Watermelon Fruit Disorders

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SUMMARY. Watermelon *(Citrullus lanatus* [Thunb.] Matsum & Nakai) fruit are affected by a number of preharvest disorders that may limit their marketability and thereby restrict economic returns to growers. Pathogenic diseases discussed include bacterial rind necrosis (*Erwinia* sp.), bacterial fruit blotch [*Acidovorax avenae* subsp. *citrulli* (Schaad et al.) Willems et al.], anthracnose [*Colletotrichum orbiculare* (Berk & Mont.) Arx. syn. *C. legenarium* (Pass.) Ellis & Halst], gummy stem blight/black rot [*Didymella bryoniae* (Auersw.) Rehm], and phytophthora fruit rot (*Phytophthora capsici* Leonian). One insect-mediated disorder, rindworm damage is discussed. Physiological disorders considered are blossom-end rot, bottleneck, and sunburn. Additionally, cross stitch, greasy spot, and target cluster, disorders of unknown origin are discussed. Each defect is shown in color for easy identification.

Growers and advisory personnel are often confronted with field problems that are difficult to diagnose. One such case in point are the many preharvest maladies affecting watermelon fruit. Although watermelon fields are frequently scouted for pest management purposes, fruit are not examined carefully until harvest begins. Accordingly, rapid diagnosis with accurate prediction of consumer acceptability is essential for marketing purposes. It is also important to know if there is likelihood of spread to unaffected fruit in transit. Where possible, we have included suggestions for amelioration of the problem, but have not included pesticide recommendations because of the advantage of current, local recommendations.

Pathogenic diseases

BACTERIAL RIND NECROSIS. Rind necrosis was first reported in Hawaii (Ishii and Aragaki, 1960). Typical rind necrosis is characterized by a light brown, dry, and hard discoloration interspersed with lighter areas (Fig. 1). The disease develops in the rind and rarely extends into the flesh. Occasionally the affected area is limited to the vascular bundles, but generally the discoloration spreads, sometimes affecting the entire rind. The causal organism was believed to be an *Erwinia* species.

The disease was first reported in the continental United States in several areas of Texas (Thomas, 1968). Fruit from some fields were completely free of the disease, whereas in other fields infected fruit ranged from few to many. Bacterial rind necrosis was often more severe in fields where plants were heavily infested with watermelon mosaic virus. This led to speculation that a common vector may facilitate the spread of both diseases. The causal organism again was narrowed down to an *Erwinia* species.

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Rind necrosis is a serious disease of watermelon that regularly occurs in California's Imperial Valley (Kontaxis and Kurupus, 1975). Results from several experiments to determine a causal agent were inconclusive.

In other experiments (Hopkins and Elmstrom, 1977), the diversity of bacterial flora isolated from healthy and diseased fruit was similar except that enterobacteria were isolated more frequently from diseased than from healthy fruit. *Erwinia, Pseudomonas, Enterobacter,* and *Bacillus* isolated from symptomatic fruit caused rind necrosis at injection sites following inoculation.

Variation among varieties in susceptibility was reported in Florida (Elmstrom and Hopkins, 1973 and Hopkins and Elmstrom, 1974). Over a 3-year evaluation period, 'Sweet Princess' and 'Jubilee' were most tolerant, whereas 'Klondike Blue Ribbon' and 'Louisiana Queen' were most susceptible. The incidence of bacterial rind necrosis varied from year to year. For example, 'Sweet Princess', the most tolerant variety, had 10.0%, 14.8%, and 21.8% affected fruit in 1972, 1973, and 1974, respectively. 'Klondike Blue Ribbon', the most susceptible variety, had 39.1%, 64.8%, and 73.7% affected fruit in those years.

Some of these same varieties were evaluated in Imperial Valley of California (Kontaxis, 1976). 'Klondike Blue Ribbon' was the most tolerant variety and 'Sweet Princess' was intermediate in susceptibility. These results are at odds with those obtained in Florida.

To summarize, bacterial rind necrosis has been reported from several watermelon growing areas, *Erwinia* sp. most often has been associated with the diseased rind areas, other bacterial organisms isolated from either diseased or healthy fruit cause typical symptoms at inoculation sites, the incidence of the disease varies among varieties and growing seasons, and varieties may not always respond to the disease in the same way.

Watermelon bacterial rind necrosis appears to be genetically controlled but its frequency is affected by an interaction with environment. For the most part, varieties in use today appear not to be as susceptible as those used in the 1970s suggesting that watermelon breeders have been successful in selecting against the disease.

BACTERIAL FRUIT BLOTCH. Bacterial fruit blotch was first reported in the Marianna Islands (Wall and Santos, 1988). Symptoms on the surface of fruit begin as small, dark-green, water-soaked lesions that rapidly expand to cover much of the fruit surface in 7 to 10 d (Fig. 2). These surface lesions do not enlarge much after harvest of the fruit. Initially, the lesions do not extend into the flesh of the melon. In advanced stages of lesion development, areas in the center of the lesion may turn brown, crack, and ooze a sticky, amber substance. Secondary organisms are ultimately responsible for decay and collapse of fruit.

The fruit blotch bacterium also produces foliar symptoms (Latin, 1996). First symptoms in watermelon seedlings consist of water-soaked areas on the lower surface of cotyledons and leaves, that become necrotic. In young seedlings, lesions in the hypocotyl can cause the collapse of the emerging plant. Leaf lesions are light brown to reddish-brown and often spread along the midrib of the leaf. Leaf lesions in the field usually are not very distinctive or severe and do not result in defoliation, but primarily serve as important reservoirs of bacteria for fruit infection.

Fruit blotch first occurred in commercial watermelon fields in the United States in Florida in the spring of 1989, with isolated severe outbreaks also occurring in South Carolina and Indiana as the season progressed (Latin and Hopkins, 1995). In some fields, losses were greater than 90%. Bacterial fruit blotch has been observed in the eastern U.S. every year since 1989, but usually occurs in only a few fields. The disease was most damaging in 1994, affecting thousands of acres of watermelons and causing extensive losses in at least 10 states.

The bacterium that causes fruit blotch of watermelon is *Acidovorax avenae* subsp. *citrulli* (formerly *Pseudomonas pseudoalcaligenes* subsp. *citrulli*). The disease cycle usually begins with contaminated seed (Latin and Hopkins, 1995). Much of the watermelon production in the U.S. is still from direct-seeded plants, but transplants are rapidly gaining in popularity. Bacteria from infested seed infect the developing seedling as the cotyledons emerge from the seed coat. Most transplant facilities employ overhead irrigation, which effectively splash-disperses bacteria to neighboring seedlings. This secondary spread in the transplant house can result in high numbers of infected seedlings reaching the field. In the field, symptom development and spread of *A. avenae* subsp. *citrulli* on foliage and fruit is most rapid during periods when the weather is hot and humid, with thundershowers (Hopkins, 1993).

The ideal control of this disease is to prevent the introduction of the bacterium into the field (Latin, 1996). This can be done by use of seeds that have tested negative for the presence of the fruit blotch bacterium, or use of plants from transplant houses in which there were no seedling symptoms of the fruit blotch disease. When symptoms are observed in the field, copper applications should begin as soon as possible and no later than the time of first flower (Hopkins, 1991). Weekly applications of the highest recommended rate of copper-containing fungicides has provided excellent control of bacterial fruit blotch of watermelon.

ANTHRACNOSE. On young fruit, anthracnose symptoms, caused by *Colletotrichum orbiculare,* consist of black spots which may

result in abortion or malformation of the melon. On older melons, small, water-soaked, elevated spots with greasy yellowish centers develop which give the fruit a bumpy appearance. These raised areas later become sunken and are covered with pink spore masses (Fig. 3). The disease may develop and spread in transit.

Anthracnose symptoms can occur on all above-ground parts of the watermelon plant (Parris, 1952). Leaf lesions are brown to black, irregularly shaped, and usually limited by leaf veins. Stem lesions are often oval shaped and tan colored with a brown margin. Leaf and stem lesions serve as a source of spores for infection of the fruit.

Anthracnose was the most destructive disease of watermelon in the United States 40 to 50 years ago (Parris, 1952). While resistant varieties and improved control strategies have reduced losses to this disease, it is still a relatively common disease of watermelon grown in humid regions throughout world. *Colletotrichum orbiculare* survives between crops on infected plant debris, volunteer plants and can be seedborne (Sitterly and Keinath, 1996a). Conidia of *C. orbiculare* are spread by wind, splashing rain, farm implements, and field workers. Humid, rainy weather is necessary for infection to occur.

Resistance to races 1 and 3 of *C. orbiculare* is present in several commercial watermelon varieties (Sitterly and Keinath, 1996a). Controls for anthracnose should include planting clean seed produced in areas where anthracnose is not a problem and suppressing primary inoculum by deep plowing of crop residue after harvest and by crop rotation. Chemical control can be attained with repeated application of protectant fungicides.

GUMMY STEM BLIGHT/BLACK ROT. On fruit, this disease caused by *Didymella bryoniae* (anamorph *Phoma cucurbitacearum* (Fr.:Fr.) Sacc.) is known as black rot whereas the foliage disease is known as gummy stem blight. Small, water-soaked spots develop on watermelon fruit, enlarge, and exude gummy material (Fig. 4). As the symptoms develop, fruiting bodies of the fungus may appear as black specks on the lesions. In the tropics, fruit infection can occur through blossom scars and begin to decay inner fruit tissue, with no visible symptoms on the surface of the watermelon. The decay eventually will progress to the stem end and reach the surface of the fruit (Sitterly and Keinath, 1996b).

Gummy stem blight can affect most above ground parts of the watermelon plant (Sitterly and Keinath, 1996b). Tan to dark brown spots occur on leaves and may rapidly enlarge as a dark brown, water-soaked lesion until the entire leaf is blighted. Symptoms on petioles and stems first appear as water-soaked spots that develop into elongated, tan lesions. A brown, gummy exudate often is produced on the surface of the lesions. Older cankers appear corky and cracked with small fruiting bodies, usually pycnidia, that appear as black specks. Stems can be girdled and plants killed by crown lesions.

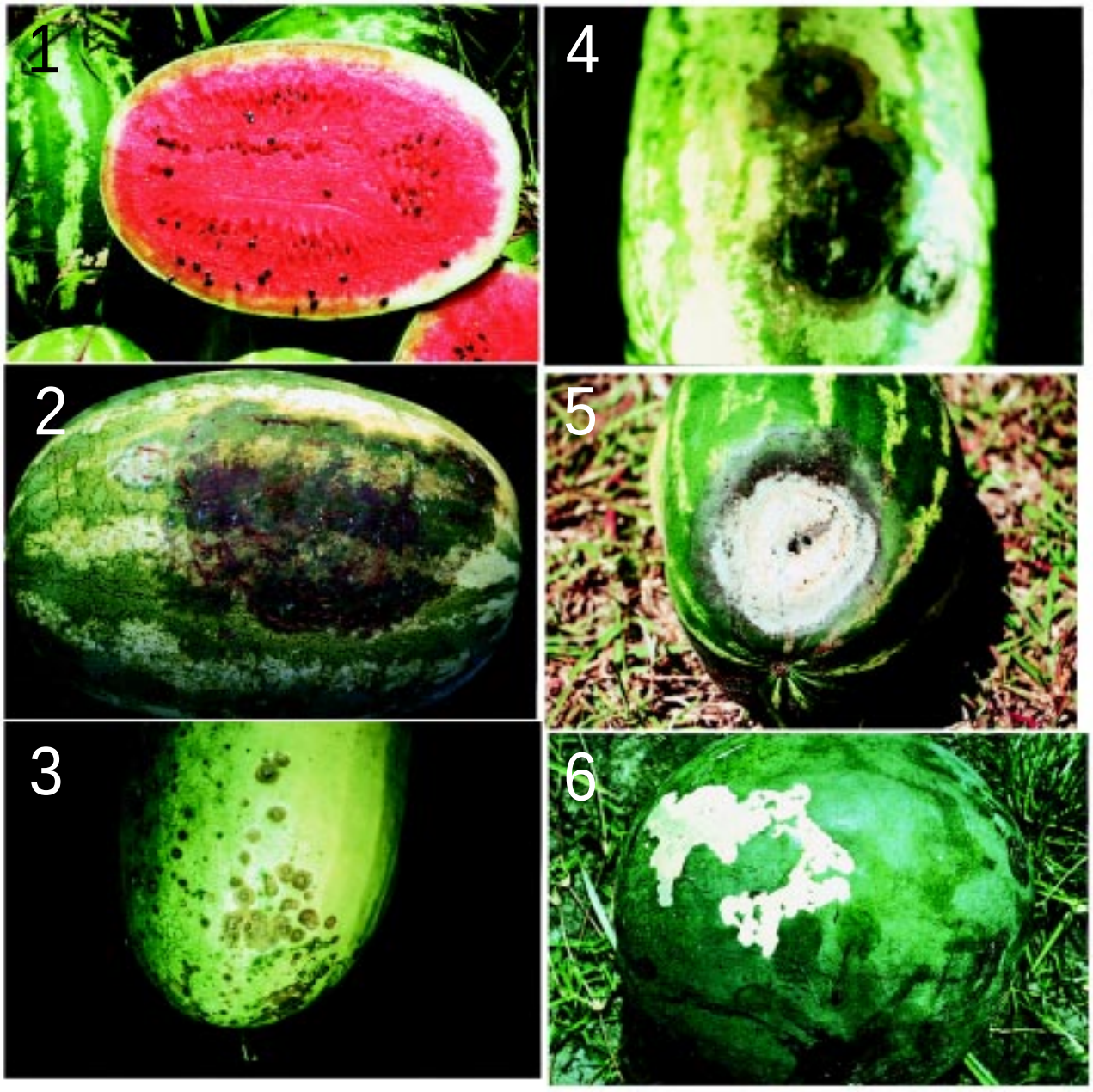
Black rot symptoms on watermelon fruit can develop in the field and result in significant losses to the grower. More frequently, severe decay of watermelon fruit from this disease occurs as a postharvest decay that develops during shipment to the marketplace, after infection occurs in the field. This can be especially severe when shipment occurs under warm, humid conditions. Black rot of watermelon was found to increase progressively from 45 to 75 °F (7 to 24 °C) (Luepschen, 1961).

In the southern U.S. and other subtropical and tropical areas of the world, gummy stem blight is one of the most important diseases of watermelon. *Didymella bryoniae* survives between seasons on crop debris in soil and on infected volunteer watermelon seedlings and wild cucurbits. Primary infection sources for a watermelon field are contaminated seed, infected transplants, and airborne spores. Airborne ascospores were detected throughout the year in Florida with the peak occurring in June–July (Schenck, 1968). Ascospores appeared to be most important in the distant spread (field to field) of the pathogen; whereas, pycnidiospores, which are spread by splashing rain, were more involved in the local plant to plant spread within a field. The pathogen often persists through adverse weather as perithecia on the crowns of watermelon plants. Warm, humid conditions favor the development of gummy stem blight and most rapid disease development occurs with frequent rains (Schenck, 1969). Free moisture on leaves for at least 1 h is required for infection and further continuous leaf wetness is needed for lesion expansion.

No commercially acceptable watermelon varieties with resistance to gummy stem blight are available (Sitterly and Keinath, 1996b). Controls for gummy stem blight should include cultural practices to reduce the primary infection as much as possible, such as using seed that have tested negative for *D. bryoniae*, and using a minimum of 2-year rotation cycles between cucurbit crops. Practices that minimize leaf wetness in the transplant house and field should be used when possible, such as drip rather than overhead irrigation. Satisfactory chemical control can be obtained by regular applications of protectant fungicides (Keinath, 1995).

PHYTOPHTHORA FRUIT ROT. Symptoms begin as a water-soaked, often depressed, spot (Fig. 5). Frequently, the area of the fruit in contact with the moist ground is affected first, but symptoms also can develop on the upper surface, following rain or overhead irrigation which provides splashing water to disperse the pathogen. In older lesions, a mass of white mycelium that contains sporangia may develop. Infected fruit can decay rapidly and collapse. Fruit decay may continue after harvest.

Phytophthora fruit rot is caused by *Phytoph-*



Figs. 1–6. Bacterial rind necrosis, bacterial fruit blotch, anthracnose, gummy stem blight/black rot, phytophthora fruit rot, and rindworm damage.

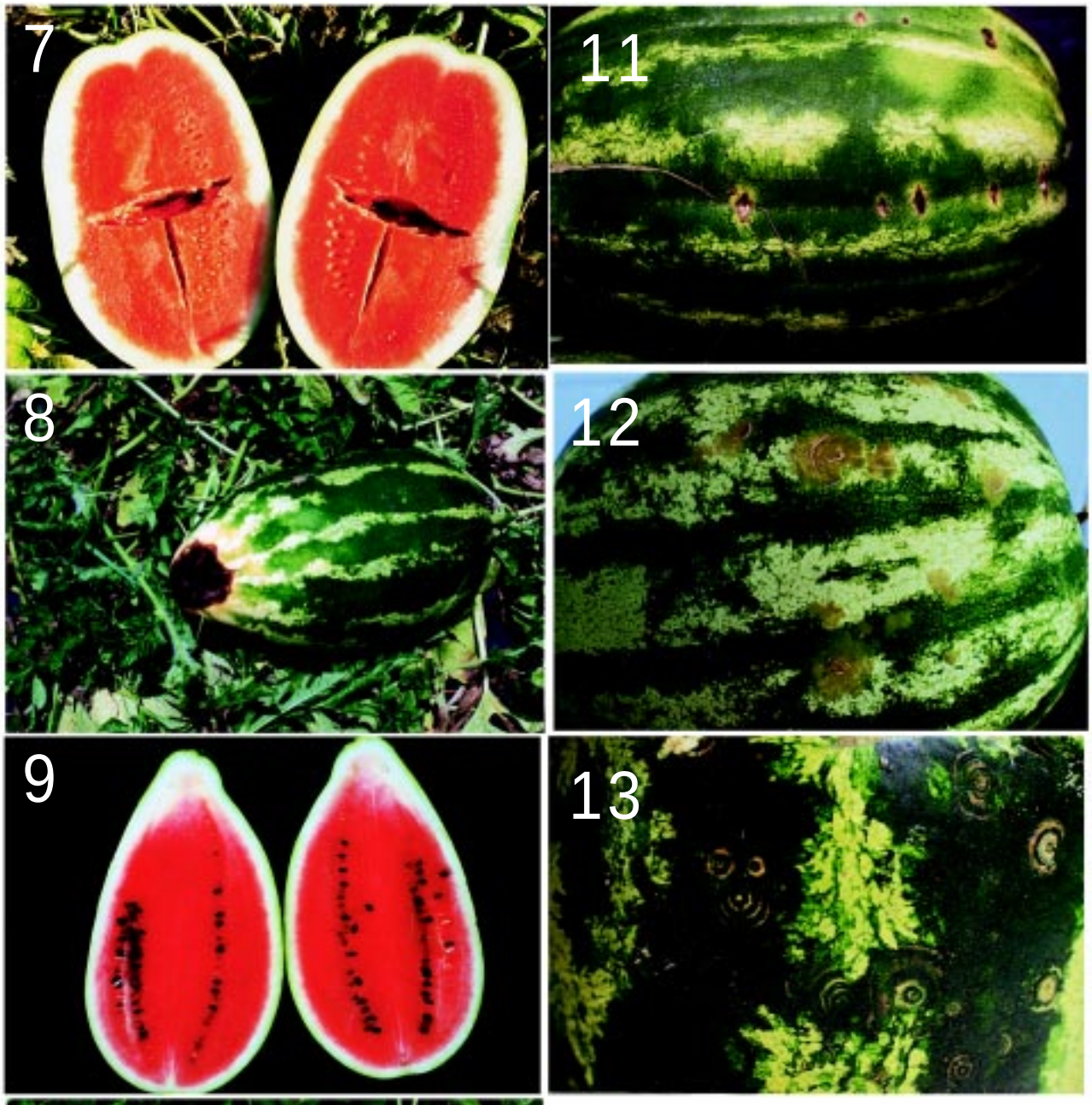
thora capsici and other *Phytophthora* sp. A wide range of vegetable crops, including all cucurbits, are susceptible to this fruit rot. All parts of the plant can be attacked, producing water-soaked lesions on leaves, stem lesions, and dieback of shoot tips in watermelon (McGovern et al., 1993). Commonly, crown rot causes entire plant wilting and death (Roberts and McGovern, 1998).

The fungus survives for at least 2 years in the soil (McGrath, 1996). Spores of *P. capsici* are spread by wind and water, in infected transplants, and through contaminated soil and equipment (McGovern et al., 1998). Surface moisture is required for motile zoospores of *P. capsici* to reach and invade the host. Therefore, phytophthora fruit rot is most severe in warm, wet weather and in low, water-logged areas of fields.

For disease control, rotation with nonhost crops is recommended, avoiding rotations with pepper, tomato, eggplant, cocoa, and macadamia (McGrath, 1996). Management of soil moisture by selecting well-drained fields, avoiding low-lying fields, and not overirrigating is an effective management strategy. Other good management practices to limit phytophthora fruit rot include the elimination of volunteer crop plants and weeds, roguing infected watermelon plants, and decontaminating equipment (McGovern et al., 1998). When combined with good cultural practices, fumigation and preventive fungicides may help suppress disease.

Insect-mediated damage

RINDWORM. Insect larvae that feed on watermelon rinds are generically referred to as rindworms regardless of their specific identities. Their feeding results in irregular trails over the rind surface (Fig. 6) which may limit salability when severe or when market conditions are poor. The most troublesome insect species that have been implicated with rindworm damage include the cabbage looper (*Trichoplusia ni* Hubner),



Figs.7–13. Hollowheart, blossom-end rot, bottleneck, sunburn, cross stitch, greasy spot, and target cluster.

tobacco budworm (*Heliothis virescens* Fabricius), and granulate cutworm (*Feltia subterranea* Fabricius). Other species occasionally may cause rind damage. Successful rindworm management has been achieved by natural control with native parasites early in the season and by chemical means later in the season (Adlerz, 1971, 1975).

Physiological disorders

HOLLOWHEART. This disorder is manifested by the separation of flesh within the fruit (Fig. 7) . It occurs in every production area but frequency and severity vary considerably among areas and seasons. Occasionally, however, loads are rejected because of hollowheart. As far as we are aware, there is no estimate of the economic impact of hollowheart although it is certain to be significant. It is very difficult to externally distinguish hollowheart fruit from fruit with an intact heart although the former tend to be somewhat asymmetrical. Noninvasive detection of hollowheart has been attempted by magnetic resonance imaging (MRI), mammography, fluoroscopy, ultrasound, and xerography (McCuistion et al., 1995). Following preliminary evaluation, fluoroscopy was found to be most

effective for distinguishing hollowheart. Operators were able to identify 20 of 23 melons with hollowheart using one view. Detection improved to 22 of 23 fruit with hollowheart when fruit were viewed from two directions. The authors suggest that commercial detection with fluoroscopy could be incorporated into packinghouse operations. The Japanese are already using nuclear magnetic resonance (NMR) in Tottori Prefecture for simultaneous determination of soluble solids and hollowheart (D. Maynard, personal observation).

Most watermelon experts agree that there is a higher incidence of hollowheart in crown-set fruit than in lateral-set fruit, that triploid (seedless) varieties have more hollowheart than diploid (seeded) varieties, and that there are differences among varieties in hollowheart susceptibility. Beyond that, there is much speculation as to the role of water and fertilizer management, temperature, pollination, and other factors.

Because of the difficulty in understanding the cause of hollowheart, little research has been reported. Results obtained in Japan (Kano, 1993) confirm that hollowheart occurs more frequently in crown-set (7th to 8th node) than in lateral-set (20th node) fruit. The crown-set fruit had fewer but larger cells than the lateral-set fruit; they also had larger intercellular spaces than the lateral-set fruit. Accordingly, cell separation occurs because the fewer, less compacted cells in the crown-set fruit cannot keep pace with the expansion rate of the rind. When growth rate was depressed by defoliation, the inner cell growth rate did keep pace with the expansion of the rind and hollowheart did not occur. These results agree with the notion held by some that watermelon plants that are "forced" are more likely to produce fruit with hollowheart (Rubatzky and Yamaguchi, 1997; Wien, 1997).

In preliminary experiments (Maynard, 1995), 'Jack of Hearts', a triploid variety, had 14% of its fruit affected by hollowheart whereas 'Sangria', a diploid variety, had 5% hollowheart fruit. The average cell separations in 'Jack of Hearts' fruit was 0.21 inch (0.5 cm) in diameter whereas the cell separation was only 0.02 inch (0.05 cm) in diameter in 'Sangria' fruit. Hollowheart in 'Jack of Hearts' was more frequent and severe when fertilized with 130N–25P–150K lb / acre (145N–29P–168K kg·ha^{-1}) than with various combinations of higher N and K rates. No relationship between fruit age (days after pollination) and hollowheart was established. However, earlier studies by Elmstrom et al., (1995) showed that there was a low incidence and severity of hollowheart in 'Jack of Hearts' and 'Crimson Sweet' watermelon at 5 and 12 d postpollination, but much greater hollowheart at 19, 26, and 33 d after pollination.

BLOSSOM-END ROT. Symptoms begin as a softening and shriveling of the blossom end of partially grown fruit and progress to a dark-brown, sunken, leathery lesion (Fig. 8). Varieties producing elongated fruit are more susceptible to blossom-end rot than those producing round fruit (Hammouda, 1987).

The incidence of the disorder is increased under low calcium regimes (Waters and Nettles, 1961). Recommendations for control of blossom-end rot include liming of soil according to soil test results and maintenance of an adequate, uniform soil moisture through irrigation management (Kucharek and Hopkins 1992).

BOTTLENECK. Constricted growth at the stem end of the fruit characterizes this disorder (Fig. 9). It is attributed to inadequate pollination either because of a low bee population or to poor conditions for bee activity such as cold, wet, or windy weather. The cause of the misshapen fruit can be verified by a cut fruit which will show an absence of seed at the stem end of the watermelon (Johnson, 1992).

SUNBURN. This disorder appears on the upper fruit surface as a gray area where the rind pigment has been destroyed (Fig. 10). High temperature and high light intensity together are essential for expression of sunburn in cucumber and pepper fruits (Rabinowitch et al., 1986), but the precise cause of sunburn on watermelon fruit has not been determined. The average watermelon rind temperature of several varieties reached 107 °F (41.6 °C) in the sun and was 97 °F (36.1 °C) in the shade when air temperature was 89 °F (31.9 °C)(Maynard, 1991 unpublished). Sunburn damage may be avoided somewhat by selection of varieties with light colored rinds which seem to be less subject to sunburn than dark-rinded varieties. The best means of circumventing sunburn damage, however, is to provide conditions for good vine growth which shades the watermelon fruit.

Disorders of unknown origin

CROSS STITCH. This disorder appears as a series of 0.5 to 1-inch (1 to 2-cm) long necrotic wounds that are perpendicular to the longitudinal axis of the fruit (Fig. 11). It was observed in Indiana in the late 1980s and early 1990s and in Florida in 1990 where the name was coined (Latin, 1993).

GREASY SPOT. Relatively inconspicuous raised, circular, olive-green areas on the rind surface characterize this disorder first described in Florida (Fig. 12). Greasy spot occurs rarely and is thought not to be of economic concern (Latin, 1993).

TARGET CLUSTER. Distinctive target-like configurations occur on the rind surface, usually in clusters of three or more targets (Latin, 1993). It has been suggested, because of their unique appearance (Fig. 13), that the causal organism may be a ringspot virus, but this has not been confirmed. The disorder is not common but it has been observed in Florida and in Australia (DNM, personal observations).

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