MARKET DISEASES OF POTATOES

AGRICULTURE HANDBOOK NO. 479

Agricultural Research Service
UNITED STATES DEPARTMENT OF AGRICULTURE

PRECAUTIONS

Pesticides used improperly can be injurious to man, animals, and plants. Follow the directions and heed all precautions on the labels.

Store pesticides in original containers under lock and key—out of the reach of children and animals—and away from food and feed.

Apply pesticides so that they do not endanger humans, livestock, crops, beneficial insects, fish, and wildlife. Do not apply pesticides when there is danger of drift, when honey bees or other pollinating insects are visiting plants, or in ways that may contaminate water or leave illegal residues.

Avoid prolonged inhalation of pesticide sprays or dusts; wear protective clothing and equipment if specified on the container.

If your hands become contaminated with a pesticide, do not eat or drink until you have washed. In case a pesticide is swallowed or gets in the eyes, follow the first aid treatment given on the label, and get prompt medical attention. If a pesticide is spilled on your skin or clothing, remove clothing immediately and wash skin thoroughly.

Do not clean spray equipment or dump excess spray material near ponds, streams, or wells. Because it is difficult to remove all traces of herbicides from equipment, do not use the same equipment for insecticides or fungicides that you use for herbicides.

Dispose of empty pesticide containers promptly. Have them buried at a sanitary land-fill dump, or crush and bury them in a level, isolated place.

NOTE: Some States have restrictions on the use of certain pesticides. Check your State and local regulations. Also, because registrations of pesticides are under constant review by the Federal Environmental Protection Agency, consult your county agricultural agent or State Extension specialist to be sure the intended use is still registered.

PREFACE

This handbook is an extensive revision of and supersedes Miscellaneous Publication 98, "Market Diseases of Fruits and Vegetables: Potatoes," by G. B. Ramsey, the late J. S. Wiant, and M. A. Smith. This handbook is one of a series designed to aid in the recognition and identification of market diseases, to provide information on factors and organisms causing the diseases, and to give control measures for reducing economic losses caused by these diseases during marketing.

These handbooks are intended for use by market inspectors, research workers, shippers, receivers, carriers, and others concerned with maintaining quality of fresh fruits and vegetables during marketing.

Other publications in this series are—

28 Market Diseases of Tomatoes, Peppers, and Eggplants. (Rev., Feb. 1968.)

155 Market Diseases of Beets, Chicory, Endive, Escarole, Globe Artichokes, Lettuce, Rhubarb, Spinach, and Sweetpotatoes. (Apr. 1959; reprinted, May 1967.)

184 Market Diseases of Cabbage, Cauliflower, Turnips, Cucumbers, Melons, and Related Crops. (Sept. 1961.)

189 Market Diseases of Grapes and Other Small Fruits. (Reprinted, Apr. 1966.)

303 Market Diseases of Asparagus, Onions, Beans, Peas, Carrots, Celery, and Related Vegetables. (Sept. 1966.)

376 Market Diseases of Apples, Pears, and Quinces. (Rev., May 1971.)

398 Market Diseases of Citrus and Other Subtropical Fruits. (Rev., May 1971.)

414 Market Diseases of Stone Fruits: Cherries, Peaches, Nectarines, Apricots, and Plums. (Rev., Dec. 1972.)

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Washington, D.C. Issued March 1978

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C. 20402 - Price $2.15
Stock Number 001-000-03516-9

MARKET DISEASES OF POTATOES

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The white potato (*Solanum tuberosum* L.) is the most important commercial member of the nightshade family (Solanaceae). Three other important members of this family are the tomato (*Lycopersicon esculentum* Mill.), the pepper (*Capsicum frutescens* L.), and the eggplant (*S. melongena* L.).

The potato tuber is a storage organ that develops from a swelling at the end of a rhizome or underground stem. When potatoes are harvested before they are fully mature, they are often called "new" or "early potatoes." Such potatoes have a thin tender skin, which is easily feathered or rubbed off during harvesting and handling. They discolor and wither rapidly at skinned places and are more susceptible to decay than mature tubers. For this reason the early crops are not suitable for long storage.

The late or main crop of potatoes, which comes chiefly from the Middle Atlantic and Northern States, is not harvested until the vines are mature or dying and the tuber skin is firm, tough, and corky. These tubers are not easily injured. If free of serious diseases and stored under proper conditions at about 40° F[2] and 85 to 90 percent relative humidity, they change very little within 3 to 6 months.

Many people handle potatoes carelessly as if they were dead. Fortunately both immature and mature tubers can develop protective and healing tissues when wounded. Under moderate temperature and humidity conditions the cells about and underneath injuries change chemically and physically so that they are impervious to water and resistant to infections. The walls of the cells become corky and additional layers of new cells are formed under and about the wound. This stops loss of tissue moisture and affords as much protection against decay-producing organisms as did the original skin.

Under optimum conditions this corky, protective tissue develops within 24 to 48 hours after the injury. However, at 50° F and below and above 95° healing does not take place rapidly enough to offer much protection against wound parasites. For this reason it is

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[2] For °C, see conversion table inside back cover.

advisable whenever possible to hold newly harvested potatoes at moderate temperatures and at as high a humidity as possible for a few days to permit wound healing before storage at low temperature.

The quality of potatoes depends not only on the variety and maturity but also on how they are harvested and handled immediately afterward. When tubers are badly cut and bruised during harvest, they discolor, wither, and decay much more rapidly than uninjured tubers, particularly early potatoes harvested during hot weather. Heat injury or scald of tubers left exposed in the field before picking up often greatly damages the early potato. Extreme heat causes death of cells with subsequent breakdown and decay by various organisms. Sublethal temperatures predispose the tubers to infection by soft rot bacteria.

Sometimes part of the northern late-potato crop is injured by low temperatures before and during harvest. Mild or severe types of freezing injury also predispose tubers to bacterial soft rot when they become warm. Unless tubers that have been injured by freezing are graded out before storage, they may be invaded by any one of several decay-producing organisms. Handling operations that avoid both mechanical injuries and temperature extremes are necessary therefore to maintain good-quality potatoes and reduce losses.

During transit and storage it is not necessary to refrigerate potatoes at very low temperatures. In fact, potatoes are of better cooking quality when they are held at 50° to 60° F than at 36° to 40° as usually recommended for storage. However, the latter temperature range is advisable for long storage periods in order to hold in check sprouting and development of decay.

BACTERIAL DISEASES

Bacterial Soft Rot (Slimy Soft Rot)

Erwinia carotovora (L. R. Jones) Holland and Other Bacteria

Bacterial soft rot is one of the most serious postharvest diseases of potatoes. It may cause severe losses during storage, transit, or marketing. No variety is immune to attack. The disease occurs wherever potatoes are grown in the world.

Symptoms

Soft rot first appears as tiny tannish or water-soaked spots on the potato skin. These spots rapidly enlarge and as the tissue beneath

decomposes, a soft blisterlike area develops on the surface of the tuber. Often a slimy or watery exudate oozes from breaks in the paper-thin skin of the blister (pl. 1, *A*). On light-skin potatoes this blister is surrounded by a brown margin; on red-skin potatoes the margin usually is black. With slight pressure on the blister the decomposed flesh beneath will squirt out.

When the tuber is freshly cut, the decomposed flesh usually is creamy, but it will soon turn pink and finally brown to black (pl. 1, *B*). The flesh is mushy, but not watery, and sharply separated from healthy tissue by a black border. The odor at this stage is almost normal. As the disease progresses, black mushy decay may consume most of the potato and the skin above the decay collapses. Then foul-smelling chains or strings of decomposing flesh and bacteria will ooze out (slimy soft rot). Finally under drying conditions the mushy tissue dries to a chalky-white foul-smelling mass (pl. 1, *C* and *D*).

Bacteria that cause soft rot also invade lenticels of potatoes swollen by long exposure in wet soils or deep washing vats. Infected areas around the lenticels may be up to one-fourth inch in diameter, slightly raised above the normal skin, and dark brown to black, particularly on red-skin varieties (pl. 1, *E*). Flesh beneath these areas normally is firm and appears yellowish to cream color or water-soaked. When such tubers are held at high temperatures, the infected flesh becomes mushy and typical bacterial soft rot develops. At low temperatures or under drying conditions these lenticel infections usually dry up, leaving a shallow spot with normal skin color but with a chalky-white deposit under the skin (pl. 1, *F* and *G*).

Causal Factors

The soft rot bacteria attack a wide variety of truck crops and are common in the soil. During harvesting and packing, the tissues are contaminated with these bacteria by means of soil, tools, hands, or wash water. Not only are the bacteria present as contaminants in the field but they cause active rots there, and much of the rot developing in transit and storage actually originates in the field.

Since the soft rot bacteria invade potato tubers chiefly through injuries, practically all infections are in tissues weakened or killed either by heat injury, freezing, mechanical injury, and enlarged lenticels or by lesions caused by other diseases. Soft rot almost invariably appears in potato tubers injured by heat in the field during harvesting and loading. Not only killing temperatures of 122° F and above but also sublethal temperatures of 109° to 113°

predispose potato tubers to soft rot. Lethal temperatures can occur in potato flesh when air temperatures are no higher than 90°. Both field and laboratory tests have shown that potatoes dug, hauled, graded, and sacked while hot are much more likely to become infected and eventually to decay than similar potatoes handled at moderate temperatures. Even potatoes subjected to sublethal temperatures develop less soft rot if they are not handled until after they become cool again.

Causal Organisms

Bacterial soft rot of potatoes can be caused by *Erwinia carotovora* (L. R. Jones) Holland and *E. atroseptica* (van Hall) Jennison. Since these organisms can liquify pectin materials, it has been suggested that they be placed in the newly created genus *Pectobacterium* (Waldee) instead of *Erwinia*. This suggestion, however, has not been generally adopted. The *Erwinia* species are gram-negative rods about 0.6 by 1.5 μ with four to six short, inconspicuous, peritrichous flagella. Although both species cause soft rot of potatoes, only *E. atroseptica* produces blackleg. On synthetic media the two species can be readily differentiated. *E. carotovora* will utilize 5 percent of ethyl alcohol and dulcitol and 1 percent of sodium hippurate, sodium malonate, and sodium urate as growth factors; *E. atroseptica* will not. *E. atroseptica* produces more acid from maltose than *E. carotovora*.

Species of *Pseudomonas* and *Bacillus* also cause soft rot of potatoes. The *Erwinia* and the *Pseudomonas* species differ in that the latter has polar flagella, will not ferment lactose, and will give a blue-white fluorescence in media containing aspargine. The *Bacillus* species differ from both of these genera since they are gram-positive rods about 0.8 by 2-3 μ, often contain spores, form long chains of cells, and grow under anaerobic conditions. Certain species of *Clostridium* are also found on rotted tubers. They are rods about 0.5-8 by 3-7 μ, which grow usually under anaerobic conditions. Generally they produce the foul odor associated with rotted potatoes.

The *Erwinia* and *Pseudomonas* species can grow and produce soft rot of potatoes from about 32° F to slightly above 90°. The optimum for growth and infection is from 70° to 80°. The *Bacillus* species on the other hand have optimum growth from 86° to 97° and will develop up to about 122°. Usually they are secondary invaders following the *Erwinia* or other organisms and may be partially responsible for the foul odor of the decayed tubers.

Bacterial soft rot organisms can spread from diseased to healthy tubers during transit, storage, or marketing.

Control

As with many other storage diseases, the control of bacterial soft rot begins in the field. It is necessary to control such field diseases as late blight, brown rot, ring rot, and blackleg, which produce lesions in tubers through which infection by the soft rot organisms may occur. Furthermore, the tubers must be protected during harvest from bruising and exposure to excessive heat or to freezing. Rotting tubers should be sorted out in packing. If possible, potatoes that have been washed should be dried before being stored or shipped. Ventilation and moderately low temperature during transit and storage are essential in controlling bacterial soft rot.

Storing tubers near 60° F for the first 4 to 5 days will allow injured surfaces to heal and will restrict the entrance of bacterial organisms. After healing, tubers should be stored at 40° in well-ventilated bins. Moderate refrigeration of early potatoes during transit in hot weather is beneficial, since decay does not develop as rapidly at 60° and slightly below as at higher temperatures.

References: *14*, *36*, *41*, *55*, *62*, *123*, *134*, *135*, *148*, *151*, *182*, *183*, *187*, *198*, *212*, *214*, *215*, *217*, *234*, *245*, *249*, *250*, *269*, *270*, *274*, *283*, *317*.

Blackleg

Erwinia atroseptica (van Hall) Jennison

Blackleg occurs wherever potatoes are grown. Its name comes from the jetblack lesion produced on the basal part of the stems. Tuber rots and discoloration of the vascular system make this an important market disease. It can be serious from a seed-certification standpoint, since infected seed pieces cause losses in stand and yield.

Symptoms

The blackleg organism enters tubers through the stolons and produces various symptoms.

The most pronounced symptoms appear on tubers infected about midseason. Jetblack slightly sunken lesions develop at the stem end, with blackened skin extending about half the length of the tubers (pl. 1, *H*). Normally the skin of intact tubers is dry and thin pieces may be peeled off. Internally the infected flesh of freshly harvested

tubers is at first cream color, then grayish, and finally black. It has the mushy texture of bacterial soft rot. Such infections spread from the stem end through the heart of the tuber in irregularly shaped patterns. A dark border separates the infected from the healthy flesh.

When infected tubers have remained for long periods in the fields or when they are stored, the blackened skin at the stem end breaks apart and the discolored flesh disintegrates, leaving irregular cavities with blackened walls, which may extend through the center of the tuber (pl. 2, *B*). These cavities differ from those caused by blackheart since they are always near or connected with the stem end. They differ from those produced by fusarium rots since no fungal growth is present.

In late-season infection, slight grayish-black skin lesions form at the stem end. The flesh beneath these lesions is yellowish to tan, is slightly water-soaked, and does not extend deep into the tubers (pl. 1, *H*; pl. 2, *A*). Regardless of the stage of tuber infection, a brownish discoloration of the vascular system often occurs (pl. 2, *B*).

The blackleg organism also may infect the lenticels, causing them to be slightly sunken and brownish to black. If the relative humidity is low, the rot around the lenticels is apt to be dry; if it is high, the rot will be moist. Lesions vary from one-eighth to one-fourth inch in diameter. Tissues beneath the lesion are brownish, disorganized, and usually dry; they extend about one-sixteenth of an inch into the healthy tissue. Sometimes the entire tuber is affected, but usually only one side.

Causal Factors

The exact conditions for severe outbreaks of blackleg are not known. The disease is tuberborne and may be transmitted from infected to healthy seed pieces by the cutting knife. Generally the disease develops most rapidly during cool, wet weather. Usually tubers infected early in the growing season decay before harvest, so that storage and seed-piece decay arise from late-season infections on tubers showing only slight blackleg symptoms. The seedcorn maggot (*Hylemya platura* (Meigen)) is reported to spread this disease in the field from infected to healthy plants.

Causal Organism

The blackleg organism *Erwinia atroseptica* (van Hall) Jennison is closely related to *E. carotovora*, which causes bacterial soft rot of

potatoes. The former is often called *Pectobacterium carotovorum* var. *atroseptica* (van Hall) Dowson. It usually does not spread from diseased to healthy tubers during storage, but it will remain dormant in tubers during storage and later cause serious seed-piece decay and plant losses. For a brief description of this organism, its distinction from *E. carotovora*, and its temperature requirements, see Bacterial Soft Rot.

Control

The best control for blackleg is to use certified seed. Since the disease-producing organism can be transmitted on a cutting knife, carefully avoid tubers with blackleg symptoms when seed pieces are cut. Tuber and lenticel rot can be controlled by low temperature and proper ventilation.

References: *16, 49, 110-112, 129, 171, 179, 183, 198, 218, 242, 270, 294, 296.*

Brown Rot (Southern Bacterial Wilt)

***Pseudomonas solanacearum* E. F. Sm.**

Brown rot may attack all varieties of potatoes with varying degrees of severity. In addition, it attacks eggplant, pepper, tomato, tobacco, peanut, and many other plants. It is primarily a disease of crops in the South Atlantic and Gulf Coast States from Maryland to Texas. For this reason, it is often referred to as southern bacterial wilt. However, the disease has been reported as far north as Massachusetts, Pennsylvania, Ohio, Michigan, Illinois, and Idaho.

Brown rot is principally a field disease, but it is sometimes serious in market shipments of the early or new potatoes from the South. The discoloration or slight decay of the vascular ring may result in reduced prices. Also, the potatoes may be invaded by secondary organisms and decay completely. Since practically none of them are stored, the disease is of little importance as a storage rot.

Symptoms

The external symptoms of brown rot may be a slight depression on the tuber where it was attached to the stolon or a grayish-brown patch on the surface (pl. 2, *C*). Sometimes no external symptoms develop. When infected tubers are cut at the stem end, a moist brown discoloration and a slight softening of the water-conducting

tissues can be seen (pl. 2, *D*). Frequently, though not invariably, yellowish-white sticky masses of bacteria ooze from the discolored vascular area without applying any pressure. At this stage there is no odor. Later, as the bacteria work toward the center of the tuber, the entire interior becomes soft and brown and only a thin crust of tissues holds the tuber together. If the rot reaches this advanced stage while the potatoes are in transit, adjacent tubers are likely to become smeared with the rotted content. In the later stages of the disease, secondary organisms gain entrance, the tissues become creamy in consistency, and a foul odor results.

The symptoms of brown rot are similar to those of ring rot. However, when a diseased tuber is cut, the presence of brown rot is indicated if white sticky droplets of bacteria ooze from the vascular ring and the decay tissues in the immediate vicinity of the ring are brown to black. Tubers affected with ring rot show no bacterial ooze from the vascular ring, but cheeselike decayed tissues can be forced out of the ring area by squeezing the tuber.

Causal Factors

The bacterium causing brown rot occurs in the soil. The disease is most severe in potato crops grown on newly cultivated land.

Warm, moist weather favors the spread of the disease. It develops at 77° to 97° F. Most rapid development is at about 90°. As the temperature is reduced, growth is retarded until it is practically stopped at 55°. Decay in transit is greatest in shipments of potatoes that have been dug during hot weather and not precooled sufficiently.

Causal Organism

Brown rot is caused by *Pseudomonas solanacearum* E. F. Sm. The bacteria are gram-negative rods 0.5 to 1.5 μ. They are motile with a polar flagellum. On agar the colonies have irregular margins and usually are small, smooth, glistening, and opalescent. At first they are white but later turn brownish. In broth a slight pellicle is formed and the broth turns brown. In culture the optimum temperature is between 95° F and 99° with a minimum of 50° and a maximum of 106°.

Control

Diseased tubers should not be stored or shipped even though infection may be only in its initial stages, because they rot very

quickly at 55° F and above. As it is practically impossible to discover and remove all tubers with brown rot in its early stages, potatoes from affected fields should be used immediately or shipped to nearby markets. Temperature during shipment should be below 55°.

References: *66, 67, 115, 203, 267, 268.*

Red Xylem and Pinkeye

Pseudomonas fluorescens (Trevisan) Migula

These diseases of potatoes are reported chiefly from the northern potato-growing areas. The discolored vascular ring and shriveling around the eyes may cause serious losses. These diseases are particularly pronounced in 'Katahdin' tubers.

Symptoms

Red xylem is most common in large tubers. A sunken but not discolored lesion occurs at the stolon scar and a cavity lined with blackened tissue may extend from this scar into the center of the tuber. The vascular ring has a thin reddish-brown line, and sometimes a network of discolored strands extends into the cortex (pl. 2, *E*). Red xylem is distinguished from stem-end browning and net necrosis since the last two discolor the phloem strands. Also, Katahdin is among the most susceptible varieties to red xylem and yet resistant to stem-end browning and net necrosis.

Pinkeye is characterized by pinkish blotches near the eyes and mostly around the blossom end of the tubers because of the pink underlying tissue. The internal tissue is firm to corky, and pink to reddish-brown and black areas and cavities may occur. During storage the discolored tissues under the eyes dry out, turn light brown, and finally become wrinkled and cracked (pl. 7, *E* and *F*). The xylem tissue of the affected tubers is about the same color as in red xylem. Pinkeye may be confused with late blight, but it does not have the brick-red granular tissue characteristic of blight.

The cause of these diseases is not truly known.

Causal Organisms

Red xylem and pinkeye are reported to be caused by *Pseudomonas fluorescens* (Trevisan) Migula, a bacterium commonly found in the soil. It apparently does not survive in stored tubers. Pinkeye in particular often is associated also with a fungus, *Verticillium*

alboatrum,[3] which causes wilting of the plants and vascular discoloration of tubers. Pinkeye is also associated with the disease caused by *Rhizoctonia solani*. Generally pinkeye occurs when the soil is very moist and is absent when it is dry.

P. fluorescens is a gram-negative rod about 1.5-2 by 0.5-0.8 μ with a polar flagellum. The rods occur singularly or in pairs. On agar plates the colonies are white to translucent, circular, and slightly raised. In a liquid medium the growth is at the surface with a pellicle formation. On agar the growth is filiform and slightly iridescent in reflected light. Growth temperatures range from about 43° to 99° F with an optimum near 80°. Strong fluorescence occurs when the organism is grown on medium with asparagine.

Control

No control methods are known

References: *20, 90, 91, 95, 98, 100, 141, 158, 242.*

Ring Rot

Corynebacterium sepedonicum (Spieck. and Kotth.) Skapt. and Burkh.

Ring rot is probably the potato grower's most feared disease and continues to be one of the most destructive since its discovery in Germany early in the 20th century. It was first reported in North America in eastern Canada in 1931 and in Maine in 1932. Because it is easily disseminated, it has since appeared in rapid succession in various parts of Canada and the United States until by now it is in all the major potato-producing States. It has caused extremely heavy losses.

Tubers with ring rot are commonly found at the terminal markets, and occasionally this disease is responsible for serious market losses. Extensive decay due to secondary invasion of soft rot bacteria also contributes to market losses.

Symptoms

The chief symptom of ring rot on tubers is a characteristic odorless decay, which is confined at first to the immediate vicinity of the vascular ring. This decay can readily be seen by slicing the tuber crosswise near the stem end. In the early stages of infection

[3] See Verticillium Wilt.

the vascular ring is cream to pale lemon yellow and approaches the color of the normal ring of a healthy tuber, but in later stages the ring is brownish. The affected areas of ring rot-infected potatoes are soft with a cheesy consistency (pl. 2, *F* and *G*). The bacterial cheesy material can readily be forced out of the affected vascular ring tissue by squeezing the tuber or by gently pressing a blunt stick or pencil against the vascular ring at the cut surface of the tuber. In more severely affected tubers the diseased tissue can readily be pushed aside, leaving a narrow cavity or cleft that extends down into the tuber and runs part way or entirely around the vascular ring (pl. 2, *H*). Dehydration follows with advanced lesions becoming dry and powdery. When these areas are invaded by soft rot bacteria, a soft mushy consistency may replace the dry powdery condition. Vascular cavities may extend to the pith or cortex and result in canker lesions on the tuber surface.

Causal Factors

The disease is spread by infected seed potatoes. The bacterium lives from season to season chiefly in tubers. It can survive in dried slime on crates, pallet boxes, bins, harvesting and grading equipment, and sacks. Contaminated cutting knives and harvesting equipment are chief sources of primary inoculum, with the causal bacterium invading the tubers through harvesting and handling bruises.

When the disease reaches the surface of the tuber near the eyes, a characteristic cracking affords an entrance for secondary soft rot bacteria, which spread through the vascular ring and eventually affect the entire tuber. The presence of secondary rots may result in a variety of discoloration symptoms near the vascular ring and thereby mask the symptoms of ring rot. Brown rot and ring rot symptoms are similar, and sometimes the two diseases are difficult to distinguish (see Brown Rot).

The incubation period for ring rot may be from 2 to 3 months. Consequently, infected tubers with no visible evidence of the disease at harvest may develop positive symptoms in storage. Although healthy tubers can be infected through fresh wounds that penetrate as deeply as the vascular ring, there is generally little, if any, spread from diseased to healthy tubers during storage. Ring rot develops in the tubers most rapidly at 64° to 72° F and only slightly at 37°.

Causal Organism

Ring rot is caused by *Corynebacterium sepedonicum* (Spieck. and Kotth.) Skapt. and Burkh. It is a nonmotile rod 0.3-0.4 by

0.8-1.0 μ. On agar the colonies are thin, smooth, translucent, and glistening white. They seldom exceed 3 mm in diameter. The organism grows best at 68° to 73° F with a minimum of 39° and a maximum of 88°.

Control

The best way to prevent ring rot is to plant disease-free tubers.

Potatoes known to contain any appreciable amount of ring rot should be moved early and promptly into marketing channels to prevent losses from advanced stages of ring rot and from secondary bacterial soft rot, which frequently follows. Storage at 38° F will control further development of ring rot and associated soft rots during the holding period.

Such varieties as Teton, Saranac, and Merrimack are resistant to bacterial ring rot.

References: *24, 29, 30, 39, 40, 103, 143, 159, 163, 170, 193, 230, 240, 265, 266, 272, 275, 279, 280.*

FUNGUS DISEASES

Armillaria Rot

Armillaria mellea (Vahl. ex Fr.) Kummer

Armillaria rot of potatoes has been reported from California, Florida, Michigan, Oregon, Washington, Wisconsin, Wyoming, British Columbia, and Australia.

Symptoms

The fungus that induces this rot produces dark-brown to black rhizomorphs or threads, which penetrate the corky layer of the tuber and generally adhere to the skin at various points. Affected tubers often have hard, brown, corky areas, which are roughened, shrunken, usually shallow, but sometimes they extend into the flesh a considerable distance. The diseased internal tissues are light brown and interspersed with convoluted, white, brown-edged plates of fungus mycelium.

Causal Factors

This disease only affects potatoes planted on newly cleared land rich in humus, and it is of little economic importance.

Causal Organism

A fungus, *Armillaria mellea* (Vahl. ex Fr.) Kummer, causes this disease. The mushroom stage is not seen on harvested potatoes. The fungus grows most rapidly in culture at 67° and 81° F. After long periods the greatest development of rhizomorphs in nonsterile potting soil was at the lowest temperatures.

Control

Control measures after harvest are not needed.

References: *8, 150, 156.*

Black Dot

Colletotrichum atramentarium (Berk. and Br.) Taub.

Although black dot is considered a minor disease of potatoes, it occurs in many potato-growing areas in the United States, Canada, Europe, Australia, and South Africa. Its symptoms, which overlap those of other potato diseases, make accurate evaluation of its importance difficult.

Symptoms

The fungus progresses down the stolons to the tubers in the soil. A common symptom is the adherence of sections of the affected stolons to the tubers at harvest. Sclerotia of the fungus are scattered or in patches on the surface of mature tubers, where their appearance is sometimes coincidental with silver scurf (pl. 3, *A*).

Causal Organism

The causal fungus *Colletotrichum atramentarium* (Berk. and Br.) Taub. has a branching, septate, slightly colored mycelium, which produces black sclerotia up to one-half mm in diameter. Acervuli, which are formed on the sclerotia, contain numerous prominent black setae or hairs, which are septate, tapered to the tip, and about 80 to 350 μ long. Conidiophores are 10 to 30 μ long, cylindrical, tapering to slightly clavate, sometimes curved, occasionally branched, and may be nonseptate to three septate. In mass the conidia are usually pink; individually they are hyaline or colorless, continuous, sometimes slightly curved, and sometimes attenuated at the basal end and round at the apical end. They are from 3 by 17 to 7.5 by 22 μ.

Control

No specific control measures are recommended.

References: *63, 210.*

Black Scurf (*Rhizoctonia*)

Pellicularia filamentosa (Pat.) Rogers

Black scurf is one of the several phases of rhizoctonia disease. It is one of the most common diseases of potato and is distributed worldwide. The causal fungus is responsible for other diseases on many species of cultivated plants, including beans, beets, cabbage, carrots, peas, and tomatoes. Damage to potato tubers results in poor appearance and a reduced market quality.

Symptoms

The most common symptom of black scurf on potato tubers is small, hard, coal-black bodies called sclerotia, which are tightly appressed to the tuber skin (pl. 3, *B*). These sclerotia are the resting or overwintering bodies of the fungus and range from as small as a pinhead to as large as a half pea.

Another symptom of this disease on potato tubers is the development of small, hard, sunken pits about the size of a pea. This condition is the result of lenticel penetration by the fungus. These areas can generally be lifted out with the point of a knife. Although this injury is common, it rarely causes serious losses. Still another symptom is a russeting of the tuber surfaces similar to that caused by the shallow form of common scab. This is often referred to as russet scab and commonly occurs in heavy soils. The disease impairs the appearance of potato tubers, particularly when they are washed, and thereby reduces their acceptance as table stock potatoes.

Following injury by the potato flea beetle (*Epitrix cucumeris* (Harris)), the causal fungus often produces deep cracks and furrows by invading the insect channels and punctures. Also, holes made by wireworms may be invaded and result in deepened pits and channels.

Although the disease seldom causes a tuber rot, a dry, punky decay has been reported at the stem end of tubers. Sometimes *Rhizoctonia* may be associated with other fungi or bacteria and a

jellylike rot may occur in the succulent pointed ends of the Russet Burbank variety.

Causal Factors

The causal fungus comprises a large number of physiologic races, or strains, which differ considerably in their environmental requirements. The disease is more severe in cool than in warm seasons. Frequently stems and roots that have few sclerotia in the summer are covered with them when the weather becomes cool. Therefore sclerotia may be abundant if tubers remain in the soil for a long period in the fall. This is particularly a problem in the northern potato-growing areas.

Rhizoctonia occurs in most soils and may live saprophytically on decaying plant debris when a living host is not available. Both the pathogen in the soil and the sclerotia carried on the seed tuber may be responsible for propagating the disease. However, there is evidence that the fungus is less virulent when propagated from sclerotia.

Causal Organism

Black scurf is caused by the basidiomycete *Pellicularia filamentosa* (Pat.) Rogers described originally as *Hypochnus solani* by Potouillard in France in 1891. Earlier the imperfect stage of this organism was termed *Rhizoctonia solani* Kuehn. The two were identified as different stages of the same organism early in the 20th century. The imperfect stage, which produces the black sclerotia on the tubers, occurs more frequently than the perfect stage. However, there is evidence that strains of the fungus from spores are more pathogenic than those from sclerotia.

The imperfect stage of the fungus, which grows well on artificial media, produces when young a hyaline mycelium, which becomes brown as the culture ages. The mycelial branches are constricted at the juncture with the main hyphae, and as the culture becomes older, the branches assume a right-angle relation to the main hyphae. On some media and under certain conditions the mycelium becomes tufted, dividing into short, ovate cells that eventually make up brown sclerotia.

The cardinal temperatures for the fungus in culture are an optimum of 77° to 86° F, a minimum of 46°, and a maximum of 88° to 95°. The sclerotia germinate at 46° to 86° with the optimum at 73°. The optimum for basidiospore germination is 70° to 77°.

Control

No completely effective control measures can be recommended for this disease. No resistant varieties are available, and chemical control is not totally satisfactory.

References: *38, 54, 94, 188, 258, 262, 284.*

Charcoal Rot

Macrophomina phaseoli (Maubl.) Ashby or *Botryodiplodia phaseoli* (Maubl.) Thirumalchar

Charcoal rot develops in potatoes grown in warm areas. In the United States serious losses from this disease occur on the market on potatoes from California, Georgia, Mississippi, Missouri, Tennessee, and Texas. Serious losses from this disease have been reported from India.

Symptoms

Externally charcoal rot appears first as black moderately firm areas near the stem end of tubers and as cloudy blotches around the eyes and lenticels. Eventually the entire skin of an infected tuber may blacken. Sometimes infected areas around the eyes and lenticels dry and form a slightly sunken, dark, dry rot. When the fungus progresses throughout the tuber, a semiwatery breakdown of the tissues occurs. Internally the freshly cut flesh is grayish or tannish yellow, but it soon changes to pinkish buff, then brown, and finally black (pl. 3, *C*). A dark or black border often separates diseased from healthy tissue. Small cavities mostly near the stem end sometimes occur. The infected flesh is firm, but with slight pressure yellowish-gray water can be squeezed from the affected areas. The decay appears somewhat like leak, but affected tissues are not as watery as leak and usually wounds are not visible.

Causal Factors

Charcoal rot occurs most often when heavy, poorly drained soils are waterlogged or excessively wet. Since high temperatures of the wet soils increase the development of this disease, the northern potato crop should remain free of the disease. Soil containing the organism and sticking to eyes or lenticels may make conditions favorable for infection.

Causal Organism

Charcoal rot is caused by *Macrophomina phaseoli* (Maubl.) Ashby, which recently was classified *Botryodiplodia phaseoli* (Maubl.) Thirumalchar. This organism will not infect potatoes below 43° to 50° F. The optimum for infection is between 89° and 97° and the maximum near 108°. On artificial media the colonies of this organism are at first grayish white but turn a blackish mouse gray at maturity. Young hyphae are hyaline and contain granular material. Older hyphae are brown gray. When pycnidia are produced, the pycnospores are at first one-celled and hyaline, but later they become two-celled and reddish brown. The organism does not spread from diseased to healthy tubers during storage or transit.

Control

Low storage or transit temperatures will prevent the development of charcoal rot, but when tubers are moved to higher temperatures, decay follows rapidly. Forced-air circulation will lower storage temperatures rapidly and help to dry the surface of the tubers.

References: *15*, *35*, *79*, *233*, *285*, *286*, *301*.

Early Blight (Alternaria Tuber Rot)

Alternaria solani (Ell. and G. Martin) Sor.

Although the early blight pathogen was first described by Ellis and Martin in 1882 and the disease was extensively studied by Jones in Vermont during 1891-1903, the tuber rot phase was not described until 1925 by Folsom and Bonde in Maine.

The disease is general in potato-growing areas, but the tuber rot phase is sometimes more serious in the northern late-season crop areas, where potatoes are harvested and stored during cool, humid weather. Early blight tuber rot is a serious storage and market disease and often predisposes tubers to secondary infection by other pathogens such as the *Fusarium* species.

Symptoms

Early blight lesions on tubers are dark brown to black, of several different shapes including circular to oblong, and about one-fourth to 2 inches in diameter. Often they are slightly sunken and have raised purplish borders (pl. 3, *D*). The lesions on a tuber vary from

one to many. The affected areas may be similar to those caused by the late blight organism, but early blight areas are shallower and sharply set off from the healthy tissue by a layer of cork. The decay does not spread irregularly into healthy tissue as does late blight. The flesh of the tubers one-eighth to one-half inch deep beneath the lesions is black and often surrounded by a yellowish zone (pl. 3, *E*). The lesions may cover the eyes of the tubers and kill the sprouts. No fungus sporulation is evident over the tuber lesions. Cracks may develop on older tubers.

Causal Factors

The causal fungus overwinters as mycelium and conidia in the soil and on potato tubers. Initial plant infection occurs on vines in the field. Fungus spores are abundantly produced in leaf lesions when rain or dew is frequent and when it is about 78° F in the field. If the disease is uncontrolled in the field, the tubers become inoculated with spores during harvest. Unbruised as well as bruised tubers are subject to infection from this inoculation at harvest since the spores, which germinate under favorable temperature and moisture conditions in storage, can penetrate directly. However, under dry storage conditions, which prevent spore germination, infection is held in check and spread to unbruised tubers is unlikely.

Bruised tubers are most likely to develop decay under moist conditions at about 55° to 60° F. The optimum for decay development is about 60°, the minimum 41° to 45°, and the maximum 77°.

Causal Organism

Early blight is caused by *Alternaria solani* (Ell. and G. Martin) Sor., which was earlier called *Macrosporium solani* Ell. and G. Martin. *A. solani* is classified in the order Moniliales of the class Fungi Imperfecti.

The mycelium is septate, branches, and darkens with age. Conidia have long beaks and are uniform and dark. They are borne singly or in chains of two in pure culture on relatively short, dark conidiophores. The conidia are formed from buds that arise from the terminal cell of the conidiophore. The conidia range from 12 by 120 to 20 by 296 μ. The fungus produces an abundant yellowish to reddish pigment, which diffuses through the substrate of artificial media. Conidia are sparse in uninjured pure culture, but abundant sporulation can be stimulated by wounding the mycelium or by

exposure to ultraviolet light. Pure cultures vary considerably in their virulence, growth rate, and sporulation.

The mycelium of the fungus remains viable in dry infected leaves for more than a year and conidia remain viable for up to 17 months at 68° to 70° F. They germinate within 1 to 2 hours in water at 43° to 93°. At an optimum of 82.5° to 86° germination will occur in 35 to 45 minutes. Cardinal temperatures for growth in pure culture are optimum 79° to 82°, minimum 34° to 36°, and maximum 99° to 113°.

Control

Chemical control of vine infections in the field is necessary to prevent tuber infection. Also, vine killing before harvest will reduce chances of tuber inoculation from infected vines.

Careful harvesting and handling will reduce tuber disease spread.

Maintaining storage temperatures below 41° F will stop decay development in storage. Forced-air ventilation will reduce or eliminate the free moisture that is necessary for spore germination and the resulting spread of infection in storage.

References: *23, 38, 89, 113, 228.*

Fusarium Tuber Rots

Fusarium spp.

Fusarium tuber rots probably cause greater storage and transit losses, particularly in late potatoes, than any other postharvest disease of potatoes. Although some types of this rot may develop on potatoes in the soil causing a stem-end decay, most infections occur through wounds during the harvesting and storing of this crop. The tuber rots develop in storage or in transit to market.

Symptoms

The symptoms of fusarium tuber rots vary greatly with different species of causal fungi. Generally at first the infected tissues are light brown and become dark brown with age. The decaying tissue becomes sunken, the skin wrinkles, and tufts of mold may appear at the eyes, lenticels, wounds, and stem end (pl. 3, *F-G*). At low storage temperatures the affected tissues are usually dry and firm, sometimes even powdery, but at high temperatures they are often moist and pliable. Secondary bacteria frequently invade the affected tissues and cause rapid breakdown of the tissues and wet rot.

In some types of fusarium tuber rot the causal organism invades the central part of the tuber, breaking down the tissues and eventually forming cavities lined with brightly colored fungal growth. This may be white, yellow, pink, rose, or bluish purple depending on the species (pl. 3, *H*). Often the external appearance of the tuber fails to give a clue as to the extent of internal rot. In the buttonhole-type rot the external symptom may be a small circular lesion approximately one-eighth to one-fourth inch in diameter (pl. 4, *A* and *B*). Sometimes it is extremely difficult to find a break in the skin and the area may be only darkened; paring reveals a small area of dry rot. These small circular dry areas may affect much of the tuber skin.

Causal Factors

Many species of *Fusarium* rot potato tubers and all are soil inhabitants. Several species may be present in all potato-growing areas, but one species is often predominant in a given soil or locality.

Most of the fusaria are wound parasites and consequently become destructive only after harvest. Rough handling during harvest, storage, or transit injures the tubers so the fungus can enter, infect, and cause decay. The mycelium and spores of *Fusarium* are commonly in the soil, on the tubers, equipment, and containers, and on the floors and walls of storage bins.

Since the fungus spores are always present and some skin breaks generally occur, temperature and moisture conditions determine whether or not infection develops. These conditions affect both the causal fungus and the potato. At 40° F wound healing proceeds so slowly that the host offers little resistance to penetration and infection. At 60°, on the other hand, wound healing is rapid enough to inhibit infection. Although most fungus species that cause tuber rot grow best between 78° and 86°, they can grow slowly and cause rotting below 50°. Near 40° infected tubers show little or no progressive decay in storage. However, at 40° the fungus is not killed but remains dormant in the wounds to become active whenever the tubers become warm. Consequently, under favorable conditions (above 70°) for the organism, potatoes may begin to exhibit visible decay within 4 or 5 days, and more than half of an average-sized tuber may be decayed in 10 to 15 days.

Causal Organisms

The genus *Fusarium* is classified in the order Moniliales of the class Fungi Imperfecti. This is a large and variable genus that is

sometimes placed in the Tuberculariaceae because some species produce sporodochia.

The mycelium of this genus is extensive and cottony, often with some tinge of pink, purple, or yellow. The color often permeates the artificial media on which the fungus is cultured. Conidiophores are variable, slender, and simple, or they are stout, short, and irregularly branched or bearing a whorl of phialides. They may be single or grouped into sporodochia. Conidia are hyaline, variable, and principally macroconidia and microconidia. Often they are held in a mass of gelatinous material. Macroconidia have from two to several cells and are slightly curved or bent at the pointed ends and typically boat shaped. Microconidia are one-celled, ovoid or oblong, and borne singly or in chains.

The *Fusarium oxysporum* Schlecht group is general throughout the U.S. potato-growing area, but it is more frequently found in the South and East. It also causes a stem-end rot of tubers and has been reported in the Northeast, East, Midwest, Southwest, and Rocky Mountain region.

F. solani (Mart.) Appel and Wr. var. *eumartii* (Carpenter) Wr. has been reported as the cause of stem-end rot from New York to Maryland and in Colorado, Idaho, Montana, Nebraska, Oklahoma, Wisconsin, and Wyoming. *F. solani* var. *radicicola* (Wr.) Synd. and Hans. causes a jelly-end rot in the Western States and a black rot of tubers in the South and East.

F. coeruleum (Lib.) Sacc., *F. sambucinum* Fckl., and *F. trichothecioides* Wr. all cause a dry rot in storage, transit, and marketing. *F. coeruleum* is common in the U.S. northern potato areas and in Europe. It causes most of the fusarium rot on potatoes in storage and often is associated with other fusaria. The species is the most vigorous wound parasite of potato tubers. *F. sambucinum* is general in the Northern and Central States to Texas and Idaho, whereas *F. trichothecioides* has been reported from Ohio and Michigan to Missouri, Arizona, California, and Washington. This species sometimes causes a field rot and wilt.

The microconidia of *F. oxysporum* are abundant and are produced in false heads. Chlamydospores are present. When grown on potato-dextrose agar (PDA), the cultures may produce a violet pigment in the agar. Blue-black sclerotia may be present. The sporodochia, when present, are from cream to salmon colored.

The microconidia of *F. solani* var. *radicicola* are nearly straight near the base, slightly curved in the upper third of their length, somewhat rounded to distinctly constricted at the apex, slightly pedicellate, mostly three-septate, and 31-40 by 4.6-5 μ. The nonseptate and one-septate microconidia are very common; the

nonseptate measure 8 by 3 μ. Chlamydospores are common, terminal and intercalary, and mostly nonseptate and one-septate. The nonseptate measure 9-10 by 8.7-8.8 μ. The aerial mycelium is well developed. The conidia range from white to olive and the substratum from pale yellow to olive on agar rich in glucose and on tuber plugs.

The microconidia of *F. coeruleum* are nonseptate and about 16 by 4.7 μ. Macroconidia are mostly of an even diameter or somewhat broader near the base, only slightly curved near the apex, somewhat rounded at the apex, never apically constricted, mostly three-septate, and 30-36 by 4.5-5.4 μ. Aerial mycelium is usually medium to well developed, feltlike with age, from white, bluish white, and olive buff to dusky slate violet on PDA rich in glucose; it is slate purple on cornmeal agar. The substratum on PDA rich in glucose ranges from deep violet to ocher red and in older culture is violet carmine. Conidia range from orange on strong acid agars to pale buff, light gray, or blue on neutral media.

F. sambucinum conidia are nearly uniform in diameter, sickle shaped, gradually attenuated, often somewhat constricted at the apex, pedicellate, mostly three- to five-septate, 28.5-41 by 4.2-5.2 μ, and are borne on aerial mycelium in sporodochia. Chlamydospores are rare and not in long chains. The aerial mycelium ranges from poorly to well developed and no red is in the mycelium or substratum.

In *F. trichothecioides* the microconidia are absent. The macroconidia are of both comma and sickle shapes, with the comma shape predominating under ordinary cultural conditions, are mostly one-septate, 14-17 by 4.2-5.4 μ, often nonseptate to three-septate, and seldom four- or five-septate. The sporodochial conidia are sickle shaped, three- to five-septate, and 24-42 by 4.5-5.5 μ. The chlamydospores are few and not prominent. Conidia are produced in powdery masses on aerial mycelium.

The prevalence of the disease in storage appears to be increasing as more mechanical harvesters are operated improperly. This coupled with the perennial stone or clod problems in some potato-growing areas contributes to small nicks, cuts, and bruises in the potato tubers, which act as entrance points for the causal fungus.

Control

Reduction of injury during harvesting, transportation, and storage is essential to control fusarium tuber rots. This can be accomplished by eliminating rocks, reducing harvesting equipment

speeds, using padded equipment, reducing the height of tuber drops, and warming potatoes before handling. However, even with the most elaborate precautions some tubers will be bruised. This emphasizes the necessity of proper curing near 60° F with high relative humidity during the initial storage period before temperatures are lowered for holding.

References: *1, 6, 7, 31, 45, 93, 105, 146, 147, 178, 186, 187, 196, 220, 227, 229, 242, 255, 272, 290, 307, 315.*

Fusarium Wilts

Fusarium oxysporum Schlecht f. sp. *tuberosi* (Wr.) Synd. and Hans. and *F. solani* (Mart.) Appel and Wr. var. *eumartii* (Carpenter) Wr.

Fusarium wilts have been reported from most potato-growing areas in the United States as well as Canada, Europe, India, Australia, and South Africa. These diseases reduce the market quality by discoloring the vascular system of the tubers. The usefulness of affected tubers for processing, especially for chip manufacture, is seriously reduced.

Symptoms

The causal organisms that enter tubers through the stolons cause different symptoms of the disease.

Tubers infected with *Fusarium oxysporum* f. sp. *tuberosi* usually have no external symptoms, but internally they show different degrees of discoloration at the stem end. The color of the xylem or woody vascular ring varies from a slight yellow or brown at the point of the stolon attachment to a dark brown to black. This discoloration extends from two-thirds to all the way through the tubers (pl. 4, *C*). Sometimes this necrosis also extends to smaller vascular elements on either side of the vascular ring and may resemble net necrosis. Frequently the vascular ring becomes hard, woody, and dark brown, and splinterlike pieces of tissue can be removed with a knife point.

Fusarium solani var. *eumartii* first causes a brownish skin discoloration at the stem end, which later becomes sunken or wrinkled and even may rot. Below this discolored or rotted area the vascular ring ranges from a yellowish brown to dark brown and is water-soaked. The band often extends throughout the tuber and is from about one-eighth to one-fourth inch wide (pl. 4, *D*). Sometimes the water-soaked brownish area causes brown flecking in

tissue adjacent to the vascular ring. In very mild cases this organism also may cause a light-brown discoloration of the vascular ring similar to that caused by *F. oxysporum* f. sp. *tuberosi*.

Causal Factors

Organisms causing fusarium wilts can live saprophytically in the soil on plant debris for many years.

The two types of fusarium wilt occur when potatoes are planted in contaminated soil or when infected seed pieces are planted. Tubers from soils with a high moisture content have more vascular discoloration than those from dry soils. Wilt caused by *F. oxysporum* f. sp. *tuberosi* is apt to be in warm soils, whereas wilt produced by *F. solani* var. *eumartii* is in cool soils. Vascular ring discolorations are present when the tubers are harvested. There is no spread from diseased to healthy tubers during transportation or storage, but the killed tissues are subject to invasion by secondary organisms. The discoloration caused by *F. oxysporum* f. sp. *tuberosi*, however, becomes more intense when tubers are stored at 50° F, and a wet rot may occur when infected tubers are stored at 60° to 70°. The disease caused by *F. solani* var. *eumartii* is favored by high storage temperatures and high relative humidity. At 41° the disease is completely checked and below 50° there is only slight growth. Above 50° with high humidity the disease increases.

Causal Organisms

Fusarium oxysporum Schlect f. sp. *tuberosi* (Wr.) Synd. and Hans. grows best on artificial medium at comparatively high temperatures with an optimum for growth between 78° and 89° F, a minimum of 41°, and a maximum of 104°. The optimum soil temperature for infection of roots is 86° or above. *Fusarium solani* (Mart.) Appel and Wr. var. *eumartii* (Carpenter) Wr., on the other hand, does not require a high temperature, with an optimum for growth near 77°, a minimum near 41°, and a maximum between 91° and 95°. The optimum soil temperature for infection of roots is 68° to 77°, the minimum is 53° to 59°, and the maximum is about 86°. The maximum soil temperature for infection by *F. solani* var. *eumartii* therefore is near the optimum for infection by *F. oxysporum* f. sp. *tuberosi*. Sometimes *F. avenaceum* (Fr.) Sacc. is also the cause of these wilts.

Control

Control consists of field rotation to reduce fusaria living in the soil and avoidance of fields known to harbor the organisms. Storage

below 50° F may lessen the development of vascular discoloration, which can occur when tubers are stored at higher temperatures.

References: *74, 75, 106, 107, 124, 126.*

Gray Mold Rot (*Botrytis*)

Botrytis cinerea Pers. ex Fr.

Gray mold rot usually occurs on potato tubers in storage, but it may also appear in the field as a shoot blight and stem girdle. Although the disease is considered to be minor in nature, it has been reported in Maine, Connecticut, Ohio, Idaho, and Alaska. The pathogen causes serious losses on other cultivated crops, such as lettuce, escarole, endive, globe artichokes, and various fruits. Under favorable conditions of low temperature and high humidity the fungus may occur in any region.

Symptoms

In the initial stage of rot a pinkish-buff to brown semiwatery breakdown of tissue may occur in any part of the tuber (pl. 4, *E*). As the rot progresses the tubers become slightly withered and flabby with grayish mycelium and masses of spores of the causal fungus appearing at the stem end or near the eyes and in bruised areas (pl. 4, *F*). Toward the end of the storage period, black irregularly shaped sclerotia may appear in the surface growth of the fungus.

Causal Factors

When a blight of the potato foliage and stems occurs in the field, tuber infection can apparently result from the fungus growing down through the stolons into the tuber stem end. This probably accounts for the commonly occurring stem-end infections. When potatoes are harvested without killing infected vines, the tubers can become contaminated. Any inoculated wounded tubers may become infected when high humidity and low temperature prevail. However, under the normal temperature-humidity conditions prevailing in most areas during harvest the bruised tubers can develop wound periderm or new cells soon enough to prevent invasion by the fungus.

Causal Organism

The causal organism *Botrytis cinerea* Pers. ex Fr. is a widespread facultative parasite, which may comprise many strains. The

mycelium, which grows rapidly in artificial culture media, is at first hyaline but becomes gray with age. The conidiophores are long and slender, arising as branches of the mycelium. They are hyaline at the tip and gray below the sporiferous region. Several branches formed just below the tip rebranch one or more times, and the apical cells enlarge and become round. Short sterigmata arise from the apical cells bearing clusters of conidia. After the conidia mature, sterigmata and branches collapse back to the main stalk, where they are cut off by a septum. A scarlike projection remains on the main trunk and marks where the branch originated. This process continues to form new conidiophores and to produce new crops of conidia. The conidia are hyaline or ash colored and appear gray in mass. They are continuous and ovate, ranging from 8 by 11 to 11 by 15 μ. Sclerotia may be produced. They are hard, black, irregular in shape, and about the size of a barley kernel or larger.

Control

Contamination of tubers at harvest can be prevented by vine killing prior to harvesting. Any tubers exhibiting stem-end infection at this time should be discarded. Proper curing of tubers at about 55° to 60° F and high humidity for 3 to 7 days before lowering the storage temperature will allow for wound periderm formation in mechanically bruised tubers. This recommended normal procedure will help to control other diseases such as fusarium tuber rots.

References: *86, 116, 232.*

Late Blight

Phytophthora infestans (Mont.) d By.

Late blight was first reported about 1830, when the disease appeared almost simultaneously in the United States and in Europe. Since then it has been reported in most areas of the world. Destruction of potatoes by this disease contributed substantially to the famine in Ireland about 1845.

Late blight is still one of the most important diseases of potatoes. During wet, cool seasons it may be responsible for serious crop losses in any potato-growing area. It is commonly found and sometimes causes serious losses of early potatoes in the Atlantic and Gulf Coast States, in the Rio Grande Valley of Texas, and in some potato-growing sections of the Pacific Coast States. Normally, however, the most serious outbreaks of late blight occur in the late potato-producing States extending westward from the New England

States through the Middle Atlantic States to and including the States bordering the Great Lakes.

Symptoms

On potato tubers, late blight appears as purplish-brown, reddish-brown, or brown areas, which, as disease development continues, become distinctively shrunken and darker (pl. 4, *G*). In storage, late blight tuber rot is moderately dry, firm, or leathery. Sometimes severely infected tubers feel "rock like," and thin layers of the dried infected skin can be peeled with a knife. This rot usually does not extend more than one-eighth to one-fourth inch into the tuber flesh. However, in newly harvested potatoes, particularly those of the early southern crop, the yellowish, brick-red, or reddish-brown granular breakdown often penetrates to a depth of one-half inch or more (pl. 4, *H*; pl. 5, *A*). The decay may also occur in the tissues at the stem end as a broad reddish-brown ring, and in advanced stages the tuber may become a shriveled mass. In newly harvested stock or in stock held under very moist conditions, white tufts of sporangiophores or fungus growth often appear at the lenticels, skin breaks, or eyes of the tubers.

Another effect of the disease in storage is the wet rot phase, which is due to the invasion of secondary bacteria following late blight development. This phase develops rapidly under warm, humid conditions and causes a very wet, soft, or slimy and foul-smelling rot. When other fungi, such as species of *Fusarium*, follow late blight as secondary pathogens, an odorless dry or wet rot may occur. The character of this rot depends on prevailing temperature and moisture conditions.

Causal Factors

Tubers are infected in the potato hill by late blight spores washed by rain from the diseased foliage into the soil. They are also contaminated with spores of the fungus if they are dug while diseased vines are still green or if they are piled in the field and covered with diseased vines.

The powdery scab organism forms sites for entrance of the late blight organism, particularly if the tubers are resistant to the blight organism.

The presence of spores on a tuber does not necessarily mean that the tuber will become infected. Infection develops only if temperature and moisture conditions are favorable. Under dry conditions below 40° F or above 86° the fungus spores will not

germinate. They may lie dormant on or in the tubers for many months and germinate and produce symptoms only when the environmental conditions become favorable. As a result, tubers infected or contaminated with spores of the late blight organism may appear healthy when they are placed in storage or in transit, but late blight may be present when they are removed from storage or on arrival at terminal markets.

During storage above 41° F with high humidity the fungus grows from diseased tubers and produces spores that can infect healthy tubers directly through the unbroken skin. At 60° or 71° with high humidity, lesions on tubers enlarge at the rate of 2 inches in 10 days. If humidity is low during storage or transit, the fungus is not apt to spread from tuber to tuber.

Potato tubers infected by *Phytophthora infestans* secrete a liquid that favors the growth of soft rot bacteria and thereby causes the wet rot phase. If the invading bacterium is pathogenic, such as *Erwinia carotovora*, the wet rot spreads beyond the fungus rot, whereas if the bacterium is saprophytic, the wet rot is confined to the zone affected by *P. infestans*.

Causal Organism

Late blight is caused by *Phytophthora infestans* (Mont.) d By., a member of the Phycomycetes.

Sporangiophores, which are hyaline, branched, and indeterminate, arise through the lenticels and eyes of the tuber. The sporangia are thin walled, hyaline, and oval with an apical papilla or beak. They range from 21 by 12 to 38 by 23 μ and are borne on the tip of a hyphal branch (sporangiophore), which, as it approaches maturity, swells slightly, proliferates, and turns the attached sporangium to the side as elongation of the sporangiophore proceeds. The sporangium may germinate directly by means of a germ tube, but most commonly the germination is indirect with cleavage within the sporangium, which forms eight biciliate zoospores that emerge together.

The sporangia are produced at 37° to 79° F with an optimum of 64° to 72°, and a minimum relative humidity of 91 percent with an optimum of 100 percent. Sporangia germinate directly by means of germ tubes at 77°, whereas indirect germination by means of zoospores occurs at an optimum of 54°. Zoospores germinate best at 54° to 59°. After germination the zoospore germ tubes grow most readily at 70° to 75°.

The fungus is generally cultured on frozen lima bean agar, on which it produces white fluffy growth at 68° F.

Control

When late blight is prevalent in potato vines, harvest should not begin until the infected tops have been killed by frost or by a herbicide vine killer, or they have died from natural maturity. Tubers with any evidence of late blight infection should be discarded during harvesting and storage.

In storage the holding temperatures should be maintained as close as possible from 36° to 40° F. The wet rot phase can be controlled with forced-air ventilation, which prevents the buildup of moisture in storage. Forced ventilation also aids in maintaining a uniform temperature throughout stored potatoes. By keeping the storage environment relatively dry, forced-air ventilation also prevents sporulation of the late blight fungus and the subsequent spread of the disease from diseased to healthy tubers.

Washed lots of tubers containing late blight had less rot when dipped in water at 120° F for 2 minutes and dried in an evaporating chamber with an air temperature of 133° for 5 minutes than those dipped in hot water only or washed in cold water.

As with all plant diseases, resistance is the ultimate in control methods. Several potato varieties resistant to late blight are available. They can be divided into (1) those possessing some resistance to the common race of the pathogen and (2) those immune to the common and certain other races of the pathogen but susceptible to some races.

In the first group are the varieties Menominee and Sebago. Although when compared with the varieties Irish Cobbler and Katahdin, these varieties display considerable resistance, but they cannot be used without protective fungicides. In the other group are the varieties Cherokee, Kennebec, and Pungo.

In some areas where late blight is an annual problem, new races of the fungus may arise suddenly. Under these conditions resistance will not hold up. Furthermore, resistant varieties are generally developed on the basis of foliage resistance, which does not necessarily give resistance in the tubers. Therefore from the standpoint of marketing problems, the value of resistant varieties is indirect by preventing foliage blight.

Although protective chemical spraying of the vines is not generally regarded as a control for blight of tubers, spraying sometimes effects a very considerable reduction in tuber infection. In some potato-growing areas the reduction in tuber infection has been as great as 50 percent.

Late blight control measures may be summarized as follows: (1) Use resistant varieties, (2) apply protective fungicide sprays in the

field, (3) do not harvest potatoes until the vines are dead or have been chemically killed, (4) cull out infected tubers before storage or transit, and (5) in storage hold potatoes at 36° to 40° F and use forced-air ventilation.

References: *1, 25, 27, 28, 46, 51, 58, 158, 166, 187, 202, 208, 220, 237, 257, 272, 289, 297, 326.*

Leak

Pythium debaryanum Hesse and *P. ultimum* Trow

Leak may occur wherever potatoes are grown, but it is of most importance in the North Central States and in California. It may cause serious losses at harvest, in storage, in transit, and on the market.

Symptoms

External symptoms of leak are usually around wounds or near the stem end. The affected area of red varieties becomes metallic gray and that of the white or dark-skin varieties turns brownish or water-soaked (pl. 5, *B*). Sometimes this area is slightly depressed. Internally the freshly cut affected tissue is grayish cream, but on exposure to air it turns reddish tan, then brown, and finally inky black (pl. 5, *C*). The diseased areas may be separated from healthy tissue by a dark-brown margin. The blackened tissue resembles blackheart, but it differs as it is extremely watery and with slight pressure a yellowish or brownish liquid is readily released. It is this characteristic that justifies the name leak. When the decay is found in stored potatoes, large quantities of juice from rotten tubers drip down on the pile.

Rarely is mold growth visible as in the wet types of fusarium rot, which may be confused with leak. Sometimes the entire inside of the potato may rot and leave an outer shell about one-fourth inch thick, even though only small external lesions are visible. This stage of the disease is called shell rot.

Causal Factors

Tubers become contaminated in the field. Infection takes place at harvest through wounds or bruises, which are often very inconspicuous. Sometimes infection can develop from the stolon. Hot, unusually wet weather favors the development of this disease. It is frequently found in tubers with sunburn or scald. Normally

leak does not spread from diseased to healthy tubers during storage or transit.

Causal Organisms

Leak is caused by *Pythium debaryanum* Hesse, *P. ultimum* Trow, and probably by other species of *Pythium*. They belong to the family Pythiaceae, the most primitive of the Peronosporales. Mycelium in the Pythiaceae has a diameter of 3 to 4 μ, rarely 7 μ, is abundantly and irregularly branched, and may be septate at maturity. On agar or on decaying produce the fluffy snow-white growth occurs. The sporangium usually is broader than the mycelium and is symmetrical. The sporangial wall is smooth, and in germination the sporangium discharges swarm spores into a thin-walled vesicle, which later ruptures allowing the fully formed spores to escape. The genus *Pythium* is closely related to *Phytophthora* but differs in the more substantial appearance of the hyphal contents and the looser branching of the mycelium. The optimum temperature for growth of these organisms is 77° to 87° F, the minimum 39°, and the maximum 104°. Entire tubers may be rotted in a few days at about 77°, but decay develops slowly between 40° and 50°.

Control

Losses from leak in storage, in transit, or on the market can be reduced by careful harvesting and handling methods and keeping the tubers as cool and dry as possible.

References: *3, 19, 109, 127, 153.*

Phoma Tuber Rot (Gangrene, Buttonhole Rot)

Phoma solanicola Prill. and Del. and Other *Phoma* Spp.

Phoma tuber rot, a storage disease at low temperature, is becoming increasingly more important in England, Ireland, and Scotland. It also has been reported in the United States, Australia, and in parts of Europe.

Symptoms

Infections occur first as numerous brownish to gray shallow depressions on the surface of the tuber (pl. 5, *D*). These circular spots may remain small and be corked off. Under favorable

conditions they may spread superficially over the surface of the tubers and remain shallow (pl. 5, *E*), or they may be restricted to a small surface area but extend deeply into the tuber and extensively rot the tissue. The internal and external affected areas are separated from healthy tissue by a dark border. Usually the rotted area is gray, dry, and hard or bony. It can be removed and leave clean cavities. The term "buttonhole rot" is applied when the sunken surface spot extends to the cavities in the flesh of the tubers. Often these cavities are lined with white fungal growth. Black fruiting bodies are in this whitish growth or on the tuber surface. Eventually a tuber may be reduced to a dry shell containing fragments of rotted tissue.

Causal Factors

Phoma rot does not occur at harvest but develops during storage. It is an important disease on tubers harvested during a wet growing season where mud sticking to the tubers contains the organisms and may result in infection. Cuts, bruises, and other injuries make excellent entrance points. Development of phoma rot also is associated with tubers infected with the powdery scab organism.

Causal Organisms

Phoma tuber rot is caused by *Phoma solanicola* Prill. and Del. and the related species *P. tuberosa* (Melhus) Rosenbaum and Schultz, *P. foveata* Foister, and *P. eupyrena* Sacc. *P. foveata* is limited to Great Britain and northern Ireland and *P. eupyrena* occurs in Europe and the United States. All these organisms produce decay more rapidly at 40° to 50° F than at higher temperatures. Decay usually will not develop when tubers are stored at 70°, possibly because the wound barriers develop rapidly at this temperature. Some of the decay caused by the dry type fusaria is believed by some workers to be primarily due to *Phoma* spp.

Phoma spp. have hyaline mycelium, which becomes septate and dark with age. Sclerotia several millimeters in diameter are produced in agar cultures. Pycnidia are dark and vary considerably in diameter from 70 to 180 μ and 175 to 420 μ. Pycnospores are hyaline, continuous, oblong, and 1.6 by 3 to 3.8 μ.

Control

Control consists of field rotations. Remove soil and other debris from tubers before storing. Store at 70° F and high relative

humidity for 7 to 10 days to stimulate development of wound barriers before storing at lower temperatures.

References: *4, 50, 84, 94, 197, 205.*

Pink Rot

Phytophthora erythroseptica Pethyb.

Pink rot is an important disease of potatoes in several European countries and in many other regions of the world. In the United States it is sporadic in areas where the soil normally is relatively cool. It can be an important rot of potatoes in storage, but it is rarely found on the market.

Symptoms

External symptoms occur usually at the stem end though sometimes eyes and lenticels are infected. In white-skin potatoes the infected area turns dark brown, whereas in red-skin potatoes the first symptom is a fading of the red. A black band occurs between the diseased and healthy tissue. Around the eyes and lenticels a blotchy purplish or black discoloration develops (pl. 5, *F*; pl. 6, *A*). The skin of the rotted portion is easily sloughed off by rubbing and exposes slightly darkened tissue with black spots beneath the lenticels. There is little shrinking of the tubers even though they are completely rotted. Rotted tubers may feel spongy or rubbery. When squeezed, droplets of liquid come from eyes and lenticels, and sometimes the droplets contain gas bubbles. Infected tubers may give off a pungent odor resembling formaldehyde.

When infected tubers are freshly cut, the tissue is granular and dull white with no demarcation line between healthy and diseased tissue. Within 15 to 20 minutes after cutting, infected tissue turns a salmon pink (pl. 5, *G*; pl. 6, *B*), then brown to black. The flesh of freshly harvested diseased tubers may be black when dug or turn black after a day or two at high temperatures. Often blackened areas are found about one-fourth inch below the surface and sometimes the central pith is blackened. The moist or watery condition of this tissue distinguishes pink rot from blackheart. Sometimes small cavities about one-eighth inch in diameter occur about one-half inch from the stem end. They do not extend deeply into the tuber and do not contain fungal mats.

Causal Factors

Pink rot occurs chiefly in cool, wet, poorly drained soils. Wet soils containing the fungus and adhering to tubers can cause eye or

lenticel infections during storage. The disease spreads rapidly when tubers are held under moist conditions in storage but does not spread if the tubers are kept dry.

Causal Organism

Pink rot is caused by *Phytophthora erythroseptica* Pethyb. The organism lives in tubers, field debris, and soil. On artificial media the organism can grow at 41° F with an optimum for growth near 77° and the maximum near 88°. Under moist conditions tuber decay develops from 43° to 86° with a maximum amount at 76°. *P. drechsleri* Tucker and *Pythium debaryanum* Hesse also can cause pink rot of tubers under certain conditions, but neither organism attacks growing plants. *P. erythroseptica* differs from many other *Phytophthora* species since it will grow at 87° and produce sexual bodies from sterile hyphae when transferred from pea broth to distilled water. The oospores are 31 μ, and growth is prevented by 1:8 million malachite green. For a more detailed description of *Phytophthora*, see Late Blight.

Control

Elimination of free moisture on tubers during storage will do much to reduce development and spread of this decay.

References: *18, 32, 42, 43, 108, 154, 155, 175.*

Powdery Scab

Spongospora subterranea (Wallr.) Lagerh.

Powdery scab was first reported in 1841 in Germany and by 1885 the disease was widely distributed in Europe. In 1891 it was found throughout its original habitat of Ecuador, Colombia, Peru, and Chile. The first report of powdery scab in North America was in 1913 from specimens in eastern Canada, and the disease was reported the same year in northern Maine. However, it was thought to have existed in Maine for 15 years before it was reported. Powdery scab has been occasionally reported since then from Alabama, Florida, Maine to Pennsylvania, Minnesota, Mississippi, Oklahoma, South Carolina, Washington, and Wyoming. In addition to North America, the disease has since become established in the U.S.S.R., South Africa, New Zealand, Australia, Rhodesia, Morocco, Algeria, Kenya, and Madagascar.

Symptoms

The first symptoms on the potato tuber are faintly brown raised areas about the size of a pinhead. In about a week these areas enlarge to about one-fourth inch, the brown disappears, and the diseased tissue becomes jellylike. In these areas under the skin the spore balls soon develop. When mature they become yellow brown to black (pl. 6, *C*), and the epidermis splits and peels back to expose the dusty spore mass formed beneath (pl. 6, *D*). Sometimes these pustules enlarge to form cankers in the tuber. In storage a dry rot develops in the pustule areas caused partially by a semisaprophytic growth of the pathogen and partially by invasion of secondary facultative parasites. No secondary infection by soft rot bacteria occurs, but severely affected tubers are likely to shrivel more rapidly in storage than normal ones.

Causal Factors

Under certain conditions the pathogen persists for undetermined periods in the soil. However, the fact that the disease has remained unimportant in the United States is good evidence that the pathogen is restricted to a rather narrow range of optimum environmental conditions. Periods of rainy, cloudy weather favor the development of powdery scab, particularly in poorly drained soil. Under warm, dry conditions the disease has reportedly failed to reappear in fields where it had occurred previously. Powdery scab has developed on tubers planted in infested greenhouse soil when the temperature was not above 57.2° F and the soil moisture was relatively high.

Causal Organism

Powdery scab is caused by *Spongospora subterranea* (Wallr.) Lagerh. Alexapoulos classified it in the order Plasmodiophorales, class Phycomycetes, and subclass Plasmodiophoromycetes. It was first termed *Erysiphe subterranea* Wallr. in 1842 and later reclassified as *Spongospora solani* Brunchorst. This classification encompasses the group of slime molds that are obligate endoparasites of vascular plants, algae, and fungi and usually cause abnormal enlargement of the host cells.

S. subterranea possesses a plasmodium and a type of motile cell similar to those of the Myxomycetes, the true slime molds. It forms no fruiting bodies.

Apparently infection originates in the ameboid stage, which results from the fusion of secondary zoospores. However, it is not

clear whether penetration of the tuber is directly through the skin or indirectly through the lenticels. Intracellular plasmodia develop after penetration by such amebae. Invaded cells are abnormally enlarged and abnormal cell division may occur. This abnormal cell growth and division form a wartlike excrescence. Generally lesion development is checked in the tuber by the formation of a periderm layer, although in some instances of early harvesting, disease development may continue to cause a dry rot.

S. subterranea spores germinate rather readily on artificial media, such as lima bean or potato agar, into single uninucleate amebae. If growth conditions become unfavorable, the amebae encyst to form a resting stage. On culture media they seem to produce saprophytic plasmodia.

Control

No satisfactory control measures have been developed for powdery scab. Since the disease is of minor importance in the United States, no resistant varieties have been developed.

References: *5, 25, 94, 118, 164, 199, 205, 231, 257, 263, 297.*

Scab

Streptomyces scabies (Thaxt.) Waks. and Henrici

Scab of potatoes has been known for over 100 years in both America and Europe and is universally distributed throughout the potato-growing regions of the world. The relationship of the causal organism to the disease was first shown by Thaxter at the Connecticut Experiment Station in 1890.

Although yields are generally unaffected, the losses from reduced marketability of the crop may be very serious. Scabby tubers are unsightly and wastage from the disease often exceeds the tolerance for the U.S. No. 1 grade. Tubers may be unsalable when the lesions are deeply pitted. The keeping quality may be impaired since rot-causing organisms often enter the scab lesions, particularly the deep pitted ones. An earthy odor and flavor are commonly present in affected tubers.

Symptoms

Three phases of the disease are commonly found on potato tubers—russet scab, shallow or surface scab, and deep or pitted scab. The severity depends on the resistance of the variety, the

physiologic race or virulence of the organism, and various cultural factors.

The first symptom of scab is the appearance of small reddish-brown spots on the surface of the young tuber. They may develop into the netted appearance of russet scab, develop more corky tissue resulting in shallow or surface scab, or develop still further into deep or pitted scab (pl. 6, *E*).

Russet scab is merely a roughening, scurfing, or cracking of the tuber skin, sometimes occurring in localized areas or sometimes over most of the tuber surface. It resembles the russeting due to *Rhizoctonia* and other causes.

Shallow or surface scab consists of superficial roughened areas, at times raised above but more often slightly below the surface of healthy skin. The lesions consist of corky tissue caused by abnormal proliferation of the cells of the tuber periderm as a result of pathogen invasion. The size and shape of the lesions vary greatly. They are usually darker than but not greatly different in color from the normal skin. This phase may grade into the deep scab phase.

Deep or pitted scab may be darker than shallow scab and consists of lesions ranging from 1 to 3 mm deep (pl. 6, *E*). As in shallow scab the tissue around the interior of the pits is corky. This phase of scab is generally believed to be due to the combined action of the causal organism, chewing insects, and other pests attracted to incipient lesions. However, some investigators have reported that the insects and other pests feed only on dead tissue.

Scab lesions may be in the form of isolated spots of various sizes and shapes or they may coalesce and form large scabby lesions on the tuber. Surface cracking generally occurs when the lesions are extensive.

Causal Factors

The pathogen is a common soil inhabitant and can be transmitted on seed potatoes. Its development is inhibited by acid soil about pH 5.0 and lower and by alkaline soil higher than about pH 8. Normally it occurs most severely in soil between pH 5.5 and 7.5. It does not infect mature tubers; consequently, the disease does not progress farther on infected tubers and does not spread to healthy tubers in storage.

Causal Organism

In relatively recent years the organism causing scab was classified as *Streptomyces scabies* (Thaxt.) Waks. and Henrici, one of the

higher or filamentous bacteria in the order Actinomycetales. Earlier this organism was classed as *Oospora scabies* Thaxt. and changed to *Actinomyces scabies* (Thaxt.) before being placed in its present nomenclature.

In culture the aerial mycelium of *S. scabies* is prostrate and branched with very slender threads. Colonies are compact, are usually small, and emit a characteristic earthy odor. Sporogenous hyphae are spiral. Minute spores are produced in chains by segmentation of hyphae. Septa are at first formed at intervals along the hyphae with final cleavage occurring when spores in the chain mature. The spores are roughly cylindrical and hyaline, ranging from 0.6 by 1 to 0.7 by 2 μ. They germinate by means of one or two germ tubes.

Cardinal temperatures for growth of *S. scabies* are an optimum of 77° to 86° F, a minimum of 41°, and a maximum of 104°. Spores germinate most rapidly at 95° to 104°. Early work showed that the growth of the organism was inhibited by high acidity, but later work pointed out that some isolates of the organism grow in culture at a pH as low as 4.8.

S. scabies is an exceedingly variable organism as shown by numerous workers who found that most isolates are unstable in pure culture, producing variants that differ in physiology, morphology, and pathogenicity. Researchers in the United States and Europe believe that some of the different strains of the organism cause the russeting and shallow scab and other strains cause deep scab. Race 1 and race 2 have been described on the basis of pathogenicity.

Control

No postharvest control methods are known except to avoid scabby seed. The ultimate control measure for common scab is the use of resistant varieties. Some U.S. varieties resistant to the common scab organism are Blanca, Cherokee, Early Gem, Menominee, Navajo, Ontario, Plymouth, Pungo, and Redskin. Some russet-skin varieties, such as Russet Burbank, Russet Rural, and Russet Sebago, have less conspicuous scab lesions than do smooth-skin varieties.

References: *2, 22, 38, 136, 158, 167, 173, 184, 185, 236, 297.*

Sclerotium Rot (Southern Blight)

Sclerotium rolfsii Sacc.

Sclerotium rot occurs in early potatoes in most of the Southern

States, but mainly in Florida and Alabama. It may be severe on southern potatoes shipped in the spring and early summer.

Symptoms

Small, partially sunken, circular spots with intact skin about one-eighth inch in diameter and with diffuse brownish margins occur anywhere on the tuber but frequently near the stem end and at the lenticels (pl. 6, *F*). As the spots enlarge, the skin ruptures and they became noticeably sunken and yellowish to tan. Internally the affected flesh at first is sunken, firm, odorless, and colorless. When the spots enlarge and deepen, the wet, transparent flesh changes to a chalky white and finally yellow.

The affected flesh has a cheesy or spongy consistency, and particularly in early infections it can be separated from healthy flesh with pressure. Later the invaded portions may dry, shrink, and collapse forming cavities or the rot may be very moist. Potatoes affected with the wet stage are called melters because of the liquid released and the nearly complete disintegration of the tubers. As the disease advances, a coarse white fungal growth containing white to brown mustard seedlike bodies can be found both externally and in the internal cavities (pl. 6, *G*).

Causal Factors

The stem portion of potato plants becomes infected near the soil line during warm, wet weather. As the soil dries, the fungus migrates below the soil level infecting roots and tubers. Tubers of plants frequently cultivated, hilled up, or ridged are apt to be invaded by this organism.

Causal Organism

The causal organism is *Sclerotium rolfsii* Sacc., a soil fungus that is widely distributed in the Southern States, where it attacks a great variety of truck crops and other plants as well as the potato. The presence of the fungus is recognized by a fine, silky, white mycelium closely appressed to the tubers. Grouping of mold filaments into coarser strands is characteristic. Later numerous sclerotia or round bodies, at first white and later brown and resembling mustard seed, develop in the mycelium. The perfect stage of this organism is *Pellicularia rolfsii* (Curzi) E. West. It consists of basidia produced on mycelial mats on the surface of the host. The basidia bear hyaline continuous basidiospores.

This fungus develops most rapidly at 86° to 95° F with a minimum of about 46° and a maximum near 98°. Growth is slow below 68°. It infects tubers in the growing stage or by contact during harvesting or storage.

Control

Early harvesting of tubers to avoid periods of hot, dry weather may afford some measure of control. After harvest the storage and transportation of tubers below 46° F will prevent development of the fungus.

References: *71, 72, 76, 132, 174, 189, 306.*

Silver Scurf

Helminthosporium atrovirens (Harz) Mason and Hughes

Silver scurf was first discovered in Austria in 1871. Since then it has been found in every potato-growing area of the world. The first report of the disease in the United States was from Connecticut in 1908, when it was called scurf by Clinton. The disease has been known by various names since its discovery—Fleckenkrankheit, Phellomyces-Fäule, scab, dry rot, gale argentee (silver scab), and finally silver scurf, as named by Melhus in 1913.

Silver scurf was considered of little importance and was relegated to the minor potato disease category. However, with the increased demand for washed potatoes in the United States, the disease has become more important to the producer. As with some of the other potato tuber blemishes, the symptoms of this disease are often masked by soil particles adhering to the skin of unwashed tubers. However, when the tubers are washed, the blemishes are exposed and are often of sufficient severity to reduce the grade of table stock potatoes and to prevent certification of seed potatoes.

Symptoms

In its early stage the disease appears on the tuber surface as rather inconspicuous circular or irregular brown lesions, which often are seen first at the stem end. Although these areas are difficult to distinguish on dry tubers, they are readily seen on washed or moistened tubers often as silvery areas, hence the name silver scurf. This silvery condition is caused by the pathogen, which penetrates the tubers through the lenticels and epidermis and is confined to the phellem tissue causing it to loosen. Air trapped

under the loosened skin causes a reflection of light and the silvery sheen (pl. 6, *H*).

As the disease progresses, the lesions become silvery brown and may coalesce to cover most of the tuber surface (pl. 7, *A*). The loosening and sloughing of the outer cork areas of the skin are responsible for excessive water loss from the tuber. This condition is followed by shriveling, wrinkling, or even a folding of the affected areas during storage.

In the early stages of secondary infection in storage the so-called brown mold stage appears as an olivaceous blackening over the affected areas. This stage is caused by the production of conidia and conidiophores of the fungus. Under favorable conditions of humidity and temperature the entire surface of the tuber may become dull black and appear sooty.

Causal Factors

The causal organism lives from year to year in the soil and on infected tubers. The primary disease cycle occurs in the soil from spores and mycelium produced in the lesions of infected seed pieces. Potato tubers become more susceptible as they mature in the soil before harvest. Tests have indicated that the primary disease cycle is initiated after the tubers have matured and while they lie in the soil before being dug.

Tubers are highly susceptible during storage. Although the secondary disease cycle may originate in the field from the primary lesions, most of the secondary infection, or spread of the disease from diseased to healthy tubers, occurs in storage. High humidity is essential for development of the secondary disease cycle. After the tubers have reached an equilibrium with the ambient air, no new lesions are formed at a relative humidity of 89 percent or below. There is little sporulation of the fungus below 95 percent relative humidity.

Unregulated temperature and humidity conditions in storage, particularly farm storage, are very favorable to the secondary spread of the disease. During initial storage after the curing process, when the storage temperature is slow in dropping to the holding temperature, there is a rapid succession of secondary disease cycles. The fungus grows rapidly at 70° to 80° F when the relative humidity is high.

Causal Organism

The causal organism *Helminthosporium atrovirens* (Harz) Mason and Hughes (*Spondylocladium atrovirens* Harz) is classified in the

order Moniliales of the class Fungi Imperfecti. It has a septate, branched mycelium, which is first hyaline but becomes brown with age. The conidia are borne in whorls from the distal end of the cells of unbranched conidiophores. Conidiophores are septate, ranging from 7 to 8 μ in width and 150 to 375 μ in length. The spores are dark and vary from 7 by 18 to 8 by 64 μ.

The temperature for growth in culture is 43° to 88° F with the optimum at 70° to 81°. Although the fungus grows best in culture with a neutral reaction, it can grow through a wide pH range. In laboratory cultures the fungus grows best at a relative humidity of 98 percent, with very little or no growth below 90 percent.

The fungus does not fruit as readily in culture as it does on tubers kept in a moist chamber, but when grown on a green bean cultural medium, conidia are abundantly produced. The first growth of mycelium can be seen in about 5 days on agar plates, with conidia appearing about the 12th day. The fungus grows slowly both on agar plates and on the host. Two types of mycelium are common in culture—the normal straight, septate, brown mycelium and the dark globular cells.

Control

Generally no direct methods are used to control silver scurf. Any limited control of the disease comes indirectly from methods used to control other diseases.

The trend toward using forced-air ventilation in potato storage presents a means of controlling the disease by manipulating the temperature and relative humidity. Proper manipulation of forced air will reduce the development and spread of secondary disease cycles during storage. Forced air is particularly useful for rapid cooling of the storage after the curing period.

The best way to control any plant disease is to eliminate the source of primary infection. For silver scurf this is on the potato seed. Better methods of handling and treating seed are needed to clean up the infected seed potatoes that often escape detection. Washing seed potatoes may be a way to prevent undetected infections.

The most effective control methods are (1) plant only disease-free seed potatoes, (2) harvest potatoes as soon as possible after maturity, (3) cool stored potatoes as rapidly as possible after the curing period, and (4) use forced-air ventilation to control temperature and relative humidity during the holding or storage period.

References: *37, 204, 252, 256, 315.*

Skin Spot

Oospora pustulans Owen and Wakefield

Skin spot is a minor disease of potato tubers in the United States. It has been reported on domestic potatoes in Maine and on imported potatoes in Pennsylvania and Washington. The report from Washington was on potatoes shipped from British Columbia.

Skin spot occurs at low temperatures and has been reported only in the cooler potato-growing regions of the world. It is a disease that develops in storage and is most destructive when infection occurs near the eyes of the tubers and hence prevents sprouting of seed pieces, which results in missing hills. In the United Kingdom the disease is considered serious on stored seed potatoes and reports of 20 to 30 percent loss of seed stock due to the disease are recorded.

Symptoms

This is one of the less conspicuous potato tuber diseases, and even with severe infection the spots are difficult to detect unless the tubers are washed. It is not unusual for apparently healthy seed potatoes in the fall to have such a high incidence of skin spot in the spring that many tubers fail to sprout. Lesions appear as small, dark, slightly raised pimples, about one-eighth inch in diameter, which are usually superficial on the tuber skin (pl. 7, *B*). The disease does not cause a rotting of the tuber and the blemishes can generally be removed by peeling. However, deep penetration of the tuber has been reported in potatoes that had previously been treated with a sprout inhibitor. This condition has been attributed to prevention of the formation of a cork barrier by the tuber. Skin spot develops extensively under these conditions and renders the affected tubers unsalable.

Causal Factors

Although slight mechanical injury to tubers at harvest can promote infection, skin spot can often establish itself without harvest bruises. Infection takes place about harvesttime through the eyes, lenticels, and slight bruises and leads to disease development in storage. This usually is not evident until the tubers are removed.

In the United States some investigators believe the pathogen is a secondary invader following powdery scab. British investigators do not share this view. The disease development is considerably affected by the prevailing storage conditions. Studies in Scotland

have confirmed that low temperature and high humidity in storage favor disease development. These workers found that potatoes stored at processing temperatures in pallet boxes in well-ventilated storage had less skin spot than potatoes stored in pallet boxes in similar storage but at a lower temperature.

British reports indicate that varieties differ in their susceptibility to skin spot, but susceptibility to general surface infection of the skin and to eye infection is not necessarily correlated. Some varieties show a much higher incidence of surface infection than others. However, those varieties high in susceptibility to surface infection may be less susceptible to eye infection.

Causal Organism

Oospora pustulans Owen and Wakefield, the causal fungus, has a hyaline septate mycelium, 2 to 4 μ in diameter, from which short, erect, branched conidiophores arise. Conidia are produced by budding on the conidiophores, where they remain attached to form a chain of spores.

O. pustulans is not difficult to isolate after thorough disinfection of the tissue surface. The organism grows well on oatmeal agar and on cooked tissue of such vegetables as potato, carrots, and parsnips. In culture, growth is best at room temperature in complete darkness.

Control

Skin spot can be effectively controlled by storing potatoes in pallet boxes under relatively dry conditions. Initial storage temperatures should be sufficiently high to properly cure the potatoes.

Combined tuber washing and chemical dip followed by complete drying is being used commercially in Scotland to control skin spot on seed potatoes.

References: *34, 64, 73, 83, 114, 128, 213, 224, 263, 297.*

Verticillium Wilt

Verticillium albo-atrum Reinke and Berth.

The seriousness of verticillium wilt varies from season to season. Generally the disease appears to be increasing both in areas affected and in severity, partially because some of our most desirable varieties are highly susceptible to the disease. The disease may escape detection in the field during some seasons and only becomes

apparent when the harvested tubers are cut. The only direct effect of verticillium wilt on tubers is the internal discoloration, which makes the potatoes undesirable to the consumer and unacceptable for certified seed.

Verticillium wilt is found in the United States chiefly in the Northeastern and Western States, but it has also been reported in Arkansas, Florida, Louisiana, Virginia, and West Virginia. It is considered to be important in eastern Canada, Maine, and western Oregon and severe in Idaho where yields are reduced.

Symptoms

The xylem or water-conducting tissues in the stem end of tubers often are yellow red to brown (pl. 7, *C* and *D*). This discoloration resembles stem-end browning, but it differs since often it extends throughout the vascular ring. Verticillium wilt is found in two of our most widely grown late varieties, Kennebec and Katahdin, which are not affected by stem-end browning. Also, internal discoloration does not always occur in infected tubers.

Verticillium wilt is often indirectly responsible for tuber breakdown in storage because of a complex called pinkeye (pl. 7, *E* and *F*). See Red Xylem and Pinkeye.

Causal Factors

Verticillium wilt is spread through infected seed. The disease apparently predisposes the tuber to infection by the pinkeye bacterium. Moderate temperatures favor the organism and the disease does not develop under extremely high temperatures. The optimum temperature at which it occurs is 77° F, the minimum 41°, and the maximum 86°.

High surface moisture and high storage temperature favor the development of the pinkeye soft rot breakdown in storage.

Causal Organism

This disease is caused by *Verticillium albo-atrum* Reinke and Berth., which was formerly called *Verticillium dahliae* Klebahn. The fungus is classified in the order Moniliales of the class Fungi Imperfecti. Conidiophores are slender with verticillate (in whorls) branches. The conidia are ovoid to ellipsoid, hyaline, one-celled, and borne singly or in small apical clusters. The mycelium is septate and hyaline when young but produces darkened branches or small sclerotia later.

The maximum temperature for growth of *V. albo-atrum* is 86° F. Spores are unable to germinate at 95°. A slight correlation exists between the temperature relations of the fungus in pure culture and its geographical distribution and seasonal occurrence. Strains of the fungus in the Northern United States have comparatively low maximum and low minimum temperature points, whereas those in the Central United States exhibit higher maximum temperature points although retaining practically the same moderate optimum temperature point.

Control

No postharvest control measures are possible for verticillium wilt. However, the disease can be prevented or reduced by using clean certified seed, rotating potatoes with millet, oats, or corn, and planting resistant varieties.

The susceptibility of *V. albo-atrum* to high temperatures suggests the possibility of heat treatment for infected seed.

References: *6, 20, 38, 90, 158, 242.*

Wart

Synchytrium endobioticum (Schilb.) Perc.

Potato wart became known in the United States in 1918, when it was found in garden plots of several villages in the hard-coal districts of eastern Pennsylvania. It has since been found in small scattered coal-mining districts of western Pennsylvania, western Maryland, western Virginia, and West Virginia. The disease originated with infected tubers imported from Europe. Because of strict quarantine regulations it is unlikely that potatoes with this disease will be found on the market. The potential seriousness of wart makes it imperative that all field and market inspectors familiarize themselves with the disease symptoms.

Symptoms

The disease causes spongy outgrowths on the tuber particularly at the eyes. At first the warts are light brown (pl. 7, *G*). With age they enlarge, darken, and eventually become black (pl. 7, *H*). They may vary from the size of a pea to that of an entire tuber. Either few or many warty outgrowths may occur on a potato. Wart can be slight and easily overlooked on potatoes at harvest but may develop in infected eye tissue during storage.

Causal Organism

Potato wart is caused by a fungus, *Synchytrium endobioticum* (Schilb.) Perc. This organism lives entirely within the cells of the potato, where it forms numerous rusty-brown resting spores, which are globoid and oval and when mature have a thick, rough, outer wall.

Control

If potatoes with this disease are found, their source should be determined and the facts reported promptly to the U.S. plant quarantine authorities.

References: *65, 125, 222.*

Xylaria Tuber Rot

***Xylaria apiculata* Cke.**

Xylaria tuber rot is a very minor potato disease. It has been reported only in southern Florida.

Symptoms

The disease causes a circular, sunken, light-tan dry rot, which generally is followed by secondary fungi and bacteria.

Causal Factors

The fungus is a common saprophyte on tree stumps and other wood debris in the soil of newly cleared land. The disease is found only on recently cleared land and generally disappears after a few years of cultivation.

Causal Organism

The causal organism is *Xylaria apiculata* Cke., an ascomycete. The fungus produces in the soil slender black rhizomorphs or coarse threads, which become firmly attached to the potato tubers.

Control

There is no practical control once infection occurs.

References: *33, 251.*

VIRUS DISEASES

The potato is subject to attack by a wide variety of virus diseases. These include several mosaics, leaf roll, spindle tuber, witches'-broom, yellow dwarf, purple top, and corky ring spot. Each is caused by a specific virus or combination of viruses. If the many strains of the viruses are considered, the number responsible for potato diseases becomes very large. Virus diseases are caused by nucleoproteins, which can replicate in plants. Only a few of the virus diseases produce tuber symptoms that are apparent on the market.

Corky Ring Spot

Under the names of sprain and internal rust spot this disease has been known for many years in the Netherlands, other European countries, the Indies, and South Africa. It was observed on the Chicago market in 1944 and 1945 in Washington-grown 'White Rose' potatoes. In 1946 it seriously damaged tubers in some fields in Florida and was given the more descriptive name of corky ring spot.

Symptoms

Affected potato tubers show brown rings, semicircles, loops, and spots on the surface (pl. 8, *A*) and similar brown rings, loops, and spots in the interior tissues (pl. 8, *B*). The skin often cracks over some of the brown slightly sunken rings. The surface markings can usually be traced to similar markings within the tuber, but many internal brown spots and semicircles appear to be independent of the surface lesions. The affected tissues are moderately firm and corky.

The characteristic ringlike external and internal markings of tubers affected with this disease distinguish it from internal brown spot and tuber necrosis caused by alfalfa mosaic.

Causal Factors

Tubers from crops infected with corky ring spot may appear healthy when harvested but develop the disease in storage. The number of tubers showing symptoms and the size of the lesions increase as the temperature is raised from 60° to 80° F. No lesions develop in potatoes stored at 90°.

Causal Agents

Corky ring spot is a virus disease caused by a combination of two soilborne viruses. One is an etching virus and the other a necrotic virus.

Control

'Sebago' and 'Red Pontiac' potatoes are very susceptible to corky ring spot. 'Merrimack,' 'Pungo,' 'Plymouth,' and 'Hunter' are resistant.

Storing potatoes below 60° F will retard development of corky ring spot after harvest.

References: *57, 65, 68-70, 190, 235, 299, 300.*

Net Necrosis (Leaf Roll)

Net necrosis is probably the most important virus disease of potato tubers. Discoloration of the vascular ring and vascular strands in the flesh of the tuber results in serious market losses. Particularly for chip manufacture, affected tubers are worthless. Discoloration of the vascular elements can become more pronounced during storage, transit, and marketing.

Symptoms

No external symptoms of net necrosis occur. The disease is most readily detected when cuts made near the stem end show a network of discolored phloem strands (pl. 8, *C*). The affected strands become hardened and turn dark brown to blackish. Their appearance varies with the angle the tuber is cut. In cross sections they show up as dots or streaks, but lengthwise cuts, after removing only a thin layer of the outer flesh, show a network of brownish phloem strands (pl. 8, *D*). These discolored strands may extend throughout the tuber to the bud end and are found in the heart of the tuber as well as in the outer flesh. Often it is necessary to cut several sides of a tuber before discolored strands can be found extending throughout the tuber.

Net necrosis can be confused with several other diseases, possibly the most important of which is stem-end browning. It differs from stem-end browning since the discolored phloem strands may extend throughout the tubers, whereas the discolored strands of

stem-end browning are limited to not more than one-half inch from the stem end. Net necrosis also is usually found in large tubers, and in cross section the cuts show more concentric zones than in stem-end browning. Net necrosis differs from the fusarium wilts and vascular browning because the latter are confined to the xylem strands. It differs from freezing injury, which may produce blue to grayish strands not distinctly defined. The discolored strands caused by freezing also are often associated with grayish areas in the flesh.

Causal Factors

Net necrosis occurs in tubers harvested from cool soils. It may or may not be present at harvest but shows up after the tubers are stored. Symptoms are more pronounced when soils have a high potassium content and are masked when soils contain high nitrogen.

Causal Agent

Net necrosis is a tuber symptom of the virus that causes leaf roll of growing plants. It occurs only when healthy plants of the current season are inoculated with the virus. Progeny of net necrosis tubers will produce plants with leaf roll, which in turn produce symptomless tubers. Storage conditions bring out tuber symptoms. Maximum development of symptoms occurs in tubers stored near 50° F. If tubers are held for 60 days at 32° to 36° or at 70° and then stored at 50°, symptoms of net necrosis are greatly reduced. Maximum development of symptoms occurs in 2 to 3 months.

In early shipments net necrosis may increase during transit and marketing. Such could arise from shipping tubers near 50° F shortly after harvest or shipping them near this temperature when the conditioning period at 32° to 36° or 70° was 30 days or less. Tubers conditioned for 60 days or longer at these temperatures may therefore have less net necrosis than tubers from the same lot shipped without proper conditioning earlier in the season.

Control

Use certified seed potatoes and proper control measures for aphids that spread the virus from diseased to healthy plants. 'Chippewa' and 'Katahdin,' although susceptible to the leaf roll virus, remain practically free of net necrosis. Whenever there appears to be extensive spread of the virus during the current season, reduction in losses from net necrosis can be obtained by conditioning the newly harvested tubers and holding them at proper

temperatures. Studies have also shown that long exposure to hot air or relatively short exposures to hot water will kill the virus in a high percentage of the tubers.

References: *56, 65, 80, 85, 87, 92, 101, 102, 119, 157, 272, 278, 293, 305, 319.*

Purple Top Wilt

Purple top wilt occurs in most areas where potatoes are grown. On the market it is important as sometimes tubers from infected plants have vascular discoloration. This discoloration is common in potatoes grown in Wisconsin, West Virginia, Pennsylvania, Maryland, and Virginia but occurs only rarely in potatoes from Maine, New York, and Nebraska.

Symptoms

The intense yellowish brown of the vascular strand occurs at the point of the stolon attachment and extends inwardly and sometimes throughout the tuber (pl. 8, *E*). Often the necrotic stolons separate from the stem end leaving a cavity at the point of their attachment. Browning is most severe in the vascular ring, but often it extends into the cortex and pith parenchyma. This discoloration of strands in the cortex and pith parenchyma distinguishes purple top wilt from other diseases that cause vascular discoloration. In addition to this symptom, tubers from plants infected with purple top wilt often are soft and flabby. Infected tubers also frequently produce weak or hair sprouts.

Causal Organism

Purple top wilt has been considered to be due to a virus that produces yellows of asters and many other plants. Recently several studies have shown that the cause of the yellows-type diseases is a mycoplasmalike organism. Most of them are spherical and range from 70 to 700 nm. Unlike viruses they can be cultivated on specialized media. They divide by binary fission and sometimes by budding. These organisms have been found in the salivary glands and alimentary tract of insects that transmit the yellows-type diseases.

Control

There are no postharvest control methods.

References: *59. 65, 145, 172, 200, 219, 254, 261, 325.*

Spindle Tuber

Spindle tuber is likely to be found on market potatoes from certain sections of the country.

Symptoms

Tubers affected with this disease are characteristically spindle shaped or cylindrical with the ends, particularly the bud end, abnormally long and pointed (pl. 8, *F*). The eyes are more numerous and shallower than those of healthy potatoes, but they appear conspicuous because of prominent eyebrows. Frequently the red-skin varieties fade. The smallness and spindle shape lower the grade of affected potatoes.

Control

Use certified seed. No specific postharvest control method is known.

Reference: *65.*

Tuber Necrosis (Alfalfa)

This disease is caused by the alfalfa mosaic virus and was first observed in 1946 in California potatoes grown adjacent to alfalfa plantings.

Symptoms

Necrosis in the tuber first develops just under the skin near the stem end. At this stage the affected areas have a purplish or silver cast and resemble late blight infection. Later the diseased areas extend into all parts of the tuber (pl. 8, *G* and *H*). The necrotic tissues are deep brown, very dry, and corky to mealy in consistency. They may occur in pockets throughout the tuber or on only one side. In later stages the affected tissues dry out, crack, become sunken, and resemble a fungus dry rot.

Control

No postharvest control methods are known.

References: *65, 223.*

Yellow Dwarf

Potatoes affected with yellow dwarf are seldom found on the market. Although not all tubers produced on diseased vines show

external symptoms of the disease, most of them are malformed and have cracks, which make it possible to grade out the affected tubers before shipment. Internal discoloration also makes them unsuitable for table use.

Symptoms

The tubers produced on plants with yellow dwarf are usually small, irregular, knobby, and cracked. The cracks, which are one-eighth to one-fourth inch deep, often radiate from the bud end and resemble growth cracks. They usually heal without infection by decay-producing organisms. When diseased tubers are cut, rusty-brown specks and spots scattered in the pith area and extending from the bud end halfway to the stem are characteristic symptoms. Discoloration is seldom found at the stem end.

References: *11, 17, 65, 120, 298.*

Other Virus Diseases

Potatoes from plants infected with the tomato spotted wilt virus sometimes are distorted and show necrotic spotting and cracking. Potatoes from plants affected with tuber blotch and Canada streak may have internal discolored, blotchy spots. With tuber blotch some of these necrotic areas may appear externally as dark sunken lesions. Neither disease has been reported on potatoes grown in the United States. Similarly the veinal mosaic virus may cause both superficial and internal necrotic areas in the tubers of some foreign varieties of potatoes.

Reference: *65.*

NEMATODES AND THEIR INJURY

Golden Nematode

The history of the golden nematode (*Heterodera rostochiensis* Wollenwebber) in the British Isles and in continental Europe indicates that it is a serious threat to the potato industry wherever it becomes established. Surveys of the U.S. potato-producing sections have revealed its presence only in New York State. It was reported first in 1941 in relatively localized districts of Nassau County, Long Island, N.Y., and was later found in adjoining Suffolk County. In 1967 the nematode was found in western New York in Steuben County near the town of Plattsburg.

Symptoms

The nematode attacks the root system of the potato plant, causing stunting, midday wilting, early dying of the vines, and a reduced yield of marketable tubers. Four to six weeks after the plants are attacked the female nematodes can readily be detected on the roots with the unaided eye. They first appear as white spherical bodies, later turning golden brown. No symptoms are evident on the tuber itself, but the orange or golden-brown spherical female cysts may be found in soil clinging to the tubers.

Control

Restrictions have been imposed by New York State to prevent further spread of the nematode and thus to prevent its introduction into other areas. In districts where it has been introduced, control becomes very difficult because it can survive long periods in the absence of a host. Some measure of control has been obtained by long rotations. Nematode populations may be reduced by applying certain nematicides. Development of commercially acceptable varieties resistant to the golden nematode offers much promise in controlling this disorder.

References: *47, 53, 191, 277, 288.*

Root Knot (Nematode Gall)

Root knot (*Meloidogyne* spp.) has been reported on potatoes from almost all sections where they are grown commercially. Affected tubers are occasionally found on the market but are usually graded out before shipment.

Symptoms

Potato tubers with advanced stages of this disorder appear roughened, irregular, bumpy, or warty because of galls on the surface (pl. 9, *A*). The skin over the galls usually remains unruptured. When such tubers are cut open, the female nematodes are abundantly scattered through the outer tissues, with most of them approximately one-eighth to one-fourth inch beneath the tuber surface. They have whitish, spherical bodies about the size of a small pinhead, each surrounded by a thin layer of brownish potato tissue (pl. 9, *B*). The infected parts of the tubers thus appear somewhat discolored with some slightly water-soaked spots sur-

rounding the infected area. Under slight infection, tubers sometimes show no external symptoms.

Causal Agents

Root knot of potato is caused by several species of the nematode genus *Meloidogyne*. Between crop seasons it maintains itself as eggs or larvae in roots, tubers, or soil. When the nematode larvae come in contact with the developing tubers, they enter these plant organs, become sedentary, and begin to enlarge as they feed on the plant tissue. The female enlarges enormously in diameter so that she eventually becomes pear shaped and visible to the unaided eye. The nematodes irritate the tissues, which develop abnormally and form the galls. Larvae hatching from eggs within the tuber may move a short distance within the tuber, initiate new galls, and continue to develop there.

Control

There are no postharvest control measures, but several nematicides afford good field control of root knot. Resistant varieties are not available.

References: *52, 53, 104, 207, 287, 288, 292.*

Rot Nematode

The only known occurrence of the rot nematode (*Ditylenchus destructor* Thorne) on potato in the United States was reported in Idaho and Wisconsin in 1943 and 1953, respectively. Elsewhere on the North American continent it has been found on Prince Edward Island and in British Columbia, Canada. In 1946, seed shipped from Prince Edward Island was intercepted at Long Island, N.Y., and found infected with the nematode. Apparently none of the infected seed pieces were planted.

Symptoms

Only tubers are infected. Small gray or brown spots of decaying tissue on the skin of the tubers are the first symptoms of nematode attack. Beneath the skin these appear as chalky areas in which the nematode can be found. Affected tissues dry out as the lesions enlarge and the skin shrinks and cracks. In more advanced stages the decaying tissues form a shallow, dark-brown layer just beneath the

epidermis (pl. 9, *C*) and deeper tissues become bluish gray. Injury may be serious in the field at the time of digging and most often increases in severity with invasion of secondary organisms in storage.

Control

The principal control measure for rot nematode in the United States is to prevent its spread into new areas in infected seed stocks. Nematicides have been very satisfactory for field control of this nematode.

References: *9, 53, 78, 288.*

INSECTS AND THEIR INJURY

Several insects attack potato tubers and cause various types of injury. The following descriptions include the insect injuries that are most apt to cause economic losses of market potatoes. In most cases if the injuries are pronounced, the tubers never reach the market. No postharvest treatments to control these insects are known.

Potato Tuberworm

The potato tuberworm (*Phthorimaea operculella* Zeller) is most damaging to the tubers after they have been dug. However, exposed tubers may be injured before digging. The larvae migrate from the stems and leaves to exposed tubers. Harvested tubers left in the field overnight may also become heavily infested by larvae moving from vines to tubers. After the eggs hatch on the tubers either in the field or in bins or sacks, the larvae at first work under the skin and later tunnel through the flesh. They often enter at the eyes, where sawdustlike heaps of material are evidence of their activity. After transit or storage the infested tubers are frequently riddled with deeply penetrating channels that make them unmarketable.

The damage by this insect is more severe in certain sections of California and the Southern States in years of low rainfall.

References: *195, 206.*

Spotted Cucumber Beetle

In the early potato-producing areas in the South the larvae of the spotted cucumber beetle (*Diabrotica undecimpunctata howardi*

Barber) sometimes tunnel into the tubers and leave surface holes. When small tubers are invaded, the remaining channel is rather deep by the time the tubers are of marketable size. In red-skin potato varieties the lining of these channels and pockets is red.

References: *195, 206.*

Tuber Flea Beetle

The larvae of the following beetles feed on potato tubers and cause injury: The tuber flea beetle (*Epitrix tuberis* Gentner) and to a less extent the western potato flea beetle (*E. subcrinita* Le Conte) and the potato flea beetle (*E. cucumeris* Harris). In feeding on or near the surface of young tubers they produce pimples (pl. 9, *D*) and channels in and under the skin. Deeper feeding results in the formation of brown splinterlike pegs extending one-eighth to one-fourth inch into the tuber (pl. 9, *E*). These corky splinters, pimples, and channels are so tough and dry that usually no secondary infections by bacteria or fungi occur.

Injury by the tuber flea beetle is severe in eastern Colorado and western Nebraska, but it is no longer a pest in most of Oregon and Washington. The western potato flea beetle causes little damage to the tubers, although it is associated with the tuber flea beetle. The potato flea beetle has a wide range, but in the Eastern States where it is most abundant, it does not usually cause much damage to tubers.

References: *195, 206.*

Wireworm

Wireworm injury caused by the larvae of the snapping or click beetles (Elateridae) is of several types. The larvae burrow into the growing tubers, the depth and size of the burrow depending on their size and length. Large worms attacking tubers late in the season bore clean-cut round holes rather deep into the tuber (pl. 9, *F*). These channels and burrows may be brown and lined with cork or may become plugged and surrounded by dead tissue caused by secondary infection with *Rhizoctonia*.

Holes made in tubers by quackgrass and nutgrass sometimes appear similar to those made by wireworms. However, the remains of these grasses in the tuber usually indicate this kind of injury.

References: *195, 206.*

NONPARASITIC DISORDERS AND INJURIES

Blackheart

Blackheart develops in tubers while they are growing or while they are in transit or storage. It occurs in all varieties of potatoes from all growing areas. It is important on the market because normally no external symptoms are apparent, but the blackened internal tissue makes the tubers worthless for culinary purposes or processing.

Symptoms

External symptoms of blackheart appear only after affected potatoes have been stored for long periods and almost the entire inner tissue is discolored. The skin of such tubers is bluish to gray blue. The discoloration sometimes covers the entire surface but normally is restricted to distinct areas.

Internally blackheart may have several symptoms. When freshly injured potatoes are cut, the affected flesh at first appears normal, but within minutes various-sized pink areas develop. In about a half hour these pink areas become jet black with a light grayish-pink center, which eventually becomes black (pl. 9, *G*). When freshly cut tubers show blackened areas, they have been injured for several days or weeks. The entire central section of the tubers may be discolored (pl. 9, *H*) or the black areas may appear as isolated pockets about one-fourth inch in diameter distributed throughout the flesh (pl. 10, *A*). The affected flesh is firm but not crisp, and usually is not separated from healthy tissue by a definite border. With prolonged holding the blackened tissue collapses, leaving hollow cavities with blackened walls.

Causal Factors

Blackheart occurs at any temperature when the supply of oxygen available to internal tissues is used up faster than it can be supplied. The affected tissue suffocates and turns black. Conditions causing blackheart can occur in the field when the soil is flooded or soil temperatures are extremely high, in storage when aeration is poor, in transit when tubers are overheated, or in prolonged storage near 32° F. Blackheart can be induced experimentally in nearly 100 percent of the tubers when they are held at 105° to 110° for 24 hours.

Control

Proper ventilation and temperature control should be provided during storage and transit to assure air circulation and prevent high temperatures. Tubers should not be left in hot, light sandy soils after the vines are dead. In hot weather after harvest the potatoes should be removed as soon as possible from the fields and placed in well-ventilated bins.

References: *12, 13, 60, 139, 264, 281.*

Browning

Browning, also called chicken skin in Maine, is used to describe the discolored skinned areas on immature tubers (pl. 10, *B*). It is most serious on skinned tubers exposed to the drying effect of winds in the field, in packing sheds, and in transit in open trucks, especially if the weather is very warm.

Slight browning is not objectionable except when large areas of the tubers are involved and excessive wilting results from loss of moisture. Severe browning affects the appearance and therefore the market value of potatoes. If browned potatoes are exposed to a moist atmosphere, the discolored areas become covered with a sticky bacterial slime and mold and finally may decay.

Control

To prevent browning before shipping, protect tubers from drying wind in the field by prompt picking and hauling, use tightly woven bags, cover loaded trucks with tarpaulins, and protect stacks of potatoes at sheds from sun and wind. Shipping in closed vans or railcars at about 60° F permits healing of the skinned areas during transit. Healed potatoes do not turn brown during marketing.

References: *10, 225, 243, 244, 271, 311.*

Bruising

Potato tubers bruised in transit are common on the market. Although there may be occasional slight injury when potatoes are pressed and rubbed together in the sack, most transit bruising is caused when the sacks come in contact with the floor racks and the walls of the railcar or truck. The injury is due to the pressure of the load, the shifting of bags at points of contact, and the jolting and jarring while in transit.

Symptoms

On newly harvested early potatoes the injured tissues are flattened or slightly sunken and usually conspicuously darkened. When the bruised tissues are black, they frequently are mistaken for chemical injury. However, transit bruising can readily be distinguished from chemical injury because there is no external softness or flabbiness of tissues beyond the borders of the flattened areas and the tissues deeper than the flattened bruise do not turn black when the tuber is cut.

Injured tissues of late tubers (pl. 10, *C*) are likewise flattened or slightly sunken, but they are seldom markedly discolored. When the bruised areas are cut transversely, they appear as a shallow layer of crushed tissue seldom more than one-eighth inch thick. At first the bruised layer is moist and somewhat tough. As it dries out, it becomes firm and starchy. In some instances deeper tissues contain cracks that radiate from the bruise (shatter bruises) (pl. 10, *D*).

Occasionally in winter shipments transit bruising is mistaken for freezing injury. The two can be distinguished readily by critically examining the affected tubers. Bruising symptoms are confined to the immediate vicinity of the flattened areas, but freezing may be recognized externally by softened or wrinkled flesh beyond the bruise and internally by softened or discolored tissues well beyond the border of the bruise.

Control

Transit bruising can be reduced by using an efficient loading method, by carefully stowing the load, and by having pads on the floor racks.

References: *291*, *310*, *314*, *321*.

Chemical Injury

Chemical injury to potatoes is rare and almost impossible to diagnose correctly. The skin of chemically injured tubers may be bleached. The injured areas are leathery and the tissue beneath is somewhat flabby. Later the flesh may become brown to black and dry. Often injured areas are invaded by decay-producing organisms.

Positive diagnosis has been made on tubers injured by incorrect or prolonged exposures to methyl bromide (pl. 10, *E*). Affected tubers may be covered with sunken or saucer-shaped lesions about one-eighth inch deep and ranging from minute spots to about

one-fourth inch in diameter. Sometimes these lesions coalesce and cover large areas of the skin. The skin is not discolored. Beneath the affected skin dry corky tissue extends less than one-eighth inch into the flesh.

Enlarged Lenticels

Enlarged lenticels detract from the appearance of the tubers and may serve as entrance points for soft rot bacteria and other decay micro-organisms.

Symptoms

Lenticels normally are inconspicuous on tubers, but under high moisture conditions they rise about one-sixteenth inch and form numerous whitish protuberances over the tuber surface (pl. 10, *F*). If tubers are held in moist atmospheres, these elevated areas remain whitish, but when the tuber dries, they become skin color, open, and somewhat depressed. They make excellent openings for micro-organisms, particularly soft rot bacteria. Once infection occurs, soft decayed tissue develops around the lenticels. If these infected tubers are held at moderate temperatures, the decay usually dries, leaving moderately large round areas over the tuber's surface. Sometimes these areas contain white starchy material. When the tubers are held at excessively high temperatures, decay may cause considerable damage.

Causal Factors

Enlarged lenticels may occur when tubers are harvested from heavy and water-saturated soils, when they are submerged too long in deep washing vats, or when they are packaged wet and held in moistureproof containers for long periods. Lenticel enlargement also can occur when the plants are growing if carbon dioxide concentrations in the soil increase to 5 percent even though adequate oxygen is present.

Control

Avoid potatoes from wet areas of fields. Do not leave tubers in deep washing vats longer than necessary. Do not package them while wet and then only in ventilated bags. Avoid conditions that may cause carbon dioxide accumulation.

Reference: *269*.

Freezing Injury

Freezing injury or frost necrosis can cause serious losses in any area of the world where below freezing temperatures are prevalent during the harvesting, storing, or shipping of potatoes. The greatest danger exists in northern areas, where early frosts or freezes are likely during harvest and where temperatures are extremely low during storage or when potatoes are removed from storage and shipped to market.

Symptoms

Since symptoms of freezing injury may overlap those of tuber diseases and disorders and since there is sometimes no external evidence, information about a given lot of potatoes usually is necessary for a correct diagnosis.

Tissues at the stem end of the tuber are more sensitive to freezing than those at the bud end, and the differentiated vascular cells, such as tracheae, sieve tubes, and tracheids, are more susceptible than are the parenchymatous cells. Because of this difference in cell sensitivity to cold, any freezing during storage and transit may manifest any or all the necrotic patterns known as ring necrosis, blotch, or net necrosis.

Ring necrosis consists of internal discoloration, particularly in the region of the vascular ring, and is more common at the stem end of the tuber (pl. 10, *G*). Blotch necrosis usually forms irregular areas of various sizes, ranging from opaque blue gray to black (pl. 10, *H*). This type of freezing injury is more difficult to identify than the other two types. Net necrosis causes blackening of the vascular elements of the tuber inner phloem, and a netlike pattern results when these vascular elements, which are scattered throughout the tuber within the vascular ring, blacken from freezing injury (pl. 10, *G*; pl. 11, *A*). The net of freezing injury usually has blotches associated with it and so differs from the net necrosis caused by the leaf roll virus. Generally the symptoms of freezing progress from the ring to net to blotch type as the freezing period lengthens. Often the different types of symptoms will overlap. The various internal symptoms of freezing injury may not occur unless the tubers are bumped, jarred, or dropped during the freezing period.

When freezing injury occurs prior to harvest, it is often referred to as field frost (pl. 11, *B*). This condition can usually be diagnosed by bluish-gray blotches beneath the skin of the more uniformly soft, flabby, or watery areas, which generally are on one side of the tuber.

Since the different types of symptoms overlap, it is often necessary to look further for clues as to where and when freezing occurred. Frozen tubers scattered throughout the load when received on the market indicate that freezing happened in the field or during storage, whereas frozen tubers only along the floor and sidewalls of the railcar or truck indicate transit freezing.

Tubers frozen solid collapse promptly on thawing and become soft and watery. The flesh is cream color or white and the eyes are often black. Such tubers are frequently called leakers. However, moisture on a tuber is not necessarily a sign of freezing, since cold tubers soon condense water from the air on their surface when brought into moist air with a temperature higher than their own. When extensive areas of a tuber are frozen, the affected tissues usually become invaded with bacteria, which cause sticky, slimy, foul-smelling rots under moist conditions. In dry air, such as in forced-air ventilated storage, the frozen tissues may become dried, tough, and leathery or may lose moisture and become granular with chalky masses of starch and tissue remnants.

Sweetness of potatoes does not indicate freezing as is commonly believed. Potatoes held for protracted periods at 40° F or lower, which is not low enough to form ice, gradually acquire a sweet taste. This condition is not due to freezing but is due to the conversion of tuber starch to sugar more rapidly than it can be used in respiration. Potatoes in this condition are not suitable for cooking, especially for frying, because of the sweet taste and since caramelization of sugar causes an undesirable color. The sweetness of some potatoes can be corrected by raising the holding temperature to about 60° for a week or more.

Causal Factors

Varieties of potatoes differ in their reaction to low temperatures. Some show serious internal discoloration after prolonged storage at several degrees above their actual freezing point, whereas others may not show injury even when exposed to 29° F, the average freezing point of most varieties. The freezing point of a given variety tends to become lower as the season progresses. Tubers stored for several weeks at low temperatures will stand about 1° lower without freezing than similar tubers stored at 50° or above.

Long exposure of tubers to temperatures just below the freezing point or moderately long exposure to very low temperatures usually results in killing of all the tuber tissues, with both internal and external symptoms becoming apparent after thawing. Moderately short exposure just at the freezing point or short exposure far

below the freezing point usually results in death of only the most susceptible tissues, and internal symptoms may not be apparent unless the tuber is cut.

Individual tubers in a given lot of potatoes differ as to the point they can undercool before ice forms, even though the temperature to which the tissue rises immediately after undercooling in each may be the same. When the temperature drops rapidly, the degree of undercooling before ice formation and freezing is generally less than when the temperature drops more gradually. Undercooled tubers may recover without injury if they are not agitated during this period.

When used for seed purposes, tubers with considerable discoloration from freezing often give poor stands, but those tubers with slight injury seldom affect stands.

Control

Freezing injury to potatoes may be avoided by providing adequate protection from temperatures below 29° to 30° F. In very cold climates or during very cold weather supplemental heat must be applied during storage and in transit. During storage this heat must be distributed through ducts of a forced-air ventilation system to prevent freezing of tubers in some areas of the building. Adequate insulation must be present in buildings used for potato storage, particularly in the northern areas.

References: *152, 241, 316, 318, 320-323.*

Greening

Potatoes exposed to light either before or after harvest gradually turn green at the surface owing to the development of chlorophyll. Greening that occurs before harvest is often called sunburn. Greening after harvest in markets or retail stores is called light burn, light struck, or simply greening. Greened potatoes are an unnatural color and may cause serious market losses. In addition, affected potatoes often have a bitter taste, which makes them unpalatable and possibly dangerous to eat since the alkaloid solanine is usually associated with the greened areas. Solanine is considered poisonous if taken in sufficient dosages.

Symptoms

Some potatoes develop greening after exposure to light for a day or two. The discoloration progresses from a light yellow to a

definite green (chlorophyll) and is generally restricted to layers immediately below the skin. If exposure to light is prolonged, chlorophyll develops deeper in the tissue and the bitter taste associated with solanine results.

Causal Factors

All potatoes will green to some extent, especially when exposed to direct sunlight. Greening occurs initially in the field when tubers are insufficiently covered with soil (pl. 11, *C*), or it may be induced by exposure to incandescent or fluorescent light during grading and in retail stores (pl. 11, *D*). Usually immature tubers green more readily than mature ones. Greening develops more rapidly at warm than at cool temperatures. Light intensities as low as 5 foot-candles produce greened tubers, and as light intensities increase, greening increases. The amount of greening is also directly proportional to the duration of light exposure. Although greening may increase under artificial light in retail stores, little if any solanine is synthesized under such light.

Control

Store potatoes in the dark and protect them from exposure to light during marketing. Avoid displays near windows or under strong fluorescent lights. Light-resistant packaging is valuable in preventing retail greening, together with package rotation and holding reserve stock in the dark.

References: *117, 121, 122, 142, 168, 177, 324.*

Heat Injury (Scald)

Heat injury occurs most frequently in the early and intermediate crops of potatoes harvested on hot days from April or May through August. The breakdown of tubers due to heat injury and the subsequent decay in the injured tissues cause excessive shipping losses. Most of the potatoes injured by heat in the field become infected by soft rot bacteria. Extensive development of this decay, usually referred to as slimy soft rot, is one of the best evidences that market potatoes were excessively warm at some period during harvesting or marketing.

Symptoms

In severe cases of heat injury the exposed surfaces of tubers become watery and blistered and juice may ooze from the lenticels.

Those with less severe heat injury may not show external symptoms of tissue breakdown for several hours. With exposure to dry air the damaged tissues not infected by bacteria lose moisture and become sunken, flat, and leathery. The condition often resembles field freezing injury (pl. 11, *E*). When first cut, the heat-injured tissues may appear white to gray, but after exposure to air they become brown and finally black as in blackheart. The skin of colored varieties is sometimes bleached if potatoes have been exposed to bright sunlight.

The term "scald spot" has been used to describe oval or irregular, sunken, brown areas on the skinned parts of freshly harvested tubers (pl. 11, *F*). When the flesh beneath this spot has a grayish to purplish watery breakdown with sometimes a purplish black discoloration on and just under the skin, the injury is called deep or sunken scald.

Causal Factors

It is not possible to judge by air temperature alone whether potatoes will be injured by heat during harvest, for on a moderately hot day the heat accumulated by tubers at their exposed surfaces may kill the tissues. The surface tissues of exposed tubers will often be between 20° and 30° F higher than the air temperature. As high as 135° has been recorded on the sunny side of tubers left exposed in the field on a hot day. The thermal death point of potato tissue is believed to be near 122°. Below this point potato tissues are not likely to break down because of death by heat alone. Such temperatures are, however, definitely dangerous to freshly dug potatoes because they are affected physiologically in such a way as to predispose them to attack by soft rot bacteria.

Prolonged exposure of tubers on or near the surface of the soil to excessively high temperatures will cause deep or sunken scald. Scald spot is caused by short exposures to high temperatures or by exposures to drying winds.

Control

To avoid heat injury, protect potatoes from prolonged exposure to the sun when harvested during hot weather, especially above 90° F. Within 15 to 30 minutes after tubers are dug, they should be hauled to the packing shed. Heat injury can occur while the potatoes are on the truck if it is left standing in the sun. Covering the load with a tarpaulin gives some protection against the sun and also prevents drying and browning of bruised and skinned areas of

tubers when hot winds and low humidity prevail. If mechanized harvesting is not used, it may be advisable in hot weather to dig the potatoes late in the afternoon, allow them to cool on the ground overnight, and pick them up early the following morning.

Since potato tubers may be injured by heat without showing external evidence of breakdown of tissues for several hours or days, use caution in shipping potatoes suspected of having been thus injured. If it is possible to hold the potatoes several days before grading and loading, a great deal of decay in transit can be avoided. Shipping the potatoes under refrigeration at 55° to 65° F can also minimize possible losses from the bacterial soft rot that always follows as a secondary effect.

References: *214, 217, 225, 272.*

Heat or Drought Necrosis

Symptoms

Heat or drought necrosis causes slate-gray to golden-yellow or brown discoloration of the water vessels of the affected tubers. It is most pronounced in the vascular system and may occur at either the stem end or the bud end. It also is in the tissues between the vascular ring and the tuber surface. Discolored tissue near the surface makes the skin appear dark, but generally there are no external symptoms.

Causal Factors

Heat or drought necrosis occurs on tubers grown in light soils in hot, arid areas. It is especially likely to appear on tubers left in the soil after the vines begin to die.

Control

There are no postharvest control measures.

References: *20, 65, 328.*

Hollow Heart

Hollow heart is a disorder of potatoes grown anywhere in the world. It can occur in potatoes growing in the field or on tubers during transit or in storage. It causes serious losses and at times seriously affects the marketability of potatoes.

Symptoms

The term "hollow heart" describes cavities that form near the center of very large tubers (pl. 11, *G*). These cavities may vary greatly in size and usually have radial cracks extending in many directions from the central cavity. The cavities usually are lined with first a pinkish and later a light-brown to brown dead tissue. The exact cause of this type of hollow heart is not known, but it is believed to be due to some type of abnormal condition that causes too rapid growth or an abnormal type of growth of the tuber.

A second type of hollow heart occurs when the tubers are exposed to very high temperatures in the fields before harvest, at the packing sheds before shipment, or during transit if they become excessively hot (pl. 11, *H*). The tubers contain from one to many small cavities distributed throughout the flesh. They may be near the center of the tuber but frequently are about a half inch beneath the skin. They are rarely over one-half inch in diameter and are lined with jetblack tissue. The condition is due to suboxidation caused by high temperatures and probably was preceded by blackheart. Tubers with this type of hollow heart symptom have been reported from Alabama, Arkansas, California, and Mississippi.

A third type of hollow heart is associated with chilling injury (see Mahogany Browning). Tubers held for long periods at or near 32° F develop both mahogany browning and blackheart. During this holding period the blackheart tissue may collapse leaving a cavity lined with jetblack dead tissue (pl. 11, *H*).

Control

Close spacing of seed pieces in the rows is reported to decrease chances of excessively large tubers and hence reduce chances of normal field hollow heart. After the vines are dead or removed during hot weather, the tubers should be harvested as promptly as possible to avoid high soil temperatures because of no vine covering. During storage and transit proper air circulation will prevent buildup of excessively high temperatures and remove possible suboxidation. Storage of tubers at 40° F and above will prevent chilling injury.

References: *139*, *140*, *160*, *176*, *308*, *309*.

Internal Black Spot

Internal black spot (black spot) of potatoes was first described in England in 1913. In Europe it is known as bruise, blue discolora-

tion, bluing, and blue spotting. It was first reported in the United States on Long Island, N.Y., in 1940. Since then it has been reported in Maine, New York, New Jersey, Pennsylvania, the Red River Valley of Minnesota, North Dakota, California, and Washington. It occurs in almost any region where potatoes are stored. The disorder has caused losses of over a million dollars a year on Long Island alone.

Symptoms

The black spots are generally limited to the outer one-fourth to three-eighths inch of tuber flesh and extend from the vascular ring outward. Occasionally the deeper tissues are affected. The spots are largely concentrated on the stem end half of the tuber and are generally on the shoulders, which are most likely to be bumped and bruised during handling. These spots are generally beneath the unbroken skin where they are not visible, even when the tuber is washed (pl. 12, *A*). This is due to a normal, uncolored thin layer of cortical cells about one-sixteenth inch thick between the discolored spots and the skin. The spots develop from a slight jolt or bruise during handling. At first they are pink, then after 4 to 6 hours they become dark red, and finally after 12 to 18 hours they are coal black. Later after the area begins to dry out, it shrinks and becomes slate gray (pl. 12, *B*). The spots are usually circular and range from a barely visible grayish tint to gray spots one-half to three-fourths inch in diameter.

External symptoms are readily visible in more severe cases of black spot. They result from a rapid drying out of the blackened tissues and the thin overlying layer. Consequently, the surface of the tuber over the black spots becomes irregularly sunken to a depth of from one-sixteenth to one-eighth inch. At first the blackened tissue is of nearly normal texture, but later it dries out and becomes starchy. Eventually it becomes sharply delimited through the formation of a thin boundary of cork tissue. This condition is generally referred to as pressure bruise or pressure spot.

Causal Factors

Black spot appears in susceptible tubers only after they have been bruised and usually only after the tubers have been removed from storage and passed over a grader. Normal handling and grading operations are sufficient to produce the discoloration by the time the potatoes reach the market.

Susceptibility of tubers to black spot seems to be dependent on

(1) cultural and environmental conditions under which the plants grow in the field, (2) the stage of tuber maturity at harvest, (3) the environmental conditions in storage and in transit to market, and (4) the duration of the storage period. Therefore the presence or absence of black spot on any single lot of potatoes indicates the degree of susceptibility or resistance at that specific time and for that specific lot.

Turgidity of tubers is a factor in their resistance to pressure bruise and black spot. There is an apparent optimum condition of turgor at which potato tubers are resistant. As water is lost during storage or in marketing and turgor of the tubers decreases, they become more susceptible to black spot. However, as the water loss continues, the cells reach a certain point of dryness at which the tubers become rubbery and once again are resistant. Black spot is apparently due to changes in physical structure and response of the tuber cells to impact.

The pressure bruise and black spot complex is becoming more common in modern potato storage facilities designed to maintain higher holding temperatures for processing potatoes. These facilities are equipped to force air through the stored potatoes so as to regulate temperatures, but they cause a greater than normal loss of tuber moisture. Unless carefully regulated, this condition plus the higher holding temperature increases the susceptibility of the tubers to internal black spot.

Although a close relationship exists between pressure bruise and internal black spot, either condition can appear without the other. The flattened or indented areas known as pressure bruise are not commercially important without black spot, for these areas tend to round out and become inconspicuous in the market channels. However, if the tubers are susceptible or predisposed to black spot, a slight pressure or bump on these areas will cause internal discoloration or black spot.

In the western potato-growing areas, a condition referred to as deep black spot is attributed to high soil temperatures. When coupled with tuber bruising, it causes a physiological rot. The bruised area at first appears as a black spot on the surface followed by a rot that penetrates to the center of the tuber. This is not true of internal black spot, for it does not cause a tuber rot.

Considerable research has been conducted to determine the effect of fertilization on black spot development. Because of conflicting and inconsistent results with nitrogen, potassium, and copper, it is impossible to prove a specific relationship between the elements and internal black spot.

We know that certain conditions can result in internal black spot in susceptible tubers, but the basic cause or causes are unknown.

Control

Avoid conditions during storage, transit, and marketing such as rapid flow of dry air, which may result in excessive loss of tuber moisture. After storage at low temperatures, warm the tubers to 55°-60° F before grading and packaging them.

References: *33, 48, 61, 144, 165, 201, 211, 221, 238, 239, 253, 259, 260, 295, 302-304, 312, 313.*

Internal Brown Spot

Internal brown spot has been noted on market potatoes from several regions. It was particularly prevalent in the Long Island and New Jersey crops in 1944, 1948, 1949, and 1953.

Symptoms

Affected potatoes have dry, firm, corky, light reddish-brown spots or blotches scattered throughout the flesh. There are no external symptoms. The spots vary from minute flecks to three-fourths inch or more in diameter and are irregular and indefinite in outline. Usually they are from one-fourth to one-half inch (pl. 12, *C*). Most of the spots are at the bud or eye end and are seldom found much beyond the middle of the tuber. Internal brown spot is very severe and appears most commonly in large, long, and irregularly shaped tubers.

Causal Factors

Internal brown spot develops before the potatoes are dug. It does not increase in severity or extent in storage. It is a physiological disorder that seems to result from extremely high temperatures and drought during the last part of the growing season.

Control

The Pontiac, Kennebec, Katahdin, and Mohawk varieties are relatively resistant to internal brown spot. There are no postharvest control measures.

References: *77, 96, 97, 169, 327.*

Jelly-End and Glassy-End Rots

Both jelly-end and glassy-end rots result in serious losses during harvesting, storage, transit, and marketing. Losses often are due to a soft watery breakdown. Sometimes apparently sound tubers make black potato chips and disintegrate during canning or other processing. Jelly-end and glassy-end rots have been considered distinct disorders, but because of strong similarities they are now considered the same.

Symptoms

Jelly-end or glassy-end rot always occurs at the stem end of tubers and often shows no external symptoms. When external symptoms occur, the stem end is soft and the skin wrinkled (pl. 12, *D*). The rotted flesh varies from colorless or glassy translucent to straw color and various shades of brown or black (pl. 12, *E* and *F*). The rotted tissue is fragile and easily broken apart, has a jellylike consistency, is devoid of fibrous texture, and is very watery. A sharp line of demarcation forms between affected and healthy tissue. During storage, water evaporates from affected areas, leaving a shriveled clump of epidermal tissue collapsed at the stem end.

Both jelly-end and glassy-end rots are most frequently found in tubers that have made secondary growth. The disorders differ mainly in the growing regions and on the varieties of potatoes they affect.

Jelly-end rot appears principally on long russet-type potatoes grown in California and the north-central region (pl. 12, *E*). It is found most frequently in tubers with malformed secondary growth, such as those with constricted stem ends (bottleneck), those with constrictions near the middle (dumbbell shaped), and sometimes long pointed types.

Glassy-end rot is found in round-type potatoes grown in the eastern and northeastern regions. It frequently occurs at harvest on bottleneck or dumbbell-shaped tubers and on tubers with abnormal stolon growth from the bud end (pl. 12, *G* and *H*). Small tubers present on these stolons at harvest do not have glassy-end rot symptoms.

In the long russet-type tubers the glassy condition extends 1 to 2 inches from the stem end. With the round-type tubers the glassiness may extend throughout the primary but not the secondary tuber. These glassy areas are devoid of starch. Absence of starch can easily be detected by failure to develop a deep purple color in the flesh when slices from the tubers are placed in potassium iodide

solutions. Both types of potatoes form a mushy translucent jelly when they are boiled.

Causal Factors

Jelly-end or glassy-end rot is caused by weather conditions that temporarily stop or inhibit the development of young tubers, followed by their rapid growth. Conditions that may cause this disorder include very high temperatures, drought followed by rainy periods, and vine defoliation, each of which may result in secondary tuber development. Growth of secondary tubers and increased glassiness may occur after the vines are dead. The disorder does not become more pronounced in storage or transit. Glassy tubers in storage, however, may lead to wet spots or in transit to spotted sacks caused by the breakdown of the glassy areas and release of water. Both conditions are ideal for development of bacterial soft rot and other disorders.

Control

Irrigation started early in the growing season and continued until near harvest will help prevent secondary growth. Plant early maturing varieties or plant regular varieties late so that crops have passed or not reached critical stages of tuber production before severe weather fluctuations occur.

No postharvest control is known. Tubers from lots containing large amounts of glassy-end or jelly-end should not be processed.

References: *99, 180, 181, 216, 226, 248, 273.*

Knobbiness and Other Malformations

Knobby or malformed tubers often occur, particularly in the long Russet Burbank-type varieties. These malformations or protuberances result in serious grade reduction.

Symptoms

Knobby tubers have secondary lateral growth from a few tuber eyes. This growth produces knoblike protuberances and sometimes new tubers form on the original ones (pl. 13, *A*).

Offtype or malformed tubers may have constrictions near the stem end (bottle shaped), or they may have constrictions near the middle (dumbbell shaped), or they may be normal with pointed ends.

No active decay occurs with any of these malformations.

Causal Factors

The specific cause of knobby tubers is not definitely known. Such factors as the following contribute to knobbiness and other malformations: High soil moisture, stem girdling by mechanical means or pathogenic organisms that interfere with water transport in the vines, and partial or complete vine defoliation by hail or other physical means, especially when tubers are partially formed.

Control

No control methods are known.

References: *21, 26, 162, 276, 282.*

Mahogany Browning

Mahogany browning was once a very important storage and marketing disorder of some potato varieties, but because of better storage conditions it has become rather rare. However, it can cause serious losses under certain conditions.

Symptoms

Normally no external symptoms appear until the tubers are very severely affected internally or when they are stored for very long periods near 32° F. Under such circumstances the skin becomes bluish gray to purple, is wrinkled or cracked, and has numerous sunken pitted areas. Such tubers may be confused with those infected with the late blight organism, particularly because of the reddish-brown flesh beneath the discolored skin (pl. 13, *B*) and because they are hard or rocklike. The reddish brown of mahogany browning may occur anywhere in the flesh of the tuber, whereas late blight usually is limited to the outer one-half inch of the flesh.

Symptoms of this disorder vary with different varieties. Katahdin, Chippewa, and Pungo show many manifestations of mahogany browning. The first symptom is a diffuse grayness occurring anywhere in the flesh. Sometime at this stage a water-soaked area or brownishness appears just under the skin. Later a firm, almost granular reddish-brown discoloration either replaces the gray or is surrounded by grayish areas (pl. 13, *B*). No sharp lines of demarcation separate this firm tissue from the unaffected flesh. In very advanced stages some blackened areas resembling blackheart develop in the reddish or gray flesh. Eventually the flesh in these

blackened areas disintegrates, leaving hollow cavities with blackened walls.

With such varieties as Sebago and Kennebec, which apparently are less susceptible to chilling injury, symptoms are less pronounced. First symptoms are yellowing and firming of affected flesh followed by the development of black areas. The flesh in these blackened areas later collapses forming cavities. Sometimes between the blackened areas the diffuse gray and reddish-brown discolorations typical of mahogany browning appear (pl. 13, *C*).

Causal Factors

Mahogany browning is an injury caused by low temperatures. It occurs when tubers are stored at 32° to 34° F for at least 3 months. With longer storage the internal symptoms become more intense and external symptoms appear. Mahogany browning should not be considered a form of freezing injury since it occurs at well above the freezing point of the tubers.

Control

Do not store potatoes below 40° F. Sometimes during storing the tuber temperature drops to near 32°. Under such circumstances it is advisable to warm them to 65° for a short period and then return them to 40° storage to avoid mahogany browning. Storage walls and bins should be properly insulated so that tubers near the walls will not be exposed to unreasonably low temperatures. Forced-air circulation should be used to keep temperatures uniform.

References: *88, 133, 139, 140.*

Mechanical Damage and Cracking

Mechanical damage to potato tubers during digging, handling, storage, and transporting probably causes more losses than any other factor. Losses can be direct from excessive damage or indirect by providing an entrance point for tuber-rotting organisms. A survey of wastage in Great Britain revealed that almost one-third of the potatoes removed from the field had been mechanically damaged to the extent that they were unfit for marketing and another one-third had some minor mechanical damage that could predispose them to tuber rots. Only 4.6 percent by weight of the total crop was absolutely free of damage. A similar situation no doubt exists sometimes in the United States.

Causal Factors

Cracking or bruising of potatoes can be caused by many conditions. Growth cracks in tubers are generally associated with an uneven water supply or incorrect use of fertilizers or both (pl. 13, *D*). These conditions cause an uneven rate of tuber growth with the resultant cracking of the rather tough skins. Pressure bruises from heavy loading in transit, particularly on tubers next to the floor racks, may cause cracks. These cracks may be severe and readily visible on the exterior or in less severe cases may be seen only after cutting the tuber. They do not extend uniformly through the tuber but present a shattered or netlike pattern in the area where excessive pressure has been applied.

Curved thumbnail cracks, which become conspicuous after grading of cold-stored crisp tubers, particularly after some drying out, result from glancing impacts of tubers with other tubers or from digging and handling equipment (pl. 13, *E*). This condition, which occurs anytime from harvest to removal from storage, is the result of mechanical shock in tubers that have developed a high internal pressure from increased water absorption. The amount of mechanical shock necessary to induce this cracking may be extremely slight.

Other cracking may be in the form of digger cuts or shattering during harvest operations. Deep cracks appearing suddenly during digging are caused by harvesting when the tubers are immature or when soil and air temperatures are too low.

Control

Handle potatoes gently at all times. Postpone digging until tubers are mature. In cold weather delay harvesting until late morning so the soil will have time to warm up.

Potatoes should be warmed before they are removed from storage, graded, or handled in any way.

References: *20, 82, 93, 130, 131, 146, 149, 187, 188, 209, 291, 310, 314, 315.*

In addition to natural russeting of such varieties as Russet Burbank and Russet Rual, there are blemishes and discolorations of smooth-skin varieties known as soil scurf, sand scurf, russet scab, and russeting. In many instances these blemishes resemble the russeting caused by the *Rhizoctonia* fungus on the tuber surface or

the superficial lesions caused by the common scab organism *Streptomyces scabies*.

Causal Factors

The exact cause of much of this russeting is unknown. However, the fact that by careful examination some *Rhizoctonia* can be found on the skin of practically all tubers has suggested that this fungus may cause most of these problems. Some studies have indicated that other causes may be responsible. A very serious type of russeting that sometimes occurs in Missouri, Kansas, and Nebraska has been reduced by some soil sterilizing agents, indicating that an organism might be responsible for the problem. Since this condition is usually more prevalent in sandy soils, it has been referred to as sand scurf. It is usually more serious in soil previously planted to potatoes, also indicating that an organism might be involved.

Any irritation or injury to the skin of growing tubers may lead to scurfy or scabby eruptions and corky discoloration, which might be called russeting. Under some conditions, tubers growing in contact with fertilizers in the soil may have rough skins that resemble russeting. Although russeting does not lead to decay in storage or in transit, the marketability of seriously russeted tubers is greatly affected.

References: *94, 252, 329.*

Stem-End Browning

Stem-end browning, which causes discoloration of the vascular system, can be considered an important disorder of potatoes and is often confused with net necrosis. It occurs mostly in Maine, New Hampshire, and Vermont but may be found in many other potato-growing regions. The vascular discoloration decreases the value of the tubers for general cooking and especially for chip manufacture. Symptoms of stem-end browning become more pronounced during storage.

Symptoms

No external symptoms of stem-end browning occur. When a thin slice is removed from the stem end of affected tubers, a dark-brown to blackish discoloration appears in the xylem and phloem strands radiating from the point of a stolon attachment (pl. 13, *F*). The discoloration rarely extends more than one-fourth to one-half inch

from the stem end (pl. 13, *G*). The depth of discoloration of the strands usually distinguishes this disorder from net necrosis.

Causal Factors

The cause of stem-end browning is unknown. Some evidence indicates that the disorder is more common when potatoes are grown for several years in the same fields with increased amounts of fertilizers, particularly those with high chloride and potassium content. There is also some evidence that it is caused by a virus. Stem-end browning may or may not be present when tubers are harvested. Maximum development occurs when tubers are stored at 50° F for about 100 days. Its incidence is greatly reduced by storing tubers from 32° to 35° or at 70° for about 60 days before permanent storage near 50°. The disorder is reported to occur when soil temperatures at harvest are too high for the development of net necrosis. Normally it is most apt to affect small potatoes. Green Mountain and Irish Cobbler are considered the most susceptible varieties to stem-end browning, whereas Katahdin and Chippewa are resistant.

Control

Conditioning potatoes for 60 days at 32° or 70° F before permanent storage near 50° will greatly reduce stem-end browning, which develops during constant storage at 50°.

References: *87, 92, 246, 247.*

Vine Killing

Killing potato vines a week or two before harvest has become a common practice in most potato-growing areas. Often tubers from these plants have varying degrees of vascular discoloration. Such discoloration can be confused with that caused by wilt diseases and by freezing and lessens the value of tubers, particularly for the potato chip industry. In this respect vine killing can be considered an important physiological disorder of potatoes.

Symptoms

Affected tubers have no external symptoms. Internally they have a brownish vascular discoloration, most pronounced at the stem end but often extending throughout the tuber (pl. 13, *H*). When the

vessels extending to each eye are affected, the peeled tubers exhibit a network of discolored vessels that resembles net necrosis. It differs from net necrosis since the discolored vessels do not extend to the inner flesh of the tubers and the browning is not as dark. Vascular discoloration from vine killing resembles that of verticillium wilt but differs usually in being dull brown rather than reddish brown.

Causal Factors

Any factor causing premature death of the vines, even frost or drought, can discolor the vascular ring. Certain chemical and mechanical methods of vine killing cause this disorder. Discoloration is more apt to occur when the vines are rapidly rather than slowly killed, when they are immature at the time of killing, and when plants are deficient in moisture when they are killed. The discoloration does not increase during storage and apparently does not make the tubers more susceptible to decay.

Control

No postharvest control methods are known.

References: *44, 81, 137, 138, 161, 192, 194.*

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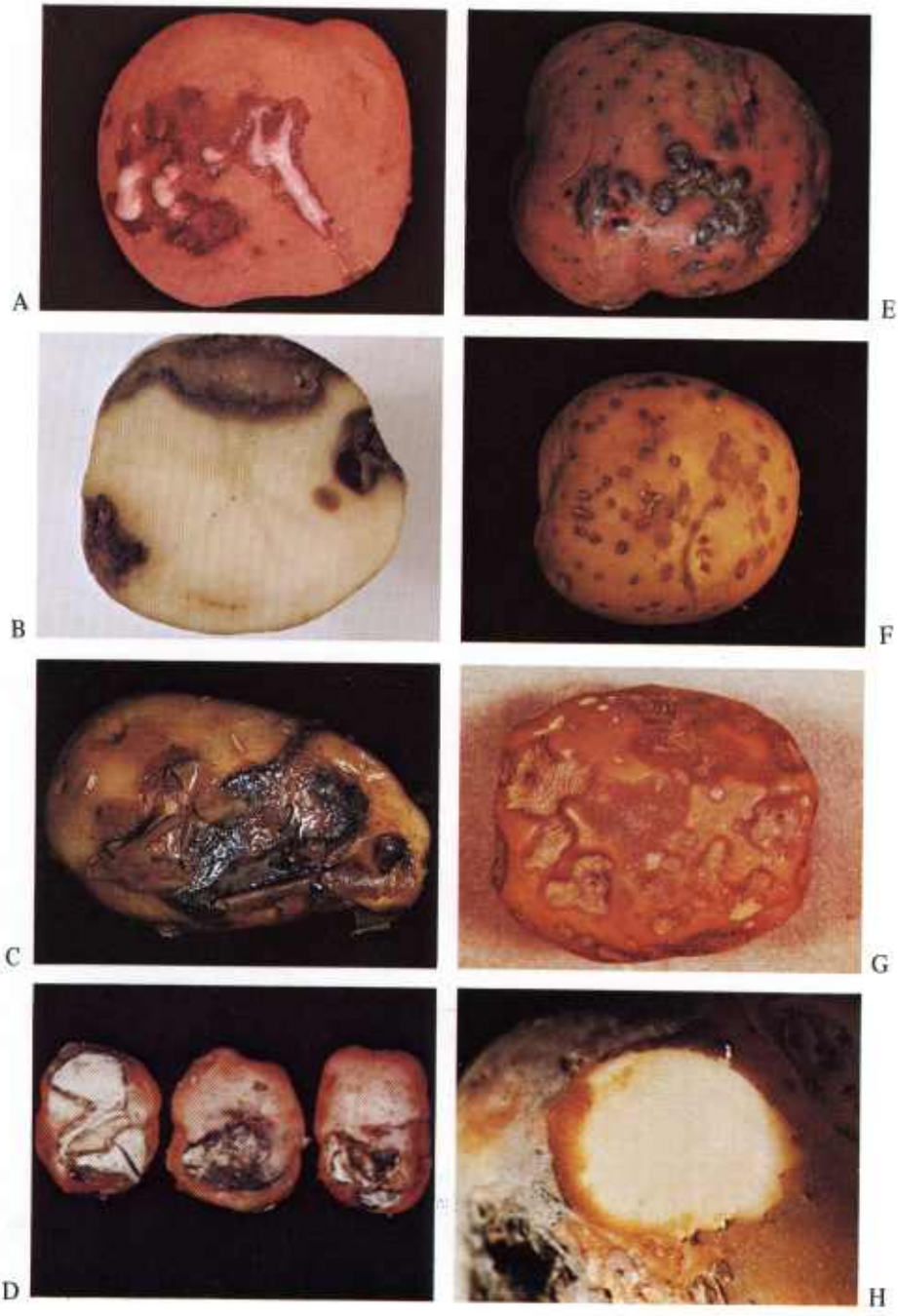
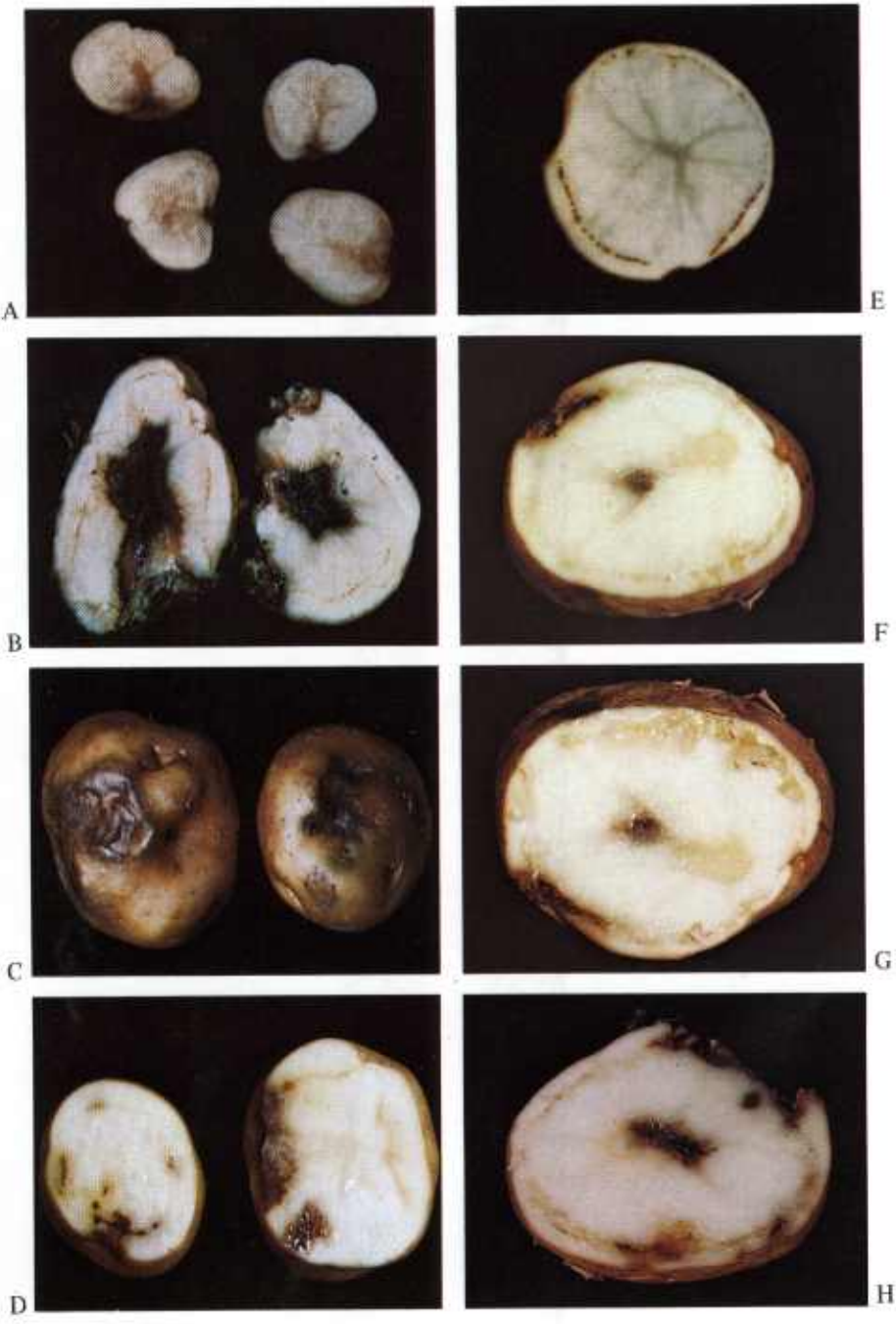
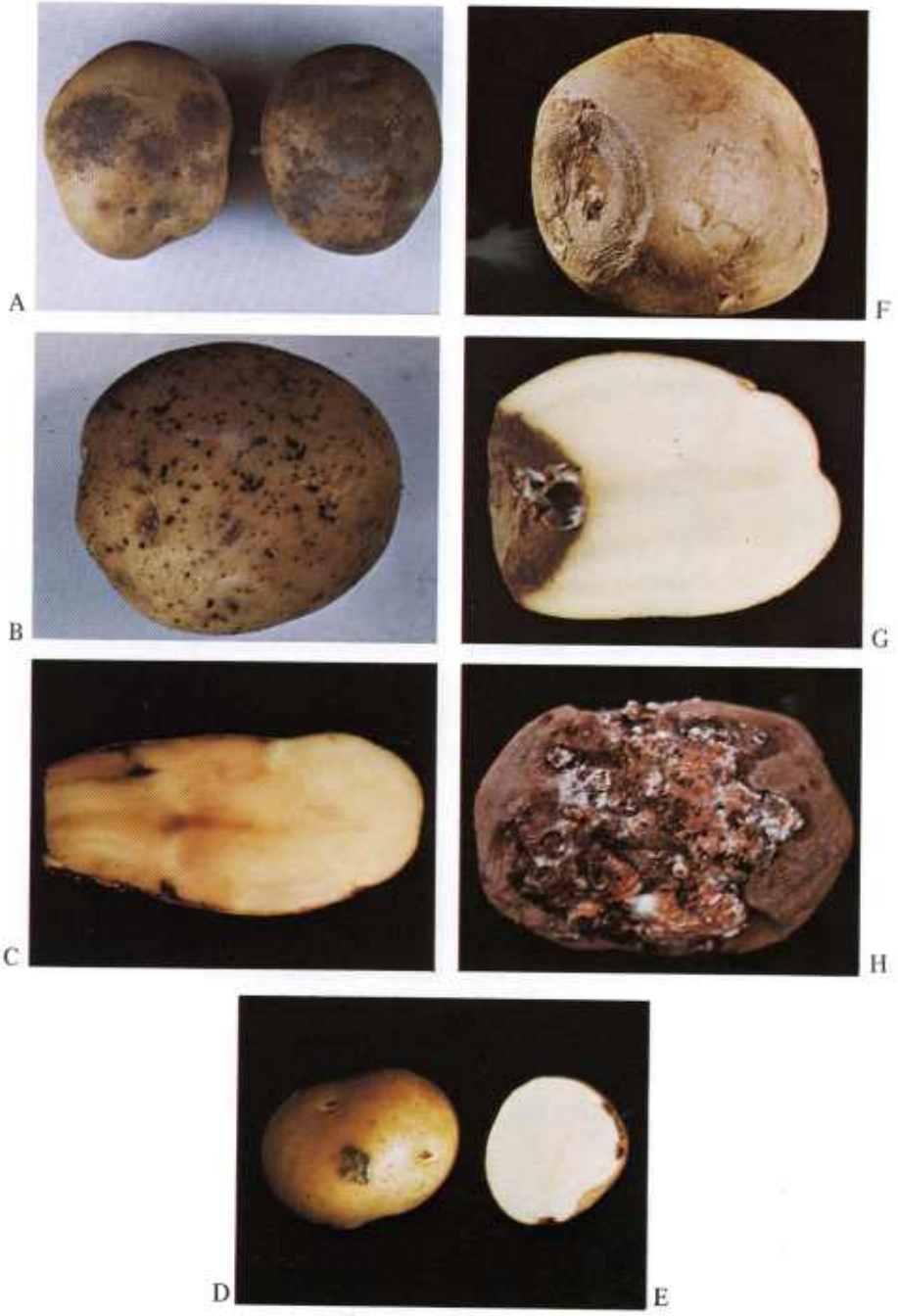


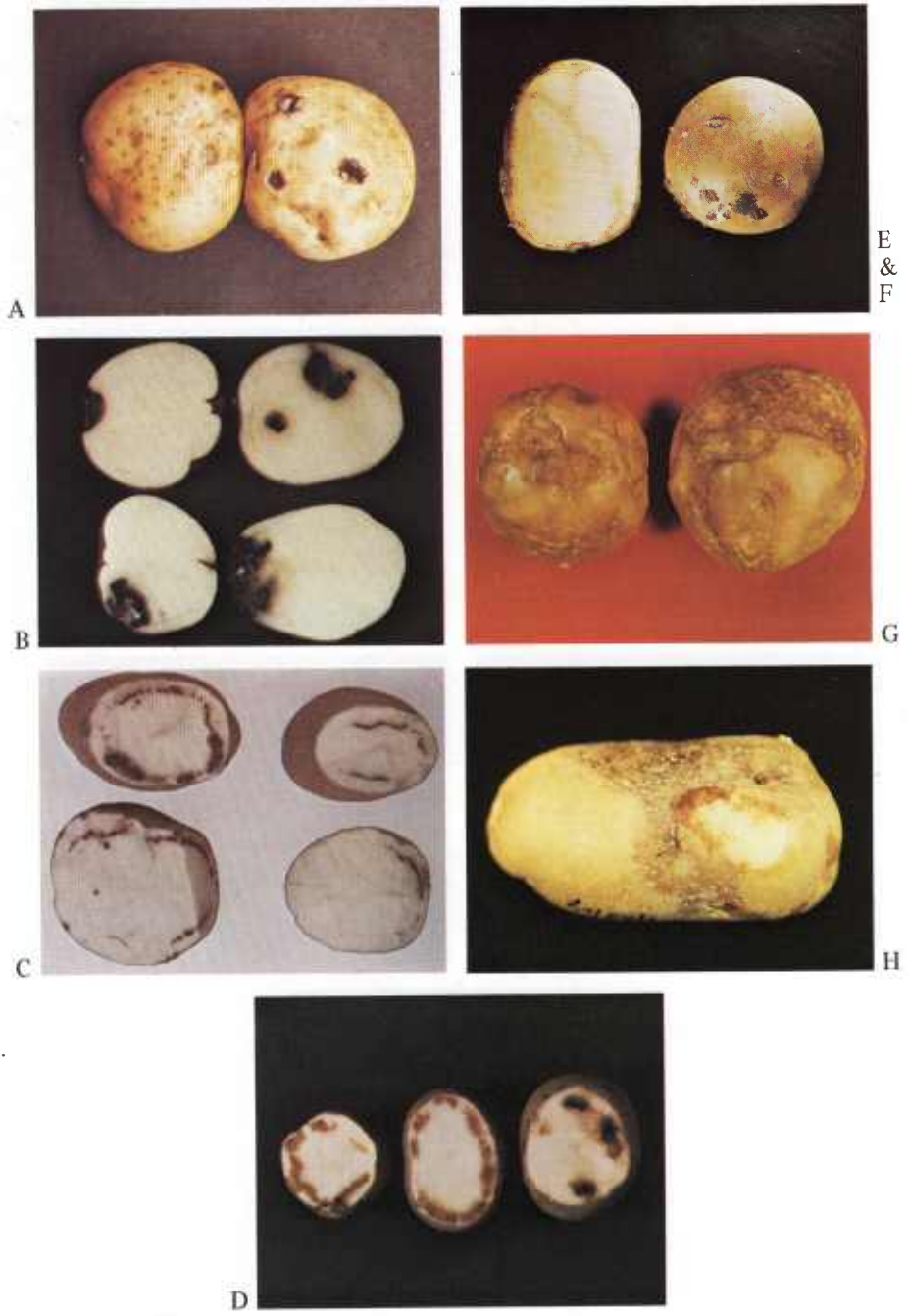
Bacterial diseases: *A-G*, Bacterial soft rot: *A*, Bacterial ooze; *B*, internal decay; *C*, advanced breakdown; *D*, dried infection; *E-G*, lenticel infection: *E*, Swollen stage; *F-G*, dried stage. *H*, Blackleg.



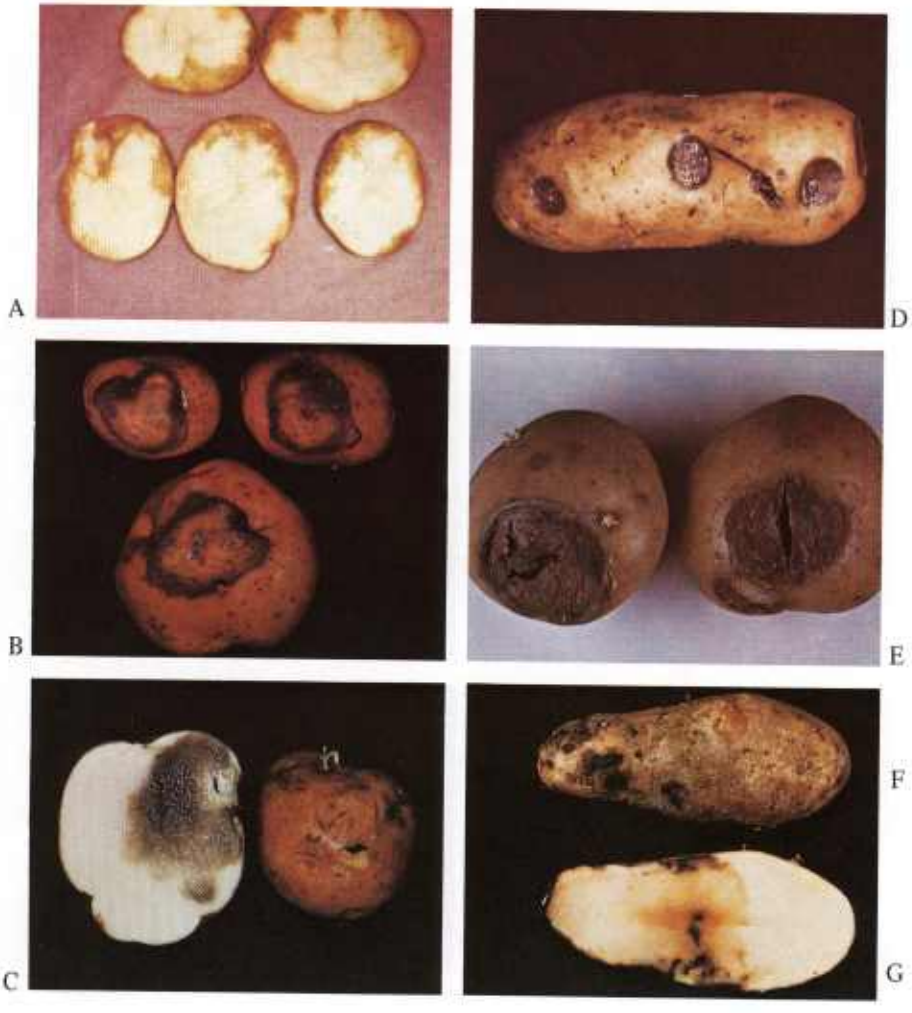
Bacterial diseases: *A-B*, Blackleg: *A*, Early field or harvest symptoms; *B*, late storage decay. *C-D*, Brown rot. *E*, Red xylem. *F-H*, Ring rot:. *F-G*, Early stage: *F*, Not squeezed; *G*, squeezed; *H*, advanced stage.



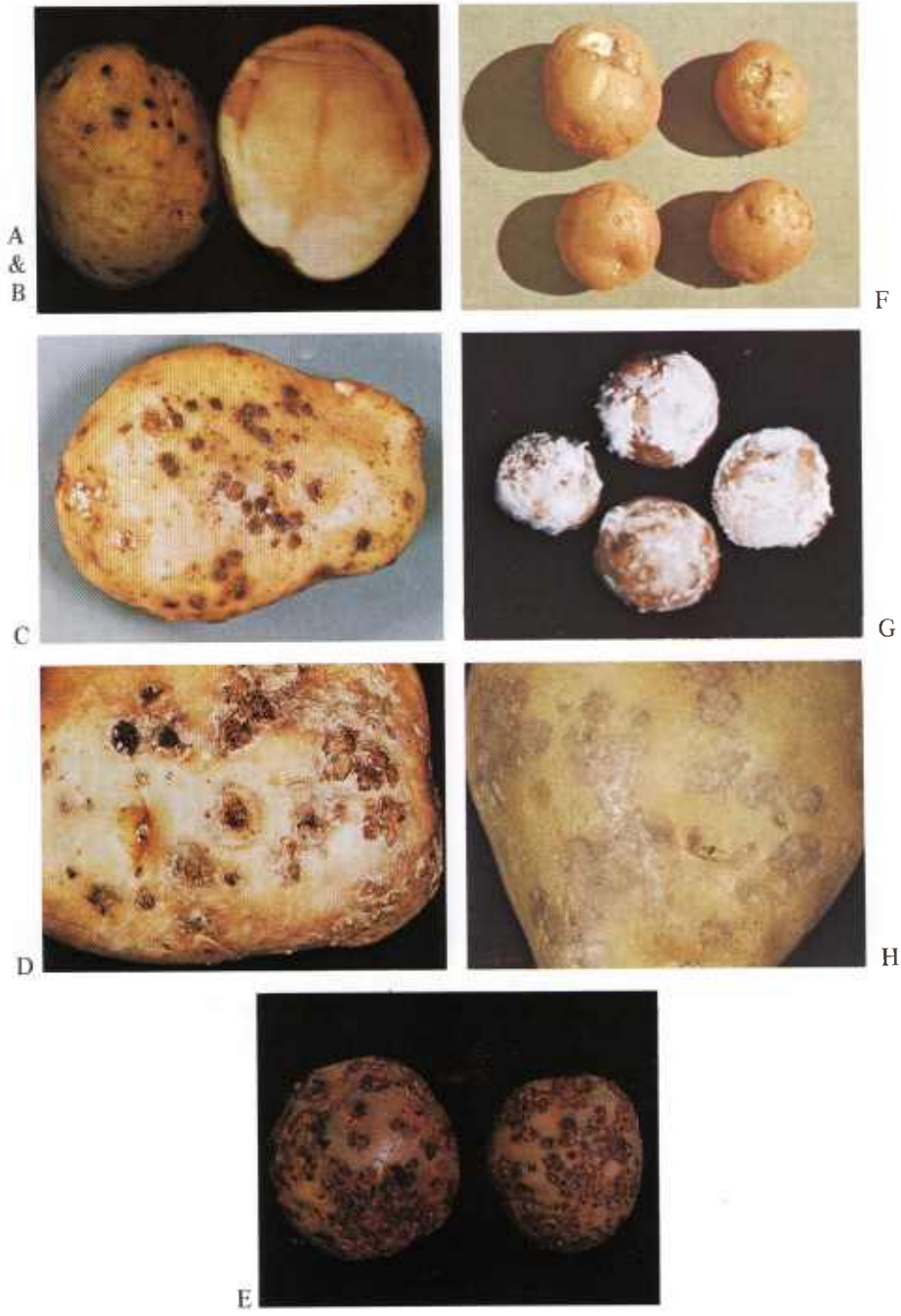
Fungus diseases: *A*, Black dot. *B*, Black scurf. *C*, Charcoal rot. *D-E*, Early blight (alternaria tuber rot). *F-H*, Fusarium tuber rot: *F-G*, Stem-end infection; *H*, advanced decay.



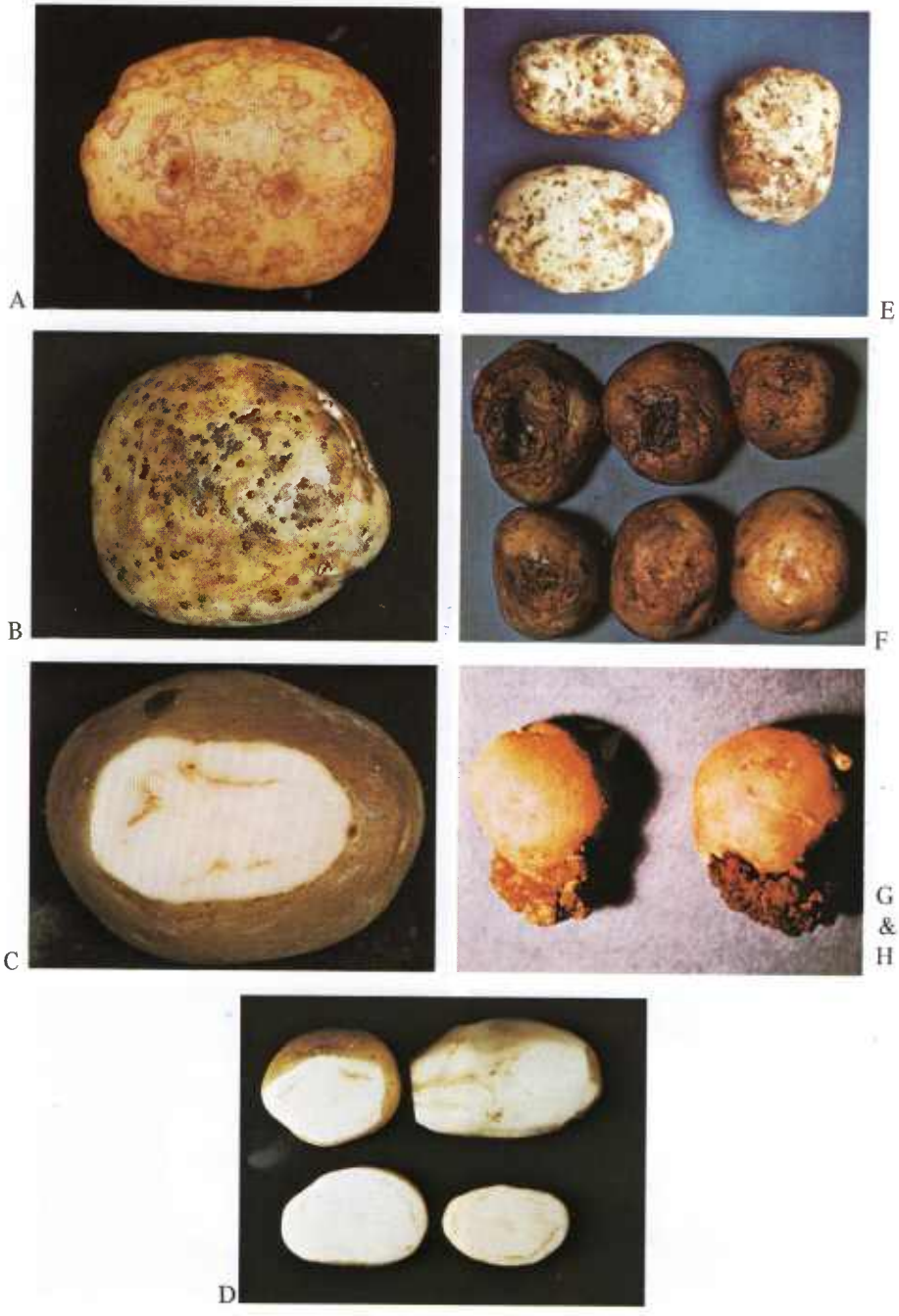
Fungus diseases: *A-B*, Fusarium tuber rot, buttonhole type. *C-D*, Fusarium wilt: *C*, *Oxysporum* type; *D*, *eumartii* type. *E-F*, Gray mold rot (*Botrytis*). *G-H*, Late blight.



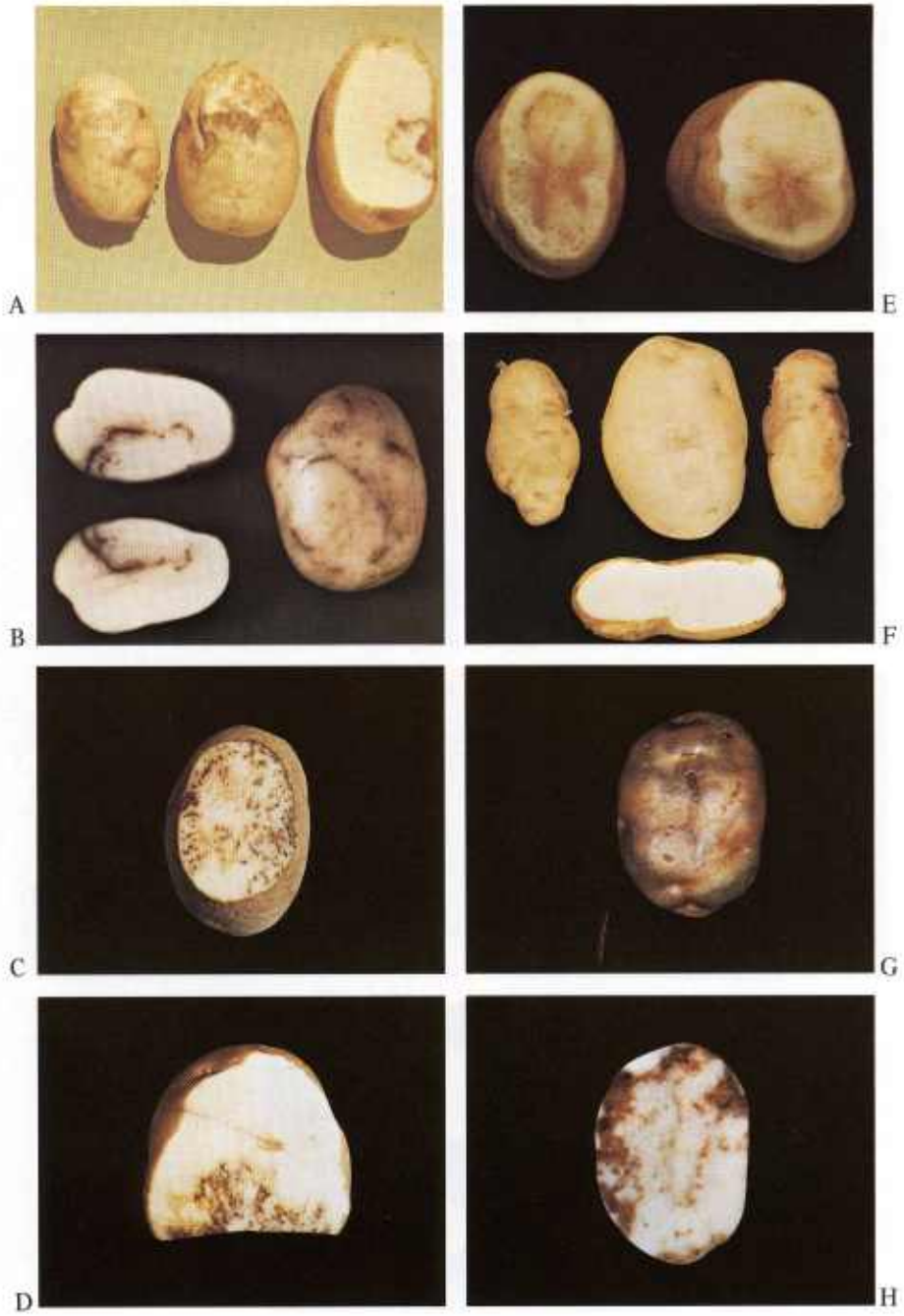
Fungus diseases: *A*, Late blight. *B-C*, Leak. *D-E*, Phoma tuber rot. *F-G*, Pink rot, western russet-type tubers.



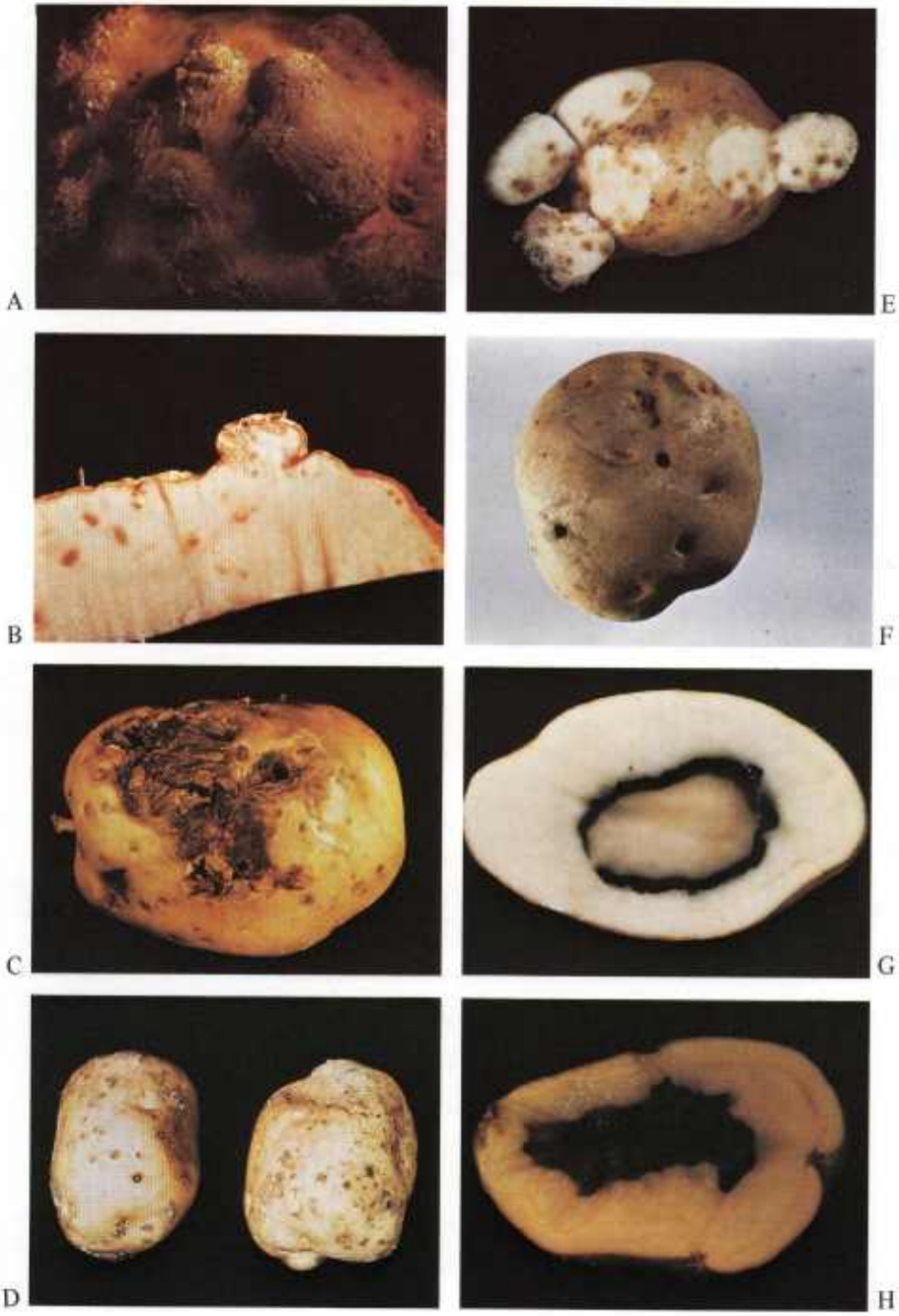
Fungus diseases: *A-B*, Pink rot, eastern round tubers. *C-D*, Powdery scab. *E*, Scab. *F-G*, Sclerotium rot. *H*, Silver scurf.



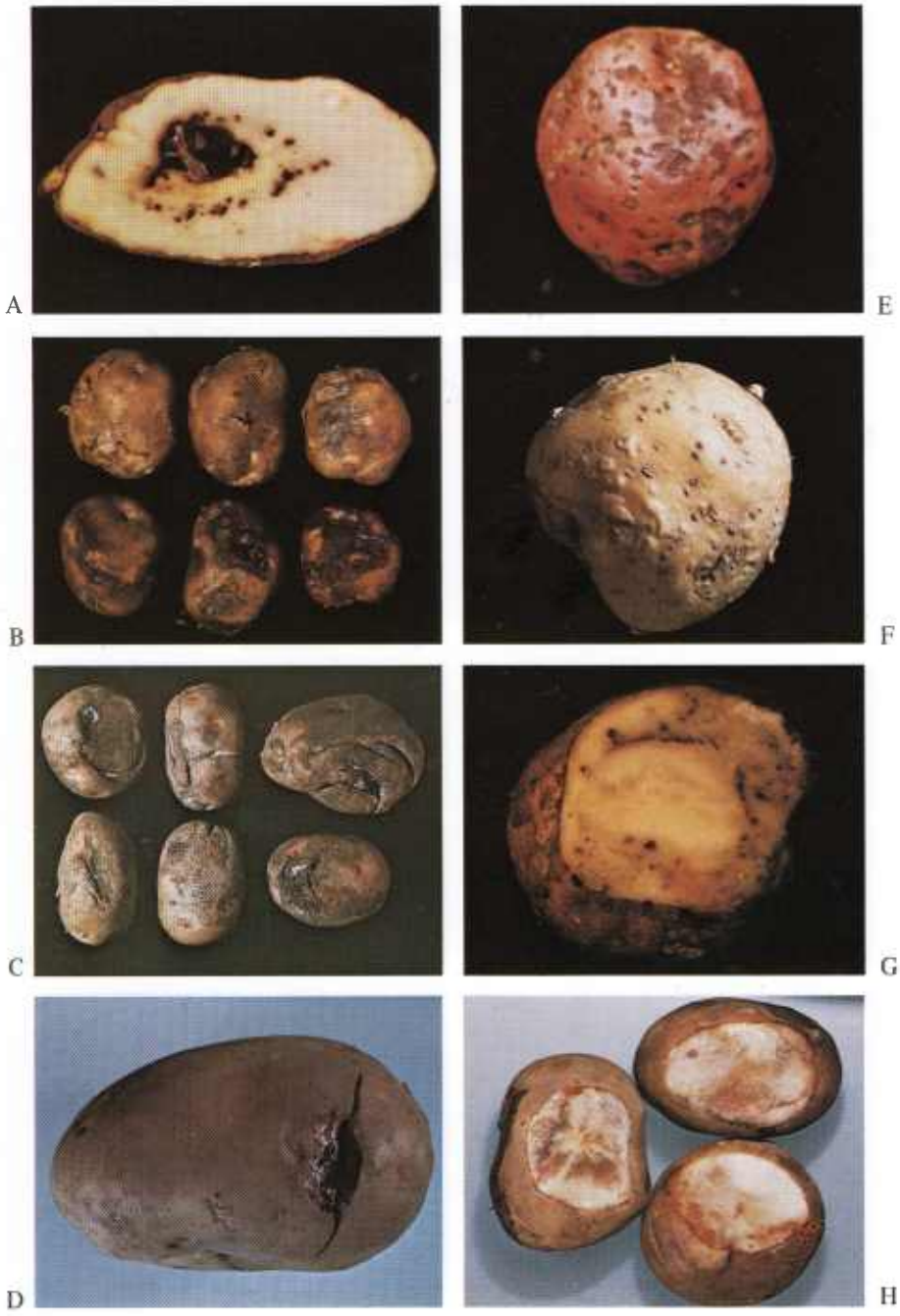
Fungus diseases: *A*, Silver scurf. *B*, Skin spot. *C-D*, Verticillium wilt. *E-F*, Pink eye. *G-H*, Wart: *G*, Early stage; *H*, advanced stage.



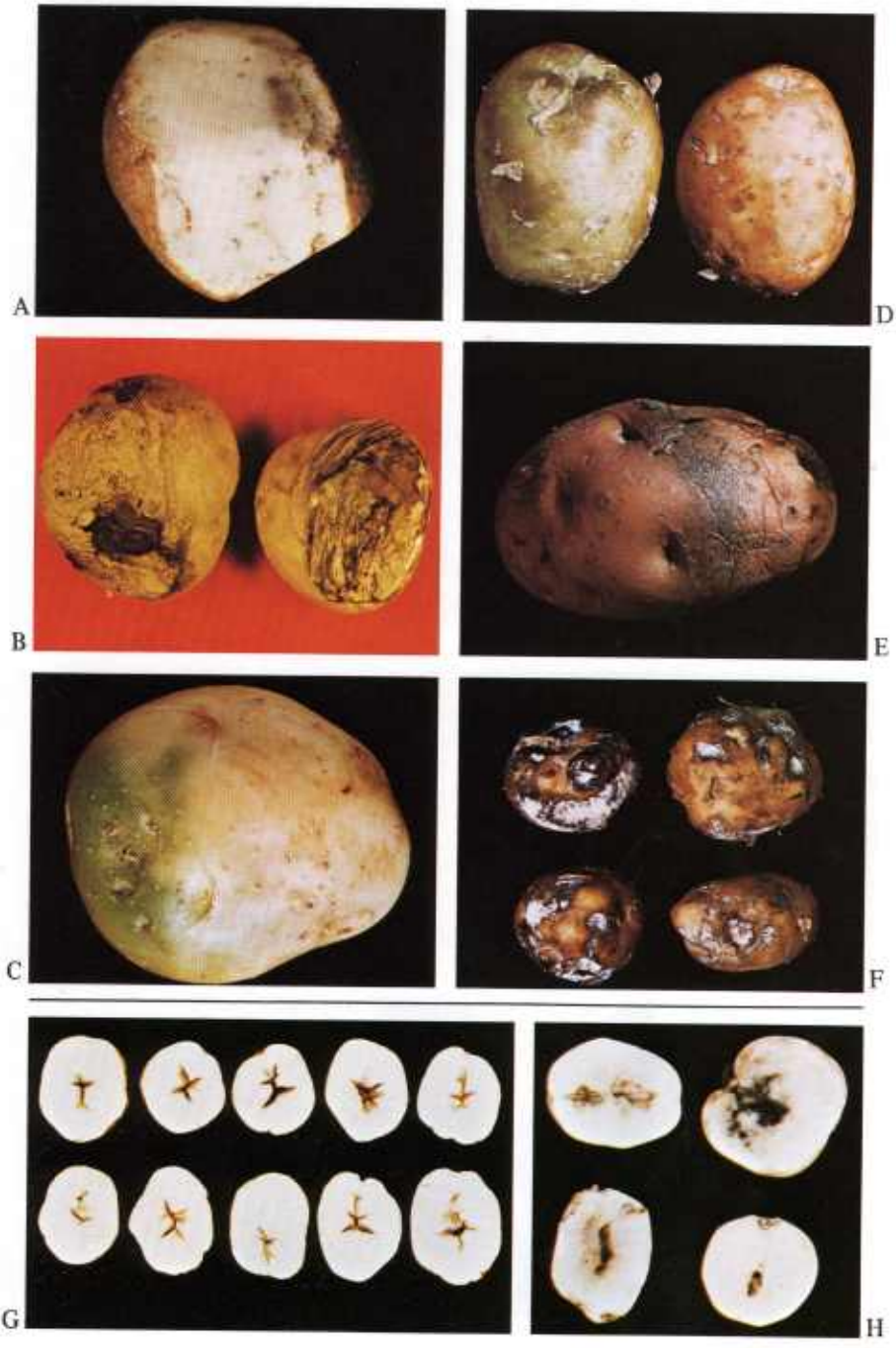
Virus diseases: *A-B*, Corky ring spot: *A*, Early stage; *B*, after storage. *C-D*, Net necrosis. *E*, Purple top wilt. *F*, Spindle tuber. *G-H*, Tuber necrosis.



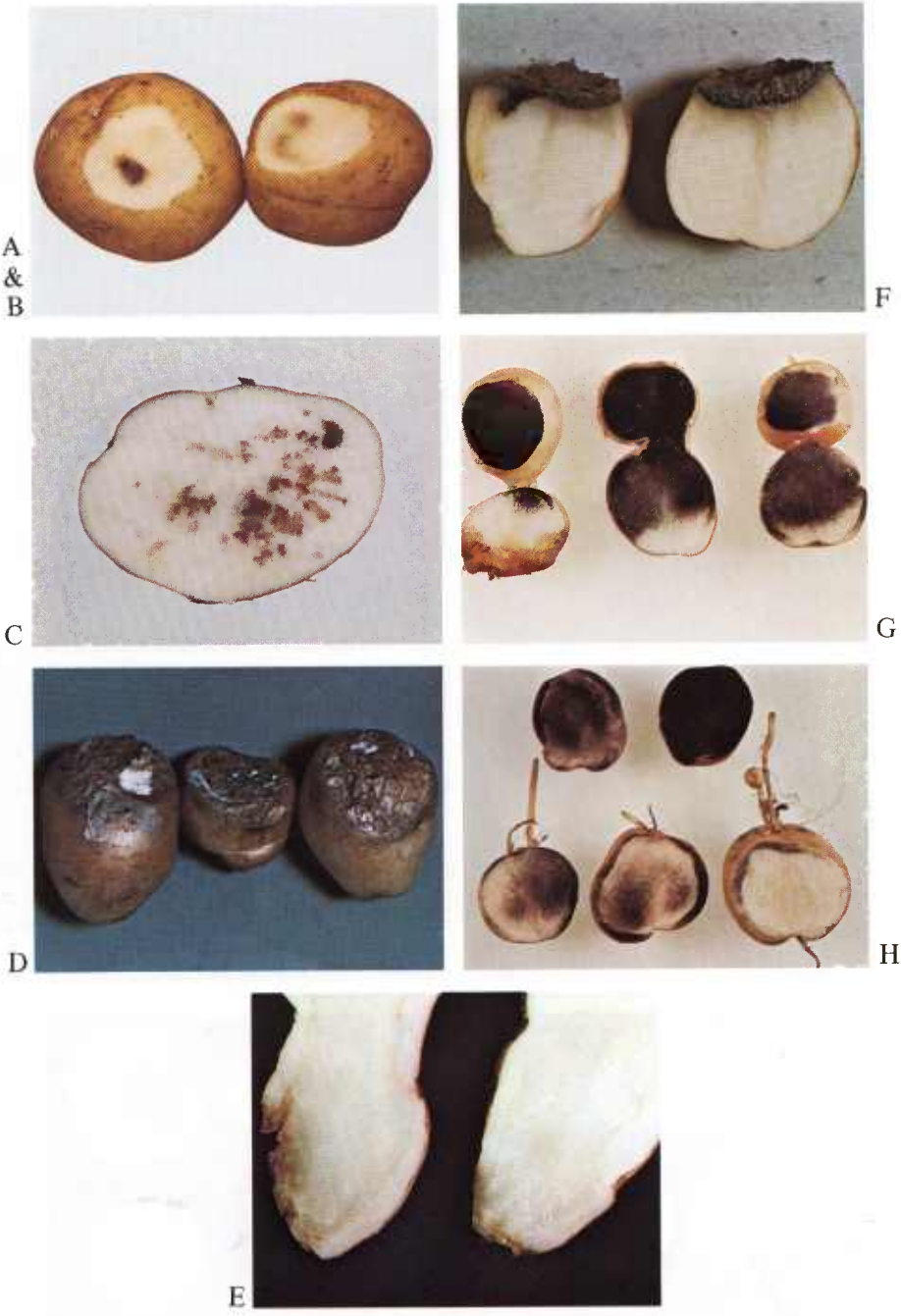
Nematodes and their injuries: *A*, Root knot. *B*, Root knot, internal view showing encysted female. *C*, Rot nematode. Insects and their injuries: *D-E*, Tuber flea beetle. *F*, Wireworm. Nonparasitic disorders and injuries: *G-H*, Blackheart.



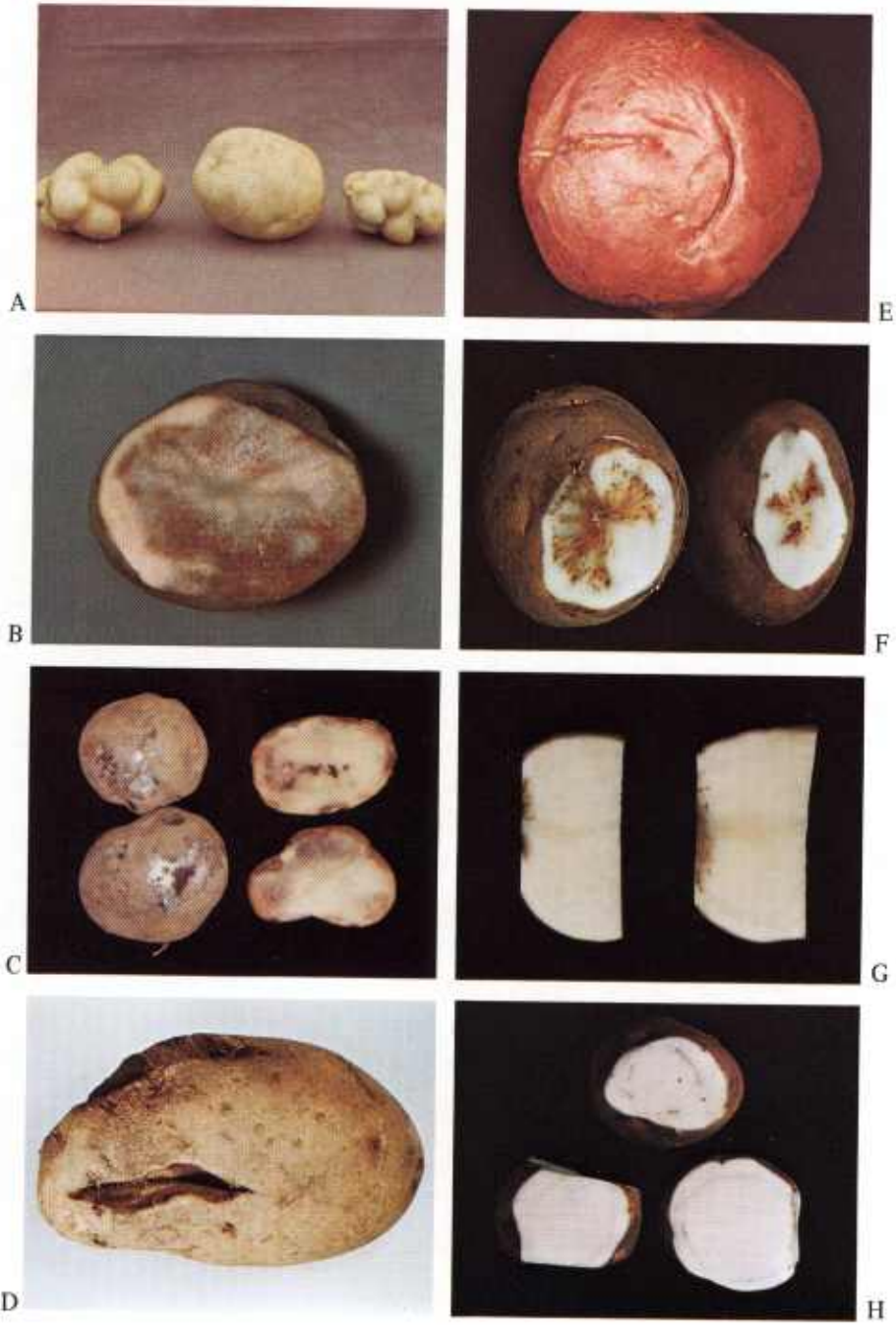
Nonparasitic disorders and injuries: *A*, Blackheart due to field heat. *B*, Browning. *C*, Floor bruising. *D*, Shatter bruising. *E*, Chemical (methyl bromide) injury. *F*, Enlarged lenticels. *G-H*, Freezing: *G*, Ring type; *H*, blotch type.



Nonparasitic disorders and injuries: *A-B*, Freezing: *A*, Internal netting; *B*, field frost. *C-D*, Greening: *C*, Field; *D*, retail display. *E*, Heat injury. *F*, Heat, deep scald. *G-H*, Hollow heart: *G*, Field; *H*, high temperatures and long storage at 32° F.



Nonparasitic disorders and injury: *A-B*, Internal black spot. *C*, Internal brown spot. *D-F*, Jelly-end rot of long russet potatoes. *G-H*, Glassy-end rot of round potatoes showing starchy areas stained with iodine and abnormal stolons from tubers.



Nonparasitic disorders and injuries: *A*, Knobby tubers. *B-C*, Mahogany browning: *B*, 'Katahdin' tuber; *C*, 'Kennebec' tuber. *D*, Growth crack. *E*, Air crack. *F-G*, Stem-end browning. *H*, Vine killing.

☆ U.S. GOVERNMENT PRINTING OFFICE : 1978 O—212-827

Conversion Table

°F	°C	°F	°C
30	-1.1	80	26.7
32	0	85	29.4
35	1.7	90	32.2
40	4.4	95	35.0
45	7.2	100	37.8
50	10.0	105	40.5
55	12.8	110	43.3
60	15.6	115	46.1
65	18.3	120	48.9
70	21.1	125	51.8
75	23.9		

Conversion:

°C = °F-32x5/9

°F = °Cx9/5+32

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