Market Diseases of Tomatoes Peppers and Eggplants

Agriculture Handbook No. 28

Agricultural Research Service
UNITED STATES DEPARTMENT OF AGRICULTURE

PREFACE

This handbook is a revision of Agriculture Handbook No. 28, "Market Diseases of Tomatoes, Peppers, and Eggplants," by Glen B. Ramsey, the late James S. Wiant, and Lacy P. McColloch. This is one of a group pertaining to market diseases of fruits and vegetables. The publications are designed to aid in the recognition and identification of pathological conditions of economic importance affecting fruits and vegetables in the channels of marketing, to facilitate the market inspection of these food products, and to prevent losses from such conditions. Other publications in this group are—

MISCELLANEOUS PUBLICATIONS

98. Potatoes. Revised January 1949.
168. Apples, Pears, Quinces. Revised November 1951. [Out of print.]
228. Peaches, Plums, Cherries, and Other Stone Fruits. Revised February 1959. [Out of print.]
498. Citrus and Other Subtropical Fruits. June 1943. [Out of print.]

AGRICULTURE HANDBOOKS

155. Market Diseases of Beets, Chicory, Endive, Escarole, Globe Artichokes, Lettuce, Rhubarb, Spinach, and Sweetpotatoes. April 1959. [Out of print.]
184. Market Diseases of Cabbage, Cauliflower, Turnips, Cucumbers, Melons, and Related Crops. September 1961.
189. Market Diseases of Grapes and Other Small Fruits. November 1960. (Reprinted April 1966.)
303. Market Diseases of Asparagus, Onions, Beans, Peas, Carrots, Celery, and Related Vegetables. September 1966.

Market Diseases of Tomatoes, Peppers, and Eggplants

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MARKET DISEASES OF TOMATOES, PEPPERS, AND EGGPLANTS

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INTRODUCTION

NOTE: Illustrations of the market diseases have been inserted in the back. Insofar as practicable each plate presents diseases similar in appearance, so that differences may be discerned more easily.

The diseases of tomatoes, peppers, and eggplants on the market are definitely associated with the vigor of the plants and the extent of diseases present in the field, with weather conditions during the time the fruits were maturing and at the time they were harvested, and with handling conditions. Very few fungi or bacteria are able to penetrate the uninjured skin of the fruit, and yet there are serious losses each year resulting from infections by a number of these organisms. Minute injuries such as broken glandular hairs, skin checks, and sand scarring are common avenues for infection on apparently sound fruits. In addition, visible blemishes and injuries such as growth cracks, faulty blossom scars, shoulder scars, and mechanical injuries frequently permit infection and decay. The majority of rots, however, are the result of infection by fungi and bacteria present on the fruits when harvested.

In general, it is advisable to cool tomatoes by ventilation or refrigeration to remove the field heat soon after loading the cars or trucks. Prompt cooling is effective in preventing soft rots and other decays caused by the rapid development of certain pathogens, and it prevents excessive ripening during transit in some seasons. Tomatoes should not be chilled, however, because chilled fruits as well as those delayed in ripening are subject to extensive decay by the slow action of weak pathogens.

Nonparasitic, or physiological, diseases are disorders brought on by abnormal conditions of the plants or fruits and are not caused by pathogenic organisms. The symptoms of nonparasitic disorders and virus diseases of the fruit are usually apparent when they are packed. Although certain of these diseases may become more pronounced after shipping, in general the presence of these diseases on the market is the result of affected fruits not being culled at time of packing. Fruits with physiological disorders and those with virus diseases may be worthless because

[1] Retired December 30, 1965.

of the blemished appearance, but these diseases do not directly result in decay.

Provided the fruits were originally sound and of good quality, diseases of tomatoes, peppers, and eggplants on the market can be greatly reduced by proper handling to avoid mechanical injury, by prompt cooling to the desired temperature in transit, and by proper handling and ripening at the market.

TOMATOES

ALTERNARIA ROT

***Alternaria tenuis*, auct.**

Occurrence and Importance

Alternaria rot is a fungus disease of tomato fruits that may occur wherever tomatoes are grown. The fungus causing this rot is a weak pathogen that is usually unable to cause an active rot on sound, uninjured, green tomatoes. Its occurrence, therefore, depends upon the condition of the fruits.

On vine-ripened tomatoes the rot nearly always follows such injuries as growth cracks, blossom-end rot, or sunscald. Because vine-ripened tomatoes are subject to extensive cracking, alternaria rot may be a serious problem in the processing industry (pl. 1, A).

On tomatoes harvested mature green, a small percentage of fruits develop alternaria rot at faulty blossom scars (pl. 1, B), but generally tomatoes do not develop serious alternaria rot in the ripening room unless they have been weakened by chilling injury.

Symptoms

Alternaria rot varies considerably in appearance, depending upon the conditions under which it develops. In general, the decayed area is brown to black in outward appearance; it may or may not have a definite margin; it has a flattened or slightly sunken surface (pl. 10, E). The lesions are firm; and the rot extends into the flesh of the fruit, producing a dark-brown to black, dry, corelike mass of decayed tissues (pl. 1, C). Dark-gray mold is often found in the cavities of the decayed tissues. Dense, velvety olive-green or black spore masses of the pathogen frequently grow over affected surfaces. Under humid conditions a dark-gray surface mold may develop. Alternaria rot frequently develops from the stem scar in V-shaped lesions. At that early stage it is impossible to distinguish the disease from similar stages of phoma rot and pleospora rot. Later, however, as the lesions enlarge and extend toward the shoulders of the tomato, the absence of pimple-like fungus fruiting bodies on the decayed surface helps distinguish alternaria from the other two rots.

After injuries such as sunscald and blossom-end rot, alternaria rot develops rapidly in the dead tissues and the lesions are soon covered by velvety spore masses of the fungus. The decay spreads along the edges of growth cracks without forming definite margins and is usually accompanied by a scanty development of dark mold. When infection occurs through faulty blossom scars, the decay generally develops internally more than on the surface (pl. 1, C). The outward appearance of the spot is often pale brown instead of black.

Tomatoes that have been chilled too long are extremely subject to alternaria rot. (See Chilling Injury.) Typical symptoms of alternaria rot on chill-injured fruits are a ring of decay around the stem scars (pl. 10, F) and numerous lesions at skin breaks over the surface of the fruits (pl. 10, G). On green fruits, either at the time the tomatoes reach the market or shortly after they are placed in the ripening room, the spots are not so dark as is usually typical of alternaria rot. Lesions around the stem scars of chilled fruits are shallow, sunken, and grayish black. Lesions over the surface of the fruits usually have definite margins, may be slightly sunken, and are tannish brown. Both types of lesions gradually become darker.

Another phase of the *Alternaria* disease is a kind of superficial scarring that disfigures the fruits, but usually does not develop into active decay (pl. 10, H). Separate grayish-brown to black scars may occur over the surface of mature-green tomatoes, during transit or in the ripening room, if harvested from the fall crop when the pulp temperature is below 60° F.[2]

Fruits subjected to a week or more of fluctuating moisture and temperatures in the field may also develop extensive scars around the shoulder area, especially if the fruits are held for a week or more at 40° F. or lower after harvest.

Causal Factors

The causal fungus *Alternaria tenuis* is ever present in nature, living on weakened plants and on plant debris. It does not affect tomato plants, and it attacks the fruits only when they have been weakened by other agents. It can grow and produce spores on the stem-scar tissue without causing decay of the fruits. Many tomato fruits show incipient infections at the margin of the stem scar that usually remain quiescent unless the fruits are subjected to adverse and weakening conditions. This no doubt is why *Alternaria* is the prevailing cause of rot on chilled tomatoes. As mentioned above, alternaria rot frequently affects tomatoes showing sunscald, blossom-end rot, growth cracks, and faulty blossom scars.

Once alternaria infection has become established the rot progresses faster at 70° to 80° F. than at lower temperatures. The infection is not entirely prevented, however, by reducing the temperature in transit. Temperatures below 60° delay ripening but

[2] Unpublished data of R. F. Kasmire, Division of Agricultural Sciences, University of California, Davis, Calif.

permit the continued, slow development of alternaria rot at skin injuries and blemishes and on weakened tomatoes. The fungus is able to grow and cause decay even at 32° if the fruits have been held for a prolonged period at that temperature. Tomatoes harvested from weakened vines near the end of the season, especially during rainy weather, usually have more blemishes and are weaker than the average of harvested tomatoes. Therefore, they are more subject to alternaria rot.

Control

The control of alternaria rot is ordinarily not a problem for good quality tomatoes that have been properly handled and promptly ripened at 60° to 70° F. Fruits showing blemishes such as sunscald, blossom-end rot, and growth cracks should be sorted out at packing time. At the end of the season fruits from weakened vines should not be shipped long distances to market. Avoid harvesting the late-fall crop if the pulp temperature is below 60° or if the crop has been exposed to chilling temperatures in the field (see p. 14). It is particularly important that chilling injury and delay in ripening be avoided. (See p. 17 for discussion of recommended temperatures.)

(See *13, 81, 82, 88, 89.*)[3]

ANTHRACNOSE

***Colletotrichum coccodes* (Wallr.) Hughes**

Occurrence and Importance

Anthracnose occurs in most of the tomato-growing regions of the United States. It is primarily a disease of ripe tomatoes; therefore, it is most serious in regions growing fruit for canning and for the local market. The fungus filaments in the flesh are often responsible for the high mold count in canning tomatoes. It is seldom important in mature-green tomatoes, although occasionally the disease is found affecting fruits as they ripen during transit and in the ripening rooms at the receiving markets.

Symptoms

In the early stage, anthracnose lesions are small, circular, slightly sunken, water-soaked spots (pl. 1, G). Later they become darker and more depressed (pl. 1, H) or develop concentric ring markings. Numerous dark pustules, which are fruiting structures, develop through the surface of the lesions. Under moist conditions, they become covered with cream to salmon-pink masses of fungus spores. The older spots are about one-half inch in diameter. During transit and marketing, the lesions ordinarily do not penetrate the flesh of mature-green tomatoes very far unless secondary organisms also are present. On vine-ripened fruit the spots become large and decay penetrates deeply into the flesh.

[3] Italic numbers in parentheses refer to Literature Cited, p. 66.

Causal Factors

Anthracnose is caused by *Colletotrichum coccodes* (*C. phomoides*). The perfect stage of *Colletotrichum coccodes* is *Glomerella cingulata* (Ston.) Spauld. & Schrenk. These fungi live from season to season on plant debris in the soil and in and on the tomato seed. Warm, wet weather favors spread of the fungus and development of decay in the fruits. Most rapid decay occurs near 80° F., but fairly rapid development of the fungus takes place at a temperature as low as 65°. Inoculation tests indicate that infection even through wounds will not occur at temperatures below 45°. Both green and ripe tomatoes may become infected without evident wounds. The susceptibility to decay greatly increases as the fruits ripen. Inconspicuous anthracnose spots on green tomatoes often increase greatly in size as the fruits turn pink and then almost double in size overnight when full-red color is attained. This rapid development of anthracnose may occur in canning tomatoes and in shipments of "pinks" without refrigeration, but it is not usual in shipments of mature-green tomatoes. Chilling of green fruits stimulates development of anthracnose.

Control

Infected tomato-plant debris in the field is one of the chief sources of infection of newly planted crops. Crop rotation should reduce this source of infection. Recommendations of the State agricultural extension service or experiment station should be followed.

(See *36*, *60*, *66*, *76*, *91*, *105*, *132*, *139*.)

BACTERIAL CANKER

Corynebacterium michiganense **(E. F. Sm.) Jensen**

Occurrence and Importance

Bacterial canker is occasionally very destructive in many tomato-growing regions. It causes wilt and death of seedlings and cankers on larger plants. It also causes a very definite fruit spot that often greatly injures the market quality of green tomatoes and reduces the value of ripe fruits for canning. The spots on mature-green tomatoes ready for shipment are usually so conspicuous that most of the blemished fruits are graded out at the packing sheds. Occasionally, however, some fruits escape notice and reach the market.

Symptoms

Affected fruits on the market show symptoms that are so characteristic that diagnosis is easy. The spots on mature-green fruits are usually 1/16 to 1/8 inch in diameter and are definitely superficial (pl. 5, E). In the early stages the spots appear as white to light-brown slightly raised areas; as they enlarge the brown center becomes more prominent and is surrounded by a characteristic white halo that does not show water soaking (pl. 6, E). The center becomes dark brown as it ages and may show a rift or slight opening; but the smooth, circular outline of the spot is maintained and the white halo usually remains visible. Although the individual spots seldom reach a greater diameter than ⅛ inch on green fruit or ¼ inch on ripening fruit, several spots often coalesce to form large, rough areas. A soft rot never develops. The fruits are sometimes infected internally by the passage of the causal bacteria from the vascular system of the diseased plant into the vascular system of the fruit; in such instances there are no external signs of disease in tomatoes of marketable size.

Causal Factors

Bacterial canker is caused by *Corynebacterium michiganense*. Contaminated seedbed soil is the principal source of infection; seed infection plays a relatively minor role. In many instances, cankerlike lesions develop on the stems of infected plants during wet weather. Bacteria from these cankers may be spread to the young fruits and other parts of the plant. There is no evidence to show that bacterial canker spots change or that new ones develop during transit or on the market.

Control

Recommendations of the State agricultural extension service or experiment station should be followed to prevent bacterial canker in the growing crop.

(See *2*, *10*, *11*, *43*, *143*.)

BACTERIAL NECROSIS

Various species of bacteria

Occurrence and Importance

A dry, firm, tissue breakdown around the shoulder area of mature-green tomatoes has been noted in shipments from Texas, California, and Florida. Losses observed on the market varied. Some were not extensive, but others amounted to 20 percent of the load. The disease is probably related to weather and growing conditions and may therefore be expected to be sporadic in occurrence.

Symptoms

Externally, the affected tissues are confined to the shoulder area. They are sharply sunken, firm, and a brownish-gray color (pl. 5, F). The affected surface may change from green to brown, but the development of red pigment in the necrotic area is suppressed.

Internally, the diseased tissues are dry and brown. The discolored tissues are confined to the pericarp and septa, but masses of bacteria collect in the seed cavities.

Causal Factors

Nothing is known about the conditions that may permit bacterial necrosis to develop in the field.

No single species of bacteria can be considered to be the cause of necrosis. Twenty-one isolates were obtained from affected tissues, eight of which caused tissue darkening and collapse. Most of the isolates causing necrosis were placed in the genus *Aerobacter;* one isolate was identified as a species of *Xanthomonas.*

The symptoms are similar to certain extreme phases of internal browning, and the possibility exists that the bacteria may be secondary to the internal browning disorder.

Control measures have not been developed.

(See *4.*)

BACTERIAL SOFT ROT

***Erwinia carotovora* (L. R. Jones) Holland, and other bacteria**

Occurrence and Importance

Bacterial soft rot occurs wherever tomatoes are grown. Although at times it may be responsible for fruit losses in the field, it is primarily a decay of tomatoes during transit and ripening. Terminal inspection records taken on the New York City market have shown it to be the most prevalent decay in rail shipments. Green fruits are more susceptible to decay from bacterial soft rot than ripe fruits.

Symptoms

The first symptom of bacterial soft rot is the appearance of a slightly depressed, water-soaked spot on the fruit. The spot enlarges rapidly to affect much of the tomato, and the affected tissues soon become very soft and watery (pl. 3, G and H). Watersoaking is particularly prominent at the borders of the lesion. The causal bacteria dissolves the material that holds together the cells of the tomato tissues. This action accounts for the extreme softness of decayed tissues. The skin is easily broken in the more advanced lesions. Sound tomatoes frequently are infected by contact with decayed adjacent tomatoes. The decay penetrates deeply into the tomato fruit. Diseased tissues are sharply delimited from healthy tissues inside the fruit by their softness and water-soaked appearance. Decayed tissues usually have a foul odor caused by secondary saprophytic bacteria.

Bacterial soft rot may at times resemble rhizopus rot and sour rot. If, however, the tissues affected with rhizopus rot are gently pulled apart, a few coarse threads of the *Rhizopus* mold can usually be seen, and the decayed tissues tend to hold together, becoming "mushy" rather than "soupy" as in bacterial soft rot. The foul odor characteristic of bacterial soft rot is not associated with rhizopus rot.

Tomatoes affected with sour rot resemble those with bacterial soft rot chiefly because of the water-soaked appearance of the spots. In sour rot, however, the affected tissues of mature-green tomatoes remain firm until the tomatoes are almost completely decayed. As the name indicates, fruits decayed by this organism have a sour, lactic acid odor. Frequently, sour rot is followed by bacterial soft rot.

Causal Factors

Massey, in 1924, identified the bacterial pathogen that causes soft rot of tomatoes as *Erwinia aroideae* (Townsend) Holland. This organism is considered by some workers to be identical with the common soft rot bacterium *Erwinia carotovora* (L. R. Jones) Holland, which causes a soft rot of a wide variety of leafy and root-crop vegetables. The latter usage is here followed. There is a strong possibility that such organisms as *Bacillus subtilis* and *B. polymyxa,* commonly found in the soil and reported to be associated with potato soft rots, may also be responsible for tomato soft rots, particularly at high temperatures.

The soft rot bacteria are found in soil and plant debris wherever tomatoes are grown. They are spread by insects, by field operations, and by the blowing, washing, or splashing of contaminated soil. Tomatoes may be inoculated with the bacteria while on the vines or, more likely, during picking and packing operations. In initial infection the bacteria enter the fruit only through weakened tissues and breaks in the skin; so high-quality, unwounded fruits are the best preventive.

Undoubtedly most of the bacterial soft rot found on tomatoes upon arrival at the market and much of that which develops in the ripening room started in field or packinghouse injuries that were not visibly infected at the time of shipment. However, there may be some spread from badly decayed fruits to adjacent fruits during transit and in the ripening room.

Warm and humid or wet weather favors the growth and multiplication of the causal bacteria. The most favorable temperature range for development of this decay is between 75° and 85° F.; at these temperatures the entire fruit may rot in 3 to 10 days. Decay may be fairly rapid at somewhat lower temperatures. Below about 65°, however, development of decay is slowed perceptibly. There is practically no development at 45° or lower, but this is too low a temperature at which to hold mature-green tomatoes.

Control

Careful handling of tomatoes during picking and packing is essential to avoid skin cuts and punctures. Picking containers should be cleaned and disinfected at frequent intervals. Good sanitation practices around packinghouses are advisable. In general, tomatoes should be picked while dry and kept as dry as possible before shipment. If tomatoes are washed, running water should be used and they should be dried before being packed. Decay is retarded by cooling the fruits rapidly to a temperature between 55° and 65° F., and maintaining that range during transit.

(See *12, 28, 48, 93, 95, 161, 163.*)

BACTERIAL SPECK

Pseudomonas tomato **(Okabe) Altsttatt**

Bacterial speck is so named because of the very small lesions produced on the fruit and elsewhere on growing tomato plants. This disease is most often found on tomatoes in the Middle Atlantic and Gulf Coast States. It is seldom of great importance, but occasionally the mature-green fruits are so badly speckled that their market quality is reduced.

The bacterium causing this disease affects the leaves, stems, and petioles of the growing plants as well as the fruit. Specks on the fruit are superficial and 1/32 to 1/16 inch in diameter; the larger ones result from infections of the fruit when very young. Most of the spots are no larger than 1/32 inch in diameter. They are dark brown, slightly raised, and have definite margins (pl. 6, F). There is no white halo about these spots, as in bacterial canker, or feathered edges, as in bacterial spot. The very early stages of bacterial spot, however, are very similar to the medium-sized spots caused by the bacterial speck organism. None of these three types of bacterial spot on tomatoes cause decay of the fruit. They do not develop or change in transit, but are merely blemishes that were present at the time of shipment. They affect only the appearance of the fruit.

Wet weather with temperatures between 73° and 77° F. favors the spread of infection and development of bacterial specks on tomatoes in the field.

Recommendations of the State agricultural extension service or experiment station should be followed.

(See *11, 27.*)

BACTERIAL SPOT

Xanthomonas vesicatoria **(Doidge) Dowson**

Occurrence and Importance

Bacterial spot of tomatoes is found in the market on fruit from many of the Atlantic Coast, Gulf Coast, and Central States and sometimes on tomatoes shipped from Mexico. In some seasons this disease causes considerable damage to tomato plants in the field and blemishes the fruits so badly that they are unmarketable. Some fruits are affected by bacterial spot each year.

Symptoms

On young fruits the lesions appear as small, dark, slightly raised dots, sometimes with a narrow water-soaked border. The smallest lesions of bacterial spot are very similar to the larger lesions of bacterial speck. The small bacterial spot lesions can usually be distinguished from bacterial speck by their slightly water-soaked border and irregular margin. On mature-green fruits received on the market the spots appear as brownish-black, elevated scablike areas (pl. 5, A and B) 1/8 to 1/4 inch in diameter (pl. 6, C), with feathered or irregular margins. Spots initiated early in the season often reach a diameter of 1/4 inch by harvest-time. They are sunken and appear gray or bleached, owing to the dry paperlike condition of the affected skin, which is usually torn and ragged (pl. 6, D). Fruits showing this stage of bacterial spot may be confused with nailhead spot (see Nailhead Spot); nail-head spot fruits seldom reach the market, however. Regardless of size, these scablike spots remain superficial and never develop into a soft rot.

Causal Factors

The causal bacterium *Xanthomonas vesicatoria* is carried on contaminated seed harvested from diseased fruit, and it is believed that the organism may also remain alive in the soil from one season to the next. Moderately warm weather (74° to 77° F.) with frequent rain favors the spread of the bacteria in the field and the production of spots on the fruit. Young green fruits are readily infected, but ripe fruits resist infection. Although wounds apparently are not necessary for infection, it is believed that sometimes insect injuries play a part in causing infections. Most infections occur while the fruits are quite young; and by the time the fruits are large enough to market, the spots are conspicuous and are easily graded out in the packing shed. There is no measurable increase in the size of the spots during transit. Those found on the market were present at time of shipment.

Control

Recommendations of the State agricultural extension service or experiment station should be followed.

(See *11*, *27*, *38*.)

BLOSSOM-END ROT

Occurrence and Importance

Blossom-end rot is a physiological disease of tomato fruits that is found wherever tomatoes are grown. It may cause serious losses during certain seasons. The disease is of considerable importance annually in the Southeastern and Gulf Coast States. Blossom-end rot commonly is found on market tomatoes from many different producing areas, although usually in small amounts.

Symptoms

The first symptom of blossom-end rot is usually the appearance of a small, water-soaked spot at or near the blossom scar of green fruits. As the spot enlarges the affected tissues dry out and become light brown to dark brown (pls. 9, E, and 8, D). The lesion thus develops into a well-defined sunken spot, with the affected tissues collapsed and leathery. The skin remains unbroken, because it is the tissues beneath that have dried out and collapsed; and the darker colored strands of the vascular system can be seen radiating from the blossom scar. Blossom-end rot is not actually a decay, although it frequently is followed by alternaria rot, bacterial soft rot, or other rots.

Although fruits of any age may become affected, blossom-end rot usually appears on fruit when it is a third to half grown. The spots vary considerably in size and may affect as much as one-half of the fruit. On slightly affected fruit the only evidence of blossom-end rot may be a slight drying out and browning of a small, superficial, irregularly shaped area near or at the blossom scar. This type of symptom is frequently seen on the market.

Causal Factors

There has been considerable experimentation and speculation about the cause of blossom-end rot. It appears that blossom-end rot may be caused by calcium deficiency since it can be greatly reduced by applying heavy dosages of gypsum and calcium nitrate to the soil and by spraying the plants with calcium chloride solution.

Because of the physiological nature of the disorder, there is no spread from fruit to fruit during transit. Under certain conditions, however, blossom-end rot may become apparent during transit and on the market in tomatoes that showed no sign of the disorder at the time of shipment. Blossom-end rot lesions already present on fruits at time of shipment change little, if any, in size unless secondary organisms are present.

Control

Blossom-end rot must be controlled by cultural practices. Recommendations of the State agricultural extension service or experiment station should be followed.

(See *31, 39, 40, 80, 117, 135, 136*.)

BUCKEYE ROT
Phytophthora spp.

Occurrence and Importance

Buckeye rot, a fungus disease of tomato fruits, has been reported from most of the important tomato-growing regions of the United States. The disease also occurs in Cuba and Puerto

Rico. Apparently all species of tomatoes are susceptible, but certain varieties and breeding lines are more resistant than others. Although buckeye rot may cause extensive losses, generally it occurs sporadically.

The occurrence and seriousness of the disease depends upon weather conditions. Prolonged periods of warm, wet weather encourage its development, regardless of geographical location. Such conditions, however, are more prevalent in the Southeastern and South Central States than elsewhere in the United States.

Buckeye rot is generally confined to fruits that are near or in contact with the soil. It attacks green fruits at any stage of maturity as well as ripe fruits. When conditions are favorable for the development of the disease, it may cause serious losses both in the field and in tomatoes shipped to market.

Symptoms

On green fruits the rot is hard and at first brown to grayish green (pl. 2, C). As the rot enlarges it remains moderately firm. The margin of the advancing rot is irregular and indefinite and has a characteristic water-soaked appearance and grayish-green color. Occasionally the entire lesion appears watersoaked, but more often it is marked by one or more fairly broad, irregular zones of alternating shades of brown and grayish brown (pl. 2, D). The surface of the well-developed rot is usually smooth, and the skin is unbroken. The typical color is chocolate brown.

The rot on ripe fruits is usually watersoaked and lacks the dark-brown color and the zoning.

Ordinarily little or no surface mold is evident, but if decaying tomatoes are held in a humid atmosphere a white, fluffy mold that becomes matted and tough when wet appears on the surface of the rot. This condition occurs in the field during rainy weather on fruits in advanced stages of decay.

Under certain conditions it is difficult to distinguish buckeye rot from late blight. (See Late Blight Rot.) In early stages of buckeye rot, when the surface of the lesion is rough, lacks the zoning, and has a paler brown color than is typical, it resembles late blight. On the other hand, when late blight spots are smooth, watersoaked, and darker brown than typical, they resemble buckeye rot. Generally, however, the surface of late blight spots is rough, or when it is smooth the margin of the spots shows a slightly roughened surface of a tan or rusty-brown color typical of late blight. Zoning is very rare in late blight.

Soil rot may be confused with buckeye rot because of the zoned appearance of the decayed spots. The soil rot lesions, however, are usually smaller and have a fairly definite margin and flat surface. The zone bands are narrow and nearly symmetrical in form. The surface of the affected skin is often cracked. (See Soil Rot.)

Causal Factors

Buckeye rot is caused by several species of *Phytophthora.* In California it is attributed to *P. capsici* Leonian and *P. drechsleri* Tucker; in Colorado only *P. capsici* has been reported as the cause. Elsewhere the cause of buckeye rot has been attributed to *P. parasitica* Dast.

These species of *Phytophthora* are soil-inhabiting fungi that do not cause disease except in warm, wet weather. Two kinds of spores are formed. The primary spores, or sporangia, develop first and these in turn give rise to swarm, or swimming, spores. The sporangia are not formed unless the soil is wet and the temperature is above 65° F. At 70° they may be formed within 24 hours. The sporangia may remain alive for days. When they become wet they burst open and discharge large numbers of swimming spores that must have free water to remain alive. It is the swimming spores that infect the tomato fruits. Infection can occur through the unbroken skin. A temperature of 80° or above is ideal for infection and development of the rot. Visible signs of the disease may appear within 20 hours, and the rot develops rapidly.

Fruits showing small spots overlooked in packing or contaminated fruits that appear sound may develop extensive decay during transit or after reaching the market. The rot enlarges at any of the temperatures desirable for handling tomatoes, but development is much slower at 55° F. than at higher temperatures. The disease may spread from decayed fruits to other fruits in contact with them, in transit or in the ripening room, especially at temperatures of 70° or somewhat higher.

Control

Tomatoes should be carefully sorted at packing to eliminate those that show buckeye rot. Buckeye rot is primarily a field disease. Most of the tomatoes that develop the disease after harvest were infected in the field, but did not show signs of the disease at time of packing. No method has been developed to prevent decay of infected fruits. A 24-hour delay in packing tomatoes suspected of having incipient infection may permit the disease to become evident so the fruits can be discarded at packing.

(See *32, 70, 107, 131, 133, 146.*)

CHEMICAL INJURIES

Sulphur Dioxide

Injury to tomato fruits caused by exposure to sulfur dioxide gas has been observed on the markets in stock shipped from California and Florida. Although this type of injury has not been observed in recent years its possible recurrence warrants mention.

The tomatoes affected show a very pronounced sinking and drying out of the tissues at the stem scar. The gas enters the fruits through the stem scar or through wounds elsewhere on the surface. The cells immediately surrounding the openings are killed and lose their water rapidly by evaporation, which causes collapse of the affected area. In some tomatoes the stem scar is sunken ½ inch and the tissues immediately around the scar are bleached to a light greenish tan, while the rest of the tomato remains normal except where mechanical injury is evident. Wherever the cuticle and epidermis of the fruit show a wound, the surrounding tissues are sunken and bleached greenish tan like those around the stem end. The sunken stem scars dry rapidly and crack, thus opening the way for various decay-producing organisms such as *Alternaria* and *Rhizopus*. This injury is somewhat similar to that caused by nitrogen trichloride; however, the strong bleaching action of sulfur dioxide causing extensive grayish-green to light-tan discoloration of the affected areas is usually sufficiently characteristic to distinguish this injury from nitrogen trichloride injury.

The injury results from using sulfur dioxide gas, as with grapes in California, to reduce the amount of decay during transit and marketing. No experimental data are available, however, to show that sulfur dioxide is of value in reducing decay in tomatoes.

(See *118*.)

Nitrogen Trichloride

Nitrogen trichloride has been used in a limited way for fumigating tomatoes to reduce decay during transit and marketing and has occasionally caused some injury to the fruit. When this chemical is used improperly, sunken tan to brown areas are evident on the fruits where there are injuries or scars that allow the gas to penetrate into the inner tissues. Gas that penetrates the stem end of the fruit causes a sinking of the tissues and a band of light-brown tissue at the edges of the stem scar. This injury is similar to that caused by sulfur dioxide; however, the affected tissues are usually darker brown, less extensive in area, and not so deeply sunken.

CHILLING INJURY

Occurrence and Importance

Chilling injury may occur in the field while the fruits are on the vine, while they are in transit, or after they reach the market if held at low, but nonfreezing, temperatures for too long a time. Tomatoes from all localities are subject to chilling injury, but susceptibility of fruits differs within a variety as well as between varieties. Mature-green tomatoes are more sensitive to chilling injury than fruits that were partially ripened on the vine and classed as "pink" tomatoes.

In general, the conditions during the normal transit period from shipping point to market are not conducive to chilling injury. Yet, until less than a decade ago chilling injury caused serious losses in tomatoes reaching the market, especially during the late fall and winter months. Although some chilling injury continues to occur, the application of research findings has brought this serious problem under control.

Symptoms

Ordinarily, chilling injury cannot be detected at the time tomatoes are removed from cars or trucks with low temperatures. The injury usually does not become apparent until the tomatoes have been in the ripening room for 2 or 3 days or longer. Tomatoes show a range of symptoms and indications of chilling injury depending on the severity of the damage. Unfortunately, the most positive physiological symptoms do not develop until tomatoes have been chilled to death. Mature-green tomatoes that have been held near 32° F. for 30 days or longer develop a dull, lifeless, pickled appearance (pl. 10, D) and feel rubbery to the touch. The internal symptoms are a watery, but not mushy, appearance of the tissues and a slightly fermented odor.

Tomatoes that are seriously injured, yet not chilled to death, may show such symptoms in the ripening room as irregular ripening or brown seeds. These symptoms are not entirely reliable if considered independently, but are useful when used as an adjunct to other indications. For example, irregular ripening may be caused by other agents than low temperature. Seeds of tomatoes tend to turn brown if the fruits are held at 32° to 40° F. for 9 days or longer. Seeds that are uniformly dark or brown seem to be a reliable symptom of chilling injury, but nonchilled tomatoes often have speckled seeds.

Alternaria rot in a pattern around the stem scar (pl. 10, E and F) and in the form of numerous small lesions (pl. 10, G) over the surface of a high percentage of tomatoes in the ripening room usually follows chilling injury and indicates that the fruits have been chilled. (See Alternaria Rot.)

Tomatoes harvested from the fall crop when the pulp temperature is below 60° may develop dark scars over their surface (pl. 10, H), during transit or ripening, owing to the *Alternaria* fungus.

Causal Factors

The effects of low, but nonfreezing, temperatures on the physiological processes of mature-green tomatoes are not well understood. It appears, however, that the fruits are unable to carry on normal metabolism at temperatures below 50° F., and as a result of prolonged holding at low temperatures their tissues gradually become injured. Tomatoes may therefore be considered as having been chilled when their physiological processes have been so impaired that ripening does not take place when the fruits are returned to temperatures that are ordinarily favorable for ripening. Chilling injury is distinct from freezing injury and occurs when tomatoes are held for prolonged periods at 32° to 45°. Fruits that have been chilled are weakened and are more subject to decay by *Alternaria tenuis*, and to a lesser extent by *Cladosporium herbarum* and *Penicillium* sp. When fruits have been seriously chilled, a high percentage show alternaria rot.

Tests with freshly harvested mature-green tomatoes have shown that tomatoes may be held at temperatures of 32° to 40° F. for 3 to 5 days and still ripen satisfactorily with little or no increase in decay. Tomatoes held for 6 to 8 days will color satisfactorily but will show an increase in decay in proportion to the length of exposure. Tomatoes are definitely weakened by holding at these low temperatures for 9 to 12 days. Ripening is unsatisfactory and decay is extensive. Tomatoes are so weakened by holding at 32° to 40° for 17 to 21 days that the entire lot usually rots without ripening. Chilling injury develops more slowly at 45° than at 32° to 40°. Definite symptoms of chilling injury do not develop, however, in tomatoes held at 45° for as much as 15 days. The nature and extent of decay that develops while the tomatoes are in the ripening room indicate that they were weakened or injured. Holding tomatoes at 45° for more than 3 to 5 days should be avoided.

Tomatoes harvested after exposure to a week or longer in the field at temperatures below 50° F., and predominantly below 40°, will likely develop serious chilling injury (*82*).

Ordinarily, the field heat in mature-green tomatoes when loaded is sufficient to require several days to cool the shipment to desirable temperatures, even by refrigeration. If the car is moved under bunker icing, however, fruits in the bottom bunker position will cool rapidly and may arrive on the market with a pulp temperature of 36° to 40° F. Under normal conditions, if the transit period does not exceed 6 days, the tomatoes would be at such temperatures for only 2 to 4 days, and these temperatures alone are not sufficient to cause chilling injury. If, however, the transit period exceeds 10 days and the load moves under refrigeration, there is danger of chilling injury in tomatoes near the bottom bunker. If the tomatoes have been exposed to temperatures of 50° or below in the field for a week or more before harvesting or stored at 40° to 45° for a week before shipping, and if they are refrigerated in transit or if the car is iced and left on the track at the market, the fruits may be at low temperatures for a sufficient time to become chilled. Tomatoes in cars with bunker icing for prolonged transit periods, because of diversion from one prospective market to another, are likely to become chilled. Chilling injury is likely to occur in cars during periods of low outdoor temperatures, and it is obvious that under these temperatures heavy icing would be undesirable. Tomatoes moving to northern markets during the winter months may need heater service to protect them against chilling injury as well as freezing injury.

Control

The recommended transit temperature for mature-green tomatoes is 55° to 65° F. Although tomatoes can withstand low temperatures for 3 to 5 days without being injured, it is not advisable for the fruit temperature to be lower than 55° during transit. This recommendation is made because of the uncertainty of the previous treatment of the tomatoes and also the uncertainty of how the load may be handled before the tomatoes are ripened.

Avoid long delays at temperatures below 55° F. Use heater service during periods of low outside temperatures to protect the tomatoes against chilling injury.

To avoid dark scars over the surface of the fruits that are associated with low temperatures, avoid harvesting mature-green tomatoes until the pulp temperature is 60° F. or above.

Although more resistant than mature-green tomatoes, pink tomatoes are subject to chilling injury and should not be held at 40° F. or below. Holding and transit temperatures depend on stage of ripeness. If fruits need to ripen they can be held at 60°. If ripening is to be retarded they should be precooled to 50° and held at 45° to 50°.

(See *82, 88, 89, 111, 130.*)

CLADOSPORIUM ROT

Cladosporium herbarum **Fr.**

Occurrence and Importance

Cladosporium rot has occasionally caused heavy losses during transit and marketing of California tomatoes during November and December and of tomatoes imported from Mexico during January. The most serious losses have occurred during long transit periods and in the ripening rooms at the receiving markets if the tomatoes were very slow in ripening. Although the causal fungus is widely distributed and of common occurrence, the decay of tomatoes is ordinarily not commercially important. Cladosporium does not cause decay of tomatoes in the field, except on discarded fruits or when associated with other types of decay.

Symptoms

On green tomatoes the first visible signs of cladosporium rot are small light-tan spots in the skin without visible surface mold. As the spots enlarge they become slightly sunken and have dark-brown to black centers with margins of tan or light brown (pl. 4, E). Most cladosporium rot lesions on fruits arriving at eastern markets range from ⅛ to ⅜ inch in diameter. In delayed shipments and in ripening rooms the spots may reach a diameter of ½ inch or more; they are shiny black and smooth at the center and have a narrow, light-brown border. Under humid conditions spots of this size often develop a fine, granular, dark-green, spore-bearing mold on their surface. The decayed tissues are usually limited to the outer wall of the fruit, but occasionally they may extend to the seed cavity. The affected tissues are moderately firm to slightly spongy, and moderate-sized lesions can be readily lifted out of the fruit with the point of a knife.

Causal Factors

Cladosporium rot of tomatoes is caused by *Cladosporium herbarum*. This fungus commonly occurs on plant debris and in the surface soil, but it is considered to be a weak pathogen. During foggy or rainy weather when the temperature is moderately low some incipient infection of the fruits may occur before harvest. In general, however, infection and decay develop after harvest during abnormally long transit or holding periods at temperatures near 40° F. Under such conditions tomatoes become weakened by chilling injury and are susceptible to cladosporium rot. The fungus appears to penetrate the weakened tissues without the aid of a skin break. Several lesions usually occur over the surface of a fruit, but they develop very slowly. Although the optimum temperature for the growth of the fungus on nutrient agar is about 70° F., cladosporium rot is not a problem on tomatoes except at low temperatures. Cladosporium is quite tolerant of temperatures of 32° to 40°, and in time it can induce decay in mature-green tomatoes held at those temperatures.

Control

The use of fans in conjunction with bunker icing, mechanically refrigerated cars and trucks, and accelerated transit schedules have done much to prevent over-refrigeration and resultant cladosporium rot. General precautions against cladosporium rot are to ship high-quality tomatoes free of field chilling injury under protective services that will provide temperatures ranging from 55° to 65° F.

(See *124*.)

CLOUDY SPOT

Cloudy spot is a blemish of tomato fruits. It is often found in small amounts on the market on fruits shipped from many tomato-growing regions, but serious loss seldom occurs because of this trouble.

On green fruits this blemish is characterized by cream-colored spots, which change to yellowish spots on ripening fruits. The spots are without definite margins and are found just underneath the skin of affected fruits (pl. 5, G and H). These light areas are made up of silvery-white cells that have a spongy texture. No decay or further discoloration of the tissues takes place after harvest. Most spots range from ⅛ to ¼ inch in diameter, but sometimes several spots coalesce and form large, irregular blotches that reduce the market value of the tomatoes.

It is believed that cloudy spot is caused by the feeding punctures of certain species of *Pentatomids* known as stinkbugs. Only certain plants in a field may show fruits affected by cloudy spot.

Usually this disease is not serious enough to warrant spraying to control the stinkbugs. Tomatoes blemished enough to affect their market value should not be packed for shipment.

(See *27, 156.*)

EARLY BLIGHT ROT

***Alternaria solani* (Ell. & G. Martin) Sor.**

Occurrence and Importance

Early blight occurs to some extent in most tomato-growing regions. It is most serious on tomatoes grown in the New England, Atlantic, and Central States, and it is usually of minor importance in the Pacific Coast States.

The early blight fungus causes a spotting of the fruits that is seen more often in the field than on the market, but it is sometimes found on vine-ripened tomatoes harvested for canning, especially late in the season.

Even though this blight is not important on mature-green tomatoes, it is included here so that it can be clearly distinguished from the more important alternaria rot that is caused by *Alternaria tenuis* (see Alternaria Rot).

Symptoms

Typical early blight spots are dark brown, leathery, and somewhat sunken. They are usually less than an inch in diameter and show concentric markings (pl. 4, D). The dark, dry decay usually does not extend more than ½ inch deep.

Early blight spots nearly always occur at the stem end of the fruit. Very often the stem becomes infected and the disease spreads to the fruit. In other fruits the lesion develops from the stem scar, but some spots develop on the shoulder apart from the stem attachment. Early blight spots have been observed on fruits of all stages of maturity on weakened plants.

Causal Factors

Early blight fruit spotting is worse in seasons of prolonged or heavy rainfall. Under such conditions the plants are weakened through the destruction of the foliage by leaf-spotting fungi and through poor aeration of the roots in waterlogged soil. The early blight spots are found on the smaller, weaker tomatoes in the cluster, but if the entire plant has been weakened, spotting may occur on many green fruits on the plant.

It is not uncommon to find *Alternaria tenuis* invading the early blight spots and causing a secondary rot in the field.

Control

Recommendations of the State agricultural extension service or experiment station should be followed.

(See *27*, *81*.)

FREEZING INJURY

Periodically, tomatoes of the late-fall crop and those grown during the winter months are injured before harvest by subfreezing temperatures. Commercial loads of tomatoes moving or standing without heater service during periods of low outside temperatures may also develop freezing injury.

Freezing ordinarily occurs when tomatoes are cooled sufficiently for their tissues to reach a temperature of 30° to 31° F. The average freezing point of ripe and green fruits has been found to be about 30.5°. The lowest point determined at which freezing first occurs was 29.7°, and the highest temperature was 31.1°. Freezing injury does not occur unless ice is formed in the tissues. It is possible to undercool tomatoes (as well as other vegetables and fruits) for short periods at temperatures several degrees below their actual freezing point without producing ice crystals or causing freezing injury if they are not disturbed. Such conditions seldom occur under commercial practice.

The chief symptom of severe freezing injury is a glassy or water-soaked appearance of the fruits. The border between uninjured tissue and frozen tissue may be quite distinct, particularly on green fruits (pl. 10, A).

Fruits frozen throughout collapse when held at ripening-room temperatures, and those with serious localized freezing injury retain the water-soaked appearance of the spots (pl. 10, B). Fruits showing either type of injury decay rapidly.

Tomatoes subjected to freezing temperatures in the field develop a wide range of symptoms. The extent of injury depends upon the vine cover and duration of subfreezing temperatures. Watersoaked areas are readily recognized as freezing injury, but many fruits less damaged may show no evidence of injury immediately after the freeze. In such fruits the frozen and normal tissues are intermingled; and when the ice crystals melt, the injured tissues tend to recover because of the presence of normal tissues. If the damage is fairly extensive the affected tissues become dry, bleached, and sunken. Sunlight hastens moisture loss and collapse. If bright sunlight follows the freeze, some damage is evident within 24 hours. If damage is less extensive, fruits gradually develop a pebbly surface over the affected area (pl. 10, C). This symptom may develop in 1 to 4 days. A slight to moderate yellowish color over the affected area is the best indication of freezing injury too slight to be accompanied by tissue collapse.

(See *83*, *128*, *159*.)

FRUIT TUMOR

Fruit tumor, also known as waxy blister, is the cause of a serious blemish that has been observed periodically on the market for the last 25 years. This physiological disorder is characterized by waxlike tumors on the surface of mature-green tomatoes. The most serious blemishes of this type occur in California tomatoes shipped during the fall and winter months. These white to cream-colored irregular blisters (pl. 8, A and B) usually range from ⅛ to ¼ inch in diameter and frequently are more than ⅛ inch high (pl. 9, A). While the tomatoes are still green these blemishes remain smooth and turgid; as they ripen, the affected tissues change from light brown to dark brown and eventually become depressed and cracked. The tissues of the tumors are made up of elongated cells in and underneath the epidermis.

Some tumors occur on tomatoes in the field, but most develop after the fruit is packed and while it is in transit. The disorder appears to be worse on fruits lacking maturity.

Rubbing the fruits while they are still green will cause tumors to develop. The slight mechanical damage apparently liberates growth hormones that incite increased cell activity and tumors. The minimum and maximum temperatures for tumors to develop are 70° and 96° F., respectively. At 91.4° the first signs of tumors may be seen within 21 hours. Ordinarily, fruit tumor does not cause consistent losses, but if it becomes a problem, fruit tumor may be controlled by careful picking and packing, and ripening at 65° F.

(See *124, 148.*)

FUSARIUM ROT

Fusarium **spp.**

Occurrence and Importance

Fusarium rot occurs on tomatoes from all producing areas of the country. It is most destructive on ripe tomatoes in the field, and it may cause serious losses in the canning crop. The *Fusarium* fungus is a weak pathogen and ordinarily does not cause much loss in tomatoes harvested mature green. Only occasionally is the rot found on tomatoes in the ripening room (pl. 4, A). During 1965, however, several shipments of tomatoes developed extensive fusarium rot a few days after they were unloaded (pl. 4, B). Losses up to 40 percent were recorded. It is believed that the tomatoes were handled in some way that weakened them, but the predisposing factors are unknown.

Symptoms

On vine-ripened fruits fusarium rot causes a slight water soaking, sinking, softening, and wrinkling of the affected tissues. There may be one or more lesions per fruit. The early appearance in the center of the lesion of a slightly raised, whitish or pinkish mold mass is the most important diagnostic characteristic of this decay. Rapid progression of the decay results in complete destruction of the fruit within a few days.

The symptoms usually found in the ripening room on tomatoes harvested at the mature-green stage differ somewhat from those on vine-ripened fruits. The decayed area appears somewhat water soaked. It gradually becomes slightly sunken, but it is quite firm unless invaded by bacteria. A tuft of cottony-white mold is usually present on the surface (Pl. 4, A). Internally the decay is a pale-brown, corelike mass that frequently can be lifted out in a single piece. White mold may be present in the cavities of the decayed tissues. The decay progresses at a moderate rate, and the lesions are usually from ¾ to 1½ inches in diameter.

Causal Factors

A number of different species of *Fusarium* can cause the decay. They are widely distributed in the soil and plant debris. Entrance to the fruit is through wounds, insect injuries, and lesions caused by other diseases. Although most of the decay seen on the market probably develops from incipient infections present at the time of shipment, tomatoes may become infected in the ripening room. Spread from diseased to healthy tomatoes is unlikely during transit. The causal fungi grow rapidly at temperatures optimum for tomato ripening and cause maximum decay at 75° F.

Specific control measures are lacking. Careful handling of fruits at all times and the culling out of tomatoes showing cracks, injuries, or disease lesions are recommended.

(See *105*.)

GHOST SPOT

***Botrytis cinerea* Fr.**

Occurrence and Importance

The blemish known as ghost spot is found occasionally on tomatoes on the market, but it is seldom serious enough to be of any consequence. This type of spot is found on tomatoes from many regions and on greenhouse fruits as well as field-grown fruits.

Symptoms

Ghost spot may occur anywhere on the surface of the fruit but is most commonly seen near the calyx end. It is characterized by a white circle, or ring, surrounding a green center (pl. 4, H). These rings of light-colored tissue are only skin deep and usually range from ⅛ to ¼ inch in diameter. The inner part of the circle is normal in color except there is often a very small brown speck in the center, which resembles an insect sting. As the fruits ripen, the circles gradually become yellow. There are no silvery-white pockets of spongy tissue underneath these spots as in cloudy spot. Decay has never been observed to develop in spots of this type. Affected fruits develop to normal size, shape, and background color, although the ghost spot lesion remains prominent.

Causal Factors

Ghost spot has been variously attributed to insect injury, lens action of sunlight on water droplets, and *Botrytis cinerea.* Investigations in this country and abroad have established the latter as the causal agent. Young, immature and mature-green fruits are the most susceptible. Botrytis spores germinating on the surface of the fruits under cool, humid conditions penetrate the epidermis creating a small brown necrotic speck. If the fruits are then exposed to warm, sunny weather the sporeling dies. Toxic materials released before its death, however, damage the surrounding cells sufficiently to create the halo characteristic of ghost spot.

Control

Recommendations of the State agricultural extension service or experiment station should be followed to prevent ghost spot in the growing crop.

(See *1, 24, 25, 33, 108, 151, 152.*)

GRAY MOLD ROT
Botrytis cinerea Fr.

Occurrence and Importance

Gray mold rot is a fungus decay commonly found in market tomatoes, but it is a problem only during cool, moist weather. The decay occurs regularly in late-fall shipments from California and during the winter on tomatoes shipped from Florida.

Symptoms

Lesions of gray mold rot on tomato fruits are conspicuous spots that occur singly or severally anywhere over the surface of the fruit, but they are most often found on the shoulder areas adjacent to the stem scar. The affected areas may have a somewhat water-soaked appearance. The chief symptom of gray mold rot, however, is the grayish-green (pl. 3, A) to grayish-brown (pl. 3, B) color of the decayed spots. The advancing edges of the lesions are regular and sharply defined.

The decay penetrates deeply and advances rapidly both on green and ripe fruits. Affected tissues are moderately soft and watery, although not so much so as those affected with rhizopus rot. They may have an earthy odor.

A grayish mold may develop where the lesions have cracked or it may develop sparingly over the surface at the center of the more advanced spots. Under humid conditions the mold develops a grayish or brownish-gray granular, velvety layer consisting of innumerable tiny clusters of spores borne on short, upraised filaments.

Causal Factors

Gray mold rot is caused by *Botrytis cinerea*, a fungus that is widely distributed and that attacks many kinds of fruits and vegetables. As indicated by the name of the rot, the causal fungus grows over the decayed areas in the form of a prominent gray mold. The fungus can enter the tomato fruit through the unbroken skin or through cuts and cracks. Frequently it infects the fruit at the edge of the stem scar. Growth of the pathogen and development of the decay are most rapid at about 75° to 80° F. Although development is less rapid at lower temperatures, the fungus continues to develop even at 32°. Tomatoes that have been exposed to moderately low temperatures for extended periods, either in the field or in transit, appear to be more susceptible to gray mold rot than those at normal temperatures. The disease may therefore be more prevalent at or following moderately low temperatures than at temperatures of 75° to 80°, which are optimum for the growth of the fungus.

Although the rot can spread from decayed to sound tomatoes in the package, it is probable that most of the decay seen on the market originated from infections occurring in the field or at the time of harvesting and packing.

Control

During the season when gray mold rot is a problem, special attention should be given to certain handling practices, including proper temperature and humidity.

Mature-green tomatoes should be shipped at 55° to 65° F. On arrival they should be unloaded and sorted promptly. Fruits showing color should be held at somewhat lower (85 percent) relative humidity than usual and all fruits should be ripened promptly at about 70°. These handling practices will aid in reducing gray mold rot which might otherwise develop from spores or incipient infection.

(See *162*.)

GROWTH CRACKS

Occurrence and Importance

Growth cracks affect tomatoes wherever grown. Losses are greater when tomatoes are ripened on the vine. On the market, growth cracks detract from the appearance of the fruit and predispose it to decay.

Symptoms

As indicated by the name, the chief symptom of this injury is rupture, or cracking, of the fruit about the stem end. Usually the cracks radiate from the stem scar and extend well down over the shoulders of the fruit (pl. 8, E); or they may develop in more or less circular fashion around the shoulder area, frequently with two or more cracks arranged concentrically (pl. 8, F). The cracks may be shallow or may extend deep into the flesh. Concentric cracks are usually shallower than radial cracks and are often healed over by corky tissue. Slight to moderate radial cracks often dry out and appear healed, but larger cracks are subject to decay. Alternaria rot commonly develops in growth cracks.

The causes of growth cracks are not well understood. The injury is usually considered to occur most often where tomato fruits have developed rapidly, particularly as they approached maturity. Periods of abundant rainfall and high temperatures favor rapid growth and seem to increase the damage from growth cracks. Fruit that has reached the ripening stage during dry weather may show considerable cracking if such conditions are followed by heavy rains and high temperatures.

Although the handling incident to picking and packing may reopen old cracks, new growth cracks do not develop during transit or on the market. Consequently, growth cracks found on fruit in the market were present at the time of shipment, but decays that develop in the cracks may not have been evident at the shipping point.

Control

During periods when cracking is serious tomatoes should not be allowed to remain on the vine longer than necessary. Tomatoes showing growth cracks should be sorted out during packing.

Crack-resistant varieties may eventually replace susceptible varieties.

(See *27*, *35*, *115*, *165*.)

HELMINTHOSPORIUM ROT

Helminthosporium carposaprum **Pollack**

Occurrence and Importance

Helminthosporium rot, a fungus disease of tomato fruits, has not been found on tomatoes grown in the United States, but occasionally it is found by inspectors at ports of entry on tomatoes imported from Mexico, Haiti, and British Guiana. The disease is included in this publication not because of its importance but as an aid to plant-quarantine inspectors in recognizing it.

Symptoms

Specimens of the rot intercepted at ports of entry are characterized by circular to irregular areas on the fruits, an inch or less in diameter, sometimes covered by a dense weft of dark-gray to almost black mycelium that completely obscures other characteristics of the rot spot.

Lesions produced on tomatoes by inoculation are flattened to slightly sunken and circular at first but becoming irregular in shape as the decay advances. Considerable variation is found in the surface coloring of the lesions, but in general it is distinct from other tomato rots. The tissue about the point of infection is usually dark or black. The most variable character is the extent of the cream-colored area surrounding the dark center. The cream color is nearly always present on infected fruits at temperatures of 60° to 80° F. This serves as a striking diagnostic character when present (pl. 4, F). An area of dark reddish brown often surrounds the cream-colored area. Occasionally there are lesions that advance slowly at 70° in which the cream-colored area never develops. Such lesions resemble those caused by *Alternaria tenuis* (see Alternaria Rot). At lower temperatures, especially at 50°, the spot is poorly defined; the fungus penetrates the tomato slowly, and at the end of a week the tissues are not completely killed but have a bronzy appearance. When this fruit is transferred to higher temperatures, the lesions gradually become like those developed at higher temperatures.

The rot consists of a firm, spongy mass of decayed tissues that extend deeply into the fruit but cause no offensive odor. When fruits with advanced decay are cut, dark-gray mycelium is evident through the necrotic tissue.

Causal Factors

The rot is caused by *Helminthosporium carposaprum.* This fungus is able to penetrate only through wounds. Once infection has occurred, the rot develops most rapidly at 70° to 80° F. Its development is greatly retarded at temperatures below 60°, but infection occurs at temperatures as low as 40°.

Control

The disease apparently does not occur extensively enough to warrant special control measures.

(See *87.*)

INTERNAL BROWNING COMPLEX

Occurrence and Importance

Similar appearing disorders of tomato fruits have been described by research workers under such names as vascular browning, brown wall, internal browning, gray wall, cloud, and blotchy ripening. Between 1949 and 1957 researchers attempted to show that such diseases as gray wall, internal browning, and blotchy ripening were distinct disorders. Between 1958 and 1965, however, because of the wide variety of causal factors reported and the inability to distinguish between the various disorders on a symptom basis, the tendency was to consider them as a group, with perhaps certain reservations.

It seems logical to place internal browning, brown wall, vascular browning, gray wall (thin wall) of California, gray wall of Florida, and blotchy ripening, which includes "cloud," in the same group.

The disorder listed as blotchy ripening appears to be partly influenced by localized high temperature injury (a kind of sunburn injury), which is not present in internal browning or the so-called gray wall of tomatoes in Florida. Blotchy ripening accompanied by necrotic internal tissues, however, is difficult to distinguish from the internal browning group.

Internal browning in some form probably occurs wherever tomatoes are grown and to some extent in most years. The disease was first observed in Florida in 1927 and has caused very heavy losses in certain years. Heavy losses have also been reported in other States along the East Coast, and in greenhouse tomatoes in Ohio. Internal browning is also a problem in Texas, Colorado, and California.

Apparently all commercial varieties of tomatoes are susceptible to the disease, but some varieties and breeding lines are much more resistant than others. There seems little doubt that internal browning is the most important physiological disease affecting tomato fruits.

Fruits become predisposed to the disease while they are on the vine. Ordinarily, the disease is readily visible at harvest and the only subsequent change is that affected tissues may become more sharply sunken. Occasionally, however, fruits may show little evidence of the disease until they are in the ripening room. During ripening, affected areas may become more discolored and sharply sunken. Diseased fruits may be either slightly or severely blemished. Although slightly affected areas on tomatoes harvested when mature green and ripened for fresh use may not cause rejection of the fruits, the blemishes will detract from their appearance and will cause wastage in their preparation. Severely blemished fruits are not marketable. The presence of the disease in tomatoes grown for canning and processing is much more serious, because all affected fruits must be discarded. The disease does not cause a rot, but the dead tissue may be invaded by rot-producing organisms that bring about secondary decay.

Symptoms

On green fruits, internal browning (including vascular browning, gray wall of Florida, and certain types of blotchy ripening) appears externally as a grayish-brown discoloration, because the brown internal tissues of the wall show through the healthy pale-green tissues and the skin. When the tissues are killed and the brown color extends to the surface, the external appearance is more brown than gray. On ripened fruits, the affected area around the lesions may fail to develop a good red color and may remain greenish or yellow. The surface color of the lesions on ripened fruits is very much like that on green fruits. The inner tissues of the fruit wall of the affected areas often appear somewhat bleached or whitish, especially on ripe fruits that are only moderately affected.

Although fruits usually are affected more on the sides and shoulders than elsewhere, in some severe cases the discolored tissues extend from the stem scar to the blossom end. Generally, only the outer wall is affected, but in some fruits the cross walls and even the basal part of the seed-bearing tissues are affected. On the surface the discolored areas appear as longitudinal, sunken streaks or as faint circular or irregular sunken areas (pl. 9, F and H). The margins of the affected areas are indistinct. In mild cases the surface is only slightly depressed; in severe cases it is sharply sunken, producing a lumpy or corrugated condition.

It is necessary to cut the fruits in cross section to examine thoroughly the internal symptoms of the disease. In milder forms of the disorder only the tissues around the vascular bundles are dead and brown, but generally a larger part of the surrounding tissues are affected and are brown, grayish brown, or somewhat bleached. If the fruits are cut and the seeds and pulp removed, it is evident that the vascular areas and the inner surface of the wall are affected first and that the disease then progresses toward the surface of the fruits. Severely affected fruits sometimes show definite ring patterns on the inner surface of the outside wall as well as irregular blotches and streaks with diffuse margins (pl. 9, G); or perhaps where tobacco mosaic virus is involved, the entire inner surface of the fruit wall may be affected and brown.

The fact that internal browning appears first in the area of the vascular bundles and on the inner surface of the tomato wall distinguishes it readily from other tomato diseases. Late blight, for example, always starts on the surface as a circular lesion and penetrates into the fruit. (See Late Blight Rot, p. 30.) Double-virus streak likewise develops on the surface, forming brown or dark-brown streaks or angular spots that may or may not be sunken. (See Virus Mottling, Double-Virus Streak, p. 49.)

Fruits may be affected at any size from ½ inch in diameter up. The disease may appear in a single fruit or a single cluster on a plant; or all fruits may be affected. Ordinarily, the first tomatoes showing internal browning are from the second or third picking. Affected plants often seem to recover from the disorder and produce normal fruits from later blooms.

In some fruits the symptoms of blotchy ripening can be readily included with those described above. Other fruits are not affected until they begin to ripen. In general, fruits with blotchy ripening have hard, green, waxy or glassy areas. These areas often occur around the stem end, but they may occur anywhere on the fruits. Affected areas are slow to lose the green color when the fruit ripens; they usually turn yellowish. Such areas may or may not be accompanied by necrotic tissues.

Causal Factors

Many causal factors have been reported from studies on vascular browning, blotchy ripening, gray wall, and internal browning. Among these are rank vine growth, deep shade, high humidity, high temperature, high transpiration rates and water deficits, high soil moisture, low temperature, excess fertility, and nutritional imbalance. In addition, certain more specific causes have been advanced, such as tobacco mosaic virus, abnormal sugar metabolism, and low nitrate/chloride ratios.

Perhaps because it is uncertain whether there is one or several disorders, no specific cause has been completely accepted. The preponderance of evidence, however, indicates that the internal browning complex is basically a physiological disease. Research has clearly shown that tobacco mosaic virus alone does not produce symptoms, but may increase the percentage of fruits affected and intensify the symptoms if the virus is present in plants also affected by other predisposing factors.

Research evidence is also being accumulated that shows that susceptibility to internal browning may be due to a genetic factor that allows some varieties to develop the disorder more readily than others when exposed to predisposing conditions.

Since 1958 the thesis has been gaining acceptance that the internal browning disorder follows an interference in the conversion of starch to sugar in the leaves and the inadequate transport of reducing sugars to the developing tomato fruits. Conditions that affect the physiology adversely also interfere with normal sugar metabolism. For example, it has been shown that the presence of tobacco mosaic virus or a deficiency of boron or potassium or shading the plants alters sugar metabolism. A disturbance in sugar metabolism is therefore viewed as a result of the same conditions that cause internal browning rather than its cause.

The disease may be viewed as a complex in which any one condition or a combination of certain conditions can cause the disorder. The loss of water from the fruits during periods of high transpiration, especially because of an imbalance between tops and roots, appears to be a basic factor, however, because many of the predisposing factors reported would be activated by a temporary water deficit.

Control

Recommendations of the State agricultural extension service or experiment station should be followed to reduce internal browning.

(See *5, 6, 8, 9, 20, 21, 22, 41, 44, 45, 50, 56, 67, 68, 97, 100, 101, 103, 125, 127, 129, 138, 141, 142, 157, 158.*)

LATE BLIGHT ROT

***Phytophthora infestans* (Mont.) DBy.**

Occurrence and Importance

Late blight may occur in any region where tomatoes are grown if weather conditions are favorable to the development of the fungus that causes the disease. In many years the conditions are favorable for late blight development in the tomato-producing areas of California, Florida, and the Rio Grande Valley of Texas during the fall, winter, or early-spring months. Late blight, which is usually sporadic in occurrence, has been widely distributed and has caused serious losses in many localities in recent years.

Symptoms

Typical late blight rot appears on the tomatoes as brown to rusty-tan blotches without definite margins; it usually develops in a circular pattern. The surface of the spots is pebbly to moderately roughened (pl. 2, A). Late blight rot is firm, but in the advanced stages other rot-producing organisms may cause it to become soft.

Although late blight rot often starts at the stem scar (pl. 2, B), it can occur anywhere on the tomato fruit. In early stages tissues affected by late blight, especially at the stem scar, appear water soaked or greasy. Such spots generally involve only one side of the fruit. In some fruits, however, the edge of the stem scar is uniformly infected and the decay spreads over the shoulder area. The surface of affected areas that start as water-soaked spots may remain smooth or may become irregular or roughened as the decay advances. When the surface of the advanced spot is smooth, the marginal area usually remains greenish and water soaked while the central area becomes brown. It is this phase of late blight rot that closely resembles buckeye rot. (See Buckeye Rot.) Late blight spots, however, are rarely zoned, and the margin of the spots nearly always shows a slightly roughened or pebbly surface of a rusty-tan color characteristic of late blight rot.

Other early stages of late blight rot appear as small, brown to rusty-tan, irregularly shaped spots intermingled with the green surface. Numerous small, slightly sunken areas soon develop on the surface of the affected area, producing a mild pebbly or moderately roughened surface. As these spots enlarge they may retain the roughened surface or eventually most of the surface may become smooth with only the margin remaining pebbly. The advanced spots vary considerably in color. They are usually darker brown than early-stage spots, and the margin is usually flecked with tan.

Causal Factors

The fungus *Phytophthora infestans* is the cause of late blight on tomatoes and potatoes. The amount of damage caused is directly related to the weather conditions during, and a few weeks before, harvest. Most serious losses occur during wet seasons when the nights are cool (50° to 60° F.) and the days only moderately warm (60° to 70°). Mean daily temperatures above 75° for 1 or 2 weeks check the disease. The spores of the fungus are killed within a few days during dry weather when the temperature reaches 80° or above.

When late blight is present on the plants in the field it causes a serious problem of unpredictable losses in tomatoes shipped to market. Tomatoes harvested from fields where late blight is prevalent may be infected and yet appear sound, because the lesions have not developed enough to be visible. It is impossible to sort out all of the infected fruits immediately after harvest, and fruits with this latent infection may develop late blight after being packed and shipped. The amount of late blight rot that develops in tomatoes during transit or after reaching the market varies with the severity of the leaf infection and the weather conditions just previous to harvesting the fruits. It has been found that nearly all infected tomatoes develop visible late blight spots in 4 to 7 days, and only a small percentage of fruits develop the disease after that time. The size of the late blight rot lesions found on tomatoes when they reach the market varies considerably, but lesions measuring 1½ to 2 inches in diameter have been found after 6 days in transit on tomatoes that appeared sound when shipped. Apparently there is very little spread of late blight rot from one fruit to another either in transit or in the ripening room.

Control

The prevention of late blight rot of the fruits depends upon the control of the disease on the plants in the field. Recommendations of the local agricultural experiment station should be followed. Variations in weather conditions in the different tomato-growing areas make it advisable for local investigators to determine the need for spraying, the kind of spray to be used, and the time of application.

When late blight is in the field, perhaps the most practical way to salvage the crop is to hold the tomatoes for 4 to 7 days after harvest to permit the late blight rot to develop so that the diseased fruits can be sorted out before being packed.

Late blight rot cannot be controlled by manipulating temperatures in transit. Its development is somewhat delayed by temperatures of 55° F. or lower, but the rot resumes development when the fruits are placed in the ripening room. Tomatoes showing the disease on arrival at the market should be sorted out, and only the fruits that seem sound should be placed in the ripening rooms. It is advisable to ripen the tomatoes at a temperature of 65° to 70°. Late blight rot develops rapidly at these temperatures, and frequent sorting to eliminate the diseased fruits will help in keeping out secondary decay such as bacterial soft rot.

(See *23*, *27*, *98*, *122*, *140*.)

MECHANICAL INJURIES

Market tomatoes may be affected by a wide variety of blemishes, scars, discolorations, and disfigurations resulting from mechanical injuries of various sorts. Dark-brown or black, sunken areas over the shoulders are one of the most common types of injury. These are often referred to as shoulder scars. The abrasive action produced by rubbing the shoulders of tomatoes against a rough board or the grading belt in the packing shed will cause this type of injury (pl. 8, G). A high percentage of similar shoulder injuries results when tomatoes that are in contact with rough unlined field boxes are hauled for some distance over unpaved roads. Similar injuries are produced elsewhere on the fruits. The abrasive action of sand adhering to the tomato may also be responsible for minute scratches in the skin that later develop into sunken pits or spots.

Unfortunately, the prevalence of mechanical injuries is usually not apparent when the tomatoes are packed. After they have been packed and shipped, however, the injured areas dry out, shrivel, and discolor, forming conspicuous pits and spots. Alternaria rot, phoma rot, and other decays frequently develop in such injuries.

Punctures and rim cuts are other forms of mechanical injuries that are usually followed by decay.

Bruising, in which the skin is not broken, is also a serious kind of mechanical injury. Bruising injury is cumulative and unless the fruits are pressed out of shape the damage is mostly obscured. Bruising injury may be caused by fixed pressure and by strong shocks or repeated impacts.

Green tomatoes under fixed pressure for 5 days or longer are permanently pressed out of shape and the internal tissues may be dry or stringy (pl. 9, C). The gelatinous tissues of tomatoes that are ripening when subjected to strong shocks or repeated impacts may be seriously damaged. The affected gel either becomes cloudy (pl. 9, D) or in more seriously affected fruits the gel tissues break down and spill out when the fruits are cut.

Handle tomatoes as carefully as possible at all times. Keep field boxes and picking containers free of sand and dirt. Avoid overfilling and careless stacking of all containers. Keep handling operations to a minimum during ripening. Avoid tossing and dropping tomatoes during sorting.

(See *46*, *78*, *79*, *84*, *96*, *102*.)

NAILHEAD SPOT

***Alternaria tomato* (Cke.) Brinkman**

Occurrence and Importance

Nailhead spot of tomatoes is a fine example of a disease that has been almost completely eliminated from domestic commercial shipments of mature-green tomatoes by the use of resistant varieties. This disease at one time caused serious blemishes on tomatoes grown in Florida and other Gulf Coast States, but in recent years it has been of no importance at the market, except in a few shipments of tomatoes received from Mexico.

Symptoms

Nailhead spot acquires its name from the characteristic small, circular, slightly sunken, tan to brown superficial spots produced on the fruit (pls. 5, C and 6, A). In the early stages the spots are tan and flat and can be identified by the time they reach a diameter ranging from $\frac{1}{16}$ to $\frac{1}{8}$ inch. The margins become more definite and dark brown to black as the spots enlarge, and the centers change to grayish brown and become definitely sunken (pls. 5, D, and 6, B). On tomatoes on the market nailhead spots range in size from $\frac{1}{16}$ to $\frac{3}{8}$ inch in diameter, the average being usually less than $\frac{1}{4}$ inch. In ripe and turning fruit the older lesions are sometimes surrounded by a halo of green tissue. Typical nailhead spots are shallow blemishes that seldom penetrate the wall of the fruit, but in some fruits these spots are invaded by *Alternaria tenuis* and *Cladosporium herbarum* and the resulting decay may extend into the seed cavity. This is likely to occur during long transit periods or in the ripening rooms when the tomatoes do not ripen promptly.

Nailhead spot can be distinguished from bacterial spot by the fact that the lesions are flat or sunken, that they continue to enlarge after packing, and that the enlarged areas are brown and have definite margins. Bacterial spot is usually raised and scablike, and the margin is narrow, brown, and feathered. Even when the bacterial spots have become sunken (pl. 6, D), they never develop the broad band of brown.

Causal Factors

The fungus (*Alternaria tomato*) that causes nailhead spot lives from season to season on plant debris, on volunteer plants in the field, on related crop plants, or on weeds. It produces spores on the tomato plants in the field that are readily disseminated by winds, water, insects, and field workers. The optimum temperature for spore germination is believed to be near 80° F. The minimum temperature for growth of the fungus on culture media is about 41°; the optimum is between 75° and 80°; and the maximum is about 93°. Under moderate temperature and moisture conditions the spores are able to germinate and infect uninjured immature-green fruit. Most infections take place in the field before the fruit reaches a diameter of 2 inches, but new infections may take place in fruits of any marketable size. The rate of development of nailhead spots is most rapid in green tomatoes; it decreases as the fruits mature. Nailhead spots that are present on tomatoes at shipping time may enlarge to some extent during transit. Experimental evidence indicates that spots $\frac{1}{16}$ inch in diameter and smaller usually almost double in size during 6 days in transit. The larger spots up to $\frac{1}{4}$ inch in diameter enlarge very little during the usual transit period unless they are invaded by secondary organisms.

Control

The best control of nailhead spot is obtained by planting resistant varieties. Tomatoes with nailhead spots should be discarded at the time of packing to avoid the development of secondary rots.

(See *110, 120, 154.*)

PHOMA ROT

Phoma destructiva **Plowr.**

Occurrence and Importance

Phoma rot is primarily a transit and market decay. Until about 1950, it was one of the most serious diseases of tomatoes after harvest. It occurred on tomatoes from many Eastern, Southern, and Gulf Coast States, but it was particularly widespread on the winter-grown southern crop. Serious losses frequently occurred in tomatoes from Florida and Mississippi and also in those shipped into the United States from islands of the West Indies. It was not found on tomatoes shipped from California or Mexico. At present (1966), the disease is rarely seen on the domestic crop during marketing.

Symptoms

The first symptom of phoma rot on tomato fruit is typically the appearance of one or several small, slightly sunken brownish spots at the edge of the stem scar (pl. 1, D). As the spots enlarge they extend somewhat radially over the shoulder of the tomato and become circular or elliptical. The centers of the spots turn brownish black and become dotted with tiny, raised brownish-black fruiting bodies, or pycnidia, of the causal fungus (pl. 1, E).

The lesions may also occur at growth cracks, mechanical injuries, or under certain conditions the fungus may enter through the unbroken skin. While they occur more often at the stem end, they may occur anywhere on the tomato. The decay advances more rapidly on turning and ripe fruits than on green ones. On riper fruits the spots are somewhat water soaked and are slightly sunken. By the time the lesions reach a diameter of ½ inch much of the sunken area is black, leathery, and firm and dotted with black pycnidia. The borders of the lesions are light brown and water soaked. In a humid atmosphere white, slimy spore masses may ooze out of the pycnidia. At times, the dry, crustlike center of the lesion cracks, and the grayish-white mycelium develops in the crevices.

Phoma rot penetrates deep into the pulp of the tomato. The affected internal tissues are brown or black and remain firm until the spots become very large or are infected with secondary organisms.

The symptoms of phoma rot are almost identical with those of pleospora rot. However, tomatoes reaching the market from California and Mexico, where pleospora rot is found chiefly, seldom if ever have phoma rot. The presence of the fungus fruiting bodies distinguishes phoma rot from alternaria rot, which it closely resembles otherwise.

The fungus causing phoma rot also produces a leaf spotting in the field.

Causal Factors

Phoma rot is caused by the fungus *Phoma destructiva.* The disease is most prevalent during seasons of moderate temperatures and high relative humidities. Great quantities of spores are exuded during wet weather from the fungus fruiting bodies (pycnidia) that develop on diseased leaves. These spores are washed onto the surface of the fruit by rain or spread by field operations when the leaves are wet with dew. Upon germination of the spores, the fungus enters the fruit through stem scars and through cracks or other wounds and injuries. The fungus may become established in the tissues of the stem scar of tomatoes that have no visible skin blemishes. Since it takes several days after inoculation before the lesion becomes visible, tomatoes inoculated in the field shortly before picking appear sound at the time of shipping. The fruits are also readily inoculated at the stem scar and elsewhere during picking and packing operations. Ordinarily, much or most of the phoma rot found in tomato shipments when they arrive on the market or in the ripening rooms was not visible when the tomatoes were packed and shipped. There is little or no spread of the rot from diseased to healthy tomatoes during transit or on the market.

Growth of the fungus and the development of decay can take place over a wide temperature range. The most favorable temperature for both is around 70° F., and there is little development of either below about 45°. When tomatoes are delayed in ripening at temperatures of 50° to 55°, however, the rot, although developing more slowly, may become more prevalent and thus may cause more loss than in tomatoes ripened promptly. Phoma rot may be worse on tomatoes harvested after extended periods of rain.

The decay progresses much more rapidly on ripe than on green fruits.

Control

The leaf-spot phase of the phoma disease must be controlled in the field if the fruit rot is to be prevented. The scarcity of the decay on tomatoes during marketing is probably due to the effective fungicides used in the field.

If phoma rot becomes a problem, the following precautionary measures will be useful. Tomatoes should not be picked while the plants are wet. They should be handled carefully during picking and packing. Particular care should be taken to avoid sand scarring and shoulder scarring by dirty or rough picking containers and field crates. Tomatoes showing cracks and injuries should not be packed, particularly during seasons when phoma rot is prevalent on the market. The tomatoes should be ripened promptly and chilling temperatures should be avoided.

For field control follow the recommendations of the State agricultural extension service or experiment station.

(See *61*, *104*, *105*, *114*, *144*.)

PHOMOPSIS ROT

Diaporthe phaseolorum **(Cke. & Ell.) Sacc. var. *sojae* (Lehman) Wehm.**

Occurrence and Importance

Phomopsis rot is a decay of minor importance that has been reported on tomatoes from New York, Hawaii, and a number of South Atlantic and Gulf Coast States. It has been reported also on tomatoes from Mexico, the West Indies, and Southern Rhodesia.

Symptoms

The first symptom of phomopsis rot is a somewhat soft and water-soaked lesion that may occur anywhere on the fruit. Usually, there is but a single lesion on a tomato. At moderate temperatures the lesions enlarge rather rapidly and eventually involve the entire fruit. As the lesions enlarge, the tissues in the center turn brown to dark brown and become very firm. Older lesions are firm over much of their area and nearly black at the center. At this stage the dark pimplelike pycnidia, or fruiting bodies, of the causal fungus break through the epidermis and become conspicuous at the center of the lesion. Further development of great masses of these bodies gives the lesion a rough, blackened appearance (pl. 4, C).

The lesions of phomopsis rot are at first smooth and very turgid. Later, the affected tissues wrinkle, shrivel, and crack.

The most prominent internal symptom is a firm spongy "core" that is composed of decayed flesh profusely permeated with the causal fungus. The core extends well into the tomato at the center of the lesion.

Causal Factors

As indicated by the name, the decay is caused by a species of *Phomopsis*. It is an imperfect (asexual) stage of the fungus *Diaporthe phaseolorum* var. *sojae* (the sexual stage). The leaves, stems and fruits are attacked in the field, but a blemish or wound is needed for infection after harvest. *Phomopsis* grows best and the decay advances most rapidly at temperatures of 80° to 85° F. Little growth and decay takes place at 40° to 45°.

Control

Elimination of fruits with scars, cuts, and other blemishes is important in the control of phomopsis rot. There would probably be little development of the decay at the recommended transit temperature range of 55° to 65°.

(See *18, 51, 77, 160.*)

PHYTOPHTHORA ROT

***Phytophthora* sp.**

A tomato fruit rot caused by a species of *Phytophthora* has recently been observed in the market on California- and Mexican-grown tomatoes. The rot occurs both on mature-green and ripening fruits.

The symptoms are distinct from those of late blight and buckeye rots. Affected areas are firm and water soaked and show little external or internal discoloration. In advanced stages the decayed surface develops an appressed, white mold (pl. 2, G), which is tough and may become wet. The decayed area finally becomes wrinkled.

Similar symptoms on Mexican tomatoes have been attributed to *P. mexicana*. It is debatable, however, whether this is a valid species. One investigator has found *P. mexicana* not sufficiently distinct from *P. erythroseptica* to stand as a separate species. Another investigator would make *P. mexicana* a synonym of *P. parisitica*. More work is needed to resolve this question.

The rot on the tomatoes in question has a striking resemblance to that on bell peppers caused by *P. capsici*. Since *P. capsici* has been reported on tomatoes in Colorado, it is entirely possible that the California and Mexican rot is due to the same species.

This is a field disease and the recommendations of the State agricultural extension service or experiment station should be followed for control.

(See *58, 69, 72, 73, 149.*)

PLEOSPORA ROT

***Pleospora lycopersici* El. & Em. Marchal**

Occurrence and Importance

Pleospora rot of tomatoes was first observed in California shipments in November 1919. Since that time the disease frequently has been observed on the eastern and middle-western markets. It has often caused serious loss in shipments of California tomatoes during November and December and in winter shipments from Mexico. So far, the disease has not been observed in tomatoes from other areas or in other seasons of the year. In some seasons carlots of tomatoes on the eastern markets have shown an average of 50 percent of the stock affected with pleospora rot. Usually, mature tomatoes of good quality ripen during transit or soon enough after arrival to escape great loss. But immature and poor-quality stock that ripens slowly and has to be held for more than a week for ripening is subject to very heavy decay by this disease.

Symptoms

Small, brown, oval spots at the edge of the stem scar or in mechanical injuries on the fruit are the first visible signs of pleospora rot. These lesions appear somewhat similar to phoma rot. But as they enlarge the brown color is retained and usually a small amount of gray or grayish-brown mycelium is noticeable, whereas in typical phoma rot lesions of the same size the affected tissues are black and there is no visible surface mold. Decayed areas ½ inch or more in diameter generally have black pimplelike perithecia at the center (pl. 1, F). The presence of these black fruiting bodies is one of the best diagnostic characters of this disease. In the absence of these fruiting bodies, it is impossible to determine without microscopic or cultural studies whether the decay is pleospora rot or alternaria rot. (See Alternaria Rot.) Under humid conditions there is usually a development of mold over the lesions. This grayish-brown mold is the *Stemphylium* stage *Pleospora*, and in mass it resembles *Alternaria*. Alternaria rot may occur wherever tomatoes are grown, whereas pleospora rot has been found only on tomatoes from California and Mexico. Consequently, if the black, pimplelike fruiting bodies are absent and the disease cannot be positively identified, it is usually advisable to call all decays of this nature alternaria rot.

Pleospora rot is inconspicuous in the tomatoes at packing time, but it is believed that the initial infection at the edge of the stem scar commonly occurs in the field, as tomatoes arriving on the market after a 5- to 8-day transit period often show large lesions.

Causal Factors

Pleospora rot is caused by the fungus *Pleospora lycopersici*, which apparently is prevalent in the tomato fields along the coastal areas of California and Mexico. No studies have been made of the development and spread of pleospora rot in the field, and consequently no data are at hand regarding its prevalence on tomato plants.

Pleospora rot develops slowly during the first 3 or 4 days in transit while the tomatoes are green, but as they ripen it progresses more rapidly. Spots ¼ to ¾ inch in diameter may develop during transit. Greenish-brown to brown, moderately firm, decayed areas involving a fourth of the fruit may develop in ripening tomatoes within 10 days after shipment.

Moisture seems to be one of the most important factors favoring infection of tomato fruits, as the seasons when this decay becomes most serious are those when foggy weather prevails or when rains keep the plants wet for several hours. The night temperatures at this time often drop to 55° to 60° F. or lower and the daytime temperatures are generally below 80°. Although tests have not been made, the decay may also be influenced by field chilling. The most rapid development of decay is between 65° and 70°. Temperatures higher than 80° are unfavorable to the growth of *Pleospora*. Experimental evidence indicates that the decay that develops during transit is the result of infections of the fruit that occur in the field and during harvesting and packing. No significant spread of the fungus to sound neighboring fruit in the package has been observed.

Control

Care should be exercised that tomatoes with infections at the stem scar are discarded when grading the fruit. During November and December in California, only the most mature and best fruit should be packed for shipment to the eastern markets. During January, Mexican tomatoes should also be graded closely for maturity and quality, as practically all shipments of these tomatoes are for distant markets.

(See *119*.)

PUFFINESS

Occurrence and Importance

Puffiness, or pockets, is a physiological trouble of tomato fruits. It may occur on tomatoes that are grown anywhere, but it is most prevalent in crops grown during the winter and early spring. It causes considerable loss in tomatoes grown in California, Florida, Mississippi, and Texas. The condition develops entirely in the field, usually at an early stage in the growth of the fruits. Seriously affected fruits are unmarketable and slightly affected fruits have reduced market quality.

Symptoms

Puffy tomatoes are easily detected because they feel hollow and are light in weight. The surface is usually flattened or sunken in the areas between the internal cross walls. Less severely affected fruits may be of normal shape. When puffy fruits are cut, large air spaces are found in the cavities normally occupied by the seed-bearing tissues (pl. 8, H). No decay or discoloration follows as a result of puffiness.

Causal Factors

Puffiness in tomatoes apparently is caused by environmental and nutritional conditions that interfere with normal pollination and thus affect the later development of the seed-bearing tissues. Normal pollination is adversely affected by temperatures above 100° F. and below 60°. Extremes such as excessive soil moisture or drought may also interfere with the normal development of the seed-bearing tissues. Unbalanced nutritional conditions created by heavy applications of nitrogen are thought to favor the development of puffiness.

Control

Because weather conditions are largely responsible for causing puffiness, definite control measures cannot be recommended. The adequate use of superphosphate together with moderate amounts of nitrogen may be of some value in reducing puffiness.

(See *34, 164, 165.*)

PYTHIUM ROT

***Pythium* sp.**

Pythium rot at times has caused serious losses in tomatoes grown along the east coast during hot, wet weather. The species of *Pythium* that causes the rot was not identified, and the distribution of the fungus in the United States is unknown.

A tomato fruit rot caused by *P. aphanidermatum* (Edson) Fitz. has been reported from Japan, South Africa, and Honduras. The tomato fruit rot in the United States occurs in the same area where *P. aphanidermatum* causes cottony leak of certain vegetables, and this species may be the cause of the tomato rot.

On mature-green tomatoes the rot appears as an extensive, firm water-soaked area (pl. 2, H) that may eventually involve the entire fruit.

No control has been developed for this rot. Careful sorting will help to hold down decay at the market, but decay may develop from infections that were invisible at time of packing.

RHIZOPUS ROT

***Rhizopus stolonifer* (Fr.) Ehr.**

Occurrence and Importance

Rhizopus rot is a fungus disease that may occur on tomatoes from any region. It may affect them when they are ripening or ripe either in transit or in the ripening room; it has been observed to cause serious losses in tomatoes shipped some distance after being prepackaged in consumer units. Occasionally it affects green fruits. Fruits affected with this decay are a total loss.

Symptoms

The symptoms of rhizopus rot are generally quite characteristic and distinct from those of other tomato-fruit rots. The lesions are usually large, with the affected areas at first somewhat distended. There is no discoloration, and the soft consistency of the rot gives it a water-soaked appearance as seen through the distended red skin (pl. 3, E). The rot develops deeply and rapidly and soon affects the entire fruit. If infection takes place through a slight wound, the skin may remain intact after decay is complete, without any external signs of mold. Frequently, however, the total decay causes the fruit to split open and to collapse into a wrinkled, soft, water-soaked, formless mass that is overrun by the causal fungus. The gray appearance of the coarse mold, because of the presence of a mixture of raised, immature white and mature black spore heads, is a characteristic symptom.

The presence of coarse mold filaments on the surface (pl. 3, F) or within the diseased tissues, along with external evidence of mold at skin breaks, serves to distinguish rhizopus rot from bacterial soft rot. Also, the diseased tissues tend to hold together, particularly in early stages, even though very soft, whereas the tissues affected by bacterial soft rot disintegrate completely. Fruits decayed with rhizopus rot have a slight fermented odor rather than the putrid odor that characterizes bacterial soft rot.

Causal Factors

The cause of rhizopus rot is *Rhizopus stolonifer*, a fungus that is responsible for serious decays of a number of different fruits and vegetables. Undoubtedly, other species of *Rhizopus* may at times be involved as causal agents.

The mycelium, or mold, of *Rhizopus* consists of coarse, whitish threads. As indicated above, the fungus fruiting bodies, in which innumerable spores are produced, are tiny, black, spherical bodies borne on raised stalks.

Rhizopus is widely distributed. It grows on plant debris and there are numerous spores of this fungus in the air. Infection usually occurs through wounds and other skin breaks. This fungus is also spread by the fruit fly (*Drosophila melanogaster*), which lays its eggs in the growth cracks on tomato fruits. It is commonly observed that the decay spreads during transit and in the ripening room by contact between decaying tomatoes and sound fruit that is ripening or ripe, even though no visible skin breaks are evident.

R. stolonifer grows best and the decay advances most rapidly at temperatures of about 75° to 80° F. The rate of development falls rapidly with a decrease in temperature to near 50°.

Control

Rhizopus rot can be reduced by careful handling to prevent mechanical injury, by eliminating fruits with serious growth cracks or other injuries at the time of grading, by cooling the load to about 55° to 65° F. as soon as possible in transit, and by sanitary practices in the packinghouses, in the canneries, and in the ripening room.

(See *3*, *16*.)

RING ROT

***Myrothecium* sp.**

Ring rot, formerly known as melanconium ring rot, is a fungus decay that has occasionally affected market tomatoes from Mexico and certain islands of the West Indies. It has also been reported from Texas and on greenhouse-grown tomatoes in Ohio and Wisconsin. The disease has not been a problem, however, since 1955.

The decay is evident as large, conspicuous, sharply defined, circular to oval, flattened lesions. One or several lesions may occur on a fruit. The affected tissues are brown to black. The decay penetrates deeply into the fruits, and the affected tissues can be removed readily as a single firm, spongy lump that separates easily from the healthy tissues. The most characteristic sign of the disease is the presence over the lesions of the spore masses of the causal fungus, *Myrothecium* sp., which develop in narrow concentric bands of alternating black spore masses and white mold (pl. 4, G).

Under moist conditions the spores germinate within a few hours at about 70° to 80° F. and are capable of infecting both green and ripe fruits through wounds. The most favorable temperature for growth of the pathogen and for development of the decay is 80°. The decay advances faster in ripe than in green fruits. Little advance occurs below a range of 35° to 40°. Although the decay is not known to spread from fruit to fruit during transit, individual lesions will continue to enlarge at temperatures above 35° to 40°.

(See *105*, *137*, *145*.)

SCLEROTIUM ROT

***Sclerotium rolfsii* Sacc.**

Occurrence and Importance

Sclerotium rot, or southern blight, is a fungus disease that affects a wide variety of vegetable and ornamental plants in the Southern States. Tomatoes shipped to market from these areas are affected occasionally with this decay, although it is seldom of much importance as a cause of transit and market losses.

Symptoms

The decayed areas of the fruit are somewhat yellowish and slightly sunken. When the spots reach a diameter of about ½ to ¾ inch the skin over them cracks. At moderate temperatures the decay advances rapidly, penetrates deeply, and may rot the entire fruit. Affected tissues are mushy soft and have a recognizable odor that resembles that of fresh, uncooked mushrooms. Under moist conditions the causal fungus grows out over the collapsing, decayed tissues in the form of an abundant, silky, white mold in which coarse strands are conspicuous. Characteristically, the mold spreads fanlike at the outer limits of growth. The resting bodies, or sclerotia, of the fungus soon develop abundantly in the mold, first appearing as small, whitish tufts that later become smooth, distinct, light-brown bodies about the size of a mustard or cabbage seed.

Causal Factors

The decay is caused by a soil-inhabiting fungus, *Sclerotium rolfsii*, which is widely distributed through the southern United States. It attacks tomato plants in the field with the result that the outer tissues of the stems decay at or near the soil line and the plants wilt and eventually die. The characteristic mold and sclerotia becomes conspicuous over the decaying stems.

Tomato fruits may become infected by contact with the ground or by becoming spattered with infested soil. The fungus is able to penetrate the skin, so wounds or cracks are not necessary for its entrance into the fruit. Practically all of this decay found on the market probably arises through infections that occurred in the field. The causal fungus is a high-temperature organism that grows most rapidly at about 85° to 95° F. It develops relatively slowly below 68°.

Control

Control of the decay in transit or on the market depends primarily upon control of the disease in the field. Careful sorting of the tomatoes at time of grading and packing to eliminate fruits showing initial spots of decay is also quite important.

(See *27*, *156*, *165*.)

SKIN CHECKS

Tomatoes are found occasionally on the market with numerous tiny cracks in the waxy coating (cuticle) over the skin. These commonly are referred to as skin checks or cuticle cracks. They frequently occur in great numbers over the shoulders as short segments of circles arranged concentrically around the stem scar. They may, however, occur anywhere on the fruit. The cracks are shallow, well healed, and somewhat darkened and for the most part are important only as blemishes that mar the appearance of the tomatoes. Where skin checks are numerous loss of moisture is rapid, and serious shriveling and discoloration follow.

Skin checks are visible at the time of packing, but they may become more conspicuous during transit and marketing because of drying out and discoloration.

The cause of skin checks is not well understood. They probably result from fluctuations of temperature and of moisture in the form of rainfall and dew.

(See *156, 166*.)

SOIL ROT

Rhizoctonia solani **Kuehn**

Occurrence and Importance

Soil rot is a fungus disease of tomato fruits that may occur anywhere tomatoes are grown, provided weather conditions are favorable. It is particularly important as a disease on tomatoes from Florida, Mississippi, California, Texas, Cuba, and Mexico that are harvested in a mature-green stage.

Symptoms

Soil rot develops on both green and ripe fruits. On green tomatoes in the field the first visible symptoms of soil rot are small, circular, brown spots on the lower half of the fruits. Spots ¼ of an inch in diameter and larger usually show very definite concentric-ring markings made up of alternating zones of light- and dark-brown tissues (pl. 2, E). As the fruits ripen and the decayed areas enlarge, the concentric zones become less evident and may even disappear. Affected tomatoes, therefore, do not always show zoned markings when they reach the market. On ripe fruits the spots are moderately firm and reddish brown, bordered by reddish-brown water-soaked areas.

Green tomatoes shipped in bulk from Florida have been observed to be affected with soil rot on the market. Such fruits showed spots that were ½ inch or more in diameter and had a uniform dark-brown color (pl. 2, F).

Typical spots are firm when less than an inch in diameter and have a definite margin and flattened surface. The skin over the affected areas is often split.

The definite margin, flattened surface, and—when present—the narrow, fairly regular concentric-zone markings, and the cracked skin on the decayed spots distinguish this disease readily from buckeye rot. (See Buckeye Rot.)

Causal Factors

Soil rot is caused by *Rhizoctonia solani*, which is a soil-inhabiting fungus that attacks not only the tomato but a wide variety of other plants as well. The amount of damage to tomatoes during any one season depends upon weather conditions. If the surface soil is wet and the plants are large enough to cover the ground and maintain a high degree of humidity underneath the vines, the fungus may attack any or all of the tomatoes that come in contact with the soil or that become spattered with soil during heavy rains. Tomatoes shipped from wet fields where the disease was abundant have been found to be as much as 60 percent infected with soil rot when cars were unloaded at the market.

Growth of the pathogen and development of the decay are rapid at temperatures favorable for good tomato ripening. The optimum temperature for the fungus and decay is 75° to 80° F. There is little decay by *Rhizoctonia* below about 50°.

Tests have shown that the fungus can enter tomato fruits through an apparently sound epidermis, although in about half the cases the organism enters through wounds or stem and blossom scars. Soil rot may develop during transit in tomatoes that were infected in the field but that showed no evidence of decay when shipped. Such lesions, developing during a 5-day transit period, may reach a diameter of ½ inch, and as many as 10 percent of the tomatoes may be affected. Small lesions, present at the time of shipment but overlooked during grading and packing, may attain a diameter of ¾ inch during a 5-day transit period and an inch or more during 7 days.

Control

The only way to control soil rot is to stake the tomato vines so that the fruit will not hang near or touch the soil. When this disease is extremely prevalent in the field it is sometimes advisable to stop packing tomatoes for a few days so the infected fruits may decay or develop spots large enough to be recognized; then the infected fruits can be sorted out before packing.

(See *42*, *105*, *121*, *156*.)

SOUR ROT

Geotrichum candidum **Pers. emend. Carmichael**

Occurrence and Importance

Sour rot (also known as oospora rot and watery rot) is a common decay of tomatoes. It occurs wherever tomatoes are grown and is equally important on mature-green and cannery tomatoes. It is regularly found affecting tomatoes in transit and on the market.

Symptoms

On green fruits sour rot lesions have a dull, greasy, water-soaked to bleached appearance (pl. 3, C). Although the decay may originate at cracks, cuts, or skin punctures, it frequently starts at the edge of the stem scar. From there, the decay may extend in a sector over the shoulder and down toward the blossom end of the tomato, or it may develop all around the stem scar and progress uniformly out over the shoulder area. The affected tissues remain fairly firm until the decay is quite advanced; they have a pickled appearance and a definite sour odor. Affected fruits either in transit or in the ripening rooms may show a dingy white, scumlike growth of the causal fungus in the stem scar or at points where the skin is broken.

Sour rot lesions on ripe or ripening fruits appear quite different from those on green fruits. The decay progresses rapidly, and the affected tissues are soft and water-soaked in appearance. The skin frequently cracks over the affected area and is usually filled with a white, cheesy, or scumlike development of the fungus (pl. 3, D). Bacterial soft rot often follows sour rot, hastening the decomposition of the fruit and altering considerably the fermented odor characteristic of sour rot.

Causal Factors

Sour rot is caused by the fungus *Geotrichum candidum*, which was formerly named *Oospora lactis* f. *parasitica*. This fungus is widely distributed in soils and on decaying fruits and vegetables. Most of the decay originates from infections in the field. Vinegar flies (*Drosophila melanogaster*) carry spores and mycelial fragments on their bodies from decaying fruits to growth cracks and wounds in healthy fruits, especially on canning stock when feeding and depositing eggs. Spores and fragments of the fungus thread also are spread by splashing rain and during picking and packing.

The fungus cannot enter through the unbroken skin; therefore, infections usually occur at stem scars, skin cracks, cuts and punctures of various sorts, and in green fruits that have been chilled for 7 to 10 days at 32° to 40° F.

The minimum temperature for germination of spores, growth of the fungus in culture, and infection of tomato fruits is approximately 36° F., the optimum is 86°, and the maximum is 101°

Control

Sour rot can be reduced by careful handling to avoid mechanical injury, eliminating tomatoes with growth cracks at time of packing, avoiding tomatoes that have been chilled in the field, and reducing the temperature of the load to 55° to 60° F. as soon as possible.

(See *13, 14, 15, 16, 17, 113, 116.*)

SUNSCALD

Sunscald may occur in any tomato-growing region if green fruits are suddenly exposed to the sun. The extent of the injury is more serious during periods of abnormally high temperatures. Fruits most subject to sunscald are those that have been exposed suddenly to the sun because of pruning, natural spreading of the plant caused by the load of fruits, or loss of foliage from leaf-spotting diseases or because the plants have been turned over by the picker. Sunscalded fruits are not marketable.

The injury is usually on the sides or upper half of the fruits, but it may occur wherever the rays of the sun strike most directly. The first symptom is a whitish, shiny, blistered area. The killed, bleached tissues gradually collapse (pl. 8, C), forming a slightly sunken area that may become pale yellowish and often wrinkled as the fruits ripen (pl. 9, B). The killed tissues encourage the development of secondary decay. Alternaria rot is the decay that is found most frequently following sunscald.

Recommendations of the State agricultural extension service or experiment station should be followed to reduce sunscald. (See *27*.)

VIRUS MOTTLING

Tomato plants and fruits are affected by a number of plant viruses and strains of these viruses. They may also be affected by more than one virus at the same time. This creates a complex problem requiring special knowledge and techniques for definite identification.

For practical purposes, only the virus diseases that have been found on the market on tomato fruits in past years are included in this handbook. They are tomato mosaic, double-virus streak, and spotted wilt. Because of the difficulty in making visual identification, they are grouped under the heading of "virus mottling," a term that has been used by the Federal-State inspection service for several years.

Because of improved production practices, such as direct seeding and sanitation, virus diseases are rarely found on tomatoes during marketing.

Tomato Mosaic

Marmor tabaci **Holmes**

Occurrence and Importance

Tomato mosaic is a virus disease that may be found in fields and greenhouses throughout the United States. It is one of the most common virus diseases of tomato plants, but its seriousness depends on the time of infection and the strain of the virus present. Fruits affected by certain strains of the virus show a superficial pattern of mottled color that is retained after they are ripened. This detracts from their appearance and perhaps causes more wastage through paring because the affected parts do not ripen thoroughly. The fruits are not necessarily a total loss, however, nor does the disease cause them to be more subject to decay. Mosaic causes very little loss in tomatoes for fresh use.

Symptoms

The symptoms of tomato mosaic on the fruits are distinct from those caused by other viruses. The difficulty of identification largely is caused by confusing marks and patterns on ripened tomatoes from other causes of blotchy or irregular ripening.

The mottled pattern caused by tomato mosaic is entirely superficial. Affected areas do not collapse, become sunken, or turn brown. The most typical diseased fruits in the field or on the market are green with cream-colored marks radiating from the stem scar over the shoulder area (pl. 7, A). These cream-colored marks may be continuous streaks, but more often they are broken up by small areas of darker green tissues. The latter condition causes the mottled or marbled appearance so typical of the disease. The margin between the green and cream-colored areas is usually distinct and not diffuse. When affected tomatoes ripen, the green areas become red, but the cream-colored areas turn yellow, thus the mottled appearance is maintained (pl. 7, B). Although shoulder markings are most typical, the mottled areas may extend over any part of the fruit, especially on fruits seriously affected. Mosaic fruits found on the market were affected when shipped, and the only change is that of color caused by the ripening of the fruits, as described above.

Causal Factors

The virus causing tomato mosaic is the same as that causing common mosaic of tobacco. It also affects peppers and eggplants. The ordinary green strain of this virus is the most abundant strain in the field. It causes various leaf symptoms on the plants, but unless infection occurs when the plants are small, no serious reduction in yield results. The green strain of mosaic does not affect tomato fruits.

Certain yellow strains of this virus cause a striking yellow mottling of leaves and may also mottle the stems. It is the yellow strains that cause the mottling of tomato fruits, and they may cause a serious reduction in yield.

The principal means of infecting tomato plants with the mosaic virus is by human transmission. Workmen get the virus on their hands and clothing when handling plants with mosaic, and they infect the healthy plants handled thereafter. The tobacco mosaic virus is commonly present in smoking tobacco, and smokers are likely to carry the virus on their hands. Aphids can transmit the virus from diseased to healthy tomato plants and possibly from virus-infected weeds that belong to the tomato family. They are not so important in spreading mosaic, however, as insect vectors are in the spread of some other viruses. The virus does not ordinarily live over winter on tomato debris in field soil, and in commercial practice, transmission of the virus by the seeds is of little or no importance.

Control

Recommendations of the State agricultural extension service or experiment station should be followed.

(See *27, 92.*)

Double-Virus Streak

Occurrence and Importance

Double-virus streak can occur in any region where tomatoes are grown. It is the most common form of streak on tomatoes. Tomato plants are severely damaged by this disease, and affected fruits are worthless. Although the disease is more common in the greenhouse than in the field, it has occasionally been found on field-grown tomatoes on the market.

Symptoms

Fruits very severely affected by double-virus streak are often rough and misshapen and have small, raised, irregularly shaped areas that may appear glassy or brown and corky. Such fruits would not reach the market. Specimens more typical of those found on the market have numerous small irregular or angular spots anywhere on the surface of the fruit; the spots are first glassy, then brown, and are usually slightly sunken (pl. 7, C and D). Independent spots often merge, forming larger irregular brown areas. Fruits on the market with double-virus streak were affected when shipped. Sinking and discoloration of affected areas increase with time and can be expected to change to some extent in transit.

Causal Factors

When plants already infected with the tomato mosaic virus *Marmor tabaci* later become infected with the so-called latent potato virus *Marmor dubium* var. *vulgare* Holmes, the disease produced by this combination is known as double-virus streak. The potato virus is present in nearly all the older varieties of potatoes grown in the United States, but it produces little or no visible mottling in the potato plant. When it is inoculated into healthy tomato plants, only a mild mottling of the leaves results and there are no fruit symptoms. The severity of the double-virus streak symptoms varies on tomatoes with the virulence of the strain of the potato virus with which they are infected. Some spotting of the leaves and streaking of the stems occur, however, with all strains.

The latent potato virus can be carried on the hands of workers who have cut potatoes before handling tomato plants. In the field it may be carried on the hands, clothing, and tools of those who have previously worked in a nearby field of potatoes, or it may be in volunteer potatoes in the tomato field. The potato virus is not spread by aphids. The combined viruses are very infectious and are spread easily by contact. If the disease develops early in the season it may be very destructive, especially in the greenhouse.

Control

Recommendations of the State agricultural extension service or experiment station should be followed.

(See *64*.)

Spotted Wilt

Lethum australiense var. *typicum* Holmes

Occurrence and Importance

The virus disease known as spotted wilt has caused serious losses in some sections of California and in Oregon. A very damaging strain of the virus there causes what is known as tip blight. This disease has been reported also from Texas. Spotted wilt occasionally occurs in the field in some of the East North Central and Atlantic States and at times has caused serious losses on tomatoes grown in greenhouses. Seriously affected fruits are worthless, and the disease is rarely seen on tomatoes shipped to market.

Symptoms

The fruits from older plants seriously affected by spotted wilt show numerous spots about ½ inch in diameter, with circular markings that appear as concentric bands of red and yellow on ripe fruit. The centers of these spots are sometimes raised, thus the spots have a roughened appearance. Such seriously affected fruits are not likely to be found on the market. Fruits with milder symptoms, such as may be found on the market, usually show a number of ring patterns over the surface of the fruits and are often accompanied by mottling caused by the lack of normal ripening in the surrounding tissues (pl. 7, E and F). The ring appearance is due to a slight sinking and shriveling of the tissues around the margin of the affected area, and this condition causes the center of the ring to appear raised. These affected areas may be practically normal in color or may appear bronzed or reddish brown. Diseased fruits found on the market were infected when shipped. Changes such as roughening of the surface and discoloration may occur during transit.

Causal Factors

Spotted wilt is caused by a virus that infects many ornamental plants, weeds, and vegetable crops, including peppers and eggplants. The virus is transmitted by flower and onion thrips. It can be transmitted mechanically, but the disease apparently does not occur to any extent unless the thrips are present in considerable numbers. The cause of serious outbreaks of the disease in tomato fields frequently has been traced to nearby plantings of infected dahlias and infected chickweed in the dahlia field.

In addition to those described, certain other virus diseases that affect tomatoes may cause mottling of the fruits during ripening (pl. 7, G and H).

Control

Recommendations of the State agricultural extension service or experiment station should be followed.

(See *26*, *27*, *112*, *143*.)

PEPPERS

ALTERNARIA ROT

***Alternaria tenuis*, auct.**

Occurrence and Importance

Alternaria tenuis is a weak pathogen that occurs in the field on dead and dying plant materials, and also on peppers that have injured areas such as blossom-end rot and sunscald. Peppers carry the spores on the surface of the pods at harvest, and at times, alternaria rot causes appreciable losses during transit and marketing. The disease may occur on peppers from any locality.

Symptoms

Typical alternaria lesions first appear as small, circular, grayish-green, water-soaked spots anywhere on the pod (pl. 12, C). The fungus appears to infect the tissues without a skin break. The margin is well defined and the spots are flattened or slightly sunken. Gradually the lesions enlarge, become tannish brown to muddy brown or may eventually become black. Advanced lesions are sharply sunken and usually have some loose gray mold over the surface (pl. 12, D). Under moist conditions the decayed surface may develop a dense growth of sporophores and spores that are olive green and velvety. Internally, old lesions are usually black and somewhat leathery and some gray mold may be present.

Causal Factors

Although alternaria rot may follow slight sunscald and blossom-end rot, these conditions occur infrequently in the commercial pack. Alternaria rot of this nature is unimportant on the market.

Under present commercial handling practices, however, alternaria rot may become a serious problem on pods with severe wounds if they are held for more than 2 weeks at moderate temperatures (50° to 55° F.) or on pods injured by chilling.

The *Alternaria* fungus grows and infects peppers slowly at 32° to 40° F. and the rot is usually not evident when the peppers are removed from low temperatures. Moderate to serious alternaria rot develops rapidly, however, at 65° to 70° on peppers that had previously been held at 32° for 15 days or longer or at 40° for 18 days or longer.

Control

Alternaria rot can be controlled by handling peppers carefully to avoid mechanical injuries, by providing a temperature range of 45° to 50° F. during transit or storage, and by avoiding delays and low temperatures during all phases of marketing. Peppers should not be held longer than 12 to 15 days from harvest. (See *85*.)

ANTHRACNOSE

Colletotrichum gloeosporioides **Penz.**

Occurrence and Importance

Anthracnose may occur on peppers from any region. On the market it seldom causes much loss unless the fruits are ripe or beginning to ripen. The common bell pepper that is marketed green rarely shows anthracnose except at wounds or following other diseases such as blossom-end rot. During some seasons anthracnose is responsible for serious losses of fruit in the field.

Symptoms

Anthracnose spots range up to an inch or more in diameter. They are small and water soaked at first; later they turn dirty green to greenish brown and become slightly sunken. The margins of the spots are definite and circular in outline. The spots become covered with raised black pustules, which are sometimes arranged in concentric rings (pl. 11, D). Under moist conditions these pustules exude creamy to pink spore masses. With one type of anthracnose (*Colletotrichum*) the spore masses become black as they age. This decay penetrates the pod walls and discolors the seed cavity and seeds.

Causal Factors

Several species of *Colletotrichum* have been reported as causes of anthracnose. In the United States *C. gloeosporioides* appears to be the only valid species. *C. piperatum* (Ell. & Ev.) Ell. & Ev. and *C. nigrum* Ell. & Hals. are presently considered synonyms of *C. gloeosporioides*. In Canada *C. coccodes* (Wallr.) Hughes is also reported as a cause of anthracnose. These fungi can invade both green and ripe pepper pods without wounds. *Colletotrichum* is a weak pathogen, however, that usually follows wounds or other diseases. These organisms usually cause most rapid decay in ripening fruit. Warm, humid or rainy weather favors the spread of the spores to the fruit in the field and the rapid development of decay. It is believed that early infections of fruit in the field, too small to be graded out at packing time, are responsible for the few anthracnose spots found on the market.

Control

Peppers shipped at temperatures ranging from 45° to 50° F. will show no appreciable development of anthracnose during transit. The disease, however, should be controlled in the field. Recommendations of the State agricultural extension service or experiment station should be followed.

(See *7, 55, 59, 134.*)

BACTERIAL SOFT ROT

***Erwinia carotovora* (L. R. Jones) Holland**

Occurrence and Importance

Bacterial soft rot is a common decay of peppers from all growing areas. During some seasons it is particularly prevalent in peppers from California, Texas, Florida, and Mexico.

Symptoms

Bacterial soft rot on peppers is characterized by water soaking and rapid softening of the tissues. Infection initiated at the stem end progresses rapidly through stem and calyx lobe tissues (pl. 11, E) into the pod. Under humid conditions and optimum temperatures (75° to 85° F.) the entire pod can be reduced to a soupy mass within 3 to 6 days after infection. Infections occurring in wounds on the pod tend to spread lengthwise on the fruit, macerating the underlying tissue and leaving a sunken, wrinkled, fragile skin covering that is easily ruptured (pl. 11, F). The foul odor that is associated with bacterial soft rot on most produce, however, is lacking on pepper fruits.

Causal Factors

Bacterial soft rot of peppers, *Erwinia carotovora* (L. R. Jones) Holland, is identical to the organism causing serious market losses in many vegetable crops. Peppers are inoculated by contact with contaminated soil and by picking and packing equipment. The organism is a wound parasite; it commonly enters by way of broken stems or other wounds incurred in the harvesting process. It is apt to be especially troublesome if peppers are harvested during warm, wet weather.

Control

Peppers should be handled carefully during picking and packing to eliminate pod wounds. Significant reduction of bacterial soft rot from stem wounds resulting from picking has been achieved in controlled tests by submerging inoculated fruits for 1½ minutes in water heated to 128° F. Hydrocooling the fruits following this treatment resulted in greater decay in inoculated fruit. It is important, however, to reduce the temperature of the pods by other means to 45° to 50°, as quickly as possible, and to maintain the fruits at this range in temperature throughout the marketing period.

(See *62, 63.*)

BACTERIAL SPOT

Xanthomonas vesicatoria **(Doidge) Dowson**

Bacterial spot on peppers is the same disease that affects tomatoes. (See Tomato, Bacterial Spot.) This disease may occur on peppers from any region, but on most markets it is more prevalent on peppers shipped from Florida and Texas. Typical spots are small, raised, brown to black scablike lesions. In time some of the oldest spots may crack and become grayish in the center.

Bacterial spot is a superficial disease that detracts from the appearance of peppers. The disease becomes seriously damaging when the spots are present in large numbers on the pods (pls. 12, F, and 14, A).

Bacterial spot must be controlled in the field. Recommendations of the State agricultural extension service or experiment station should be followed.

(See *37, 52, 57, 155.*)

BLOSSOM-END ROT

Blossom-end rot is a physiological disease that frequently occurs on peppers on the market. It is serious because it disfigures the fruit and opens the way for secondary bacterial and fungus invaders that cause extensive decay. Alternaria rot is the most common secondary decay that follows blossom-end rot, but in many peppers anthracnose (see Anthracnose) follows in this broken-down tissue.

Small, water-soaked spots on the lower half of the fruit near the blossom end are the first symptoms of this disease. As the spots enlarge they become light brown to brownish black and dry, so that the affected tissues are more or less leathery in texture (pl. 14, F). No soft rot is produced unless secondary organisms invade the diseased tissues; however, *Alternaria* or some other secondary organism is present on most of the peppers that show blossom-end rot on the market.

Blossom-end rot of peppers is similar to, if not actually the same as, the blossom-end rot of tomatoes. (See Tomato, Blossom-End Rot.) It is associated with calcium deficiency and can be greatly reduced by soil applications of gypsum.

Recommendations of the State agricultural extension service or experiment station should be followed for control of blossom-end rot.

(See *47, 53, 75, 99.*)

CHARCOAL ROT

***Macrophomina phaseoli* (Maubl.) Ashby**

Charcoal rot of peppers is seldom found on the market. This disease, however, causes damage to peppers in the fields of New Jersey, Texas, California, Georgia, and Kansas.

Pepper fruits affected with charcoal rot show very little external evidence of the disease except a shriveling of the epidermal tissues. The interiors of the pods are discolored, however, and a great number of small black specks, sclerotia, are visible within the tissues and on the seeds. The fungus will enter through wounds and become established within the tissues in 4 days. Affected fruits are completely decayed in about 2 weeks. No control measures are known.

(See *94*, *150*, *167*.)

CHILLING INJURY

Occurrence and Importance

Bell peppers are subject to chilling injury and should not be over-refrigerated or held beyond a normal marketing period. Chilling injury has been observed primarily in shipments during the late-fall and winter months, and during warm weather when mixed loads of vegetables, including peppers, were top iced. Although losses are not as serious as in past years, the problem continues to warrant attention.

Symptoms

Peppers that have been injured by low, but nonfreezing, temperatures may develop such physiological disorders as sheet pitting and surface scald or certain kinds of decay.

Sheet pitting develops on the most tender part of the pods. Slightly affected pods may develop only a few scattered pits, but those seriously affected may have one half or more of the surface covered in a sheet effect. Sheet pitting appears first as water-soaked spots, which may be scattered or close together. Affected areas are grayish green, and individual spots are flat or slightly sunken. En masse, affected areas have a dappled appearance (pls. 12, A and 14, C). Affected tissues lose moisture, sink irregularly (pl. 14, D), and often appear pebbly. The pattern of sheet pitting varies according to the spacing of the pits. Sheet pitting develops while the peppers are at 32° F.

Surface scald does not develop until after the peppers have been removed from low temperature. Affected pods are a dull grayish brown.

The calyxes of peppers are seriously injured by holding the pods at 32° to 40° F. When placed at higher temperatures the calyxes deteriorate rapidly and develop alternaria rot with visible mold (pl. 12, B).

Numerous lesions of alternaria rot over the surface of the pods are also an indication of chilling injury. (See Alternaria Rot.)

The occurrence of numerous lesions of gray mold rot that have developed without the aid of visible skin breaks indicates chilling injury perhaps of a less serious nature than that described for alternaria rot. (See Gray Mold Rot.)

Causal Factors

In tests, sheet pitting developed on pods held at 32° F. 4 days or longer. Sheet pitting has been observed in peppers that were in transit during periods of low outside temperatures, and during warm weather in mixed loads of vegetables that were top iced. Although sheet pitting is a symptom of chilling, its presence does not necessarily indicate serious chilling injury; that is, extensive decay does not always follow.

Surface scald, dying and moldy calyxes, and numerous lesions of alternaria rot follow serious injury, which occurs in 12 to 15 days at 32° F. and 18 days or longer at 40°.

Control

Chilling injury can be prevented by avoiding temperatures below 45° F. in transit, storage, and at the market. Peppers should not be held longer than 12 to 15 days from harvest. A holding temperature of 48° to 50° is best.

(See *85*.)

CLADOSPORIUM ROT

Cladosporium herbarum **Fr.**

Occurrence and Importance

The *Cladosporium* fungus is a weak pathogen, and although widespread, it does not cause serious losses in peppers except under special conditions.

Most of the cladosporium rot seen on the terminal markets has developed in California peppers shipped from the fall and early-winter crops. Peppers that become susceptible usually develop extensive decay.

Symptoms

The lesions of cladosporium rot develop slowly; they never become large; and from the early stages they have characteristics that set them apart from other rots.

Lesions no larger than the head of a pin are slightly depressed and a light-tan color. Although the fungus may enter through a wound, most of the numerous lesions on pods show no sign of a skin break. As the spots enlarge they become more sunken and the center is dark brown in contrast to a light-brown border. Finally the central area becomes jet black and shiny (pl. 12, E) and the border remains brown. The lesions are shallow and can be readily lifted out on the point of a knife. The decayed tissues are spongy and grayish brown. Lesions that have been developed for a long time eventually penetrate the pod wall and may cause some internal discoloration. Under moist conditions short spore-bearing stalks and spores form a dense, velvety, dark olive-green mass over the old lesions.

Causal Factors

Shipments that are unloaded after only 6 days en route usually show little evidence of infection. Peppers that are shipped under refrigeration and are delayed and those that have been refrigerated and held on track or held in storage at 36° to 40° F. for a week or more develop serious decay.

Damp, foggy weather in the field has previously been considered to be an important predisposing factor in cladosporium rot. Moist weather would cause an increase in the *Cladosporium* spore population, but the weakening effects of chilling injury appear to be the principal cause of pods becoming susceptible.

Control

Maintain peppers at 45° to 50° F. while in transit or storage. Avoid prolonged exposure to temperatures below 45° during marketing.

(See *123*.)

CLOUDY SPOT

Cloudy spot on peppers is the same kind of blemish as that on tomatoes and has the same general symptoms (pl. 14, B).

Cloudy spot does not occur as extensively, however, on peppers as on tomatoes. (See Tomato, Cloudy Spot.)

GRAY MOLD ROT

***Botrytis cinerea* Fr.**

Occurrence and Importance

Gray mold rot, also called botrytis rot, may occur on peppers from any source, but it has often been a sporadic problem on peppers shipped from California during the fall and winter. Although conditions are favorable for the spread of the fungus in the field during cool, moist weather, there is little decay after harvest if the pods are carefully handled and not over-refrigerated. Peppers that are over-refrigerated and delayed in reaching the market often develop extensive gray mold rot.

Symptoms

If pods are weakened by over-refrigeration, numerous lesions may develop anywhere over the surface without the aid of skin breaks. At temperatures of 40° to 45° F. gray mold rot starts as small, cream-colored specks with a tendency to first spread in a rhizoid pattern. These specks gradually develop into round water-soaked lesions (pl. 11, A). As the spots enlarge they develop a grayish to grayish-brown color (pl. 11, B and C).

Decayed tissues are wet, but relatively firm. Under humid conditions a mass of brownish-gray mold with a surface layer of granular spores of the same color are characteristic.

Causal Factors

Freshly harvested unwounded bell peppers are quite resistant to gray mold rot at 50° and 55° F. even at high relative humidity. Similar pods can be expected to become susceptible at 45° and to develop visible lesions in about 15 to 18 days. Pods become more susceptible at 40° than at 45°, but require about 18 to 21 days for lesions one-fourth inch in diameter or larger to appear.

Nonchilled peppers are readily infected by *Botrytis* through wounds in the pod or through the cut or broken stem end.

Chilling injury is cumulative and near-freezing temperatures in the field may predispose peppers to gray mold rot if the peppers are over-refrigerated after harvest.

Control

Gray mold rot can be controlled by providing a temperature range of 45° to 50° F. during a normal period of transit and by avoiding delays and low temperatures during all phases of marketing. Peppers should not be held longer than 12 to 15 days from harvest. A holding temperature of 48° to 50° is best.

(See *90*.)

PHYTOPHTHORA ROT

***Phytophthora capsici* Leonian and *P. parasitica* Dast.**

Phytophthora rot of peppers seldom causes much loss on the market. *Phytophthora capsici* has been associated with a blight of the plants and a fruit rot in most of the growing areas. *P. parasitica* has been reported as a fruit rot on peppers from Illinois, Indiana, and New Jersey.

The causal fungus migrates from the blighted plant through the fruit pedicel and invades the fruit at the stem end. The first symptom of this disease is the presence of small, green, water-soaked spots that tend to elongate down the sides of the pod as they enlarge. Usually by the time one-fourth of the fruit is decayed a superficial, fine, grayish-white to tan mold is evident over the lesion (pl. 11, G). This decay eventually involves the whole pod, and under humid conditions mold develops extensively over the entire surface. Peppers on the market seldom show more than one-fourth of the fruit pod decayed. Apparently the rot found on the market develops from inconspicuous infections that are not observed when the fruit is packed. The usual refrigeration temperatures in transit retard the development of the decay.

Control measures for this disease on harvested peppers have not been developed. For field control, consult the State agricultural extension service or experiment station.

(See *71, 74, 150, 153.*)

RHIZOPUS ROT

Rhizopus stolonifer **(Fr.) Ehr.**

Occurrence and Importance

Rhizopus rot is a soft decay of peppers that is found occasionally on the market and may be responsible for serious losses.

Symptoms

Rhizopus rot, bacterial soft rot, and gray mold rot are sometimes confused. Rhizopus lesions are softer to the touch than those of gray mold, and the lesions tend to be stretched and flattened, in contrast to gray mold lesions, which follow the natural contour of the fruit. Both rhizopus and bacterial soft rot decays are very soft, but the tissues invaded by *Rhizopus* hold together somewhat and tend to be "mushy" in consistency, whereas tissues attacked by the bacterial soft rot organism are "soupy." If affected tissue is gently pulled apart the coarse threads of the *Rhizopus* mold may be observed. A whitish surface mold may appear at the point of original entry of the fungus or at any break on the lesion surface. Shortly thereafter the fruiting stage appears, consisting of raised stalks each with a small spore ball at the end. These spore balls are white when immature and shade into black with maturity. This blend of immature to mature fruiting bodies lends a grayish cast to the coarse mold growth and provides the surest means of identification of rhizopus rot.

Causal Factors

Rhizopus is a fungus of worldwide distribution, and inoculum is present throughout the harvest and postharvest period. It is a wound parasite that enters through any skin break. On peppers it is often noted on fruits broken through crushing. This fungus is capable of spreading by contact from diseased to healthy fruits.

Control

Control of rhizopus rot depends mainly upon careful handling of the pods to avoid injury. Since little growth of this fungus occurs below 50° F., cooling the peppers as quickly as possible to 45° to 50° F. and maintaining that temperature throughout the marketing period will minimize losses from rhizopus rot.

RIPE ROT

Colletotrichum capsici (Syd.) Butl. & Bisby

Ripe rot is the most serious and destructive disease of pimento peppers in certain areas in Georgia, where they are grown for canning. In these areas the growers sometimes lose 50 percent of their crop. It also has been found on ripe fruits on the market. Fruits may become infected at any stage of their development, but decay does not become apparent until they begin to ripen. Warm, wet weather favors the spread of the causal fungus and the rapid development of fruit decay. Seemingly sound fruits harvested from infested fields have been observed to deteriorate overnight to such an extent that more than half of them had to be discarded at the cannery.

The first symptoms appear on ripening pods as yellowish to light-pink circular spots about ⅛ to ¼ inch in diameter. As these spots enlarge they become water soaked and soft, with dirty-brown to black centers and reddish-brown borders. Over the central areas of the larger spots masses of pink spores appear. The spore masses and other general characteristics of the decay are very similar to anthracnose. (See p. 52.) Ripe rot extends through the pod wall and affects the seeds.

The optimum temperature for growth of *Colletotrichum capsici* in culture is 84° F.

Ripe rot must be controlled in the field. Recommendations of the State agricultural extension service or experiment station should be followed.

(See *19, 54, 134.*)

SUNSCALD

Occurrence and Importance

Sunscald is caused by the sudden exposure of fruits to direct sunlight. Partial defoliation by the bacterial spot or the cercospora leaf-spotting organisms may result in considerable loss of the fruit from sunscald. This is a disease of minor importance on the market.

Symptoms

Sunscald on peppers appears as a light-colored, soft, sometimes wrinkled area on the surface exposed to the sunlight. As the injured tissues dry out the lesion sinks somewhat, becoming bleached and papery (pl. 14, E). This type of sunscald rarely reaches the market. There is, however, another type of injury that is not apparent at packing called delayed sunscald, wherein the bleaching characteristic is lacking. The affected tissues have a slightly water-soaked appearance and eventually become desiccated and somewhat brown (pl. 11, H). This manifestation of sunscald on peppers is not easily recognized while they are being packed and may appear on the market.

Cultural practices to promote the health and vigor of the plants, and a spray program to control bacterial spot and cercospora leaf spot should reduce sunscald.

(See *27.*)

VIRUS MOTTLING

Peppers from most of the important shipping regions sometimes show various types of mottling of the fruit similar to virus mottling of tomatoes. Virus diseases such as mosaic, cucumber mosaic, spotted wilt, and tobacco etch are serious problems in the production of peppers. Stunting, distortion, yellow and green mottling, and definite rings or loops on the pods are characteristics of these virus diseases (pl. 13). Pods with ring markings are rarely seen on the market, but pods showing a slightly puckered side with pale yellowish longitudinal streaks are frequently seen.

Recommendations of the State agricultural extension service or experiment station should be followed for the control of virus diseases.

(See *7.*)

EGGPLANTS

ALTERNARIA ROT

***Alternaria tenuis*, auct.**

As indicated in the discussion of alternaria rot of tomatoes and peppers, the fungus is present in the field as a saprophyte or weak parasite. While alternaria rot may occur on eggplant fruits from any locality, the extensive occurrence of the disease depends on chill-injured fruits.

In the early stages, the lesions range from ⅛ to ½ inch in diameter. They are shallow, circular spots with definite margins and rich brown color. Decay usually develops around the calyx and over the surface of the fruits (pl. 16, B), without visible skin breaks. In advanced stages, the spots often coalesce to form larger areas which penetrate the flesh ½ inch or more. The decayed flesh is spongy and tan to grayish tan. Surface mold often develops on the old spots. The mold may be dark gray or, if the fungus is sporulating heavily, the mass will appear olive green and velvety.

Eggplant fruits that have been in contact with top ice during transit for 6 days and those that have been held at 32° to 40° F. for 10 days or longer can be expected to develop a serious amount of alternaria rot after removal to higher temperatures.

To control alternaria rot maintain a transit temperature of 45° to 50° F. Avoid shipping eggplant fruits in mixed loads of vegetables with top ice. Avoid prolonged holding and chilling temperatures.

(See *86.*)

BACTERIAL SOFT ROT

***Erwinia cartovora* (L. R. Jones) Holland**

Bacterial soft rot on eggplant may develop through slight skin breaks especially following periods of hot, rainy weather.

The decay develops rapidly at temperatures of 70° F. or above. Extensive lesions are a dingy, grayish brown (pl. 15, A) with tender skin. The rot is soft and watery and lacks the foul odor that accompanies bacterial soft rot on tomatoes.

On the basis of size and color of the lesions this disease might be confused with rhizopus rot. Bacterial soft rot may be distinguished, however, by the watery nature of the decay, the absence of coarse mold filaments within the decayed tissues, and the lack of external mold growth.

Bacterial soft rot can be controlled by careful handling and by holding the fruits at temperatures of 45° to 50° F. during the brief period of transit and marketing.

CHILLING INJURY

Eggplant fruits are subject to chilling injury if held at temperatures below 45° F. for a week or more.

The problem of chilling injury is not extensive, but occasionally shipments during the winter show some degree of injury. Chilling injury has been noted especially on eggplant in mixed loads of vegetables that were top iced.

The symptoms of chilling injury are pronounced, but usually do not show until after the fruits are removed from low temperature. Fruits held at 32° F. for 10 days or 40° for longer periods may develop extensive areas of scaldlike browning of the skin (pl. 16, A) and may also show browning of the flesh when cut. Other signs of chilling injury are the decline, decay, and loosening of the capstems, and the occurrence of numerous spots of alternaria rot (pl. 16, B) over the surface of the fruits.

To prevent chilling injury, eggplants should be marketed promptly and held at temperatures of 45° to 50° F. during the brief marketing period. Avoid the use of top ice as a refrigerant. (See *86*.)

COTTONY LEAK

***Pythium aphanidermatum* (Edson) Fitz.**

Cottony leak may be found on market eggplants, particularly during wet seasons. Seldom, however, is it of great importance.

The first symptoms of decay are a bleaching of the purple color, the development of a tan discoloration, and a slight wrinkling of the skin (pl. 15, B). The affected tissues underneath are very watery and are light brown. Slight pressure causes a brownish liquid to flow freely from the diseased tissues. The chief distinguishing feature, however, is the abundant cottony, white mold developed by the causal fungus, which covers the surface of the decayed spot. This is most conspicuous under humid conditions. It is the characteristic, together with the "leaky" nature of the decay, that suggests the name "cottony leak."

The decay is caused by the soil-inhabiting fungus *Pythium aphanidermatum*. Wet weather favors its development. Fruits that are in contact with the soil or that become splattered with infested mud may become affected with the decay, which usually originates at the blossom end. Wounds are not necessary for entrance of the fungus.

Since the decay progresses rapidly, fruits showing even slight spots should be discarded at the time of packing. All fruits affected at shipping time are sure to decay, and adjacent fruits in the package may likewise become infected during transit or on the market.

(See *29*.)

GRAY MOLD ROT

Botrytis cinerea **Fr.**

Gray mold rot may occur wherever eggplants are grown, but the disease ordinarily causes very little loss during marketing. The fungus flourishes in the field during cool, moist weather. Fruits harvested following periods of weather favorable to the fungus may occasionally develop the decay through wounds or old blemishes. But if the fruits are held at 45° F. for 10 days or longer they may develop gray mold rot anywhere on the fruits without the aid of skin breaks.

Decayed tissues are bleached from dark purple to medium brown with a grayish cast. Actively developing lesions usually have a reddish-purple margin (pl. 15, D).

The lesions are relatively firm and those of 2 to 3 inches in diameter may penetrate the flesh for ½ to 1 inch. The decayed tissues separate readily from the healthy flesh and, if a cut is made around the margin of the lesion, the decayed mass can be lifted free from the fruit.

To control gray mold rot move eggplant fruits rapidly through the marketing channels. Avoid delays at 40° to 45° F.

PHOMOPSIS ROT

Phomopsis vexans **(Sacc. & Syd.) Harter**

Occurrence and Importance

Phomopsis rot, or fruit rot, is the most common and the most destructive transit and market decay of eggplants. It affects eggplants from all regions, but it causes greatest damage on those from the Southern States, Puerto Rico, and the Philippines. Important losses of marketable fruits frequently occur in the field, and a serious blight of the plant often reduces fruit production and quality.

Symptoms

The decay appears on eggplant fruits in the form of small, more or less circular, tan to light-brown, slightly depressed spots. These may occur singly or severally anywhere over the fruit, but frequently the decay originates under or at the edge of the calyx lobes. The decayed spots become darker brown with age and somewhat zoned in appearance; they enlarge rapidly (pl. 16, C), sometimes to a diameter of 2 or 3 inches. Often two or more spots coalesce to cover much of the fruit. The decay is soft and spongy and penetrates throughout the tissues of the fruits, causing a light-brown discoloration of the flesh. The borders of the decayed tissue are distinct on the surface, but internally they are not always sharply defined from the healthy flesh.

The presence of the fungus fruiting bodies (pycnidia) helps identify the decay (Pl. 16, D). They appear as tiny black pimples that develop in abundance just under the epidermis. When the pycnidia break through the surface, their tiny beaks are readily visible. In advanced stages of decay the pycnidia literally cover the surface.

Causal Factors

Phomopsis rot is caused by the fungus *Phomopsis vexans*. It attacks not only the fruits but the leaves and stems of the plant as well. The decay is greatly increased by a rainy season. Rots found in transit or on the market originate from infections that occurred in the field. In most instances, however, there may be no visible evidence of decay at the time of shipment.

Growth of the causal fungus and development of decay are greatest at temperatures of about 80° to 85° F. There is little development of either at 45° or lower.

Control

Transit temperatures of about 45° F. should be maintained. Eggplant fruits showing evidence of spotting should be discarded at time of shipment. For field control the recommendations of the State agricultural extension service or experiment station should be followed.

(See *30*, *49*, *106*, *109*.)

PHYTOPHTHORA ROT
***Phytophthora* spp.**

This disease occurs in the field as a blight and fruit rot, and sometimes infected fruits are found on the market. The decayed areas of the fruits are dark brown, with conspicuously lighter colored borders. The decay extends well into the flesh. Vascular tissues extending between the decayed spots and the stem may appear as darkly discolored strands. The decay is caused by soilborne species of the fungus *Phytophthora*, similar to or identical with those responsible for buckeye rot of tomatoes. (See Tomato, Buckeye Rot.)

(See *65, 126.*)

RHIZOPUS ROT

Rhizopus stolonifer **(Fr.) Ehr.**

Occasionally, eggplants on the market are affected with rhizopus rot. The lesions are usually extensive, as the decay progresses rapidly at moderate temperatures. The affected area of the skin is brown (pl. 15, C). The brownish, soft, wet decay penetrates deeply into the fruit. This decay can be distinguished from phomopsis rot by the soft and watery nature of the diseased tissues, and from that of bacterial soft rot by the coarse strands of mold that can be exposed by gently pulling apart the decayed tissues.

(See Tomato, Rhizopus Rot.)

LITERATURE CITED

(1) AINSWORTH, G. C., OYLER, ENID, and READ, W. H.
1938. OBSERVATIONS ON THE SPOTTING OF TOMATO FRUITS BY BOTRYTIS CINEREA PERS. Ann. Appl. Biol. 25: 308–321.

(2) ARK, P. A.
1944. STUDIES ON BACTERIAL CANKER OF TOMATO. Phytopathology 34: 394–400.

(3) BAKER, K. F.
1946. AN EPIPHYTOTIC OF RHIZOPUS SOFT ROT OF GREEN-WRAP TOMATOES IN CALIFORNIA. U.S. Dept. Agr. Plant Dis. Rptr. 30: 20–26.

(4) BERAHA, LOUIS, and SMITH, M. A.
1964. A BACTERIAL NECROSIS OF TOMATO. U.S. Dept. Agr. Plant Dis. Rptr. 48: 558–561.

(5) BERRY, J. W., CAROLUS, R. L., JR., and DEDOLPH, R. R.
1964. THE RELATION BETWEEN LOCULAR SEED DENSITY AND THE EXPRESSION OF BLOTCHY RIPENING IN TOMATO. Amer. Soc. Hort. Sci. Proc. 85: 497–501, illus.

(6) BEWLEY, W. F., and WHITE, H. L.
1926. SOME NUTRITIONAL DISORDERS OF THE TOMATO. Ann. Appl. Biol. 13: 323–338.

(7) BOSWELL, V. R., DOOLITTLE, S. P., PULTZ, L. M., TAYLOR, A. L., and CAMPBELL, R. E.
1959. PEPPER PRODUCTION, DISEASE AND INSECT CONTROL. U.S. Dept. Agr. Farmers' Bul. 2051, 33 pp.

(8) BOYLE, J. S., and WHARTON, D. C.
1957. THE EXPERIMENTAL REPRODUCTION OF TOMATO INTERNAL BROWNING BY INOCULATION WITH STRAINS OF TOBACCO MOSAIC VIRUS. Phytopathology 47: 199–207.

(9) BREWER, P. H., KENDRICK, J. B., and GARDNER, M. W.
1926. EFFECT OF MOSAIC ON CARBOHYDRATE AND NITROGEN CONTENT OF THE TOMATO PLANT. Phytopathology 16: 843–851.

(10) BRYAN, M. K.
1930. STUDIES ON BACTERIAL CANKER OF TOMATO. U.S. Dept. Agr. Jour. Agr. Res. 41: 825–851.

(11) ———
1933. THREE BACTERIAL SPOTS OF TOMATO FRUIT. U.S. Dept. Agr. Cir. 282, 2 pp.

(12) BURKHOLDER, W. H., and SMITH, W. L., JR.
1949. ERWINIA ATROSEPTICA (VAN HALL) JENNISON AND ERWINIA CAROTOVORA (JONES) HOLLAND. Phytopathology 39: 887–897.

(13) BUTLER, E. E.
1959. FUNGI AND ROTS IN CALIFORNIA CANNING TOMATOES. U.S. Dept. Agr. Plant Dis. Rptr. 43: 187–192.

(14) ———
1960. PATHOGENICITY AND TAXONOMY OF GEOTRICHUM CANDIDUM. Phytopathology 50: 665–672, illus.

(15) ———
1961. TRANSMISSION OF GEOTRICHUM ROT OF TOMATO FRUIT BY DROSOPHILA MELANOGASTER. Phytopathology 51: 250–255, illus.

(16) ——— and BRACKER, C. E.
1963. THE ROLE OF DROSOPHILA MELANOGASTER IN THE EPIPHYTOLOGY OF GEOTRICHUM, RHIZOPUS AND OTHER FRUIT ROTS OF TOMATO. Phytopathology 53: 1016–1020.

(17) CARMICHAEL, J. W.
1957. GEOTRICHUM CANDIDUM. Mycologia 49: 820–830.

(18) CEPONIS, M. J., and FRIEDMAN, B. A.
1955. THE OCCURRENCE OF PHOMOPSIS ON TOMATOES IN NEW YORK. U.S. Dept. Agr. Plant Dis. Rptr. 39: 989–990.

(19) CHOWDHURY, S.
1957. STUDIES ON THE DEVELOPMENT AND CONTROL OF FRUIT ROT OF CHILLIES. Indian Phytopathology 10: 55–62.
(20) CONOVER, R. A.
1949. VASCULAR BROWNING IN DADE COUNTY, FLORIDA GREEN-WRAP TOMATOES. U.S. Dept. Agr. Plant Dis. Rptr. 33: 283–284.
(21) COOPER, A. J.
1957. BLOTCHY RIPENING AND ALLIED DISORDERS OF THE TOMATO: A CRITICAL REVIEW OF THE LITERATURE. Glasshouse Crops Res. Inst. Ann. Rpt. 1956, pp. 39–47.
(22) COTTER, D. J.
1961. THE INFLUENCE OF NITROGEN, POTASSIUM, BORON AND TOBACCO MOSAIC VIRUS ON THE INCIDENCE OF INTERNAL BROWNING AND OTHER FRUIT QUALITY FACTORS OF TOMATOES. Amer. Soc. Hort. Sci. Proc. 78: 474–479.
(23) CROSIER, W.
1934. STUDIES IN THE BIOLOGY OF PHYTOPHTHORA INFESTANS (MONT.) DE BARY. N. Y. (Cornell) Agr. Expt. Sta. Mem. 155, 40 pp., illus.
(24) DARBY, J. F.
1954. RECENT DEVELOPMENTS IN THE CONTROL OF THE MAJOR DISEASES OF UNSTAKED TOMATOES GROWN ON THE SANDY SOILS OF SOUTH FLORIDA. Fla. Hort. Soc. Proc. 66: 103–107.
(25) ———
1955. A PROGRESS REPORT ON GRAY MOLD AND GHOST SPOT OF TOMATOES AND THEIR CONTROL. U.S. Dept. Agr. Plant Dis. Rptr. 39: 91–97.
(26) DOOLITTLE, S. P., and SUMMER, C. B.
1934. PROBABLE OCCURRENCE OF AUSTRALIAN SPOTTED WILT OF TOMATOES IN WISCONSIN. Phytopathology 24: 943–946.
(27) ——— TAYLOR, A. L., and DANIELSON, L. L.
1961. TOMATO DISEASES AND THEIR CONTROL. U.S. Dept. Agr. Handb. 203, 86 pp., illus.
(28) DOWSON, W. J.
1949. MANUAL OF BACTERIAL PLANT DISEASES. 183 pp., London.
(29) DRECHSLER, C.
1926. THE COTTONY LEAK OF EGGPLANT FRUIT CAUSED BY PYTHIUM APHANIDERMATUM. Phytopathology 16: 47–50, illus.
(30) EDGERTON, C. W., and MORELAND, C. C.
1921. EGGPLANT BLIGHT. La. Agr. Expt. Sta. Bul. 178, 44 pp.
(31) EVANS, H. J., and TROXLER, R. V.
1953. RELATION OF CALCIUM NUTRITION TO THE INCIDENCE OF BLOSSOM-END ROT IN TOMATOES. Amer. Soc. Hort. Sci. Proc. 61: 346–352.
(32) FELIX, E. L.
1953. SUSCEPTIBILITY TO TOMATO BUCKEYE ROT FUNGUS IN TENNESSEE. Phytopathology 43: 290.
(33) FERRER, J. B., and OWEN, J. H.
1959. BOTRYTIS CINEREA THE CAUSE OF GHOST SPOT DISEASE OF TOMATO. Phytopathology 49: 411–417.
(34) FOSTER, A. C., and TATMAN, E. C.
1937. ENVIRONMENTAL CONDITIONS INFLUENCING THE DEVELOPMENT OF TOMATO POCKETS OR PUFFS. Plant Physiol. 12: 875–880.
(35) FRAZIER, W. A.
1935. A STUDY OF SOME FACTORS ASSOCIATED WITH THE OCCURRENCE OF CRACKS IN THE TOMATO FRUIT. Amer. Soc. Hort. Sci. Proc. (1934) 32: 519–523.
(36) FULTON, J. P.
1948. INFECTION OF TOMATO FRUITS BY COLLETOTRICHUM PHOMOIDES. Phytopathology 38: 235–246, illus.
(37) GARDNER, M. W., and KENDRICK, J. B., SR.
1921. BACTERIAL SPOT OF TOMATO AND PEPPER. Phytopathology 13: 307–315.

(38) GARDNER, M. W., and KENDRICK, J. B., SR.
1921. BACTERIAL SPOT OF TOMATO. U.S. Dept. Agr. Jour. Agr. Res. 21: 123–156.
(39) GERALDSON, C. M.
1956. THE USE OF CALCIUM FOR CONTROL OF BLOSSOM-END ROT OF TOMATOES. Fla. State Hort. Soc. Proc. (1955) 68: 197–202.
(40) ———
1957. EVALUATION OF CONTROL METHODS FOR BLACKHEART OF CELERY AND BLOSSOM-END ROT OF TOMATOES. Fla. State Hort. Soc. Proc. (1956) 69: 236–241.
(41) ———
1960. NUTRITIONAL FACTORS AFFECTING THE INCIDENCE AND SEVERITY OF BLOTCHY RIPENING OF TOMATOES. Fla. State Hort. Soc. Proc. 73: 111–114.
(42) GONZALEZ, L. C., and OWEN, J. H.
1963. SOIL ROT OF TOMATO CAUSED BY RHIZOCTONIA SOLANI. Phytopathology 53: 82–85.
(43) GROGAN, R. G., and KENDRICK, J. B., SR.
1953. SEED TRANSMISSION, MODE OF OVERWINTERING AND SPREAD OF BACTERIAL CANKER OF TOMATO CAUSED BY CORYNEBACTERIUM MICHIGANENSE. Phytopathology 43: 473.
(44) HAENSELER, C. M.
1949. INTERNAL BROWNING OF TOMATOES IN NEW JERSEY. U.S. Dept. Agr. Plant Dis. Rptr. 33: 336–337.
(45) HALL, C. B., and DENNISON, R. A.
1955. ENVIRONMENTAL FACTORS INFLUENCING VASCULAR BROWNING OF TOMATO FRUITS. Amer. Soc. Hort. Sci. Proc. 65: 353–356.
(46) HALSEY, L. H.
1955. PRELIMINARY STUDIES OF BRUISING OF "TURNING" AND "PINK" TOMATOES CAUSED BY HANDLING PRACTICES. Fla. State Hort. Soc. Proc. 68: 240–243.
(47) HAMILTON, L. C., and OGLE, W. L.
1962. THE INFLUENCE OF NUTRITION ON BLOSSOM-END ROT OF PIMENTO PEPPERS. Amer. Soc. Hort. Sci. Proc. 80: 457–461, illus.
(48) HARRISON, A. L., and BROOKS, A. N.
1943. CONTROL OF BACTERIAL SOFT ROT OF TOMATOES. Fla. Agr. Expt. Sta. Press Bul. 592, 2 pp.
(49) HARTER, L. L.
1914. FRUIT ROT, LEAF-SPOT, AND STEM-BLIGHT OF THE EGGPLANT CAUSED BY PHOMOPSIS VEXANS. U.S. Dept. Agr. Jour. Agr. Res. 2: 331–338, illus.
(50) HAYSLIP, N. C., and ILEY, J. R.
1965. N-K RATIO MAY INFLUENCE TOMATO GRAYWALL. Better Crops With Plant Food. March-April, 28–29.
(51) HENDRIX, J. W.
1950. A PHOMOPSIS DISEASE OF TOMATOES IN HAWAII. Phytopathology 40: 966.
(52) HIGGINS, B. B.
1922. THE BACTERIAL SPOT OF PEPPERS. Phytopathology 12: 501–516.
(53) ———
1925. BLOSSOM-END ROT OF PEPPER (CAPSICUM ANNUUM L.). Phytopathology 15: 223–239.
(54) ———
1930. A PEPPER FRUIT ROT NEW TO THE UNITED STATES. Ga. Agr. Expt. Sta. Bul. 162, 10 pp., illus.
(55) ———
1934. IMPORTANT DISEASES OF PEPPER IN GEORGIA. Ga. Agr. Expt. Sta. Bul. 186, 20 pp., illus.
(56) HOLMES, F. O.
1950. INTERNAL BROWNING DISEASE OF TOMATO CAUSED BY STRAIN OF TOBACCO MOSAIC VIRUS FROM PLANTAGO. Phytopathology 40: 487–492.

(57) HORSFALL, J. G., and MCDOWNELL, A. D.
1940. VARIETAL SUSCEPTIBILITY OF PEPPERS TO BACTERIAL SPOT. U.S. Dept. Agr. Plant Dis. Rptr. 24: 34–36.

(58) HOTSON, J. W., and HARTGE, LENA.
1923. A DISEASE OF TOMATOES CAUSED BY PHYTOPHTHORA MEXICANA SP. NOV. Phytopathology 13: 520–531.

(59) ILLMAN, W. I.
1960. IDENTITY OF AN INCITANT OF ANTHRACNOSE IN SWEET PEPPERS. Canada Dept. Agr. Canadian Plant Disease Survey 40: 45.

(60) ——— LUDWIG, R. A., and FARMER, JOYCE.
1959. ANTHRACNOSE OF CANNING TOMATOES IN ONTARIO. Canadian Jour. Bot. 37: 1237–1246, illus.

(61) JAMIESON, C. O.
1915. PHOMA DESTRUCTIVA, THE CAUSE OF A FRUIT ROT OF THE TOMATO. U.S. Dept. Agr. Jour. Agr. Res. 4: 1–20.

(62) JOHNSON, H. B.
1964. EFFECT OF HOT WATER TREATMENTS AND HYDROCOOLING ON POSTHARVEST BACTERIAL SOFT ROT IN BELL PEPPERS. U.S. Dept. Agr. AMS-517, 6 pp.

(63) ———
1966. BACTERIAL SOFT ROT IN BELL PEPPERS—CAUSE AND COMMERCIAL CONTROL. U.S. Dept. Agr. Mktg. Res. Rpt. 738, 6 pp.

(64) JONES, L. K., and BURNETT, G.
1935. VIRUS DISEASES OF GREENHOUSE-GROWN TOMATOES. Wash. State Agr. Expt. Sta. Bul. 308, 36 pp.

(65) KENDRICK, J. B., SR.
1923. PHYTOPHTHORA ROT OF TOMATO, EGGPLANT, AND PEPPER. Ind. Acad. Sci. Proc. (1922) 38: 299–306, illus.

(66) KENDRICK, J. B., JR., and WALKER, J. C.
1948. ANTHRACNOSE OF TOMATO. Phytopathology 38: 247–260, illus.

(67) KIDSON, E. B., and STANTON, D. J.
1953. "CLOUD" OR VASCULAR BROWNING IN TOMATOES. New Zeal. Jour. Sci. and Technol., Sec. A, 35 (4): 368–374.

(68) ——— and STANTON, D. J.
1963. "CLOUD" OR VASCULAR BROWNING IN TOMATOES. New Zeal. Jour. Agr. Res. 6 (5): 376–381.

(69) KREUTZER, W. A., BODINE, E. W., and DURRELL, L. W.
1940. CUCURBIT DISEASES AND ROT OF TOMATO FRUIT CAUSED BY PHYTOPHTHORA CAPSICI. Phytopathology 30: 972–976.

(70) KREUTZER, W. A., and BRYANT, L. R.
1946. CERTAIN ASPECTS OF THE EPIPHYTOLOGY AND CONTROL OF TOMATO FRUIT ROT CAUSED BY PHYTOPHTHORA CAPSICI LEONIAN. Phytopathology 36: 329–339, illus.

(71) LEONIAN, L. H.
1922. STEM AND FRUIT BLIGHT OF PEPPERS CAUSED BY PHYTOPHTHORA CAPSICI SP. NOV. Phytopathology 12: 401–408.

(72) ———
1925. PHYSIOLOGICAL STUDIES ON THE GENUS PHYTOPHTHORA. Amer. Jour. Bot. 12: 444–498.

(73) ——— and GEER, H. L.
1929. COMPARATIVE VALUE OF THE SIZE OF PHYTOPHTHORA SPORANGIA OBTAINED UNDER STANDARD CONDITIONS. U.S. Dept. Agr. Jour. Agr. Res. 39: 293–311.

(74) LEYENDECKER, P. J., JR.
1947. AN EPIPHYTOTIC OF PEPPER BLIGHT CAUSED BY PHYTOPHTHORA CAPSICI IN SOUTHERN NEW MEXICO. U.S. Dept. Agr. Plant Dis. Rptr. 31: 421–422.

(75) ———
1950. BLOSSOM-END ROT OF PEPPER (CAPSICUM ANNUUM L.) IN NEW MEXICO. Phytopathology 9: 497–520.

(76) LINN, M. B., and EMGE, R. G.
1949. DEVELOPMENT OF ANTHRACNOSE AND SECONDARY ROTS IN STORED TOMATO FRUITS IN RELATION TO FIELD SPRAYING AND FUNGICIDES. Phytopathology 39: 898–906.

(77) LUTTRELL, E. S.
1947. DIAPORTHE PHASEOLORUM VAR. SOJAE ON CROP PLANTS. Phytopathology 37: 445–465.

(78) LUTZ, J. M.
1942. FACTORS INFLUENCING SHOULDER BRUISES OF GREEN-WRAP TOMATOES. Amer. Soc. Hort. Sci. Proc. 40: 455–459, illus.

(79) ———
1944. MATURITY AND HANDLING OF GREEN-WRAP TOMATOES IN MISSISSIPPI. U.S. Dept. Agr. Cir. 695, 12 pp., illus.

(80) LYON, C. B., BEESON, K. C., and BARRENTINE, M.
1942. MACRO-ELEMENT NUTRITION OF THE TOMATO PLANT AS CORRELATED WITH FRUITFULNESS AND OCCURRENCE OF BLOSSOM-END ROT. Bot. Gaz. 103: 651–667, illus.

(81) MCCOLLOCH, L. P.
1951. ALTERNARIA TENUIS THE CAUSE OF A TOMATO FRUIT ROT. U.S. Dept. Agr. Plant Dis. Rptr. 35: 234–236.

(82) ———
1955. EFFECT OF LOW FIELD TEMPERATURES ON QUALITY OF TOMATOES. Fla. State Hort. Soc. Proc. 68: 188–192, illus.

(83) ———
1960. SALVAGING TOMATOES FROM FROZEN VINES. U.S. Dept. Agr. Mktg. Res. Rpt. 423, 11 pp., illus.

(84) ———
1962. BRUISING INJURY OF TOMATOES. U.S. Dept. Agr. Mktg. Res. Rpt. 513, 31 pp., illus.

(85) ———
1962. CHILLING INJURY AND ALTERNARIA ROT OF BELL PEPPERS. U.S. Dept. Agr. Mktg. Res. Rpt. 536, 16 pp., illus.

(86) ———
1966. CHILLING INJURY OF EGGPLANT. U.S. Dept. Agr. Mktg. Res. Rpt. 749, 5 pp., illus.

(87) ——— and POLLACK, F. G.
1946. HELMINTHOSPORIUM ROT ON TOMATO FRUITS. Phytopathology 36: 988–989.

(88) ——— and WORTHINGTON, J. T.
1952. LOW TEMPERATURE AS A FACTOR IN THE SUSCEPTIBILITY OF MATURE-GREEN TOMATOES TO ALTERNARIA ROT. Phytopathology 42: 425–427, illus.

(89) ——— and WORTHINGTON, J. T.
1954. WAYS TO PREVENT CHILLING MATURE-GREEN TOMATOES. Pre-Pack-Age 7: 22–25, Feb., illus.

(90) ——— and WRIGHT, W. R.
1966. BOTRYTIS ROT OF BELL PEPPERS. U.S. Dept. Agr. Mktg. Res. Rpt. 754, 9 pp., illus.

(91) MCLEAN, D. M., and BJORNSETH, E. H.
1946. CONTROL OF ANTHRACNOSE ON CANNERY TOMATOES. Mich. Agr. Expt. Sta. Quart. Bul. 28: 287–293, illus.

(92) MACNEILL, B. H.
1962. A SPECIALIZED TOMATO FORM OF THE TOBACCO MOSAIC VIRUS IN CANADA. Canad. Jour. Bot. 40: 49–51.

(93) MADHOK, M. R., and FAZAL-UD-DIN.
1943. BACTERIAL SOFT ROT OF TOMATOES CAUSED BY A SPORE FORMING ORGANISM. Indian Jour. Agr. Sci. 13: 129–133.

(94) MARTIN, W. H.
1917. SCLEROTIUM BATATICOLA, THE CAUSE OF A FRUIT ROT OF PEPPERS. Phytopathology 7: 308–312.

(95) MASSEY, A. B.
1924. A STUDY OF BACILLUS AROIDEAE, TOWNSEND, THE CAUSE OF A SOFT ROT OF TOMATO, AND B. CAROTOVORUS JONES. Phytopathology 14: [460]–477.

(96) MASSEY, L. M., LABELLE, R. L., and ROBINSON, W. B.
1962. MECHANICAL HARVESTING EFFECTS ON TOMATO FRUITS. N. J. State Hort. Soc., Hort. News, 44: 194–195.

(97) MAYNARD, D. N., GERSTEN, B., and MICHELSON, L. F.
1959. THE EFFECTS OF BORON NUTRITION ON THE OCCURRENCE OF CERTAIN TOMATO FRUIT DISORDERS. Amer. Soc. Hort. Sci. Proc. 74: 500–505.

(98) MILBRATH, D. G.
1928. LATE BLIGHT OF TOMATO. Calif. Dept. Agr. Monthly Bul. 27: 271–274.

(99) MILLER, C. H.
1960. THE EFFECTS OF LOW AND HIGH LEVELS OF NUTRIENT ELEMENTS ON BELL PEPPERS. Assoc. South. Agr. Workers Proc. 57: 179.

(100) MINGES, P. A., NICKLOW, C., and WILKINSON, R.
1959. TEMPERATURE AS A POSSIBLE FACTOR IN THE DEVELOPMENT OF BLOTCHY RIPENING OR GRAY WALL IN TOMATO FRUITS. Amer. Soc. Hort. Sci. Proc. Abst. p. 36.

(101) ——— and SADIK, SIDKI.
1964. BLOTCHY RIPENING SYMPTOMS OF TOMATOES AND PROCEDURES FOR RATING. Fla. State Hort. Soc. Proc. 77: 246–247.

(102) MORRISON, W. W.
1962. PREPARING FRESH TOMATOES FOR MARKET. U.S. Dept. Agr. Mktg. Bul. 19, 14 pp., illus.

(103) MURAKISHI, H. A.
1960. COMPARATIVE INCIDENCE OF GRAYWALL AND INTERNAL BROWNING OF TOMATO AND SOURCES OF RESISTANCE. Phytopathology 50: 408–412.

(104) NIGHTINGALE, A. A., and RAMSEY, G. B.
1935. DEVELOPMENT OF PHOMA ROT OF TOMATOES IN TRANSIT AND STORAGE. U.S. Dept. Agr. Cir. 371, 8 pp.

(105) ——— and RAMSEY, G. B.
1936. TEMPERATURE STUDIES OF SOME TOMATO PATHOGENS. U.S. Dept. Agr. Tech. Bul. 520, 36 pp.

(106) NOLLA, J. A. B.
1929. THE EGGPLANT BLIGHT AND FRUIT ROT IN PORTO RICO. Porto Rico Dept. Agr. Jour. 13: 35–57.

(107) OBRERO, F. C., and ARAGAKI, MINORU.
1965. SOME FACTORS INFLUENCING INFECTION AND DISEASE DEVELOPMENT OF PHYTOPHTHORA PARASITICA DAST. ON TOMATO. U.S. Dept. Agr. Plant Dis. Rptr. 49: 327–331.

(108) OWEN, J. H., and FERRER, J. B.
1957. STUDIES CONCERNING THE GHOST SPOT DISEASE OF TOMATO. Phytopathology 47: 530–531.

(109) PALO, M. A.
1936. THE PHOMOPSIS DISEASE OF EGGPLANT AND ITS CONTROL. Philippine Jour. Agr. 7: 289–315.

(110) PAULUS, A. O., and POUND, G. S.
1955. EFFECT OF AIR TEMPERATURE ON INITIATION AND DEVELOPMENT OF GRAY LEAF SPOT AND NAILHEAD SPOT OF TOMATO. Phytopathology 45: 168–174.

(111) PENTZER, W. T., and MCCOLLOCH, L. P.
1953. REFRIGERATION OF TOMATOES IN TRANSIT. Indus. Refrig. 125(3): 24–28, Sept., illus.

(112) PIRONE, P. P.
1935. SPOTTED WILT OF TOMATOES AND PEPPERS IN NEW YORK. U.S. Dept. Agr. Plant Dis. Rptr. 19: 244.

(113) POOLE, R. F.
1922. A NEW FRUIT ROT OF TOMATOES. Bot. Gaz. 74: 210–214, illus.

(114) PORTE, W. S.
1934. NOTES ON THE CONTROL OF TRANSIT AND STORAGE DECAYS OF TOMATOES BY THE USE OF CHEMICAL WASHES. Phytopathology 24: 1304–1312.

(115) PRASHAR, D. P., and LAMBETH, V. N.
1960. INHERITANCE OF RADIAL FRUIT CRACKING IN TOMATOES. Amer. Soc. Hort. Sci. Proc. 76: 530–537, illus.

(116) PRITCHARD, F. J., and PORTE, W. S.
1923. WATERY-ROT OF TOMATO FRUITS. A PHYSIOLOGICAL FORM OF OOSPORA LACTIS; EFFECT ON THE HOST; PENETRATION OF THE CELL WALLS BY ENZYMIC ACTION. U.S. Dept. Agr. Jour. Agr. Res. 24: 895–906, illus.

(117) RALEIGH, S. M., and CHUCKA, J. A.
1944. EFFECT OF NUTRIENT RATIO AND CONCENTRATION ON GROWTH AND COMPOSITION OF TOMATO PLANTS AND ON THE OCCURRENCE OF BLOSSOM-END ROT OF THE FRUIT. Plant Physiol. 19: 671–678.

(118) RAMSEY, G. B.
1932. SULPHUR DIOXIDE INJURY OF TOMATOES. Phytopathology 22: 267–268, illus.

(119) ———
1935. PLEOSPORA ROT OF TOMATOES. U.S. Dept. Agr. Jour. Agr. Res. 51: 35–43.

(120) ——— and BAILEY, A. A.
1929. DEVELOPMENT OF NAILHEAD SPOT OF TOMATOES DURING TRANSIT AND MARKETING. U.S. Dept. Agr. Jour. Agr. Res. 38: 131–146.

(121) ——— and BAILEY, A. A.
1929. THE DEVELOPMENT OF SOIL ROT OF TOMATOES DURING TRANSIT AND MARKETING. Phytopathology 19: 383–390, illus.

(122) ——— and BAILEY, A. A.
1931. TOMATO LATE-BLIGHT ROT, A SERIOUS TRANSIT AND MARKET DISEASE. U.S. Dept. Agr. Cir. 169, 11 pp., illus.

(123) ——— and HEIBERG, B. C.
1952. CLADOSPORIUM ROT OF PEPPERS. U.S. Dept. Agr. Plant Dis. Rptr. 36: 481–482.

(124) ——— WIANT, J. S., and MCCOLLOCH, L. P.
1952. MARKET DISEASES OF TOMATOES, PEPPERS, AND EGGPLANTS. U.S. Dept. Agr. Handb. 28, 54 pp., illus.

(125) RAYCHAUDHURI, S. P.
1952. STUDIES ON INTERNAL BROWNING OF TOMATOES. Phytopathology 42: 591–595.

(126) REITSMA, J., and SLOOFF, W. C.
1947. A DISEASE OF EGGPLANT FRUITS CAUSED BY PHYTOPHTHORA PARASITICA DASTUR AND PHYTOPHTHORA PALMIVORA BUTLER. Chron. Nat. 103: 60–63. Indonesia.

(127) RICH, SAUL.
1958. FERTILIZERS INFLUENCE THE INCIDENCE OF TOMATO INTERNAL BROWNING IN THE FIELD. Phytopathology 48: 448–450.

(128) ROSE, D. H., WRIGHT, R. C., and BRATLEY, C. O.
1944. FREEZING INJURY OF FRUITS AND VEGETABLES. U.S. Dept. Agr. Cir. 713, 31 pp.

(129) SEATON, H. L., and GRAY, G. F.
1936. HISTOLOGICAL STUDY OF TISSUES FROM GREENHOUSE TOMATOES AFFECTED BY BLOTCHY RIPENING. U.S. Dept. Agr. Jour. Agr. Res. 52: 217–224.

(130) SEGALL, R. H., HAYSLIP, N. C., and WALTER, J. M.
1963. EFFECT OF POSTHARVEST TEMPERATURES ON SEVERAL TOMATO VARIETIES HARVESTED AT THE MATURE-GREEN STAGE. Fla. State Hort. Soc. Proc. 76: 172–177.

(131) SHERBAKOFF, C. D.
1917. BUCKEYE ROT OF TOMATO FRUIT. Phytopathology 7: [119]–129, illus.

(132) SHOEMAKER, R. A., and CREELMAN, D. W.
1958. THIRTY-SEVENTH ANNUAL REPORT OF THE CANADIAN PLANT DISEASE SURVEY, 1957. Canada Dept. Agr. 132 pp.

(133) SIMONDS, A. O., and KREUTZER, W. A.
1944. INFECTION PHENOMENA IN TOMATO-FRUIT ROT CAUSED BY PHYTOPHTHORA CAPSICI. Phytopathology 34: 813–817, illus.

(134) SMITH, R. W., and CROSSAN, D. F.
1958. THE TAXONOMY, ETIOLOGY, AND CONTROL OF COLLETOTRICHUM PIPERATUM AND COLLETOTRICHUM CAPSICI. U.S. Dept. Agr. Plant Dis. Rptr. 42: 1099–1103.

(135) SPENCER, E. L., and BECKENBACH, J. R.
1949. BLOSSOM-END ROT OF TOMATOES. Fla. Agr. Expt. Sta. Cir. S–6, 7 pp., illus.

(136) SPURR, A. R.
1959. ANATOMICAL ASPECTS OF BLOSSOM-END ROT IN THE TOMATO WITH SPECIAL REFERENCE TO CALCIUM NUTRITION. Hilgardia 28(12): 269–295.

(137) STEVENSON, J. A., and MCCOLLOCH, L. P.
1947. MYROTHECIUM AS A TOMATO FRUIT ROT ORGANISM. U.S. Dept. Agr. Plant Dis. Rptr. 31: 147–149.

(138) STONER, W. N., and HOGAN, W. D.
1950. A REPORT OF GRAYWALL OR INTERNAL BROWNING OF TOMATOES IN SOUTH FLORIDA. U.S. Dept. Agr. Plant Dis. Rptr. 34: 379–380.

(139) SWANK, G.
1953. THE PERFECT STAGE OF COLLETOTRICHUM PHOMOIDES ON TOMATO. Phytopathology 43: 285–287.

(140) TAUBENHAUS, J. J., and EZEKIEL, W. N.
1931. LATE-BLIGHT ROT OF TOMATOES AND POTATOES. Tex. Agr. Expt. Sta. Cir. 60, 15 pp., illus.

(141) TAYLOR, G. A.
1956. THE INFLUENCE OF SOME ENVIRONMENTAL AND NUTRITIONAL FACTORS ON THE INCIDENCE OF INTERNAL BROWNING OF TOMATO. Ph.D. Thesis, Pa. State Univ., 90 pp.

(142) ————
1958. INTERNAL BROWNING OF TOMATOES. N. J. State Hort. Soc., Hort. News 39(5): 3333–3335.

(143) THYR, B. D.
1965. BACTERIAL CANKER AND SPOTTED WILT OF TOMATO IN THE WEST-CENTRAL GREAT PLAINS. U.S. Dept. Agr. Plant Dis. Rptr. 49: 386–387.

(144) TISDALE, W. B., and HAWKINS, S. O.
1937. EXPERIMENTS FOR THE CONTROL OF PHOMA ROT OF TOMATOES. Fla. Agr. Expt. Sta. Bul. 308, 28 pp.

(145) TISDALE, W. H.
1916. A MELANCONIUM PARASITIC ON THE TOMATO. Phytopathology 6: 390–394.

(146) TOMPKINS, C. M., and TUCKER, C. M.
1941. BUCKEYE ROT OF TOMATO IN CALIFORNIA. U.S. Dept. Agr. Jour. Agr. Res. 62: 467–474, illus.

(147) TOMPKINS, D. R., and STARK, F. C.
1964. FACTORS ASSOCIATED WITH INTERNAL BROWNING OF TOMATO FRUITS. Md. Agr. Expt. Sta. Bul. A–137, 29 pp.

(148) TRESHOW, MICHAEL.
1955. THE ETIOLOGY, DEVELOPMENT, AND CONTROL OF TOMATO FRUIT TUMOR. Phytopathology 45: 132–137.

(149) TUCKER, C. M.
1931. TAXONOMY OF THE GENUS PHYTOPHTHORA DE BARY. Mo. Agr. Expt. Sta. Res. Bul. 153, 208 pp.

(150) UNITED STATES DEPARTMENT OF AGRICULTURE.
1960. INDEX OF PLANT DISEASES IN THE UNITED STATES. U.S. Dept. Agr. Handb. 165, 531 pp.

(151) WALTON, C. L.
1937. THE CAUSE OF A "SPOTTING" OF TOMATO FRUITS (STIGMONOSE). Gardener's Chronicle 101: 12–14.

(152) WEBB, R. E.
1962. AN UNUSUAL LOCAL EPIDEMIC OF TOMATO GHOST SPOT. U.S. Dept. Agr. Plant Dis. Rptr. 46: 260.

(153) WEBER, G. F.
1932. BLIGHT OF PEPPERS IN FLORIDA CAUSED BY PHYTOPHTHORA CAPSICI. Phytopathology 22: 775–780.

(154) ———
1939. NAILHEAD SPOT OF TOMATOES. Fla. Agr. Expt. Sta. Bul. 332, 54 pp.

(155) ———
1940. BACTERIAL SPOT OF PEPPERS. Fla. Agr. Expt. Sta. Press Bul. 549, 2 pp.

(156) ——— and RAMSEY, G. B.
1926. TOMATO DISEASES IN FLORIDA. Fla. Agr. Expt. Sta. Bul. 185, pp. 60–138.

(157) WHARTON, D. C., and BOYLE, J. S.
1957. THE PATHOLOGICAL HISTOLOGY OF THE INTERNAL BROWNING DISEASE OF THE TOMATO. Phytopathology 47: 208–212.

(158) WHITE, H. L.
1938. FURTHER OBSERVATIONS OF THE INCIDENCE OF BLOTCHY RIPENING OF THE TOMATO. Ann. Appl. Biol. 25(3): 544–557.

(159) WHITEMAN, T. M.
1957. FREEZING POINTS OF FRUITS, VEGETABLES, AND FLORIST STOCKS. U.S. Dept. Agr. Mktg. Res. Rpt. 196, 32 pp.

(160) WIANT, J. S.
1941. PHOMOPSIS FRUIT ROT OF TOMATO. Phytopathology 31: 24.

(161) ——— and BRATLEY, C. O.
1948. SPOILAGE OF FRESH FRUITS AND VEGETABLES IN RAIL SHIPMENTS UNLOADED AT NEW YORK CITY, 1935–42. U.S. Dept. Agr. Cir. 773, 62 pp.

(162) WILSON, A. R.
1963. SOME OBSERVATIONS ON THE INFECTION OF TOMATO STEMS BY BOTRYTIS CINEREA. FR. Ann. Appl. Biol. 51: 171.

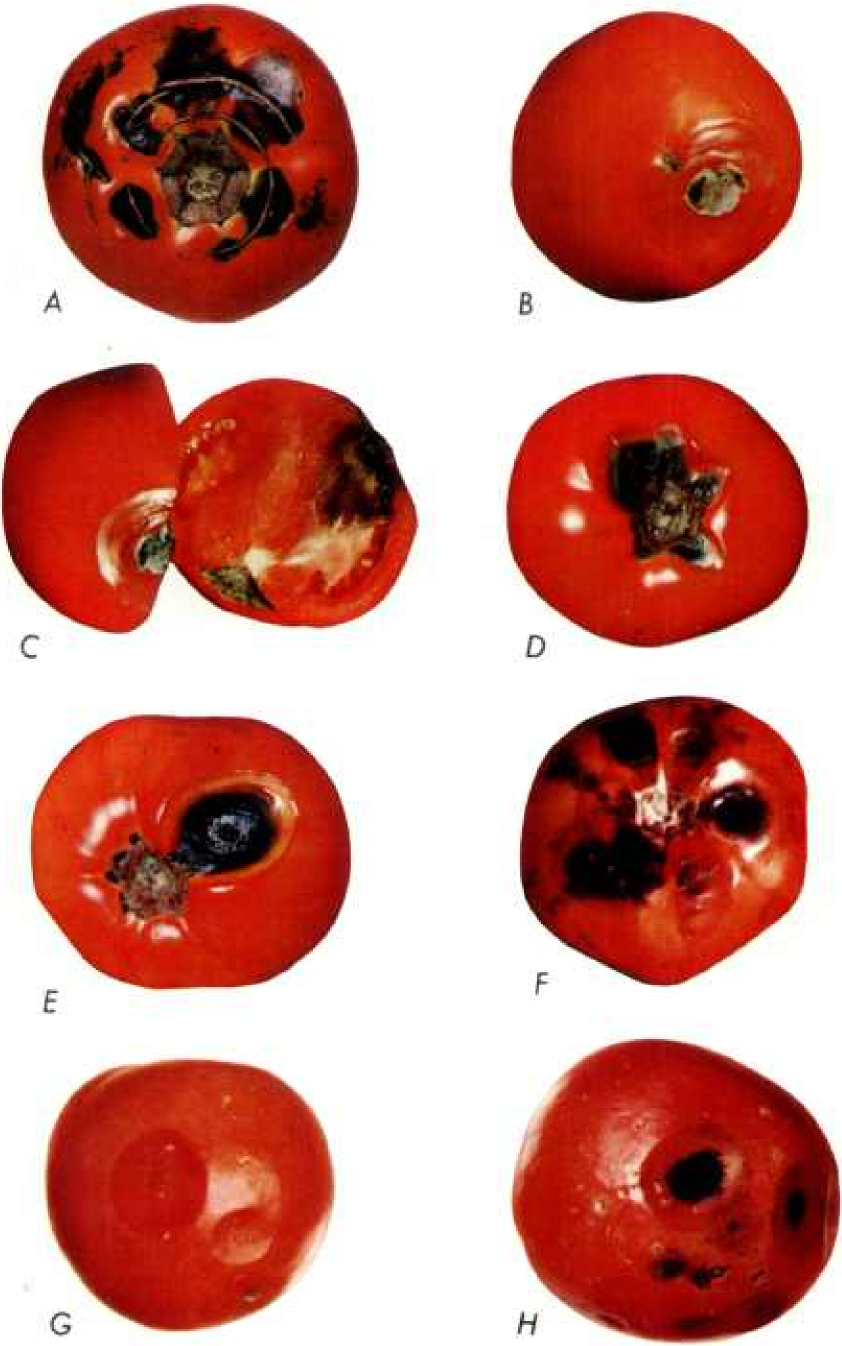
(163) WINGARD, S. A.
1924. BACTERIAL SOFT ROT OF TOMATO. Phytopathology 14: [451]–459.

(164) YARNELL, S. H., FRIEND, W. H., and WOOD, J. F.
1937. FACTORS AFFECTING THE AMOUNT OF PUFFING IN TOMATOES. Tex. Agr. Expt. Sta. Bul. 541, 64 pp., illus.

(165) YOUNG, P. A.
1946. TOMATO DISEASES IN TEXAS. Tex. Agr. Expt. Sta. Cir. 113, 66 pp.

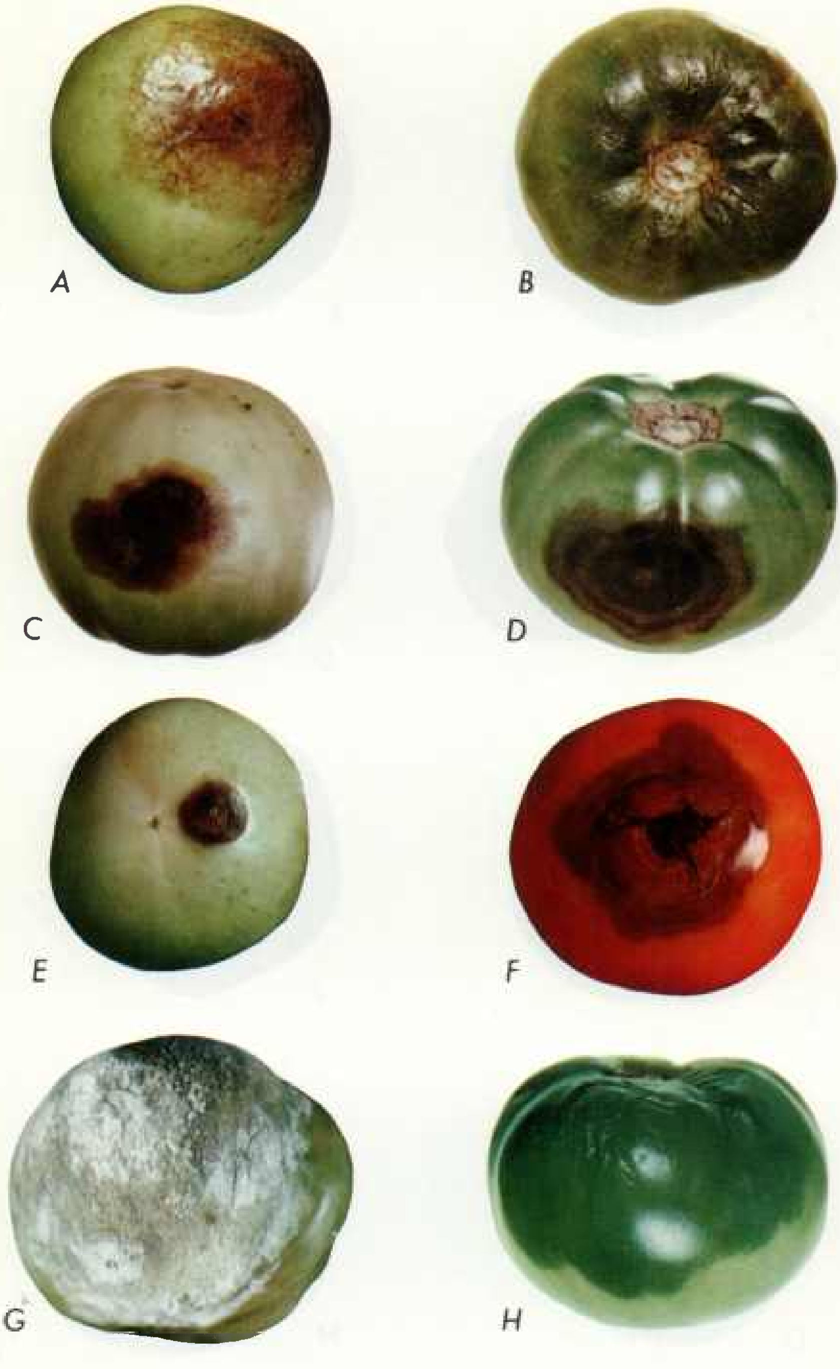
(166) ———
1947. CUTICLE CRACKS IN TOMATO FRUITS. Phytopathology 37: 143–145.

(167) ———
1948. CHARCOAL ROT IN PEPPER ROOTS. U.S. Dept. Agr. Plant Dis. Rptr. 32: 482–483.



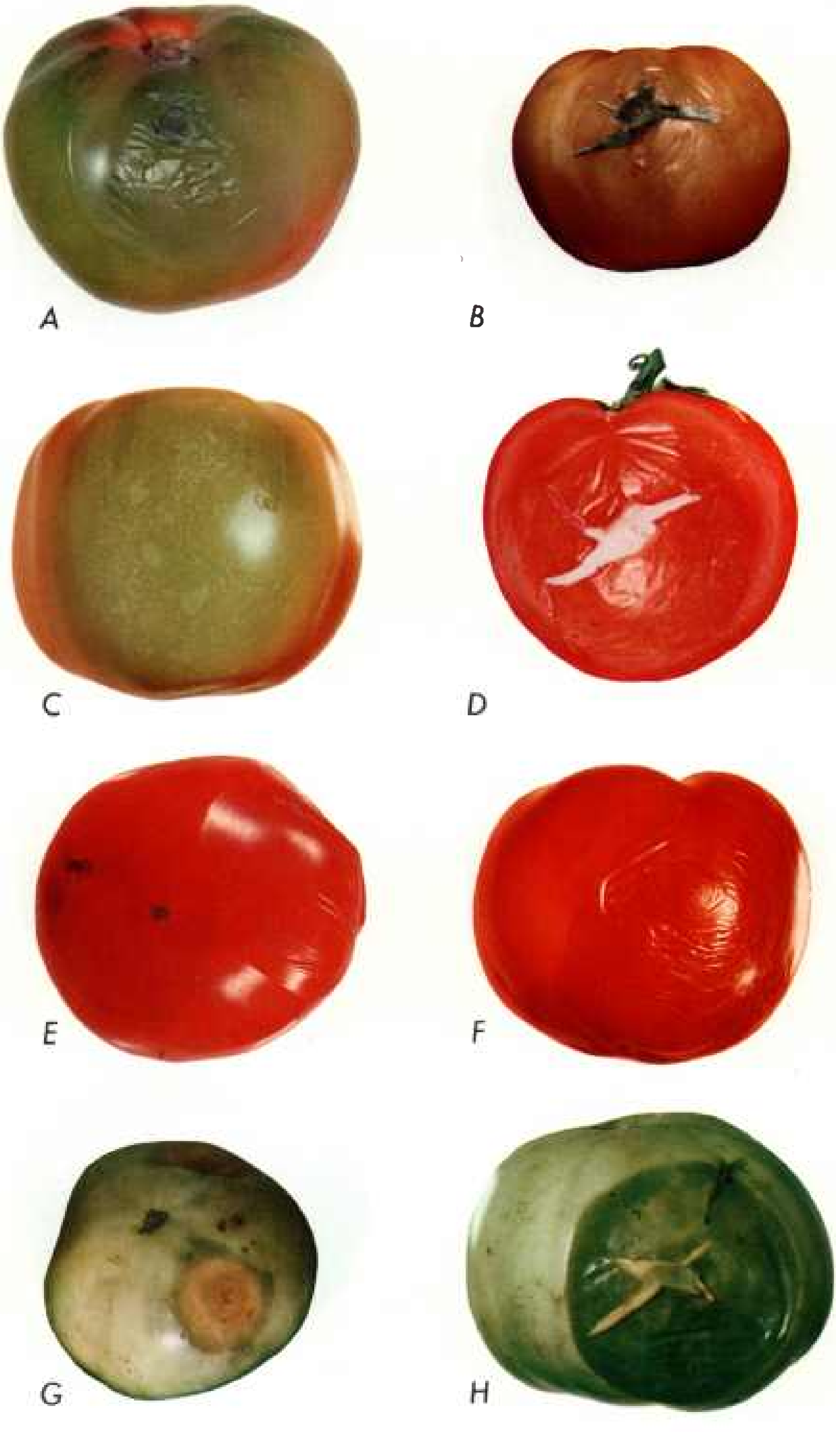
FUNGUS DISEASES

A, Alternaria rot following growth cracks; *B* and *C*, alternaria rot at faulty blossom scar; *D* and *E*, phoma rot; *F*, pleospora; *G*, anthracnose (early stage); *H*, anthracnose (advanced stage).



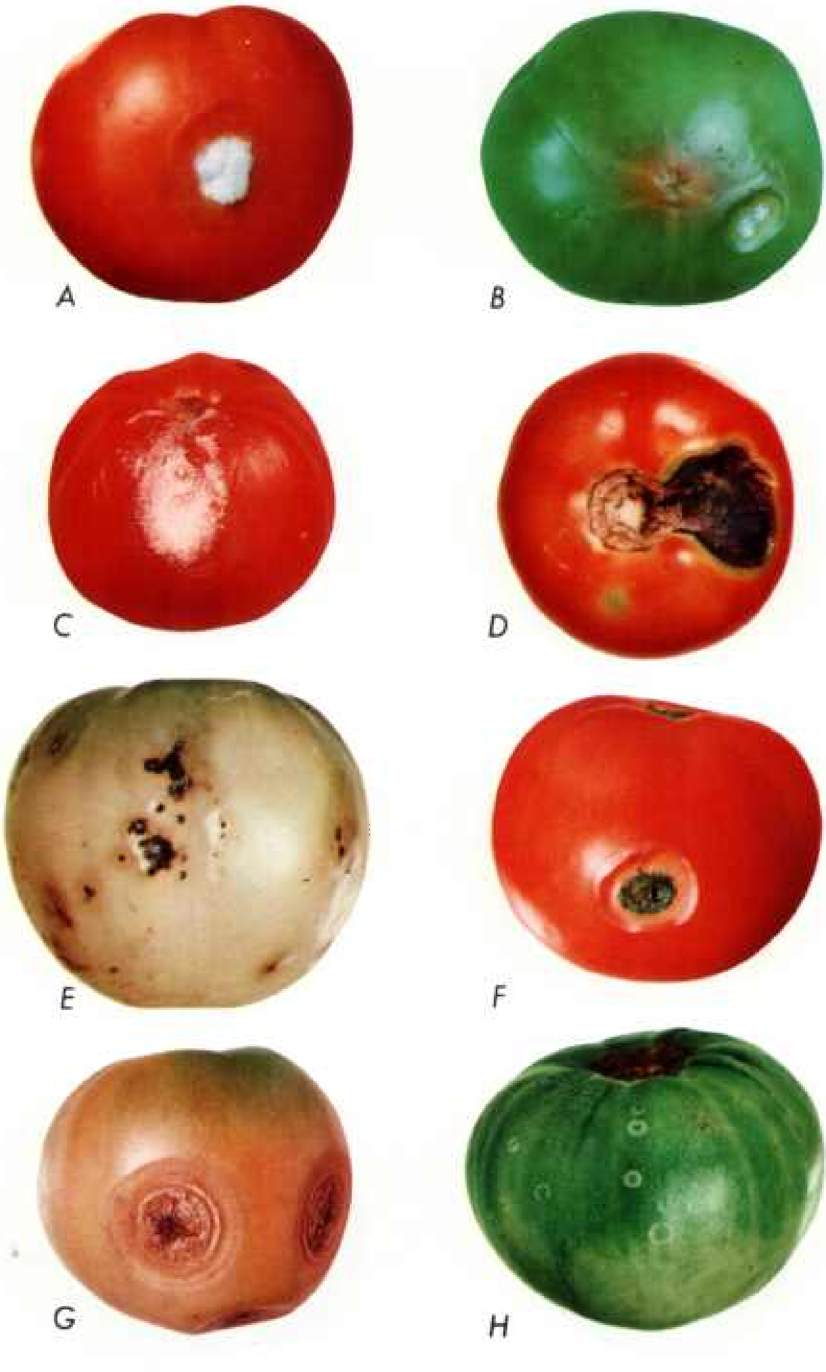
FUNGUS DISEASES

A and *B*, Late blight rot; *C*, buckeye rot (early stage); *D*, buckeye rot (zoned stage); *E* and *F*, soil rot; *G*, phytophthora rot; *H*, pythium rot.



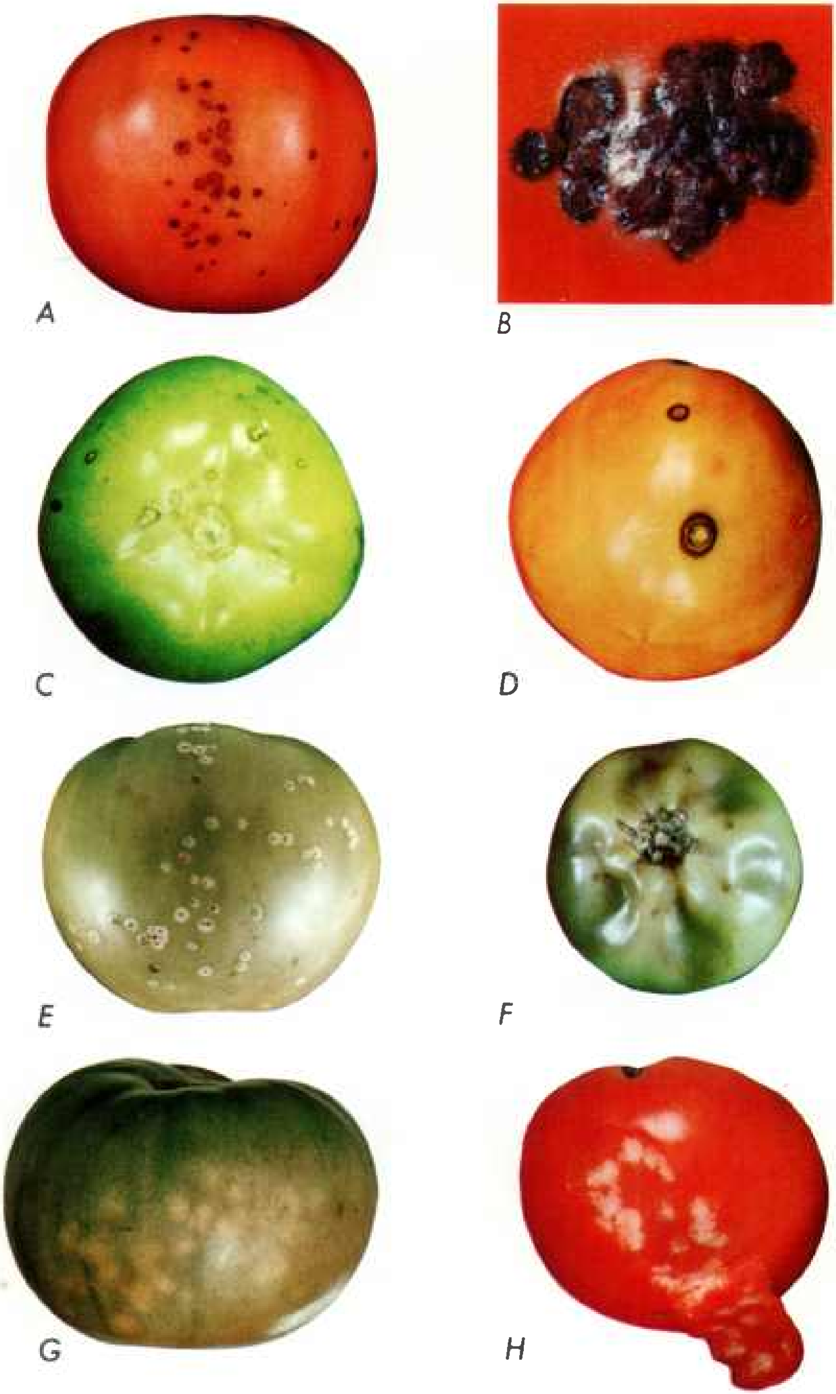
FUNGUS AND BACTERIAL DISEASES

A and *B*, Gray mold rot; *C*, sour rot initiated on green fruit; *D*, sour rot on vine-ripened fruit; *E*, rhizopus rot (early stage); *F*, rhizopus rot (advanced stage); *G*, bacterial soft rot (early stage); *H*, bacterial soft rot (advanced stage).



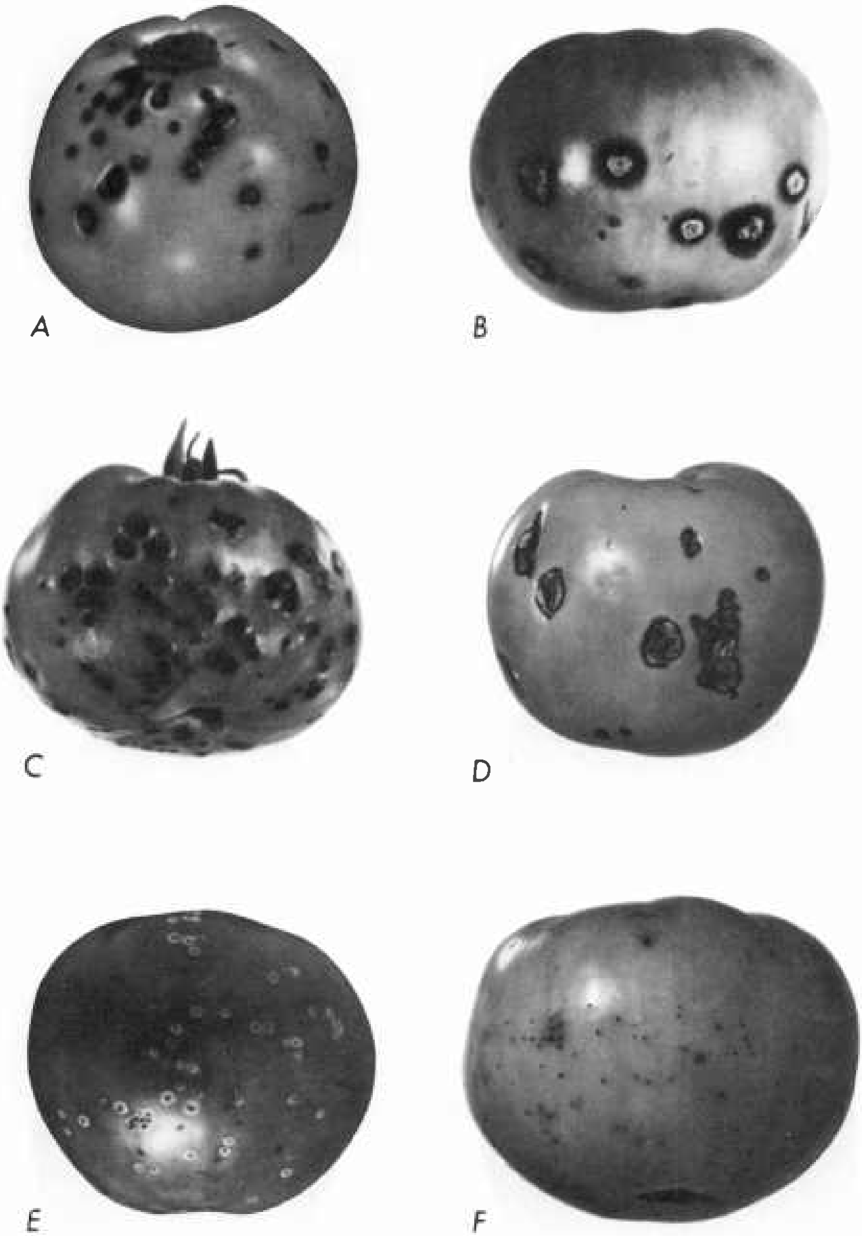
FUNGUS DISEASES

A and *B*, Fusarium rot; *C*, phomopsis rot; *D*, early blight rot; *E*, cladosporium rot; *F*, helminthosporium rot; *G*, ring rot; *H*, ghost spot.



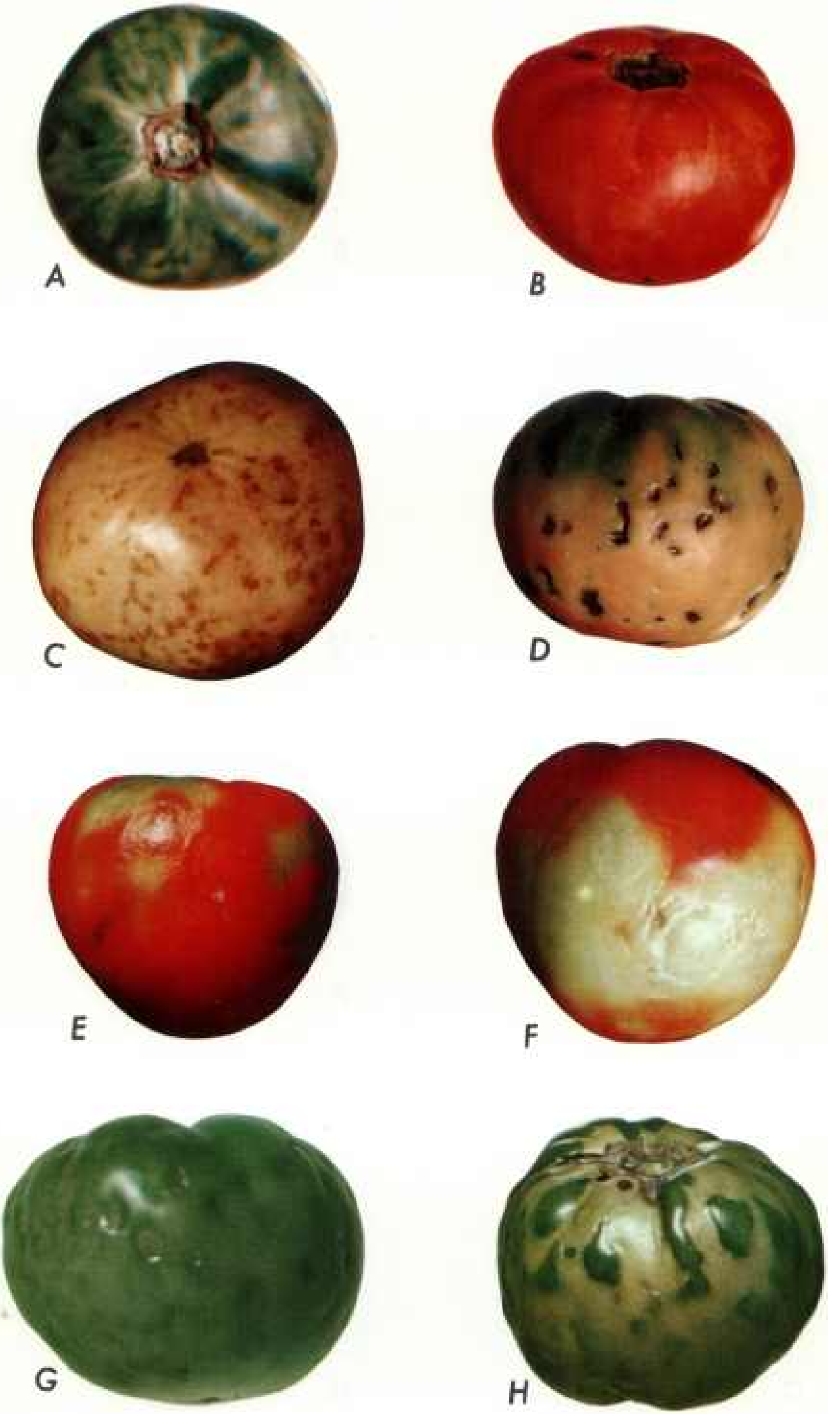
BACTERIAL AND FUNGUS DISEASES AND INSECT INJURY

A and *B*, Bacterial spot; *C* and *D*, nailhead spot; *E*, bacterial canker; *F*, bacterial necrosis; *G* and *H*, cloudy spot.



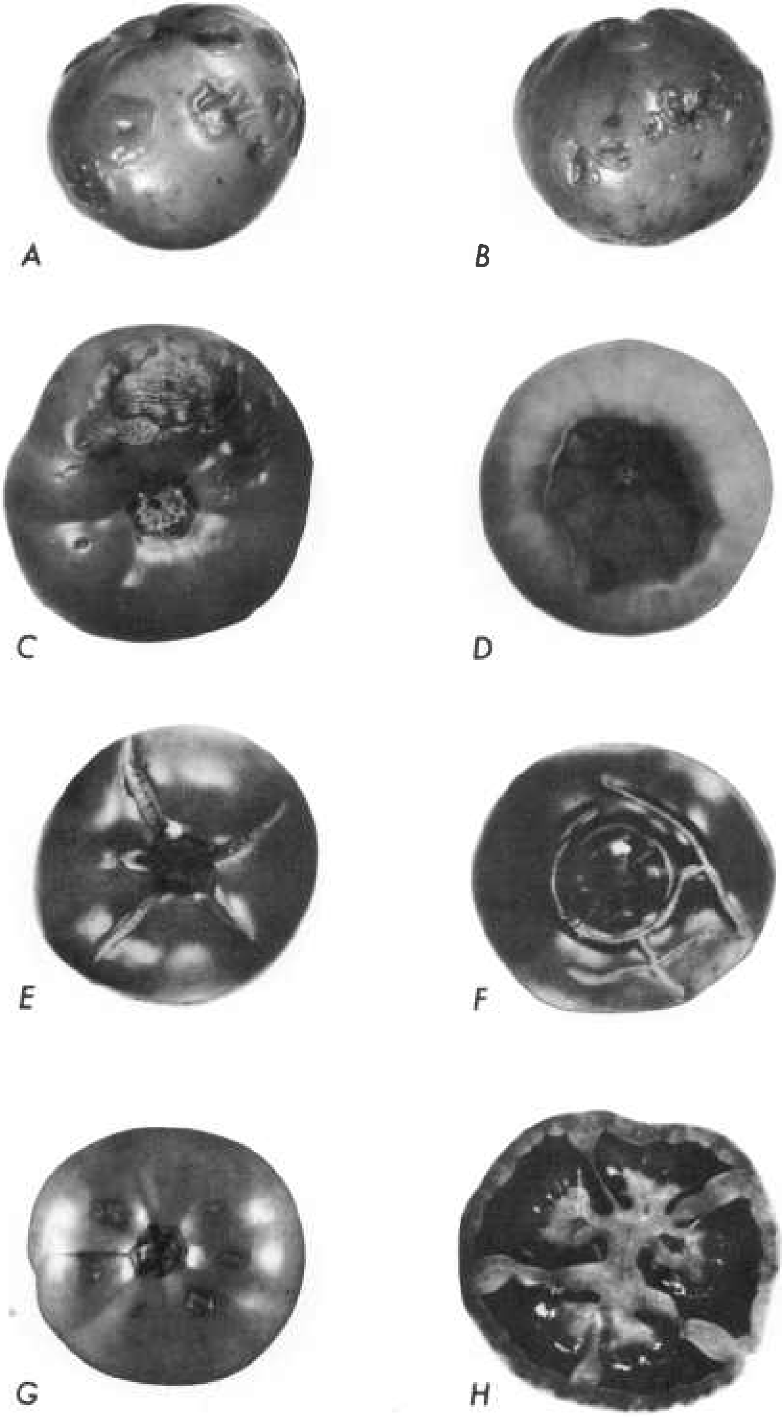
FUNGUS AND BACTERIAL DISEASES

A and *B*, Nailhead spot; *C* and *D*, bacterial spot; *E*, bacterial canker; *F*, bacterial speck.



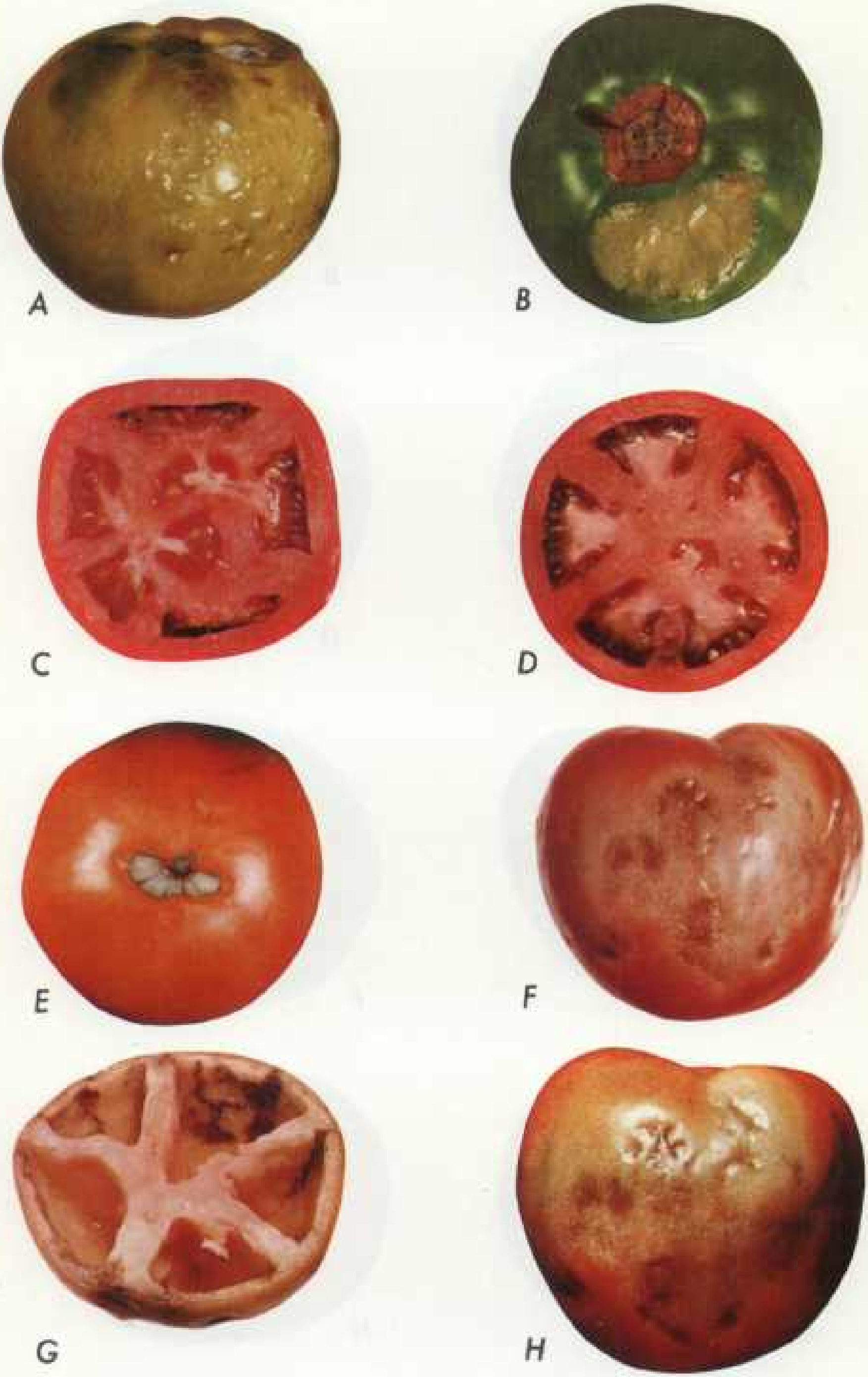
VIRUS MOTTLING

A and *B*, Tomato mosaic; *C* and *D*, double-virus streak; *E* and *F*, spotted wilt; *G* and *H*, unidentified virus mottling.



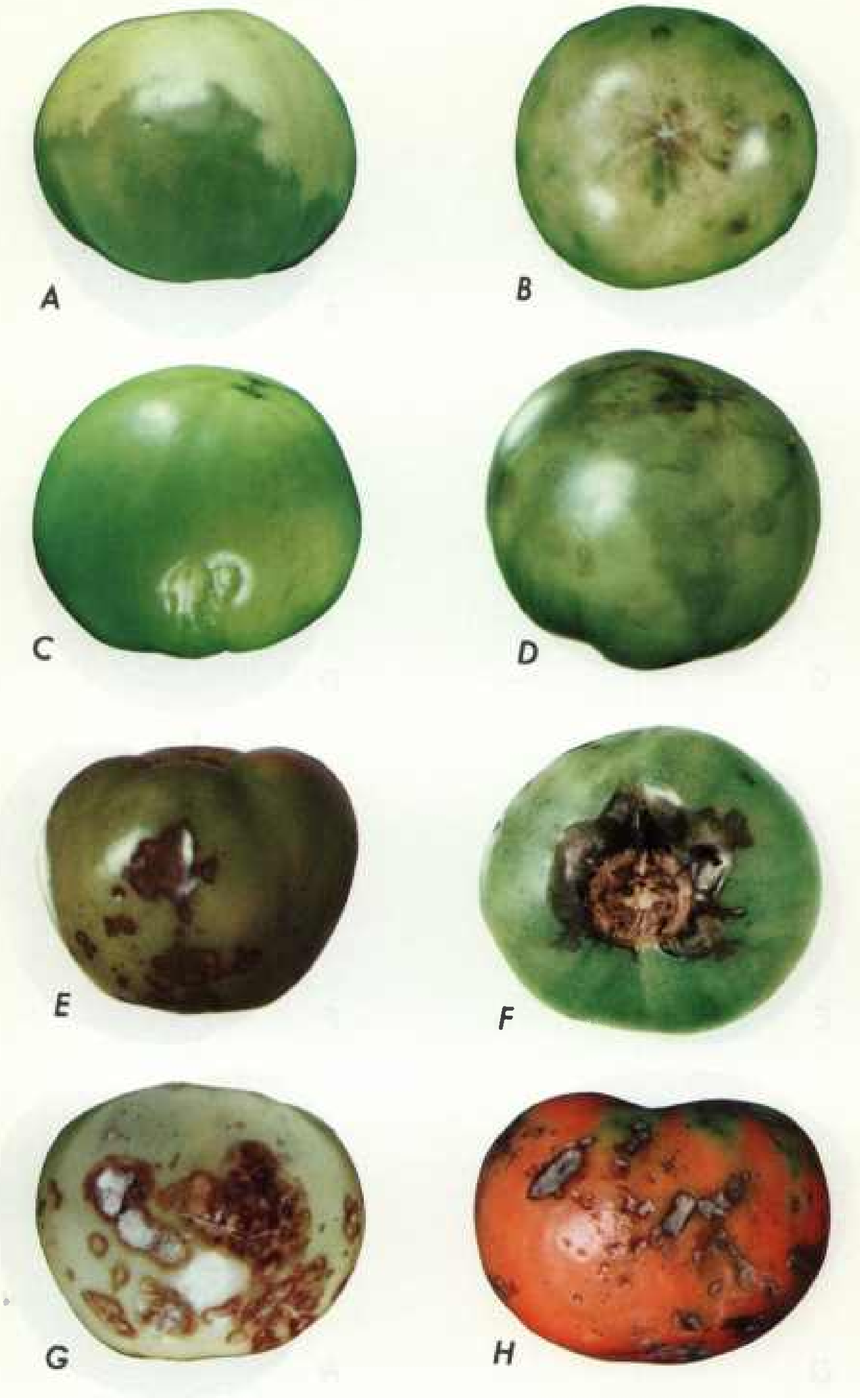
PHYSIOLOGICAL DISEASES, INJURIES AND DEFECTS

A and *B*, Fruit tumor (waxy blister); *C*, sunscald; *D*, blossom-end rot; *E*, growth cracks (radial); *F*, growth cracks (concentric); *G*, shoulder scars; *H*, puffiness.



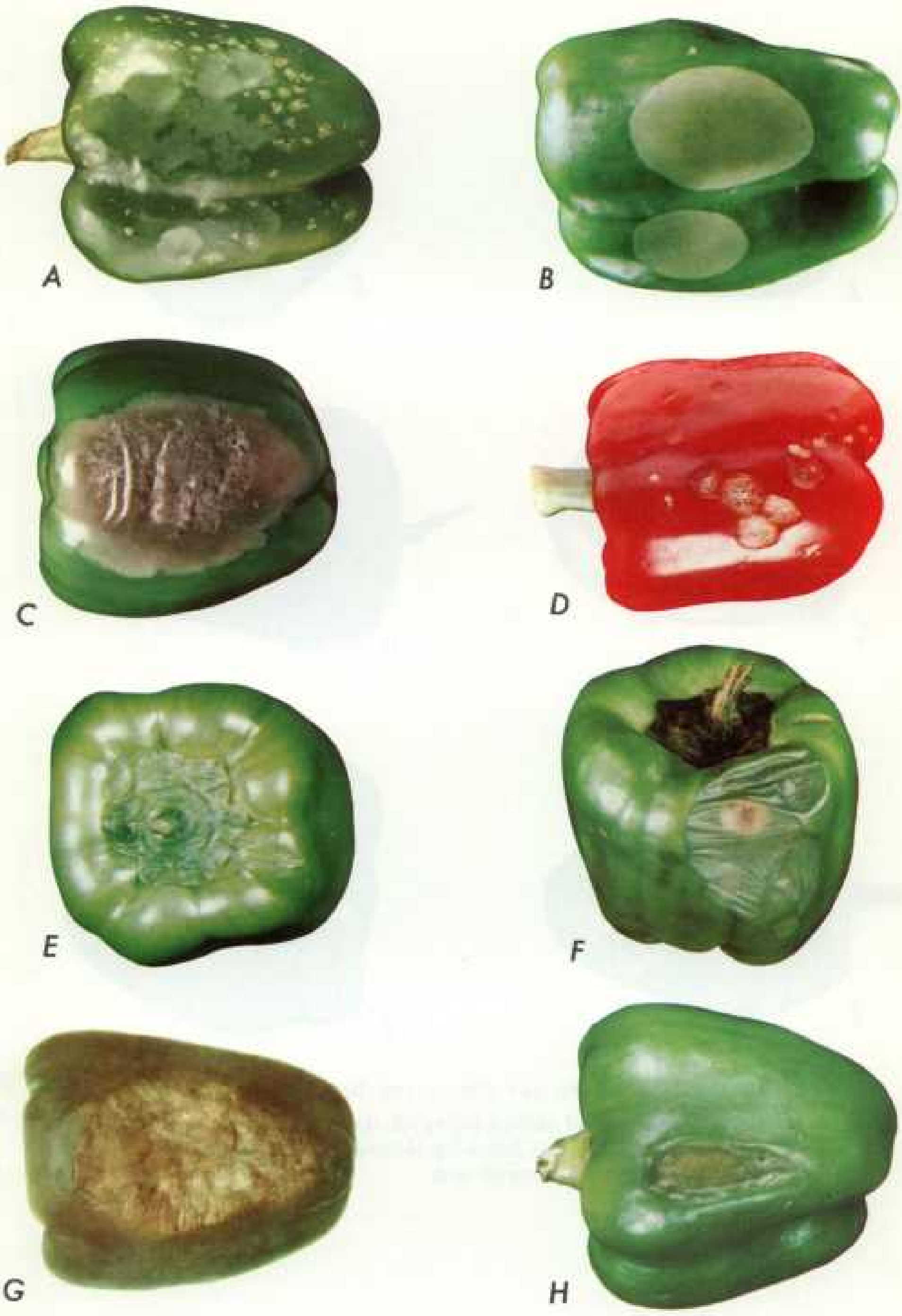
PHYSIOLOGICAL DISEASES AND INJURIES

A, Fruit tumor (waxy blister); *B*, sunscald; *C*, pressure bruise; *D*, impact bruise; *E*, blossom-end rot; *F*, *G*, and *H*, internal browning.



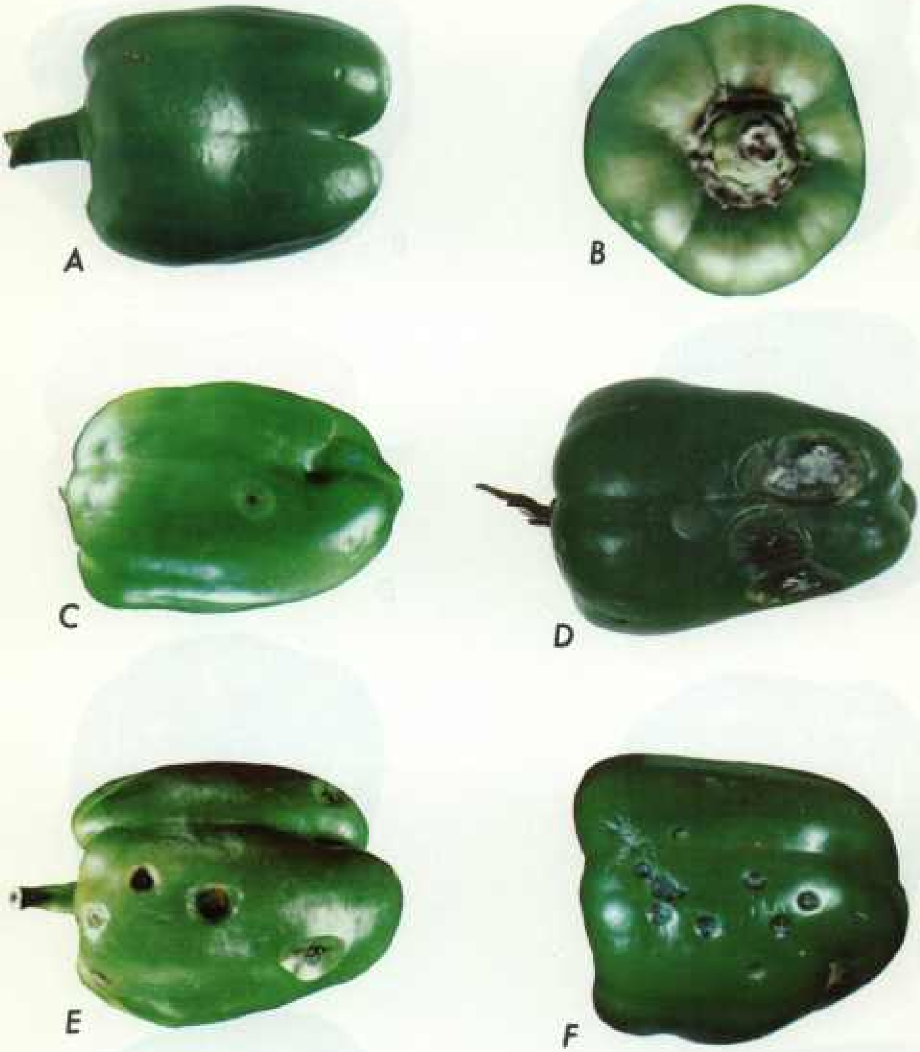
FREEZING AND CHILLING INJURY AND DECAY

A, Freezing injury (severe); *B*, freezing injury (moderate); *C*, freezing injury (slight); *D*, chilling injury (severe); *E* and *F*, chilling injury with alternaria rot following; *G*, severe chilling injury with alternaria rot following; *H*, chilling injury with superficial scars caused by *Alternaria*.



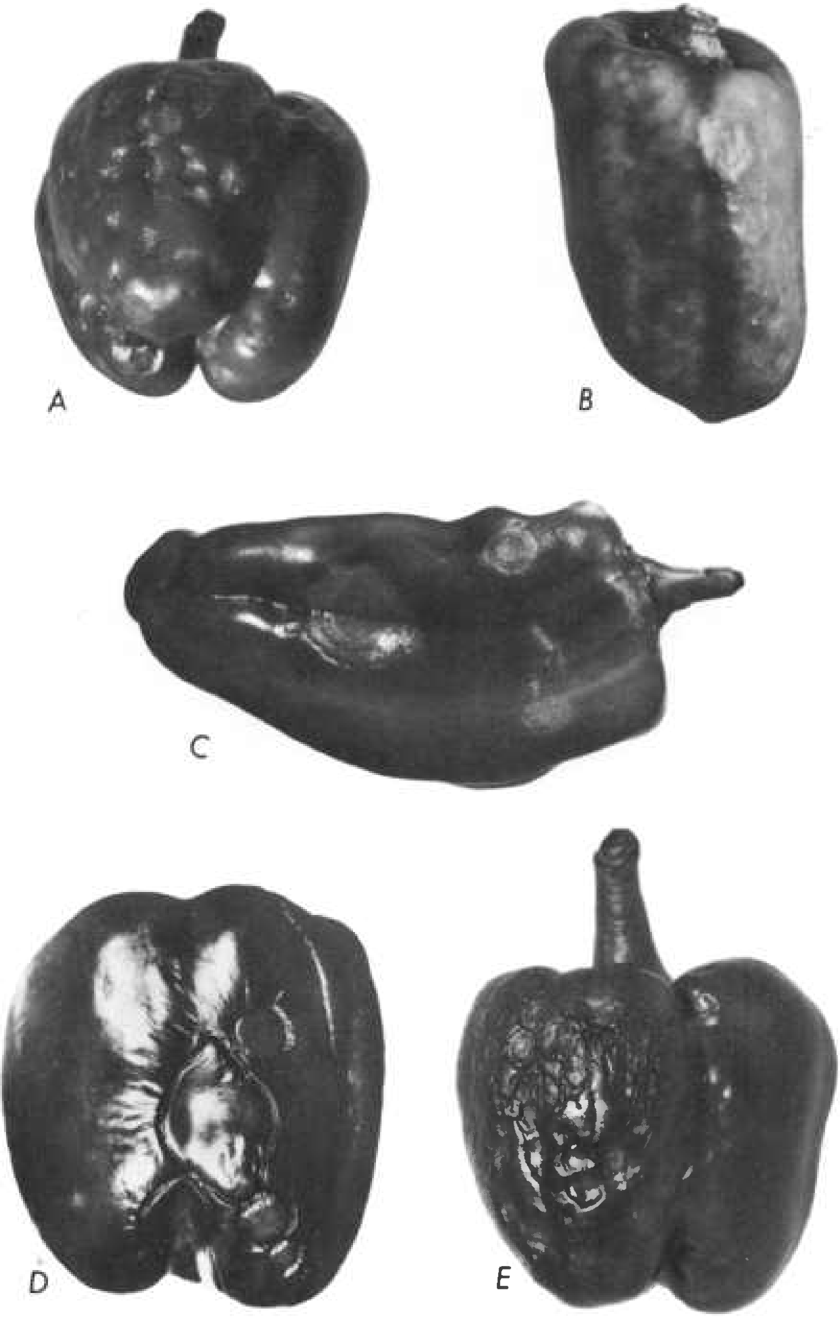
FUNGUS AND BACTERIAL DISEASES AND INJURY

A, Gray mold rot (early stages); *B* and *C*, gray mold rot; *D*, anthracnose; *E*, bacterial soft rot at calyx; *F*, bacterial soft rot on pod; *G*, phytophthora rot; *H*, sunscald.



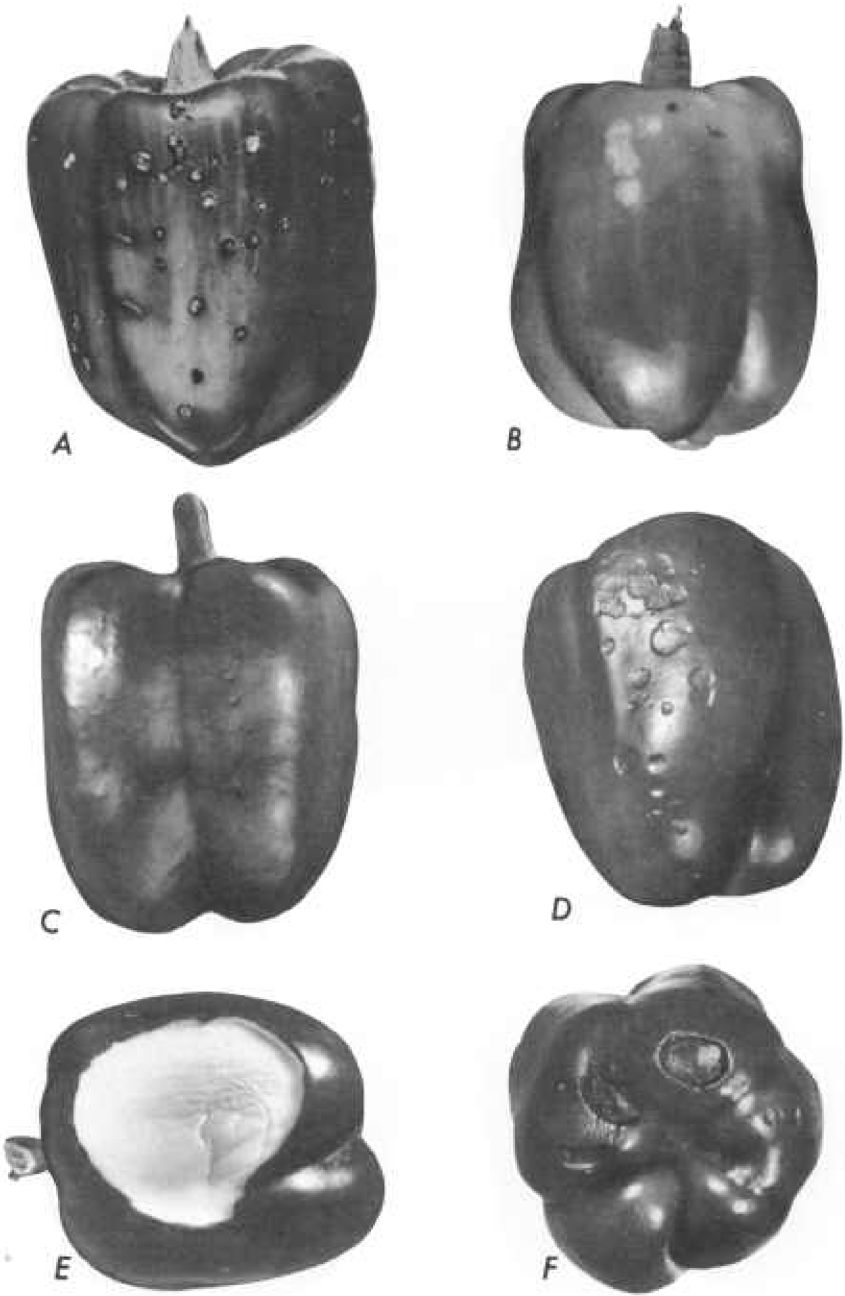
CHILLING INJURY AND DECAY AND BACTERIAL SPOT

A, Sheet pitting, a symptom of chilling injury; *B*, death of calyx following chilling injury; *C* and *D*, alternaria rot following chilling injury; *E*, cladosporium rot following chilling injury; *F*, bacterial spot.



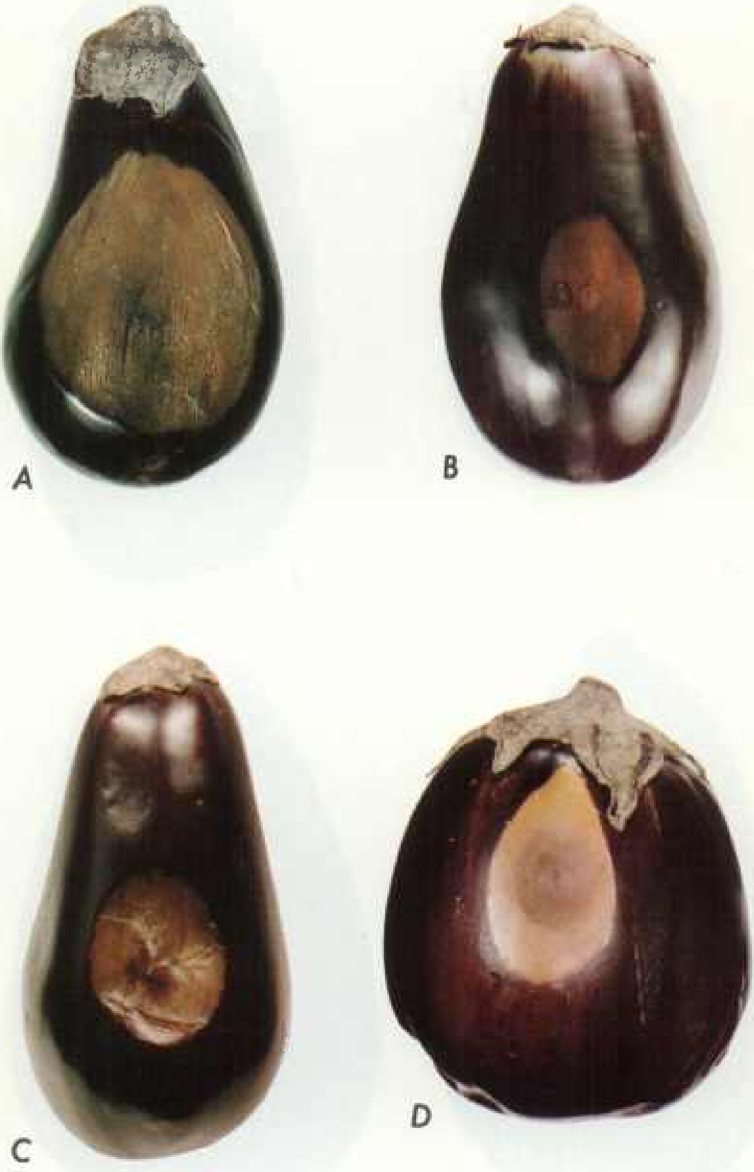
VIRUS MOTTLING

Various kinds of virus mottling.



BACTERIAL AND PHYSIOLOGICAL DISEASES AND INJURIES

A, Bacterial spot; *B*, cloudy spot; *C* and *D*, sheet pitting, a symptom of chilling injury; *E*, sunscald (severe); *F*, blossom-end rot.



FUNGUS DISEASES

A, Bacterial soft rot; *B*, pythium rot (cottony leak); *C*, rhizopus rot; *D*, gray mold rot.

INJURIES AND FUNGUS DISEASES

A, Pitting and scald following chilling injury; *B*, alternaria rot following chilling injury; *C*, phomopsis or fruit rot (early stages); *D*, phomopsis or fruit rot (advanced stage showing fruiting pustules).

CAUTION

If pesticides are handled or applied improperly, they may be injurious to humans, domestic animals, desirable plants, and pollinating insects, fish, or other wildlife, and may contaminate water supplies. Use pesticides only when needed and handle them with care. Follow the directions and heed all precautions on the container label.

PROTECT

Use Pesticides Safely

FOLLOW THE LABEL

U.S. DEPARTMENT OF AGRICULTURE

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