercuts the roots, and B, lifts carrots by gripping the tops.

and *A. zinniae* on *Zinnia* spp. (16). Since the morphologies of many members of the porri species-group are very similar (robust conidium with a long filiform beak) and the host range of each species is very restricted, several species have been considered as formae speciales of a single species, most notably *A. porri* f. sp. *dauci* as a synonym of *A. dauci* and *A. porri* f. sp. *solani* as a synonym of *A. solani* (38).

Host range. The established host range of *A. dauci* is cultivated and wild carrot (12,57). However, *A. dauci* has also been reported to cause disease on wild parsnip and *Fumaria muralis* (61), as well as on celery and parsley (38). The report of *A. dauci* infections on noncarrot hosts is not without controversy, as the application of Koch's postulates on recovered, putative isolates of *A. dauci* has rarely been reported. Moreover, the occurrence of infection on noncarrot hosts also is rarely reported, suggesting conditions for putative infection are quite restrictive. For example, in Israel, field crops of parsley, celeriac, and fennel planted next to carrot with active *Alternaria* leaf blight outbreaks were regularly inspected for *A. dauci* infections, but none were observed (39). Interestingly, in the same study, three wild Umbelliferous hosts, *Daucus maximus*, *Ridolfia segetum*, and *Caucalus tenella*, were observed to have typical *Alternaria* leaf blight infections. Putative isolates of *A. dauci* were obtained from symptomatic tissue and when reinoculated onto carrot leaflets produced typical *Alternaria* leaf blight symptoms (39).

Disease symptoms. Foliar symptoms of *Alternaria* leaf blight appear 8 to 10 days after infection as small, greenish-brown, water-soaked lesions. The lesions enlarge and the infected tissue becomes dark brown to black and may be surrounded by a chlorotic halo. Under favorable conditions, lesions coalesce and may cause the entire leaf to collapse. Older leaves are infected first. Lesions are more common on the margins of the leaflets, but may

Fig. 2. Morphology of conidia of *Alternaria* spp. pathogenic on carrot. A, *A. dauci*, B, *A. radicina*, C, *A. carotiincultae*, and D, *A. petroselini*.

occur on any part of the leaf blade or petiole (Fig. 3A). Lesions on the petioles are similar to lesions on the leaf blades, except they often become elongated. Numerous or large petiole lesions can kill the entire leaf, and heavy infestations may cause complete defoliation (Fig. 3B) (41).

Although foliar symptoms are the most common, *A. dauci* can also infect the inflorescences, seeds, and developing seedlings (41,60). Infection of the buds and flower parts can lead to infection of the developing seeds (60). Flowers infected early in development produce nonviable seed, but those infected later may produce viable seed. The fungus remains in the pericarp and does not grow into the embryo or endosperm (63). Infected seed can transmit *A. dauci* into new carrot production areas or may spread new strains of the fungus into existing production areas (55,63,66). Carrot seedlings may be infected by mycelium growing out of the pericarp or from

soilborne inoculum. Seedling infection typically occurs near the hypocotyl-root junction, at or just below the soil line. The infected region becomes constricted, then shrivels and turns gray to black (41). Under favorable conditions, infected seedlings may produce abundant conidia, which serve as secondary inoculum.

Disease cycle. The disease cycle of *Alternaria* leaf blight is similar to that of other foliar *Alternaria* diseases. Sources of inoculum include mycelium in overwintering carrot debris, diseased volunteer or wild carrots, and infected seed. In warm temperate areas, overlapping production seasons allow conidia produced in more mature fields to serve as inoculum for nearby more recently planted fields. Mycelium overwintering in carrot debris is apparently the most common source of inoculum. The fungus survived in infected carrot debris in the field for up to 30 weeks in Florida and 52 weeks in California (47).

In a greenhouse experiment, *A. dauci* was recovered from infected carrot debris for 30 weeks in soil that was regularly watered and for 52 weeks in dry soil (47). Conidia are produced on the surface of infected carrot debris, volunteer or wild carrots, or necrotic seedlings originating from infected seed, and are aerially dispersed. Conidiophores and conidia were produced at temperatures from 8 to 28°C and in the presence of 96 to 100% relative humidity or free water on the plant surface (62). Conidia are released and become airborne as the relative humidity drops soon after daylight and winds are above 2 to 3 m/s (18,62). In a study of spore dispersal patterns in Ontario, the number of airborne spores detected displayed a circadian periodicity with peak dispersal from 1100 to 1400 h (27). Spore dispersal increased with decreasing relative humidity, transition from wet to dry leaves, and increases in temperature and wind speed. Spore dispersal patterns can be interrupted by prolonged high winds, long periods of leaf wetness, and cool temperatures (27).

Conidia deposited on leaf surfaces will germinate and infect under appropriate temperature and leaf wetness conditions. At 24°C, damage generally increases with increasing hours of leaf wetness from 8 to 56 h (65). In addition, infection rate was markedly reduced at night temperatures below 12°C (65). Multiple germ tubes are produced from each conidium. Germ tubes penetrate the host cell wall directly to establish an infection site (13). *A. dauci* produces the phytotoxin zinniol, which degrades cell membrane structure and chloroplasts (5). Zinniol is produced in germ tubes and young hyphae and apparently is part of the pathogenesis mechanisms of the fungus (5). Application of purified zinniol to carrot leaves results in

symptoms similar to those produced by *A. dauci* infection (5). During colonization, carrot leaf cells adjacent to mycelium lose chlorophyll and become necrotic. Under favorable conditions of moderate temperatures and high relative humidity, a new crop of conidia can be produced 8 to 10 days after infection (65). Prolonged environmental conditions conducive to disease development can result in rapid development of Alternaria leaf blight epidemics and may result in severe yield loss.

Disease management. Control of Alternaria leaf blight is necessary to prevent destruction of the photosynthetic surface area of the plants and to allow for efficient mechanical harvest. Healthy leaves are necessary for harvest since leaves weakened by blight will break, leaving the root in the soil. Alternaria leaf blight is controlled through integrated use of clean seed, sanitation, crop rotation, cultivar selection, and fungicide applications. Under high disease pressure, no single control measure is sufficient to manage the disease adequately.

Planting seed with little or no *A. dauci* contamination is an important first step in disease management. Fungicide applications to the seed crop can minimize seed-borne infections, resulting in fewer early-season infections in production crops. Infected seed can be treated with hot water or fungicides to reduce, but not eliminate, contamination (31,64,69). Heat treatments are not widely used since the temperature and duration of treatment necessary to eliminate *A. dauci* may reduce the percent germination of seed (69). Dusting seed with thiram or iprodione can significantly reduce *A. dauci* contamination (31,64). However, in one study, 0.01 and 0.43% of seed treated with iprodione and thiram, respectively, were still contaminated after

treatment (64). At seeding rates of 500,000 to 3,000,000 per ha, 0.01% contaminated seed translates into 50 to 300 potential inoculum sources per hectare.

Crop rotation, plowing, and sanitation reduce the amount of inoculum that persists into the next carrot cropping cycle. Crop rotations must be of sufficient duration to allow carrot debris to decompose, thereby eliminating the fungus. Fungal sporulation was reduced more rapidly on buried carrot leaf tissue than on leaf tissue left on the soil surface (47). This suggests that incorporating debris into the soil will promote the reduction of inoculum through more rapid decomposition of buried debris. Infected carrot debris may also be carried from recently harvested fields to new fields on irrigation pipe and equipment. In California, growers pressure wash solid-set sprinkler pipes between crops to remove necrotic leaf debris as a way to reduce early-season inoculum (J. J. Nunez, *personal communication*).

Carrot inbreds and cultivars differ in their resistance to infection by *A. dauci* (3,8,49,59). Heritability of resistance to Alternaria leaf blight was estimated at 0.40, based on half-sib progeny tests indicating a medium to low heritability of resistance (8). Analysis of diallelic crosses revealed that resistance to Alternaria leaf blight is predominately additive (horizontal) but with some dominant gene action (59). Some reciprocal crosses of inbreds resulted in different resistance levels in the progeny, and the performance of the inbreds was only partially predictive of the resistance of the hybrids (59). In addition, carrot somaclones regenerated from callus tissue culture had differing levels of chlorophyll loss when exposed to *A. dauci* (13,14). In these studies, chlorophyll loss was used as an indicator of susceptibility

Fig. 3. Alternaria leaf blight symptoms: A, on an individual leaf, and B, extensive defoliation in the field (41).

to *Alternaria* leaf blight. Selections from somaclone regeneration could be used to provide enhanced disease resistance in carrot breeding.

Host resistance also is influenced by cultural practices, such as fertilization rates. Fertilization at one-half the recommended rate resulted in 23 to 30% more foliar blight than fertilization at the recommended rate (73). In Georgia, increasing nitrogen fertilization by 29% had no effect on disease (28), but in Israel doubling fertilization rates reduced disease by 10 to 15% (73).

In most carrot production areas, routine applications of fungicides are necessary to adequately control *Alternaria* leaf blight. Decisions regarding selection of fungicide chemistry, the disease threshold necessary to initiate applications, and whether to time applications based on disease forecasting or calendar schedules are integral to the efficacy of control. Iprodione and chlorothalonil have been the fungicides of choice in the United States in recent years. In the past few years, azoxystrobin and pyraclostrobin have shown excellent control in field evaluations and have been labeled for control of *Alternaria* leaf blight of carrot in the United States (1,21,28,33,48). Azoxystrobin and pyraclostrobin need to be alternated or tank mixed with fungicides with different modes of action in order to minimize the possibility of selecting for fungicide resistance in populations of *A. dauci*.

Threshold levels of *Alternaria* leaf blight that are used by growers to initiate fungicide applications differ among carrot production areas. In many areas, growers initiate fungicide applications after the disease is first detected in the field. Recent studies in Michigan confirmed that initiating sprays at first detection of the disease gave comparable control to initiating sprays 30 days after planting (9). Under the higher disease pressure found in Israel, fungicide applications are initiated 30 days after planting for the autumn crop and 70 days after planting for the spring crop (6). Delaying the time of first fungicide application compromised the level of control (6). In California, attempts to forecast disease outbreaks using the TOM-CAST model failed due to a lack of accumulated disease severity values (J. J. Nunez, *personal communication*). In contrast, use of the TOM-CAST model in Michigan effectively scheduled fungicide applications at 15 disease severity value intervals, resulting in disease control comparable to a 7-day calendar-based schedule and three to four fewer sprays per season (9).

In recent years, there has been interest in nonfungicidal treatments for control of *Alternaria* leaf blight. Although field trials have demonstrated a lack of efficacy for a *Bacillus subtilis*-based biological control product (Serenade), a harpin elicitor protein (Messenger), and a systemic acquired

resistance activator (Actigard), the plant hormone gibberellin was demonstrated to reduce disease severity (21,48,54). Under relatively low disease pressure in California, 20 to 40 mg of gibberellic acid per liter applied in 280 liters of water per hectare 4 to 8 weeks after planting gave comparable control to iprodione (54). In production fields, gibberellic acid application rates must be calculated carefully, as high rates can result in increased foliar growth and decreased root growth.

Alternaria Black Rot

Black rot of carrot was first described in 1888 as a common disease of carrot in Denmark and other northern European countries (50). At that time, the disease was mistakenly attributed to the fungus *Sporidesmium exitiosum* (Kühn) v. *dauci* (Kühn) 1855 (syn. *Alternaria dauci*), causal agent of *Alternaria* leaf blight. In the United States, the disease was first reported in 1918 on Long Island, NY, and in Washington, DC, where it caused significant losses in carrots held in cold storage and in transit (29,34). In subsequent studies, the causal agent was correctly determined to be a new species of *Alternaria* (51,57).

Since these initial observations, black rot has been reported from all major carrot-growing regions of the world (57). In the United States, the disease has been reported in most of the primary carrot-producing states (16). Although statistics for annual losses to black rot are not available, field-based surveys provide evidence of considerable damage. Coles and Wicks (11) reported up to 88% disease incidence on mature plants in select fields in South Australia. Pryor et al. (43) reported disease incidence of 63 to 99% on mature plants in a survey of eight fields in the Cuyama Valley of California. In some of the California cases, entire fields were abandoned before harvest due to excessive petiole decay. Estimates of disease incidence from storage houses and markets also reveal the prevalence of black rot. For example, in studies conducted between 1918 and 1926, carrot shipments from New York farms revealed infection rates as high as 62% (34). Surveys conducted in Europe and Asia revealed that the average incidence of black rot on stored carrots ranged from 1 to 50% (17,74).

Causal agent. Black rot of carrot is caused by the fungus *Alternaria radicina* Meier, Drechsler, & Eddy 1922 (syn. *Stemphylium radicinum* (Meier et al.) Neergaard 1939, *Pseudostemphylium radicinum* (Meier et al.) Subramanian 1961) (15). Reproduction occurs by means of asexual conidia produced in abundance on erect conidiophores, each with one to three geniculate conidiogenous sites. Conidia are borne singly, or occasionally in chains of two, and are typically dark olive-brown to natal brown, broadly ellipsoid to ovoid, 20-

50 × 10-25 µm, with two to five transepta and one to three longisepta in any or all segments, except basal and apical segments, which usually are free of septa (Fig. 2B). Less frequently, conidia mature into a long, narrow form, broadly ellipsoid to obclavate, 50-65 × 15-20 µm, with seven to eight transepta and one to two longisepta in most of the segments (20,37,51). Sexual reproduction by *A. radicina* is not known to occur.

A. radicina is the representative species for the *radicina* species-group of *Alternaria* fungi (46,57). Other plant pathogens are included in this group, such as *A. carotiincultae* (Fig. 2C), also pathogenic on carrot, *A. petroselini* (Fig. 2D), *A. smyrnii*, and *A. selini*, all pathogenic on parsley, celery, and/or other Umbelliferous plants (38,57). Many morphological characteristics of these fungi are very similar, making identification to species difficult if one relies solely on morphology for diagnostic criteria. Differentiating *A. radicina* and *A. carotiincultae* is particularly difficult considering they are both carrot pathogens. *A. carotiincultae*, described only recently from wild carrot in Ohio (57), differs slightly from *A. radicina* in having: (i) a greater average conidium length; (ii) a lower percentage of obovoid and subspherical conidia; and (iii) a greater number of conidia formed in chains of two, and sometimes three (57). Later studies revealed that *A. carotiincultae* can also infect carrot roots, causing black rot, and is in fact a more aggressive pathogen on carrot than is *A. radicina* (46). Despite its noted pathogenicity to carrot, *A. carotiincultae* is not common, and the vast majority of *Alternaria* isolates obtained from black rot tissue are *A. radicina* (46).

Host range. *A. radicina* is known primarily as a pathogen of carrot, responsible for root and crown disease but also causing foliar blight under certain conditions. However, the fungus also has been reported to cause foliar blight of parsley, stalk/root rot of celery, and is pathogenic on caraway, dill, fennel, and parsnip (15,71,72,77). In at least one study, the causal organism originally reported was later determined to be misidentified. Wearing (77) identified a root and crown disease of celery in Australia caused by *A. radicina*. Re-examination of the type material revealed morphological characteristics and pathogenicity consistent with that of Neergaard's *A. radicina* type c (46). However, sequence analysis of the nuclear internal transcribed spacer rDNA and mitochondrial small subunit rDNA clearly revealed that the fungus was, in fact, a variety of *A. petroselini*, a notable pathogen of both parsley and celery (46). Further support of possible misidentifications of parsley and celery pathogens as *A. radicina* comes from recent host range studies comparing foliar symptoms produced by *A. radicina*, *A. carotiincultae*,

and *A. petroselini* (46). *A. radicina* and *A. carotiincultae* were strongly pathogenic on carrot and weakly pathogenic or nonpathogenic on parsley, celery, cilantro, and fennel. In contrast, *A. petroselini* was weakly pathogenic or nonpathogenic on carrot and moderately to strongly pathogenic on parsley, celery, cilantro, and fennel. This study highlights the potential for misidentification of *A. radicina* and related *Alternaria* species.

Symptoms and disease cycle. Black rot of carrot is characterized by dry, black, sunken lesions on carrot roots. The disease is most damaging on carrots kept in cold-storage, where lesions can quickly expand, decay the entire root, and spread to adjacent carrots (30,32,50). However, *A. radicina* can infect most carrot tissues. Since the fungus is seed-borne, the disease cycle often begins with the planting of *A. radicina*-infested carrot seed (36). As seed germinates, *A. radicina* infects the hypocotyl, resulting in a black necrosis of the developing seedling and preemergence damping-off (35,36). Post-emergence damping-off occurs as emerged seedlings are infected at or near the soil line. The fungus then sporulates, and spores are spread by wind, irrigation water, or rain to other seedlings, resulting in reduced stands and yields (38,51).

Under favorable conditions, *A. radicina* can infect carrot plants at any stage of growth. Once past the seedling stage, vigorously growing carrot plants tend to be more resistant to infection (19,59). As plants reach marketable size, they again become susceptible to infection by *A. radicina*. Older, senescing petioles are particularly susceptible to infection and colonization by *A. radicina* and provide an avenue for infection of the carrot crown (19). This results in a black ring of decay at the points of petiole attachment (Fig. 4),

a characteristic symptom that has led to the term “black crown” of carrot. In the field, *A. radicina* infections often remain restricted to the carrot crown and petioles. Although these infections do not necessarily result in a reduction of the quality of the carrot root, decay of the crown and petioles can interfere with mechanical harvesting. Petiole infections may become so severe that entire fields are abandoned prior to harvest.

Under optimal disease conditions, *A. radicina* spreads from the crown to the canopy and may cause a foliar blight (19). Symptoms begin as small necrotic spots that may be surrounded by a chlorotic margin. As lesions expand and coalesce, entire leaflets or leaves may develop a black necrosis. Such blighted carrots have reduced photosynthetic capacity, which results in a reduction in root size and subsequent yield (19,72).

Crown infection may also lead to infection of the carrot root. In these cases, the upper portion of the root becomes necrotic, and necrosis can progress down into the lower portions of the root. Carrot root infections can also be initiated belowground, but storage roots appear to be fairly resistant to direct penetration by *A. radicina* (19,30). Belowground infections are facilitated by injuries, such as mechanical or insect damage (19). If root infections occur early in the growing season, infected carrots will be culled during sorting due to the obvious presence of large black lesions. Carrots infected later in the season may not have developed visible lesions by harvest, resulting in potential inoculum sources during storage.

The primary economic damage caused by *A. radicina* occurs during storage of carrots (30,32,72). In cooler climates, carrots are grown during the warmer months and then stored for various periods ranging

from 50 to 200 days. *A. radicina* can infect carrot tissue at temperatures as low as -0.6°C , provided humidity remains high (30). Thus, the cold, moist conditions associated with long-term storage of carrots provide a suitable environment for growth of *A. radicina* and the development of black rot. In severe cases, all of the carrots within a storage bin may become infected.

A. radicina can survive between carrot crops in association with crop debris (32,51,67). Under favorable conditions, the fungus sporulates on infested debris and spores initiate infections in new carrot crops. *A. radicina* can also persist in soil as conidia long after the infested plant debris has decayed (43). For example, carrots developed black rot when grown in *A. radicina*-infested soils that had not been planted to carrots for as long as 8 years (31,32).

Infection of carrot umbels and seed by *A. radicina* completes the disease cycle. As carrot is a biennial plant, seed is produced in the second year of growth. In many, but not all, seed production areas, carrot plants are harvested after the first season and stored until the following spring when they are replanted, often in different areas. After a short period of vegetative growth, these transplanted carrots (stecklings) bolt and produce seed. If stecklings are infected with *A. radicina*, the resulting seed crop probably will be infected (32,72). Stecklings are also more susceptible to infection from soilborne inoculum than are first-year carrots due to wounding associated with harvest, storage, and replanting (32). Thus, if healthy stecklings are planted in *A. radicina*-infested soil, the resulting seed crop will likely become infected.

A. radicina is a serious pathogen of carrot seed crops because infections may lead to significant seed losses (7,38,72). Symptoms of umbel blight caused by *A. radicina* are similar to foliar blight caused by this fungus. If infection is severe, the entire umbel can become necrotic, resulting in no seed production. If seed is produced, *A. radicina* contamination usually occurs as conidia on the seed surface, although histological studies have demonstrated the presence of mycelia in the inner layers of the pericarp or, occasionally, in the testa (30,60). The fungus has not been detected in the endosperm or in the embryo. The level of seed contamination can be very high. Grogan and Snyder (19) reported that all of 38 commercial seed lots assayed in 1950 were infested with *A. radicina* at levels ranging from 1 to 20%. Maude (30) found that 13 of 19 commercial seed lots tested were contaminated with *A. radicina* at levels ranging from 0.25 to 30%. Of 313 commercial seed samples assayed for *A. radicina* in Poland from 1984 to 1988, 99% were contaminated, with infestation levels ranging from 0.5 to 82.5% (72). In a 1999 survey of 19 commercial seed lots in Australia, 18 were infected with *A.*

Fig. 4. Black rot symptoms on the petiole bases and crown of carrot (40).

radicina, with the highest infection rate being 35% (11). *A. radicina* was also recovered from 11 of 16 samples of seed treated with thiram and/or iprodione, at levels of 0.2 to 14% (11). These reports highlight that contamination of carrot seed by *A. radicina* is a continuing problem.

A. radicina infection during seed formation may also affect seed vigor, and in severe cases, infected seed may not germinate (19,37,71). Seeds that germinate slowly are more susceptible to damping-off pathogens and less able to compete with weeds, resulting in poor stand and reduced carrot yields (37,38,71).

The long-term consequence of planting *A. radicina*-infested seed is the introduction of the fungus into new fields and/or production areas (38,55). In soil surveys conducted in the Cuyama Valley of California between 1993 and 1995, *A. radicina* was recovered from 83% of commercial carrot fields tested (43). In the Cuyama Valley, black rot is common and can be found in almost every carrot field but has not been found in any native, nonagricultural soils tested (43).

Disease management. Control of black rot of carrot is difficult for several reasons. The fungus is commonly seedborne, persistently soilborne, and can spread via windblown dust and crop residue. In the field, black rot symptoms on the carrot crown often become apparent only after the canopy closes, making detection difficult and chemical applications ineffective. However, black rot can be effectively managed using an integrated approach.

Fungicides applied for the control of other *Alternaria* species are usually effective against *A. radicina* as well. These include the fungicides chlorothalonil, iprodione, and azoxystrobin. However, targeting the fungicide applications toward the carrot crown is difficult and generally ineffective. As such, the use of fungicides to control black rot generally occurs before planting or after harvest. Fungicide seed treatments are commonly used to control seedborne *A. radicina* as well as *A. dauci* (see above). Control of black rot on harvested carrots can be achieved by application of fungicides during sorting and prior to storage (17,78). However, the expense associated with these treatments usually precludes their use. Simple hot water treatment (steam) prior to storage has been shown to significantly reduce the incidence of storage rots of carrots, including black rot, and may be an important method of disease management for organic carrot production (2). However, if bacteria, such as *Erwinia carotovora* subsp. *carotovora*, are present in sufficient numbers on harvested carrots, then steam treatment of the carrots prior to storage may actually increase the incidence of storage rots (2).

The ability of *A. radicina* to survive in soil for long periods of time (years) is an important component of black rot epidemi-

ology. Prior to a comprehensive survey in six carrot production areas in California from 1993 to 1995 (43), little data existed on soil population densities of *A. radicina*. In the survey, soil population densities, determined using soil dilution plating and an *A. radicina*-selective medium (ARSA), ranged from 0 to 316 CFU/g soil (43). More importantly, the incidence of black rot at harvest was positively correlated with soil population densities at planting (43). This suggested that determining *A. radicina* population densities in soil prior to planting could be used to predict the potential for black rot development in fields.

Crop rotation may play an important role in the management of soilborne *A. radicina*. In the Cuyama Valley of California, restricted water availability limits crop rotation options in many fields (40). Therefore, fallow rotation is commonly used between carrot crops, with the fallow fields remaining dry for much of the year. This practice may favor survival of *A. radicina*, a hypothesis supported by the fact that the highest population densities of *A. radicina* in the Cuyama Valley (>100 CFU/g soil) were recovered in fields with fallow rotations between carrot crops, whereas much lower population densities were detected in fields in which crops such as beans, alfalfa, and wheat were grown in rotation with carrot (43). However, *A. radicina* can survive for long periods in the absence of a host, and although short-term rotation (2 to 4 years) to nonhost crops may lower population densities, extended rotations (>8 years) may be necessary to manage soilborne inoculum in heavily infested fields.

Deep-tillage has been used as a method to control soilborne disease inoculum in several cropping systems (70). In California, deep-tillage was evaluated as a means to control *A. radicina* in carrot fields (40). Within the vertical soil profile in a heavily infested field, approximately 50% of the total soil inoculum occurred in the upper 10 cm of soil. Approximately 30 and 20% lay at depths of 10 to 20 cm and 20 to 30 cm, respectively. Following moldboard plowing to a depth of 30 cm, populations of *A. radicina* in the top 10 cm were reduced to approximately 40% of the total, and populations in the 10 to 20 cm depth were increased to approximately 40%. Little change in population density occurred below 20 cm. Reduction in soilborne inoculum in the top 10 cm of soil resulted in a small but significant reduction (6.1%) in the incidence of black rot at harvest. This reduction in disease incidence was not economically justified considering the costs of deep-tillage.

Carrot cultivars resistant to *A. radicina* infection are a promising tool for improved control of black rot. Cultivated carrot has been grown for over 1,000 years, and extensive germ plasm is available for selection (4). In addition, the genus *Daucus*

includes 17 species and is a considerable source of phenotypic and genetic variation (4,58). Only recently have carrot breeding programs begun to assess black rot resistance in cultivated and wild carrot germ plasm (58). Studies conducted in Europe and Asia have indicated that some carrot germ plasm possesses resistance to *A. radicina* (23,75,76). Carrot lines evaluated in these studies were primarily types grown commercially in Europe and Asia, such as Berlinikum, Amarak, Nantes, and Chantenay, and black rot resistance was found in representatives of all types. In the United States, susceptibility to direct infection by *A. radicina* was evaluated in three field trials for 46 commercial carrot cultivars, including 41 Imperator types, using a toothpick inoculation method (44). A number of cultivars appeared to possess some level of resistance to infection by *A. radicina* (i.e., significantly reduced lesion sizes). Nine cultivars also were inoculated with *A. radicina*-infested toothpicks and maintained in cold-storage for 10 weeks (44). Lesion development was much greater in cold-storage than in the field, but the relative ranking of cultivars in terms of resistance to *A. radicina* was similar, with Caro-pak and Panther being the most resistant. The identification of Caro-pak as resistant to *A. radicina* is interesting because this cultivar is known to be highly susceptible to *A. dauci* (J. J. Nunez, *personal communication*). It is possible that distinct genetic mechanisms may govern resistance or susceptibility to infection of root versus leaf tissues by these *Alternaria* spp.

The use of pathogen-free (tested) seed is the first, and perhaps the most important, step toward the integrated management of black rot of carrot, particularly in new production areas. Seed treatments, such as hot water and/or fungicides (e.g., thiram), can be used to eradicate seedborne fungi, including *A. radicina*, from infested seed (32,72). While these treatments are generally effective, small populations of the pathogen may persist on treated seed (11,42). Furthermore, because these treatments can be expensive and/or reduce seed germination, they may not be used commercially. In some cases, growers simply over-seed to compensate for reduced stands resulting from damping-off (7,72). However, with the increasing use of precision planting to obtain uniform stands, even low levels of *A. radicina* infestation can lead to unacceptable stands.

Because of difficulties with application of fungicide to control *A. radicina* in the field, little effort has been placed in developing effective chemical control of black rot in the field. However, interest in biological control of plant diseases has been ongoing. Most efforts have focused on the use of bacterial agents that exhibit antifungal activity, such as, *Burkholderia* (*Pseudomonas*) *cepacia* and *Bacillus subtilis*.

The effects of *B. cepacia* in promoting carrot seedling emergence and controlling disease were equivalent to 100 ppm iprodione, with both applied as seed treatments (10). In addition, *B. cepacia* significantly reduced the development of black rot on carrot foliage compared with the control. Similar results of improved seedling emergence and disease reduction in carrots were recorded in studies utilizing *B. subtilis* (24). In stored carrots, isolates of *Trichoderma viride* were antagonistic to two storage rot pathogens, *A. radicina* and *Sclerotinia sclerotiorum*, and offer potential for management of storage rots of carrot (56).

Detection of *Alternaria* Species on Carrot Seed

Testing of treated seed is critical to ensure the efficacy of seed treatments and to determine whether infestation levels are at, or below, economic thresholds. Several methods are available for detection of *A. radicina* and *A. dauci* on carrot seed.

These include seedling grow-out, malt agar, and freeze-blotter assays (22). The most commonly used method for detecting *Alternaria* species on carrot seed is the freeze-blotter method. This involves placing seed on moistened blotter paper, freezing the seed to prevent germination, and then incubating seed to allow for fungal growth and sporulation. Seeds are examined microscopically for pathogenic *Alternaria* species (22,42).

More recently, new methods of detecting *A. radicina* on carrot seed have been developed based on semiselective media. *A. radicina* selective agar (ARSA) was developed specifically for the detection of *A. radicina* based upon characteristic mycelia development (Fig. 5) (42). More recently, *Alternaria dauci-radicina* selective medium (ADRSM) was developed for detection of both *A. radicina* and *A. dauci*; however, the medium can be optimized for either one or the other (68). These agar methods have the advantage over the freeze-blotter method in that pathogenic

Alternaria species can generally be differentiated from saprophytic fungi on the basis of distinctive mycelium and colony morphology rather than more laborious microscopic examinations.

Despite efforts to use certified pathogen-free (tested) seed, the continued occurrence of seedborne *A. radicina* and *A. dauci* impacts commercial carrot production worldwide (38,42,55). Part of the problem is that cultural methods used to assay seeds for these pathogens are time-consuming, laborious, and rely on visual identification of the fungi on seeds. Moreover, there are technical constraints to the number of seeds per lot that can be practically evaluated. In order to effectively detect and reduce levels of inoculum on seed in an increasingly global commercial seed market, new methods for detection of seedborne pathogens are needed that are rapid, specific, and sensitive enough to detect low levels of seed infestation.

Polymerase chain reaction (PCR)-based seed assays have been developed for the detection of *A. radicina* and *A. dauci* from infested carrot seed and offer the possibility of accomplishing these objectives. Pryor and Gilbertson (45) developed PCR primer pairs and a PCR protocol for detection of *A. radicina* on infested carrot seed. The *A. radicina* primer pair directed amplification of the target DNA sequence from as little as 200 fg of total genomic DNA, and the PCR-based seed assay detected *A. radicina* in seed lots with natural infestation rates as low as 0.3% (45). In seed samples prepared by mixing *A. radicina*-infested seed with increasing amounts of noninfested seed, the PCR-based seed assay detected the pathogen at infestation rates as low as 0.1% (Fig. 6).

Subsequent research on PCR-based detection of *Alternaria* in carrot seed, by Konstantinova et al. (25), has led to the development of PCR primers specific for three *Alternaria* species known to occur on carrot: *A. radicina*, *A. dauci*, and *A. alternata*. The primers were highly sensitive and were able to differentiate among the three *Alternaria* spp. Moreover, the primers were used to detect the fungi in samples of carrot tissue and carrot seed, highlighting their potential utility in detecting the fungus in seed production fields before seed infestation occurs. Thus, PCR-based detection of *Alternaria* spp. pathogenic to carrot may become a standard technique in the management of both black rot and *Alternaria* leaf blight.

Summary and Future Research

Alternaria diseases of carrot are, and will continue to be, challenging plant health problems. Since the pathogens causing these diseases are seedborne, *Alternaria* leaf blight and black rot occur in most areas where carrots are grown. No single control method is sufficient for either disease. Therefore, disease control is

Fig. 5. Distinctive growth and appearance of *Alternaria radicina* on *A. radicina* semi-selective agar (ARSA) after recovery from soil. A, Single colony showing characteristic growth of black hyphae. B, Soil dilution plated to ARSA showing distinctive colonies of *A. radicina* compared with those of other soil fungi (43).

Fig. 6. Detection of *Alternaria radicina* from carrot seed lots with different levels of infestation using a polymerase chain reaction-based seed assay (45). Lanes 1 to 12 show results from two independent DNA extractions for each seed lot having the indicated rates of *A. radicina* infestation: lanes 1 and 2 = 20.00%, lanes 3 and 4 = 4.00%, lanes 5 and 6 = 1.00%, lanes 7 and 8 = 0.5%, lanes 9 and 10 = 0.25%, and lanes 11 and 12 = 0.10%. Lane 13 is a positive control showing the amplification of the approximately 900-bp target DNA fragment amplified from *A. radicina* total genomic DNA. Lane M contains a 1-kb DNA ladder (Gibco BRL, Gaithersburg, MD).

dependent on integrating the most appropriate methods for the specific production region.

Current strategies to control *Alternaria* leaf blight and black rot involve multiple methods, including planting of pathogen-free seed, seed testing, seed treatments, crop rotation, scouting, computer forecasting models, applications of gibberellin and/or fungicides, and appropriate postharvest handling of carrots. Each of these methods has been well researched and optimized, but less frequently have the methods been evaluated in combination (e.g., forecasting and fungicide use). Future research should focus on the integration of multiple control methods to maximize yields and minimize inputs. Using multiple disease control methods may reduce or even eliminate the need for fungicide use. Alternatively, optimum use of multiple methods of disease management may allow production of carrots when production circumstances preclude the use of one specific method. For example, growers may not always be able to use crop rotation to effectively decrease *Alternaria* inoculum, and organic growers do not use synthetic fungicides. Under these circumstances, optimal integration of the available control options should be examined in detail.

A. dauci and *A. radicina* initially infect senescing carrot foliage, but healthy foliage is necessary for efficient mechanical harvest. Considering these factors, it may be possible to decrease losses to *Alternaria* leaf blight and black rot of carrots by delaying foliar senescence. Methods for accomplishing late-season delay in foliar senescence and their effects on disease severity, harvestability, and yield need to be explored.

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James J. Farrar

Dr. Farrar is an assistant professor of plant pathology in the Department of Plant Science at California State University, Fresno. He received a B.S. in plant pathology from the University of Wisconsin, Madison, an M.S. in plant pathology from the University of California, Davis, and a Ph.D. in botany from the University of Wisconsin, Madison. His research focuses on control of vegetable diseases in the San Joaquin Valley of California.

Barry M. Pryor

Dr. Pryor is an assistant professor in the Division of Plant Pathology and Microbiology at the University of Arizona, Tucson. He received a B.S. in biology from the University of California, Santa Cruz, an M.S. in plant protection and pest management from the University of California, Davis, and a Ph.D. in plant pathology from the University of California, Davis. His research interests include biological and cultural control of disease in field, tree, and vegetable crops, phylogenetic analysis and species concepts in fungi, secondary fungal metabolites, and environmental mycology.

R. Michael Davis

Dr. Davis is a professor of plant pathology and Specialist in Cooperative Extension in the Department of Plant Pathology at the University of California, Davis. He received a B.A. in biology from California State University, Long Beach, and a Ph.D. from the University of California, Riverside. His current research focuses on the epidemiology and control of diseases of vegetables and field crops, especially those caused by fungal and bacterial pathogens.