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Economically Important Diseases of Spinach

Spinach (*Spinacia oleracea* L.) is an economically important leafy vegetable crop in many countries, and approximately 14,000 ha (35,000 acres) are grown annually throughout the United States (25) for fresh and processed (frozen and canned) markets. The annual crop value in the United States is approximately $70 million (25). Major spinach production states include California, Texas, Arkansas, Oklahoma, Maryland, Virginia, New Jersey, and Colorado (32).

Spinach apparently is native to central Asia and has been cultivated for more than 1,300 years (46). Spinach is a member of the Chenopodiaceae and is related to Swiss chard, sugar beet, and table beet. The plant produces a compact rosette of leaves and bolts (i.e., produces a seed stalk) in response to temperature and photoperiod. Spinach is usually dioecious, producing either male or female flowers. The majority of the spinach seed used in the United States is produced in the Pacific Northwest, where long days correspond with moderate temperatures.

Spinach typically is grown as a direct-seeded crop, with planting and management strategies dictated by the market destination of the commodity. Cultivars can vary from "flat-leaf" to "highly savoy" or wrinkled types. Spinach is grown year-around in California but is a fall, winter, or spring crop in other locations in the United States. Most fresh-market spinach is hand-harvested as a loose-leaf pack or bundled in containers. Spinach for processing is mechanically harvested and in some locations is repeat-harvested after regrowth.

As with most agricultural commodities, diseases impose significant production constraints affecting both yield and overall quality of spinach. An integrated disease management approach, including the use of disease-resistant cultivars, crop rotation, careful irrigation, fertility management, and fungicides, is often necessary to produce a high-quality product. Although many diseases have been reported on spinach (14,36,41), this discussion is limited to fungal and viral diseases that are generally recognized as economically important. Bacterial diseases of spinach in the field are noticeably absent. Aster yellows, caused by a mycoplasma, is occasionally diagnosed (36), and bacterial soft rot has been observed as a postharvest disease of leaves and on overly mature plants in the greenhouse.

Downy Mildew

Downy mildew (or blue mold), caused by *Peronospora farinosa* (Fr.:Fr.) Fr. f. sp. *spinaciae* Byford (= *P. effusa* (Grev.) Ces.) (6) is probably the most widespread and potentially destructive disease of spinach worldwide. Initial symptoms are slightly yellow, irregular, chlorotic lesions on leaves (Fig. 1A). Lesions frequently expand and coalesce and may become necrotic. Heavily infected leaves can appear curled and distorted. Under wet conditions and/or high relative humidity, blue-gray sporangia and sporangiospores are produced and can be seen in mass on the underside of the leaf and occasionally on the upper leaf surface (Fig. 1B). Under environmental conditions favorable for the fungus, sporulation can often be observed in the absence of lesion development.

P. f. spinaciae is a heterothallic obligate fungal pathogen belonging to the Peronosporales (18). Although it is morphologically similar to the downy mildew pathogen that attacks beets, the two apparently are physiologically distinct, with *P. f. spinaciae* infecting only the genus *Spinacia* and several species of *Chenopodium* (9). Four physiologically distinct races of *P. f. spinaciae* have been reported (Table 1) (6).

Under cool, wet conditions, sporangia can germinate directly or release zoospores in 2–6 hours on leaf surfaces (31). Germination of sporangia and germ tube elongation can occur between 2 and 25 C, with an optimum temperature of 9–12 C. Lesion development is favored by temperatures of 15–25 C, and resporulation can occur 6–12 days after infection. Sporangia are readily dispersed by wind and rain splashing but can rapidly lose viability when desiccated or exposed to sunlight (31,45). Under conditions of prolonged leaf wetness and cool temperatures, epidemics can progress very rapidly and an entire crop can be lost in a short period of time. As lesions mature, the fungus can produce oospores that serve as a resistant survival structure. Oospores and mycelium of the downy mildew pathogen have been recovered from spinach seed, and contaminated seed have been shown to give rise to infected seedlings (19). Oospores can survive in soil and may represent an important source of primary inoculum (45). Windborne sporangia from sur-

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rounding spinach crops may represent an important source of primary inoculum in locations with successive spinach crops.

Historically, downy mildew has been controlled by planting cultivars with single-gene resistance to a given race (6,20,34,35). Single-gene resistance to downy mildew is relatively easy to identify and manage in a breeding program and can be incorporated into horticulturally acceptable cultivars relatively quickly. However, when new races of the downy mildew pathogen appear, as recently occurred in California (6,8,10), it may be several years before a new commercial cultivar with single-gene resistance becomes available. Consequently, additional management practices, including the use of fungicides, are necessary (23).

White Rust

White rust, caused by *Albugo occidentalis* G.W. Wils., is a very important disease in all U.S. spinach production areas east of the Rocky Mountains. The pathogen was first reported on *Chenopodium capitatum* (L.) Aschers. from Colorado in 1903 and was recognized as an economically important disease on spinach in Virginia in 1907, in Texas in 1937, and in the Arkansas River Valley in 1945 (30,43). White rust does not occur on spinach in western production areas and has not been reported on spinach outside the United States.

Initial symptoms of white rust are small chlorotic lesions on the leaf surface. As lesions develop, small glassy white pustules (sori) are produced, frequently in concentric rings, on the underside of the leaf and occasionally on the upper leaf surface (Fig. 2A). Lesions often coalesce, and white rust pustules can cover the entire leaf surface. As lesions mature, dark oospores are frequently produced, giving the lesion a grainy appearance prior to necrosis (Fig. 2B).

The biology of the spinach white rust pathogen is similar to that of the more thoroughly studied white rust pathogen of crucifers, *Albugo candida* (Pers.) Kunze. Like the downy mildew pathogen, *A. occidentalis* is an obligate fungal pathogen in the Peronosporales. Its host range is limited to *Spinacia* and several species of *Chenopodium* (30,43). Unlike the case with *A. candida*, physiological races of *A. occidentalis* have not been reported. Dry sporangia are hyaline and discoid and measure approximately 10 × 14 μm. When hydrated, sporangia become spherical to ellipsoid (Fig. 3A) and measure 10–19 × 20–22 μm. Oospores are finely reticulated (Fig. 3B) and 44–62 μm in size (30).

Sporangia typically germinate indirectly but occasionally directly (30) and produce six to nine biflagellate motile zoospores that encyst and produce a germ tube which directly penetrates the spinach leaf. Sporangia can germinate at 2–25 C, with an optimum temperature of 12–16 C. Approximately 22 C is optimum for sporulation, and higher temperatures accelerate lesion development. Sporangia are produced in the leaf pustules, or sori, and are released when the epidermal tissue covering the sori ruptures. A copious amount of sporangia can be released from a heavily infected leaf. In Texas, severe epidemics of white rust apparently are favored by cool nights with heavy dew alternating with warm, dry sunny days (30).

Oospores are produced abundantly in mature lesions and are an important

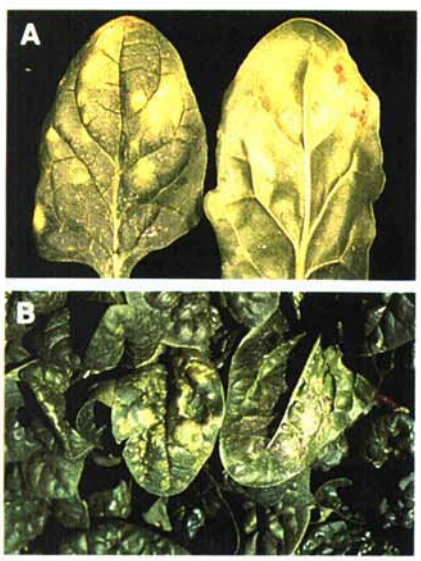

Fig. 1. Symptoms and signs of downy mildew of spinach caused by *Peronospora farinosa* f. sp. *spinaciae* include (A) diffuse chlorotic lesions and (B) blue-gray sporulation of the pathogen.

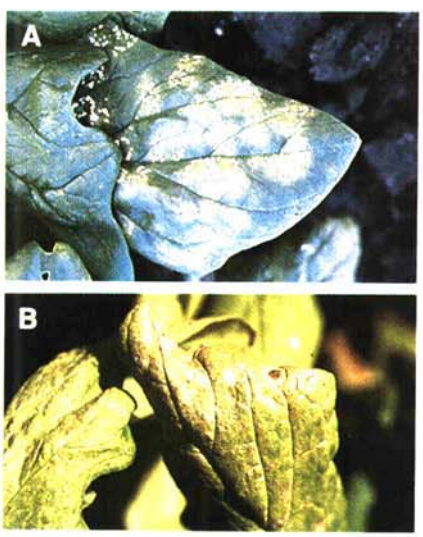

Fig. 2. Symptoms and signs of white rust of spinach caused by *Albugo occidentalis* include (A) white rust pustules (sori) that may occur in concentric rings and (B) a dark grainy appearance as lesions mature and oospores are produced.

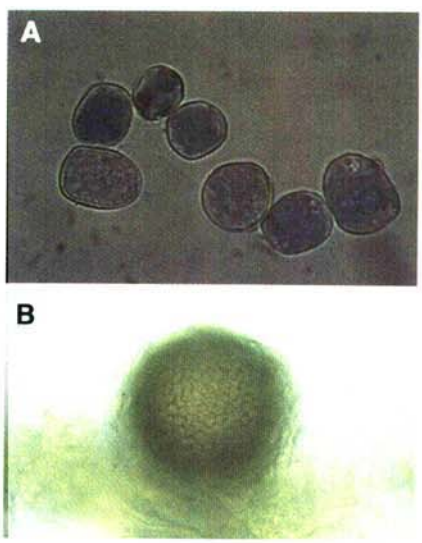

Fig. 3. (A) Sporangia and (B) oospore of *Albugo occidentalis*.

Table 1. Disease reactions of differential spinach cultivars to the downy mildew pathogen, *Peronospora farinosa* f. sp. *spinaciae*

Race	Cultivar reaction[a]				
	Viroflay	Nores	Califlay	St. Helens	Polka
1	S	R	R	R	R
2	S	R	S	R?[b]	R
3	S	S	R	R	R
4	S	S	S	S	S

[a] S = >90% incidence on cotyledons and >85% incidence on true leaves; R = <10% incidence on cotyledons and <15% incidence on true leaves (6).
[b] Disease incidence on cotyledons and true leaves was 40 and 2%, respectively (6).

Fig. 4. Spinach disease breeding nursery in Uvalde, Texas. Highly susceptible "spreader" rows appear lighter because of severe white rust disease.

survival structure. Higher temperatures favor production of oospores more than production of sporangia. Oospores probably function as a source of primary inoculum to initiate epidemics. Oospores of *A. candida* can germinate directly or produce a sporangium to initiate infection (39,40). There are no descriptions of oospore germination of *A. occidentalis*.

A recurrent selection breeding strategy has been used to develop spinach cultivars with field, or horizontal, resistance to white rust (Fig. 4) (4). With horizontal resistance, latent periods are prolonged and both lesion development and sporulation are reduced (7,13). Several cultivars with excellent levels of white rust resistance have been released in recent years (5,17). However, such cultivars can be severely damaged under favorable environmental conditions and high disease pressure. Integrated management practices such as crop rotation, use of resistant cultivars, a preplant treatment with the fungicide metalaxyl, and foliar fungicides have been effective in managing this disease (3,12). Certain spinach cultivars with a relatively high level of white rust resistance have also been shown to have some field resistance to races 3 and 4 of the downy mildew pathogen (7).

Leaf Spot Diseases

Several fungi can cause leaf spot diseases on spinach. Anthracnose, caused by *Colletotrichum dematium* (Pers.) Grove f. sp. *spinaciae* (Ellis & Halst.) Arx (= *C. spinaciae* Ellis & Halst.), can be particularly damaging to spinach foliage. Anthracnose has been reported from most spinach production areas in the United States (11,22). Initial symptoms are small, circular, water-soaked lesions on both young and old leaves (Fig. 5A). Lesions often enlarge and become chlorotic or necrotic (Fig. 5B). Older lesions turn tan and tissues become thin and papery. Leaves look entirely blighted as lesions coalesce (Fig. 5C). Acervuli with dark setae are often observed in anthracnose lesions and are useful diagnostic characteristics (Fig. 5D). Also, *C. d. spinaciae* is a common secondary pathogen on lesions caused by *A. occidentalis* (Fig. 5E) or *Cercospora beticola* Sacc. (11). During white rust epidemics, anthracnose often can be more of a problem than the white rust because the fungal setae give the lesions a dark appearance.

C. d. spinaciae can survive in infected plant debris as dormant mycelium. This is probably the most important source of primary inoculum. There is some debate as to whether or not this fungus is seedborne. Acervuli can produce many rain-splashed and airborne conidia that serve as secondary inoculum. Anthracnose epidemics are sporadic and are favored by very wet conditions. Dense plantings, poor air circulation, and low plant fertility also can increase the severity of epidemics.

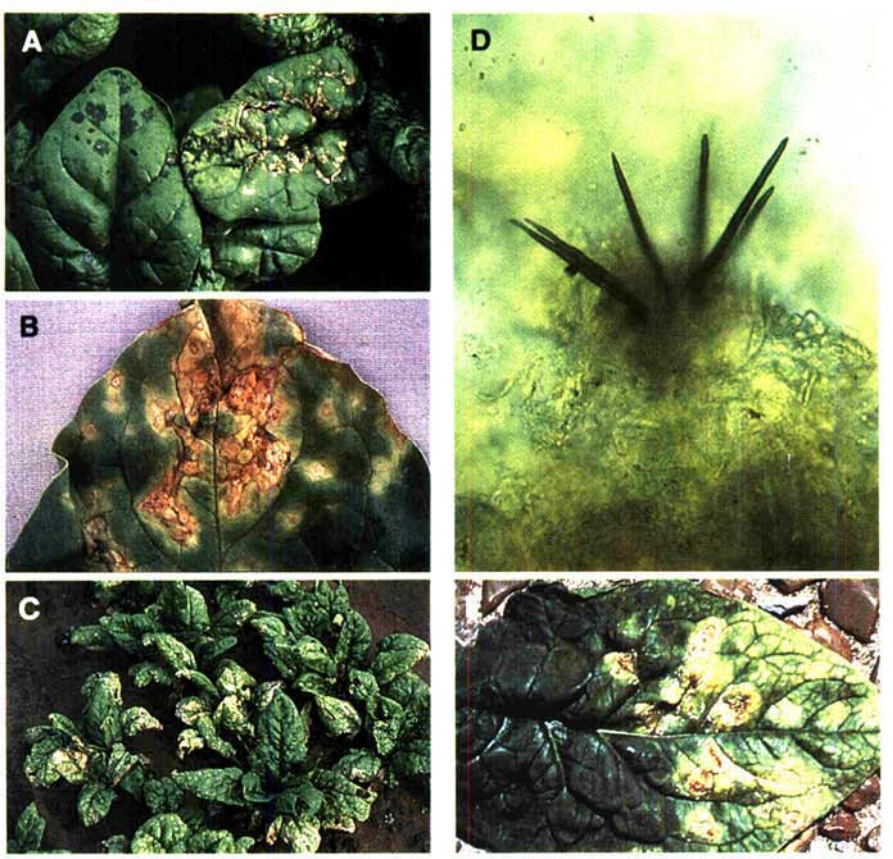

Fig. 5. (A) Initial symptoms of anthracnose of spinach caused by *Colletotrichum dematium* f. sp. *spinaciae* are water-soaked lesions that progress into (B) chlorotic and necrotic lesions. (C) Leaves under heavy disease pressure appear blighted. *C. d. spinaciae* produces (D) a reproductive structure (acervulus) with dark setae on infected leaves and (E) also can infect leaves previously attacked by other pathogens such as *Albugo occidentalis*.

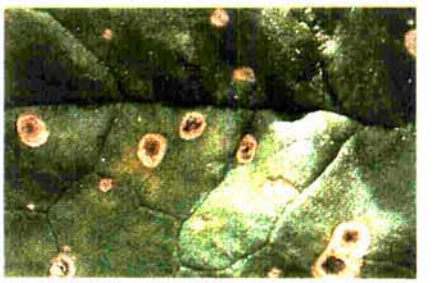

Fig. 6. Lesions on spinach leaf caused by *Cladosporium macrocarpum*.

Fig. 7. Leaf lesions caused by several fungi, including *Cladosporium macrocarpum*, *Alternaria* sp., and *Stemphyllium* sp., on mature spinach plants being used for seed production.

Fig. 8. Pre- and postemergence damping-off of spinach seedlings and young plants caused by a complex of fungal pathogens.

Management practices such as irrigation and fertilization can reduce the severity of anthracnose epidemics. Resistance to anthracnose has been reported but has been difficult to document quantitatively (11,17). Several copper fungicides have been used to slow anthracnose epidemics, but they generally have been ineffective, particularly under very wet conditions.

Other leaf-spotting fungi that have been observed on spinach include *C. beticola, Cladosporium macrocarpum* G. Preuss, and *Alternaria* sp. (36). Symptoms of infection by *C. macrocarpum* have been observed on spinach under very wet conditions (Fig. 6). *C. macrocarpum, Alternaria* sp., and *Stemphyllium* sp. also can be very damaging to foliage of mature plants grown for seed production (Fig. 7).

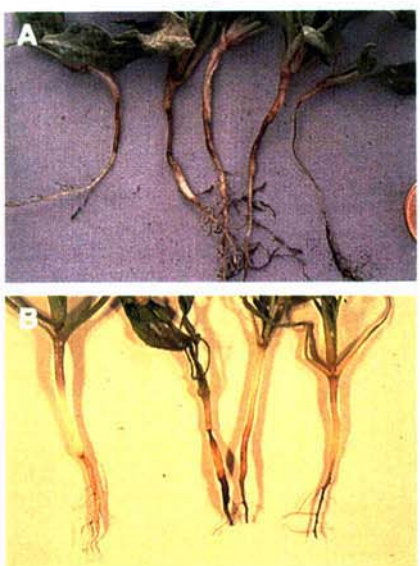

Fig. 9. (A) Girdling of spinach seedling roots near the soil line as a result of infection by *Pythium* sp. and (B) taproot tip necrosis caused by *Fusarium oxysporum* and *Rhizoctonia solani*.

Soilborne Diseases

There are several economically important soilborne diseases of spinach. Damping-off is problematic in spinach production areas throughout the world. Severity is influenced by cultivar, soil temperature, soil moisture, and disease pressure. Fungi that cause diseases of seedlings include *Fusarium oxysporum* Schlechtend.:Fr., *Fusarium* sp., *Pythium aphanidermatum* (Edson) Fitzp., *P. irregulare* Buisman, *Pythium* sp., *Rhizoctonia solani* Kühn, and *Aphanomyces cochlioides* Drechs. (24,36,37). These fungi can cause pre- and post-emergence damping-off (Fig. 8). Roots of infected seedlings can appear water-soaked, the upper taproot may be girdled by a necrotic lesion (Fig. 9A), or the tip of the taproot may be necrotic (Fig. 9B).

Severe damping-off of spinach is associated with warm, wet soils with a history of frequent spinach production. Management practices typically include the use of a seed-treatment fungicide effective against pythiaceous fungi, *F. oxysporum*, and *R. solani*.

Fusarium wilt of spinach, caused by *F. o. spinaciae* (Sherb.) W.C. Snyder & H.N. Hans., is a very important disease of spinach worldwide (2,24). In Arkansas, *F. o. spinaciae* is an important component of the damping-off complex in spinach planted in the fall in warm soils. In spring-planted spinach, *F. o. spinaciae* causes the more typical vascular wilt symptom on mature plants because of increasing temperatures as the plant matures (16). Fusarium wilt has not been reported to be a problem on spinach in Texas. The older leaves of symptomatic plants may wilt and become chlorotic (Fig. 10A). Often, the vascular system of an infected plant is darkly discolored and the tip of the taproot is necrotic (Fig. 10B). Fusarium wilt can also kill mature plants being grown for seed production (Fig. 10C).

Single-gene resistance to *F. o. spinaciae* has not been reported. However, several U.S. plant introductions and cultivars have been reported to have some resistance to the pathogen (17,27).

Several *Phytophthora* species, including *P. megasperma* Drechs. and *P. cryptogea* Pethybr. & Lafferty, have been reported to cause root rot of spinach (14,36). In addition, an unidentified *Phytophthora* sp. can attack spinach leaves and petioles in contact with wet soil (Fig. 11A) and progress into the plant crown (Fig. 11B).

Several species of nematodes also attack spinach roots (28). In the United States, the southern root knot nematode (*Meloidogyne incognita* (Kofoid & White) Chitwood) can cause galling on spinach roots (Fig. 12), particularly in sandy soils.

Virus Diseases

Although 14 naturally occurring virus diseases have been reported on spinach

Fig. 10. Fusarium wilt of spinach caused by *F. oxysporum* f. sp. *spinaciae*: (A) General plant collapse and chlorosis of older leaves, (B) vascular discoloration and taproot tip necrosis, and (C) death of plants being grown for seed production.

Fig. 11. *Phytophthora* sp. infection (A) of spinach leaf and petiole in contact with the soil surface and (B) advancing into the plant crown.

Fig. 12. Roots of spinach plants damaged by the southern root-knot nematode (*Meloidogyne incognita*).

in the United States and Europe (38,44), only a few are considered to be economically important in the United States. Cucumber mosaic virus (CMV), beet western yellows virus (BWYV), and beet curly top virus (BCTV) are three of the more common. The economic impact of these three viruses has been described on sugar beet (42) but has not been well documented on spinach. In one field test in which all plants were inoculated with CMV, yields were reduced by 23–47%, depending on the cultivar (44).

CMV is capable of infecting many agricultural hosts and has even been referred to as the "influenza" virus of the plant world. On spinach, CMV causes the disease known as spinach blight. Symptoms vary greatly according to cultivar, plant age, temperature, and virus strain. These include a mild general chlorosis of younger leaves progressing to a severe blighting of the growing point and plant death. Younger leaves of infected plants often have a chlorotic mottle and are narrow and severely wrinkled with veinal distortions (Fig. 13A). Leaves also can have an inward rolling of margins. In advanced stages of disease, the plants often appear stunted (Fig. 13B) and the crown leaves may become completely blighted, killing the growing point (Fig. 13C).

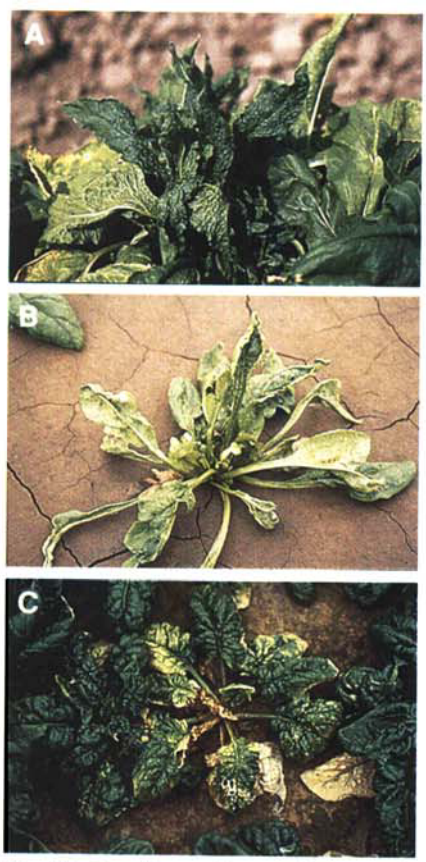

Fig. 13. Symptoms of cucumber mosaic virus infection on spinach include (A) narrow and "puckered" young leaves, (B) stunting and leaf distortion, and (C) leaf chlorosis and necrosis of the growing point.

CMV is an icosahedral single-stranded RNA virus and a member of the cucumovirus group. Many strains of CMV have been reported (21), including several from spinach (15,47). The epidemiological significance of the various strains of CMV on spinach is not known.

Although other vectors may exist, CMV on spinach apparently is vectored predominantly by aphids in a nonpersistent manner. Typical CMV symptoms developed on spinach within 9 days of exposure to viruliferous green peach aphids (*Myzus persicae* (Sulzer)) (Fig. 14) (44).

Resistance to some strains of CMV on spinach has been shown to be controlled by a single dominant gene in the cultivar Virginia Savoy (29). Single-gene resistance to CMV has been incorporated into many contemporary spinach cultivars but may not be complete and is known to break down at higher temperatures (>28 C). Consequently, CMV can still be problematic in spinach. In addition to use of resistant cultivars, management practices include cultural and chemical control of weeds that may serve as reservoirs of CMV. Efforts to reduce populations of aphid vectors should also be made, although there is no evidence that insecticides targeting aphid vectors reduce the incidence of CMV in spinach. Recently, CMV was shown to be seedborne in spinach (47). In some cases, seed may serve as an important source of primary inoculum. However, we do not know how widespread CMV is in seed of commercial spinach cultivars, if the seedborne CMV is a unique strain, or if only certain cultivars are prone to seed transmission.

BWYV infection is widespread in spinach. Initially, interveinal and leaf margin chlorosis appear on older leaves (Fig. 15A). As the disease progresses, chlorosis becomes much more intense until the older leaves near the soil line become completely chlorotic (Fig. 15B). The older symptomatic leaves are frequently invaded with secondary fungal pathogens such as *Alternaria* sp. (42), resulting in reddish brown to bronze-colored lesions. These secondary invaders may often be more important in reducing overall leaf quality than BWYV alone. Leaf quality can decrease drastically within 1 or 2 days when clear, sunny weather follows several overcast days.

BWYV is an icosahedral single-stranded RNA virus like CMV but is a member of the luteovirus group. BWYV has a very broad host range that includes several important agricultural crops such as broccoli, cauliflower, lettuce, pea, potato, radish, and sugar beet. Many weed hosts, particularly crucifer weeds,

Fig. 14. Green peach aphids (*Myzus persicae*) are vectors of cucumber mosaic and beet western yellows viruses on spinach.

Fig. 15. With beet western yellows virus infection of spinach, (A) initial interveinal chlorosis is followed by (B) severe chlorosis of older leaves and often secondary infection by fungal pathogens such as *Alternaria* sp.

Fig. 16. (A and B) Stunted, compact, and chlorotic spinach plants infected with beet curly top virus.

can serve as reservoirs of inoculum. The virus is vectored by several aphid species in a persistent or circulative manner (42) and can persist in aphids for relatively long periods of time (4–6 weeks). On spinach, the green peach aphid apparently is the most common vector (26).

BWYV is difficult to control. Reservoirs of inoculum and aphid populations should be reduced by cultural and chemical control of weed hosts. Spinach should not be planted adjacent to susceptible agricultural hosts if possible. In southwest Texas, where spinach fields are repeat-harvested, early harvests help to minimize quality problems in fields with a high incidence of BWYV. No resistance to BWYV has been reported in spinach.

BCTV disease of spinach can be very destructive. Symptoms initially appear 3–4 weeks after infection (26). Infected plants initially appear stunted and chlorotic. Younger leaves in the center of the rosette are often very chlorotic, extremely curled, and rigid (Fig. 16). Plants usually die a few weeks after symptoms appear. BCTV can be particularly severe in spinach fields with poor stands or low plant populations (26).

BCTV is a single-stranded DNA virus in the geminivirus group and has an extensive host range capable of infecting over 300 plant species (42). On spinach, BCTV is vectored in a persistent manner by the beet leafhopper (*Circulifer tenellus* (Baker)). In Texas, primary infections occur on spinach in the fall after migrating leafhoppers acquire the virus from weed hosts (26). Secondary spread of BCTV in spinach has not been documented. Again, weeds that may serve as vector reservoirs should be controlled. In addition, efforts should be made to ensure uniform stands. Several spinach plant introductions have been reported to have some resistance to BCTV (33).

Meeting a Goal

Spinach continues to increase in popularity as a leafy vegetable crop highly regarded for its nutritional value. The use of pesticides on spinach can be greatly reduced by exploiting disease-resistant cultivars and employing proper disease management strategies. The development of new strategies, such as virus resistance using virus coat-protein transformed spinach (1,47), and improvement of techniques, such as soil solarization to reduce soilborne pathogens and weed pests, may allow spinach to meet one of the goals of modern agriculture: production of a nutritious and profitable crop with little or no use of pesticides.

Acknowledgments

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