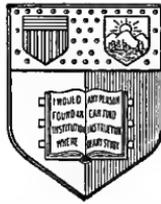


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**Office of Cotton, Truck, and Forage Crop Disease Investigations
Washington, D. C.**

**HANDBOOK
OF THE DISEASES OF VEGETABLES
OCCURRING UNDER MARKET,
STORAGE, AND TRANSIT CONDITIONS**

By

GEORGE K. K. LINK and MAX W. GARDNER

Prepared under the direction of W. A. ORTON and W. M. SCOTT

Preliminary Edition

Printed for the use of the Food Products Inspectors of the Bureau of Markets

Not available to the public

May 24, 1919

May 20, 1933

Mr. W. W. Ellis,
Librarian, Cornell University,
Ithaca, New York.

Dear Mr. Ellis:

Your letter of May 2 requesting a copy of the Handbook of the Diseases of Vegetables Occurring under Market, Storage, and Transit Conditions," by G. K. K. Link, has been referred to this Division for attention. This was published in 1919 for the use of food-products inspectors. No copies were placed on sale through the Superintendent of Documents and the publication is now out of date. We have, however, a very limited number of copies in our reserve office files and take pleasure in sending you a copy for your library.

Miscellaneous Publications 98 and 121, entitled "Market Diseases of Fruits and Vegetables: Potatoes," and "Market Diseases of Fruits and Vegetables: Tomatoes, Peppers, Eggplants," respectively, are the first of a series intended to take the place of the above-mentioned Handbook. We assume that your library was included in the official distribution of these and that you have a single copy of each.

Yours very truly,



W. W. Gilbert,
Senior Pathologist.

HANDBOOK OF THE DISEASES OF VEGETABLES OCCURRING UNDER MARKET, STORAGE AND TRANSIT CONDITIONS

By GEORGE K. K. LINK and MAX W. GARDNER. Prepared under
the direction of W. A. ORTON and W. M. SCOTT.¹

PREFACE

This handbook is designed primarily to aid the inspectors of the Bureau of Markets in the detection and identification of plant diseases as they occur in vegetables under transit, storage, and market conditions.

The entire inspection service and the survey and study of vegetable crop diseases under transit and market conditions, were begun but little more than a year ago, consequently this handbook, an outgrowth of that survey and study, is necessarily incomplete. The text and illustrations are the property of the United States Department of Agriculture, and are not to be used in any published form whatsoever.

The description of the signs, symptoms, or effects of each plant disease is followed by a statement of the geographic distribution and the seasonal or climatic relations of the disease so far as they are known, since some diseases occur only in certain districts and often only under certain conditions.

A statement is made as to the time and place of inception, development, and spread of the disease. This is especially important from a market point of view. For example, in placing the responsibility for the poor condition of a shipment caused by bacteria or fungi, it is essential to bear in mind that some diseases are field infections and do not develop nor spread in transit or storage; that others are field infections which develop or progress but do not spread in transit or storage; that others are field infections which develop and spread under transit and storage conditions; and finally that a number are transit and storage infections of stock perfectly sound when harvested.

It is fully realized that at present not all decay can be referred to clearly defined causes. However, by specifying whenever possible the disease responsible for the decay or defect in a consignment, the inspector can further the con-

¹ Dr. V. B. Stewart was consulted freely in the preparation of the text. In addition, the following men were consulted with reference to special portions as enumerated below: Beans, Mr. W. W. Gilbert and Mr. G. A. Meckstroth; cabbage and other crucifers, Dr. L. L. Harter and Dr. L. O. Kunkel; celery, Dr. I. C. Jagger; cucumber, Mr. W. W. Gilbert and Mr. W. N. Ankeney; potato, Dr. H. A. Edson, Prof. L. R. Jones, Dr. L. O. Kunkel, Dr. W. A. Orton, Mr. M. Shapovalov, Mr. E. S. Schultz, and Dr. H. G. MacMillan; onion, Dr. J. C. Walker; lettuce, spinach, radish, etc., Dr. I. C. Jagger, Mr. J. B. Norton and Mr. L. P. Byars; tomato, Mr. G. H. Godfrey, Mr. F. J. Pritchard, Dr. J. Rosenbaum and Mr. W. B. Clark; watermelon, Mr. F. C. Meier; sweet potato, Dr. L. L. Harter and Dr. J. L. Weimer.

trol and prevention of diseases, with the resultant improvement of American agriculture and the elimination of waste.

Inspectors should make it a point to send in material concerning which they are in doubt. By doing this they will aid themselves in acquiring a better acquaintance with the various diseases, and will aid in the detection of new diseases or of the occurrence of diseases in regions from which they have not been previously reported. Cartons and franked tags will be supplied the inspectors for use in sending such material. Many specimens can best be sent wrapped in paper (newspaper does admirably). It is very important that material be mailed without delay.

INTRODUCTION.

THE NORMAL PLANT.

Plants and Plant Parts Are Living.

VEGETABLES and fruits are plants or plant parts and are alive. Few people realize that a potato, an onion, an apple, or other fruit or vegetable is alive and is therefore subject to injury, disease, and death. Failure to realize this fact is responsible for much of the loss in the handling of fruits and vegetables.

The Structure of Plants.

Our agricultural plants are composed of members or organs, such as the root, stem, leaf, flower, and fruit. These organs in turn are composed of tissues, the stem, for example, usually being composed of pith, ring, and bark tissue. Tissues, finally, are made up of cells, which are microscopic in size.

Plant tissues often are classified according to their functions. There are, for example, the protective tissues, the supporting tissues, the conductive tissues, the food-making tissues, and the storage tissues.

The Plant Cell and Tissues.

The cell is the unit of composition, structure, and function of all plants. It is composed of the living substance protoplasm, together with its products and inclusions. One of the products of the jelly-like protoplasm is the rigid cell wall in which it encases itself and which gives shape and rigidity to the cell and to the tissues. Tissues and cell walls often remain intact long after the protoplasm has died. In some diseases, the cementing substance which holds adjoining cells together is dissolved out, and disintegration of the tissue results. In other cases the cell walls are punctured and the protoplasm is killed. Chemical analysis of plant tissues, which are, of course, composed of cells, yields principally water, proteins, carbohydrates, fats, and salts. Water is by far the most abundant single constituent, most plant tissues containing 70-75 per cent water.

The structure of plant tissues is roughly comparable to that of honeycomb except that the cells of plant tissues are not as closely packed as those of honeycomb. There are spaces between plant cells, called intercellular spaces, which are most marked in leafy tissues. These constitute the ventilation system of the plant, and through them the cells are in communication with the air.

The epidermis or outermost protective layer of cells of leaves and young stems and roots has many minute openings or breathing pores, the stomata. Through these, water

vapor from the moist intercellular spaces escapes into the air, and carbon dioxide and oxygen exchange between the cells and the air takes place. The loss of water by the living plant through evaporation is known as transpiration.

When unbroken, the epidermis prevents excessive loss of water from the moist tissues underneath and keeps out foreign organisms such as fungi and bacteria. In older stems and roots, such as the Irish potato, the protective covering is composed of cork layers, in which the breathing pores are present as lenticels.

The supporting tissues, such as the veins of leaves or the fibers of stems, are composed of woody or thickened cells. The conducting tissues, found in the veins of leaves or the ring tissues of stems or roots, translocate the water and minerals taken in by the absorptive tissues of the roots, and the food made in the food-making tissues.

The green color of vegetation is due to a substance known as leafgreen or chlorophyll. By virtue of chlorophyll, and with the aid of sunlight, the green tissues of plants are able to manufacture carbohydrates (sugars and starches) from the carbon dioxide of the air and the water of the soil.

These carbohydrates are the basic substances from which the other principal food of plants and animals, such as fats, oils, and proteins, are manufactured by green and non-green cells.

Many plants possess tissues in which they accumulate excess food materials. These are the storage tissues. The Irish potato and the sweet potato are to a large degree composed of storage cells in which starch and sugar, respectively, are stored.

Digestion and Respiration.

The stored food of the potato is in the form of starch grains. In the spring when the potato sprouts, the insoluble starch is changed to soluble sugar, to which is due the sweet taste of potatoes at that season. In ripening plant tissues, insoluble, unpalatable substances, such as starch and tannins, become soluble, aromatic, and palatable.

Another process which goes on in all protoplasm, plant or animal, is respiration. The making of food out of simple materials is a constructive process, whereas respiration is destructive. In its simplest form, it involves a consumption of oxygen, a burning or oxidation of the substances of the cell, such as sugar, starch, fat, and proteins, and a liberation of carbon dioxide gas, water, and energy. Part of this energy is manifested as heat. The so-called "heating" of crops in transit and storage generally is due to the heat evolved in respiration.

The rate of respiration is greatly influenced by temperature. At high temperatures, respiration proceeds at a rapid rate, and the food substances are soon burned up. At low temperatures, the rate of respiration is so retarded that it

becomes practically negligible. Proper aeration is essential for the continued life and respiration of vegetables. The carbon dioxide liberated must be removed, and the necessary oxygen must be supplied. If respiring cells are not properly aerated, they die, and spoil.

The fundamental reason for the refrigeration of fruits and vegetables is based upon the fact that refrigeration retards respiration and digestion; retards not only the ripening processes of fruits and vegetables, but also their decay, which is due to the activity of bacteria and fungi.

Relation of the Plant to Its Environment.

The continued existence of protoplasm and therefore of plants themselves is dependent upon external factors which constitute the environment of plants. Some of these are non-living, such as moisture, heat, light, and the air; others are living, such as other plants, or animals. The environment of a plant may vary within certain limits without injury to the plant. However, if these limits are exceeded for a sufficient period of time the plant becomes abnormal or diseased and eventually may die.

When plants or plant parts are shipped and stored as fresh food products, they are subjected to conditions very unlike those prevailing in the field, and care must be exercised to make these such that the life of the plant or plant parts is still possible. This means that the external conditions must be controlled and kept within the narrow range tolerated by the plant in question. Each crop must be handled in a manner suited to its peculiar requirements. This will depend upon the climatic conditions prevailing during the growing season and at harvest; upon the methods employed for culture and harvesting; upon the presence of climatic or mechanical injuries; upon the maturity at harvesting; upon the method of packing or loading; upon the type of container; upon the length of the transportation and storage periods; upon the season, and upon the climatic conditions to which the crop will be subjected in transit. Failure to provide proper conditions for plants or plant parts in transit or storage results in the loss, annually, of thousands of cars of fruits and vegetables. It may result merely in an inferior product so far as appearance and taste are concerned, or in partial or complete decay.

The Relation of Plant Functions to Keeping Quality.

It is well known that vegetables and fruits shrink in transit and storage. Some of the shrinkage is due to readjustment in the pack or load. There is in addition to this, however, a real decrease in the weight and volume of the crops as can be determined by weighing and measurement.

Usually there is a very considerable shrinkage immediately following the harvesting of a crop, due mainly to the

loss of water vapor by the plant through its stomata, or transpiration, and by evaporation from cut surfaces. The loss of water by plant organs is illustrated very strikingly by the phenomenon commonly known as "sweating," which is the result of condensation of this water vapor.

The first result of excessive water loss is known as wilting. Wilting is especially frequent in leafy crops with large transpiring surfaces, such as celery, lettuce, or spinach. The compact, heavy crops such as deciduous and citrus fruits, potatoes, and onions are made up of storage tissues and are much less subject to water loss than the more leafy ones. They lose water much more slowly because of their rather thick protective skin and fewer stomata. They also have a greater bulk of water available in their tissues, and a larger volume of tissue relative to their transpiring surface.

Drying of the surfaces of plants, with a slight wilting of leafy plants, before they are shipped or stored is advisable. Wet plant surfaces favor the development of bacteria and fungi and, when stomata are filled with water, easy ingress is provided for bacteria. Many crops are sweated, cured, or kiln-dried before they are put into storage or shipped, to avoid undue shrinkage by loss of water, and to dry and harden the tissues to render them less subject to attack by bacteria or fungi which flourish on tender tissues under moist conditions. Continued loss of water by plant tissues soon results in their death after which they may become the prey of bacteria and fungi.

Not all of the decrease in weight of fruits and vegetables in storage is due to loss of water. A thoroughly cured sweet potato loses weight and shrinks in storage even when stored in a very humid atmosphere, especially if the temperature is high. A part of this loss is due to transpiration, the remainder to respiration. At low temperatures, the processes go on very slowly, but ultimately lead to physiological breakdown. In other words, there is a time limit to the storage of all living plant parts. The period is especially brief in the case of succulent crops. Each crop has its period beyond which it is not advisable to store it. For example, a Jonathan apple cannot be stored as long as a Winesap.

Proper aeration and refrigeration are essential in storage places. The former prevents the accumulation of water vapor and attendant sweating, provides oxygen, and removes carbon dioxide. The latter decreases the rate of digestion and respiration in the plant tissue, thus lowering the rate at which the latter consumes itself. More important, however, is the effect of these factors upon the bacteria and fungi which, because of their power to cause decay, are the chief enemies of stored fruits and vegetables. Bacteria and fungi cannot attack plants when the plant surfaces are dry. Furthermore, at low temperatures their activity is so reduced that they seldom attack plant tissues.

DISEASE IN PLANTS.

Disease in plants is any deviation from the normal condition of their functions or tissues. The majority of plant diseases as they occur in the market are localized in their effects and differ therein from most animal diseases, which usually are systemic, and affect the whole body because of the presence of a nervous and a circulatory system. For example, the outer leaves of a celery plant may be rotted severely while the inner leaves or heart are in no way injured and are fit for food. It must be remembered, however, that the sound part of a fruit and vegetable may in some cases acquire a disagreeable odor or flavor from the diseased part. At times even sound fruits or vegetables are affected in taste and flavor by nearby decaying fruits and vegetables.

Types.

Diseases may occur in the growing plant, checking and preventing its growth or its production of normal parts or even killing it outright. They may also occur in fruits and vegetables in transit and storage. We may therefore distinguish between field and transportation diseases, and shall attempt to differentiate as much as possible between diseases which originate in the field and those which originate in transit and storage.

For example, infection of sweet potatoes with soft rot or peaches with brown rot may occur either in the field, in transit, or in storage, whereas infection of sweet potatoes with scurf or peaches with scab occurs only in the field. The first two diseases, however, may develop or progress in the field, in transit, or in storage, whereas the latter two probably do not develop or spread in transit or in storage.

Symptoms.

The signs or characteristics of a disease which mark it and by which its presence is discerned, are known as the symptoms of the disease. During the progress of a disease, its symptoms may change. Frequently there are early and advanced symptoms. The early symptoms of watermelon anthracnose, for example, are raised welts on the rind, while the advanced symptoms are deeply sunken lesions with a salmon pink covering of spores.

Not all the symptoms of a disease necessary for its diagnosis may appear on one specimen. As a matter of fact, it is often impossible to diagnose a disease by examining a single specimen, and a correct diagnosis often involves an examination of the whole field or the entire lot in the car or in storage. In a shipment of celery, for example, some plants may show a watery rot. This condition alone may be insufficient for a correct and complete diagnosis. Other plants in the same shipment may show a white cottony

growth of mold and no rot, while others may show the mold and black masses of the fungus. The presence of all of these symptoms in a package or car, however, furnish the basis for a correct diagnosis of the rot as watery soft rot due to *Sclerotinia*.

Very frequently the symptoms of several diseases appear on the same specimen or in the same lot. A single potato may show symptoms of black scurf, black heart, and *Fusarium* tuber rot at the same time. Sometimes the symptoms of one disease are followed immediately by those of another, as in black heart of potatoes followed by slimy soft rot.

Diseases Due to Living, Parasitic Agencies.

Most plant diseases are caused by one of two types of minute plant life, commonly known as germs and molds and often referred to collectively as "microorganisms."

Host and Parasite.

The plant attacked and at whose expense the bacteria or fungi feed, and upon and in which they live, is known as the host. Bacteria or fungi living upon and at the expense of a living plant or animal are known as parasites. When these live upon dead plant or animal matter they are classed as saprophytes. Some bacteria and fungi are restricted to a single host; others can attack a variety of hosts. Diseases due to the latter type of organisms are more likely to originate in transit and storage than those due to the former type.

Bacteria and Fungi.

Bacteria are exceedingly small, single-celled, non-green plants which multiply by simple division. Under proper conditions of temperature, moisture, and food supply, bacteria multiply with astounding rapidity.

Some fungi also are composed of single cells, but most of them consist of many cells united end to end to form threads. A single thread is known as a hypha (plural hyphae) and the mass of hyphae of a fungus is known as the mycelium. Generally the mycelium grows within the tissues of the host and is invisible, although under favorable conditions it may also grow out on the surface. At times the mycelium of some fungi form thickened, hardened masses known as sclerotia.

Fungi generally reproduce by the formation and liberation of special cells or groups of cells called spores. In function, these spores are the equivalent of the seed of higher plants, and are distributed as these are, by man, animals, wind, and water. They are, of course, distributed very much more readily and widely because they are so small and light. The spores of some fungi, such as "blue mold" or "bread mold" are found everywhere.

Fungi bear their spores in various ways. In the simpler types the spores are borne on the surface of the attacked plant as a powdery or dusty covering such as one sees in blue mold (*Penicillium*) and gray mold (*Botrytis*). Some, such as *Rhizopus nigricans*, bear their spores in special cases called sporangia, (singular, sporangium). The summer spores of many fungi are known as conidia (singular, conidium).

In other fungi, spores are produced in slimy heaps on little pads or cushionlike bodies known as acervuli (singular, acervulus). These are especially marked in the anthracnoses. Still other fungi bear their spores in special flask-shaped bodies on or at the surface of the diseased portion of the attacked plant. These appear as dots, or pimple-like bodies and are known as pycnidia (singular, pycnidium). They are conspicuous in the late blight spots on celery, the fruit rot of egg plant, the Phoma rot of tomatoes, and the black rot of apples. The special spore-bearing structures of fungi are known as fruiting bodies. The fruiting bodies which result from fertilization and contain the winter spores do not occur generally under market and storage conditions.

There are four classes of fungi, Phycomycetes, Ascomycetes, Fungi Imperfecti, and Basidiomycetes. Most of the fungi with which we will deal belong to the third of these groups.

The Life Processes of Bacteria and Fungi.

The protoplasm of bacteria and fungi lacks chlorophyll and therefore cannot manufacture its own food, but must obtain it from green plants, either directly or indirectly. It obtains its food and energy by digestive and respiratory processes from the tissue attacked. This digestive and respiratory action of bacteria and fungi is popularly known as fermentation. Bacterial rots generally are accompanied by putrefactive odors due to the breaking down of protein compounds.

The attack upon living tissues by parasites leads to a disturbance in the functions, structure, and composition of the attacked plant, in other words, to disease. The diseased areas produced by parasites or other causes are known as lesions. At present only those produced by the action of parasites will be considered. A lesion may be a leaf or pod-spot, a wart, a soft or dry-rot, or a canker, depending upon the parasite, the external conditions, and the nature and response of the affected host tissues. Soft rot of the carrot, for example, consists of lesions caused by *Bacillus carotovorus*. These bacteria secrete ferments or enzymes which digest or dissolve the cementing substances between the carrot cells and disintegrate the tissue into a soft, slimy mass.

In other diseases the lesions arise in a different manner. In anthracnose of the cucumber, for example, the hyphae

of the fungus, *Colletotrichum lagenarium*, penetrate the cell walls and feed upon the protoplasm. As a result, the affected cells die and lose their water, consequently one of the first symptoms of the disease is a water-soaked spot on the fruit. Since the water evaporates rapidly from this spot, it dries out and becomes sunken. As more cells are killed and the lesion enlarges, cavities result from the shrinkage and rupture of the killed tissue. In other diseases, such as late blight of the potato, the fungus grows between the cells and sends suckers into the cells, thereby securing its nourishment, and eventually killing the host protoplasm.

The activity of the bacteria and fungi, like that of other plants, is greatly influenced by temperature. Low temperatures inhibit the germination and growth of these parasites, consequently are useful in controlling many diseases. Low temperatures also retard digestion and respiration in these organisms, consequently decrease their destructive activities. These facts are fundamental reasons for the refrigeration of fruits and vegetables. Control of the life processes of these organisms by low temperatures probably is more important than the effect of refrigeration upon the life activities of the stored fruits or vegetables. Some fungi and bacteria can grow well at moderate temperatures and consequently are very active even in iced cars. *Sclerotinia*, for example, grows well at moderate temperatures and therefore causes a rot of celery in iced cars.

Active bacterial and fungus cells do not possess the effective protective tissues of higher plants, consequently their protoplasm is very subject to drying out. In the desiccated state these organisms are inactive or resting, and cannot resume growth or develop so long as they are dry. In combating diseases caused by parasites, it is fundamentally important to recognize the fact that a moist environment is essential for their growth and spread. Dried or slightly wilted plant tissues, when shipped or stored in a well aerated placè, are not attacked readily by bacteria and fungi. Loading or storing wet fruits or vegetables often leads to disastrous results because of the action of micro-organisms.

Inception of Diseases Due to Bacteria and Fungi.

While bacterial cells and fungus spores are everywhere present in great numbers on the exposed surfaces of all normal uninjured plant parts, the interior tissue is usually absolutely sterile; that is, free from all foreign organisms.

Before disease can occur infection must take place. By infection we mean the entrance of the disease-producing organism into the host tissue and its development therein. Plants are protected against the entrance of foreign organisms by their epidermis, by their cork layers, and by the nature of their protoplasm. Foreign organisms may gain entrance through the unbroken skin, or through natural openings in the epidermis such as stomata and water pores.

However they generally enter plant tissues through wounds or abrasions of the protective layers. This type of infection is the most important in transportational diseases. Injured or dead areas offer an excellent foothold for rot-producing organisms.

Diseases Due to Non-living, Non-parasitic Causes.

A very considerable number of plant diseases are due not to parasitic organisms but to other unfavorable factors in the environment of the plants. Water core and Jonathan spot of apples, black heart, hollow heart, and sunburn of potatoes, tip-burn of lettuce and other crops, and scald and freezing or chilling injury of vegetables and fruits are excellent examples of disease due to non-parasitic causes.

Tip-burn of lettuce and other plants, water core and Jonathan spot of apples, and blossom-end rot of tomatoes and watermelons probably are due to irregularities in the water relations of these crops. The nature of the plants themselves is also a very important factor in such troubles. For example, with the same soil and climatic conditions, the same water supply, and the same treatment of trees of various varieties, the Jonathan and the Grimes Golden alone may show Jonathan spot. Black heart of potatoes is a good example of the evil effects of disturbed respiration in plants.

Under normal conditions, oxygen enters the internal tissues of the potato at a rate sufficiently rapid to maintain normal respiration. However, if the temperature is raised to 100° F., the rate of respiration is so increased that oxygen cannot penetrate to the inner cells rapidly enough to supply their needs. As a result the internal tissues of the tuber become asphyxiated, die, and turn black.

If potatoes are kept at normal temperatures but are not well aerated and do not have a normal supply of oxygen, black heart also results. In this case the death and discoloration of the cells is not restricted to the heart of the potato.

Chilling and freezing injury are good examples of the effects of excessively low temperature upon plant tissues, while sunscald is an example of the effect of extremely high temperature. Either extreme kills the tissues.

Tolerance, Resistance and Predisposition to Disease.

Many plants have to a certain degree an ability to tolerate unfavorable climatic, soil, and cultural conditions, and to withstand or tolerate the attacks of bacteria and fungi. This tolerance varies with the age and variety of the plant and with the conditions under which the plant is grown.

Resistance to parasitic attack may be due to the nature and thickness of the protective covering of the plant, to a scarcity of natural openings, to the rapidity with which wounds heal over (with wound cork), to the time of ma-

turity of the plant, to the acidity of its tissues, or more commonly to some unknown factor in the composition of the protoplasm of the cells themselves.

Disease Control.

By disease control is meant the elimination, the checking, or the prevention of a disease. Plant diseases usually are controlled by prevention rather than cure. A very generally used method in the control of diseases due to parasites is the spraying, dusting, or dipping of plants or plant parts to kill the fungi and bacteria present on the surfaces, or to prevent the germination of spores that may lodge thereon subsequently. There are a number of other practical methods of disease control, such as proper cultural methods, crop rotation, soil and seed sanitation, planting of disease-free stock or disease-resistant varieties, and the variation of the planting date.

In the control of transportational diseases, icing, refrigeration, ventilation, precaution not to pack wet plants or plant organs, drying of plants, and careful handling are important means of disease control.

LITERATURE CITED.

In addition to these special references, the following books will be found very helpful and should be freely consulted:

Freeman, E. M. Minnesota plant diseases. 1905.

Duggar, B. M. Fungous diseases of plants. 1909.

Stevens, F. L., and Hall, J. G. Diseases of economic plants. 1910.

Hesler, L. R., and Whetzel, H. H. Manual of fruit diseases. 1917.

Coons, G. H., and Nelson, R. The plant diseases of importance in the transportation of fruits and vegetables. Am. Ry. Perishable Freight Assn. Cir. 473-A. 1918.

- (1) Bartholomew, E. T.
1913. Black heart of potatoes. In *Phytopathology*, v. 3, No. 3, p. 180-182, pl. 19.
- (1a) Bessey, E. A., and Byars, L. P.
1915. The control of root-knot. U. S. Dept. Agr. Farmers' Bul. 648, 19 p.
- (2) Brandes, E. W.
1918. Anthracnose of lettuce caused by *Marssonina panattoniana*. In *Jour. Agr. Research*, v. 13, No. 5, p. 261-280, 4 fig., pl. C, 20. Literature cited, p. 280.
- (3) Brooks, Charles.
1914. Blossom-end rot of tomatoes. In *Phytopathology*, v. 4, No. 5, p. 345-374, 4 fig., pl. 24-26. References, p. 372-373.
- (4) Brown, Nellie A.
1915. A bacterial disease of lettuce.* A preliminary report. In *Jour. Agr. Research*, v. 4, No. 5, p. 475-478. Literature cited, p. 478.
- (5) Burger, O. F.
1913. Lettuce drop. Fla. Agr. Exp. Sta. Bul. 116, p. 27-32, illus.
- (6) ———
1914. Cucumber rot. Fla. Agr. Exp. Sta. Bul. 121, p. 97-109, fig. 37-42.
- (7) Carpenter, C. W.
1915. Some potato tuber-rots caused by species of *Fusarium*. *Jour. Agr. Research*, v. 5, No. 5, p. 183-210, pl. A-B, 14-19.
- (8) Carsner, Eubanks.
1918. Angular-leafspot of cucumber: dissemination, overwintering and control. In *Jour. Agr. Research*, v. 15, No. 3, p. 201-220, pl. 13-16. Literature cited, p. 220.
- (9) Chittenden, F. H., and Orton, W. A.
1918. How to increase the potato crop by spraying. U. S. Dept. Agr. Farmers' Bul. 868, p. 13-16.
- (10) Cook, M. T., and Lint, H. C.
1915. Potato diseases in New Jersey. N. J. Agr. Exp. Sta. Circ. 53, 23 p., 9 fig. Substitute for Circ. 33, now out of print.
- (11) Coons, G. H., and Levin, Ezra.
1916. The *Septoria* leaf spot disease of celery, or celery blight. Mich. Agr. Exp. Sta. Spec. Bul. 77, 8 p., 9 fig.

*Papers marked with a star are exhausted so far as the Department supply is concerned. Copies may be purchased from the Superintendent of Documents, Government Printing Office, Washington, D. C.

- (12) Doolittle, S. P.
1916. Cucumber scab caused by *Cladosporium cucumerinum*. In 17th Rpt. Mich. Agr. Acad. Sci. [1915]: 87-116. Literature, p. 116.
- (13) ———
1916. A new infectious mosaic disease of cucumber. In *Phytopathology*, v. 6, No. 2, p. 145-147.
- (14) Edgerton, C. W.
1910. The bean anthracnose. *La. Agr. Exp. Sta. Bul.* 119, 55 p., 14 pl. Bibliography, p. 51-54.
- (15) ——— and Moreland, C. C.
1913. The bean blight and preservation and treatment of bean seed. *La. Agr. Exp. Sta. Bul.* 139, 43 p., 6 pl.
- (16) Edson, H. A.
1915. *Rheosporangium aphanidermatum*, a new genus and species of fungus parasitic on sugar beets and radishes. In *Jour. Agr. Research*, v. 4, No. 4, p. 279-292, pl. 44-48.
- (17) ———
1915. Seedling diseases of sugar beets and their relation to root-rot and crown-rot. In *Jour. Agr. Research*, v. 4, No. 2, p. 135-168, pl. 16-26. Literature cited, p. 165-168.
- (18) Erwin, A. T.
1916. Late potato blight in Iowa. *Iowa Agr. Exp. Sta. Bul.* 163, p. 287-305, 8 fig.
- (19) Galloway, B. T.
1895. The growth of lettuce as affected by the physical properties of the soil. In *Agr. Sci.*, v. 8, p. 302-316.
- (20) ———
1895. Commercial lettuce forcing. In *Amer. Gard.*, v. 16, p. 135.
- (21) Gardner, M. W.
1918. Anthracnose of cucurbits. *U. S. Dept. Agr. Bul.* 727, 68 p.
- (22) Gilbert, W. W.
1916. Cucumber mosaic disease. In *Phytopathology*, v. 6, No. 2, p. 143-144, pl. 5.
- (23) Harter, L. L.
1914. Fruit-rot, leaf-spot, and stem-blight of the eggplant caused by *Phomopsis vexans*. In *Jour. Agr. Research*, v. 2, No. 55, p. 331-338, 1 fig., pl. 26-30.
- (24) ———
1916. Sweet potato diseases. *U. S. Dept. Agr. Farmers' Bul.* 714, 26 p., 21 fig.
- (25) ———
1917. Podblight of the lima bean caused by *Diaporthe phaseolorum*. In *Jour. Agr. Research*, v. 11, No. 10, p. 474-504, 11 fig., pl. 42-43. Literature cited, p. 502-504.
- (26) ——— and Jones, L. R.
1918. Cabbage diseases. *U. S. Dept. Agr. Farmers' Bul.* 925, 30 p., 13 fig.
- (27) ———, Weimer, J. L., and Adams, J. M. R.
1918. Sweet potato storage rots. In *Jour. Agr. Research*, v. 15, No. 6, p. 337-368.
- (28) Hawkins, L. A.
1916. The disease of potatoes known as "leak."* In *Jour. Agr. Research*, v. 6, No. 17, p. 627-640, 1 fig., pl. 90.
- (29) ———
1917. Experiments in the control of potato leak. *U. S. Dept. Agr. Bul.* 577, 5 p.

- (30) Humbert, J. G.
1918. Tomato diseases in Ohio. Ohio Agr. Exp. Sta. Bul. 321, p. 157-196, illus.
- (31) Jagger, I. C.
1916. Experiments with the cucumber mosaic disease. In Phytopathology, v. 6, No. 2, p. 148-151.
- (32) Jamieson, Clara O.
1915. Phoma destructiva, the cause of a fruit rot of the tomato.* In Jour. Agr. Research, v. 4, No. 1, p. 1-20, pl. 1-6 (2 col.). Literature cited, p. 19-20.
- (33) Jones, L. R.
1901. A soft rot of carrot and other vegetables. In Vt. Agr. Exp. Sta. 13th Ann. Rpt. 1899-1900, p. 299-332, illus.
- (33a) Jones, L. R., and Bailey, Ernest.
1917. Frost necrosis of potato tubers. In Phytopathology, v. 7, No. 1, p. 71-72.
- (34) ———, Giddings, N. J., and Lutman, B. F.
1912. Investigations of the potato fungus *Phytophthora infestans*.† U. S. Dept. Agr. Bur. Plant Indus. Bul. 245, 100 p., illus., 10 pl. (2 col.), map.
- (35) Kunkel, L. O.
1919. Wart of potatoes; a disease new to the United States. U. S. Dept. Agr. Bur. Plant Indus. Office Cotton, Truck, and Forage Crop Diseases, Circ. 6.
- (36) Levin, Ezra.
1917. Control of lettuce rot. In Phytopathology, v. 7, No. 5, p. 392-393.
- (37) Link, G. K. K.
1916. A physiological study of two strains of *Fusarium* in their causal relation to tuber rot and wilt of potato. Nebr. Agr. Exp. Sta. Research Bul. 9, 45 p., illus.
- (38) Lutman, B. F.
1913. The pathological anatomy of potato scab. In Phytopathology, v. 3, No. 5, p. 255-264, illus. Bibliography, p. 264.
- (39) ——— and Cunningham, G. C.
1914. Potato scab. Vt. Agr. Exp. Sta. Bul. 184, 64 p., 6 fig., 12 pl. Bibliography, p. 62-64.
- (40) McKay, M. B., and Pool, Venus W.
1918. Field studies of *Cercospora beticola*. In Phytopathology, v. 8, No. 3, p. 119-136, illus.
- (41) MacMillan, H. G.
1918. Sun-scald of beans. In Jour. Agr. Research, v. 13, No. 12, p. 647-650, pl. 64-66.
- (42) Meier, F. C.
1916. Watermelon stem-end rot. [Preliminary report.] In Jour. Agr. Research, v. 6, No. 4, p. 149-152, pl. 17.
- (43) Melhus, I. E.
1913. Silver scurf, a disease of the potato.† In U. S. Dept. Agr. Bur. Plant Indus. Circ. 127, p. 15-24, 4 fig. Bibliography, p. 24.
- (44) ———
1914. Powdery scab (*Spongospora subterranea*) of potatoes.† U. S. Dept. Agr. Bul. 82, 16 p., 3 pl. Bibliography, p. 15-16.
- (45) ———
1915. Hibernation of *Phytophthora infestans* in the Irish potato.* In Jour. Agr. Research, v. 5, No. 2, p. 71-102, 3 fig., pl. 4-8. Literature cited, p. 100-102.

†Papers marked with a dagger are out of print.

- (46) Morse, W. J.
1914. Powdery scab of potatoes. Maine Agr. Exp. Sta. Bul. 227, p. 87-104, fig. 44-52.
- (47) Muncie, J. H.
1914. Two Michigan bean diseases. Mich. Agr. Exp. Sta. Spec. Bul. 68, 12 p., 2 fig., 1 pl. (col.).
- (48) Munn, M. T.
1917. Neck-rot disease of onions. N. Y. State Agr. Exp. Sta. Bul. 437, p. 361-455, 11 pl. Bibliography, p. 450-455.
- (49) Orton, C. R.
1916. The diseases of the potato. Penn. Agr. Exp. Sta. Bul. 140, 37 p., 23 fig.
- (50) Orton, W. A.
1913. Potato tuber diseases. U. S. Dept. Agr. Farmers' Bul. 544, 16 p., illus.
- (51) ————
1917. Watermelon diseases. U. S. Dept. Agr. Farmers' Bul. 821, 18 p., 11 fig.
- (52) Osmun, A. V., and Anderson, P. T.
1915. Ring-spot of cauliflower. In *Phytopathology*, v. 5, No. 5, p. 260-265, 4 fig. Literature cited, p. 265.
- (53) Pratt, O. A.
1916. Control of the powdery dryrot of western potatoes caused by *Fusarium trichothecioides*.* In *Jour. Agr. Research*, v. 6, No. 21, p. 817-831, pl. 108.
- (54) ————
1916. A western field-rot of the Irish potato tuber caused by *Fusarium radicolica*. In *Jour. Agr. Research*, v. 6, No. 9, p. 297-310, pl. 34-37.
- (55) Rogers, S. S.
1911. The late blight of celery. Calif. Agr. Exp. Sta. Bul. 208, p. 83-115, illus., 1 pl.
- (56) Rosenbaum, Joseph
1918. The origin and spread of tomato fruit rots in transit. In *Phytopathology*, v. 8, No. 11, p. 572-580, illus., pl. 4.
- (57) Schultz, E. S.
1916. Silver-scurf of the Irish potato caused by *Spondylocium atrovirens*.* In *Jour. Agr. Research*, v. 6, No. 10, p. 339-350, pl. 45-48. Literature cited, p. 350.
- (58) Sheldon, J. L.
1904. Diseases of melons and cucumbers during 1903 and 1904. W. Va. Agr. Exp. Sta. Bul. 94, p. 119-138, pl. 1-5.
- (59) Sherbakoff, C. D.
1917. Buckeye rot of tomato fruit. In *Phytopathology*, v. 7, No. 2, p. 119-129, 5 fig.
- (60) ————
1917. Some important diseases of truck crops in Florida. Fla. Agr. Exp. Sta. Bul. 139, p. 191-277, fig. 76-112.
- (61) Smith, Erwin F.
1911-14. Bacteria in Relation to Plant Diseases, v. 2, 1911; v. 3, 1914. Washington, D. C. (Carnegie Inst. Washington Pub. 27, v. 2-3.)
- (62) ————, and Bryan, Mary K.
1915. Angular leaf-spot of cucumbers.* In *Jour. Agr. Research*, v. 5, No. 11, p. 465-476, pl. 43-49.
- (63) Spaulding, Perley, and Field, Ethel C.
1912. Two dangerous imported plant diseases.† U. S. Dept. Agr. Farmers' Bul. 489, 29 p., 3 fig.
- (64) Stakman, E. C., and Tolaas, A. G.
1912. Potato diseases. Minn. Agr. Exp. Sta. Ext. Bul. 35, 15 p., 7 fig.

- (65) Stevens, F. L.
1911. A serious lettuce disease (lettuce sclerotinose). N. C. Agr. Exp. Sta. Bul. 217, p. 7-21, illus.
- (66) ———, and Hall, J. G.
1911. A serious lettuce disease (sclerotinose) and a method of control. N. C. Agr. Exp. Sta. Tech. Bul. 8, p. 89-143. Bibliography, p. 144.
- (67) Stewart, F. C.
1899. Notes on various plant diseases. I. A bacterial rot of onions. In N. Y. State Agr. Exp. Sta. Bul. 164, p. 209-212, pl. 1-2.
- (68) ———, and Mix, A. J.
1917. Blackheart and the aeration of potatoes in storage. N. Y. Agr. Exp. Sta. Bul. 436, p. 319-362, 10 pl.
- (69) Taubenhaus, J. J.
1918. Pox, or pit (soil rot), of the sweet potato. In Jour. Agr. Research, v. 13, No. 9, p. 437-450, pl. 51-52. Literature cited, p. 449-450.
- (70) ———, and Manns, T. F.
1915. The diseases of the sweet potato and their control. Del. Agr. Exp. Sta. Bul. 109, 55 p., 65 fig. Literature on potato diseases, p. 48-51.
- (71) Thaxter, Roland.
1890. The "smut" of onions. Conn. Agr. Exp. Sta. Ann. Rpt. 1889, p. 129-154, pl. 1-3.
- (72) ———
1890. The onion *Vermicularia* (*Vermicularia circinans*). Conn. Agr. Exp. Sta. Ann. Rpt. 1889, p. 163-165.
- (72a) Tisdale, W. H.
1916. A *Melanconium* parasitic on the tomato. In Phytopathology, v. 6, p. 390-394.
- (73) Van Hook, J. M.
1906. Blighting of field and garden peas, chiefly due to seed infection. Ohio Agr. Exp. Sta. Bul. 173, p. 229-249, 12 fig.
- (74) Van Pelt, Wayne.
1917. Black mold of onions. Ohio Agr. Exp. Sta. Mo. Bul., v. 2, No. 5, p. 152-156, illus.
- (75) Whetzel, H. H.
1906. Some diseases of beans. N. Y. Cornell Agr. Exp. Sta. Bul. 239, p. 195-214, illus.
- (76) ———
1908. Bean anthracnose. N. Y. Cornell Agr. Exp. Sta. Bul. 255, p. 429-447, fig. 217-222.

GRAY MOLD ROT

OF ARTICHOKE, BEET, CABBAGE, CARROT, CAULIFLOWER, CITRON, LETTUCE, ONION, PARSNIP, RHUBARB, RUTABAGA, SALSIFY, SHALLOTS, TURNIP, AND OTHER VEGETABLES.

Cause: A fungus (*Botrytis*).

This is a semi-watery to watery soft rot characterized externally by a gray velvety fungous growth. Frequently there is a slight tinge of green or brown in the gray color of this mold. At times gray to black solid masses known as sclerotia are produced on the affected tissues as in the case of the gray mold rot of onions generally known as "neck rot."

Gray mold rot can be differentiated from the watery soft rot caused by *Sclerotinia* by the gray color of the mold and by the absence of a cottony mycelium.

Gray mold rot can be differentiated from slimy soft rot by the gray fungous growth and by the absence of offensive odors. *Rhizopus* rot can be differentiated from gray mold rot by its abundant stringy and coarse mycelium, its sporangia, and its sour odor.

In case of doubt as to the identity of a given rot, it is advisable to collect and incubate some material in a warm damp-chamber secured either by wrapping it in paper or by placing it in covered pans. If a mycelium develops within 24 hours, the rot is not slimy soft rot. If a white cottony mycelium develops which later becomes matted, the fungus is probably *Sclerotinia*. A grayish, powdery fungous growth indicates *Botrytis*, and a stringy mycelium with sporangia, *Rhizopus*. The presence of sclerotia will further indicate either *Sclerotinia* or *Botrytis*.

Often the watery soft rot due to *Sclerotinia* and the gray mold rot due to *Botrytis* occur either together or in close succession on the same specimen.

Gray mold rot does not progress as rapidly as watery soft rot and, like slimy soft rot, may be checked by drying affected tissues, while watery soft rot (due to *Sclerotinia*) progresses rapidly even under dry atmospheric conditions.

Infection takes place in the field, in transit, and in storage since the spores of the fungus seem to be everywhere present. The fungus may develop in the field if moist weather prevails.

The rot occurs commonly in mature tissues kept in a very humid atmosphere. It attacks frozen or bruised tissues very readily.

Affected stock may be made marketable if trimmed and kept in a dry, well-ventilated place. It is not safe to store affected stock because it is a menace to healthy stock, the fungus spreading readily from affected to healthy tissues.

Little is known about the control of this rot, but soil aeration and proper ventilation in transit and storage are preventive measures. The neck rot type of this rot found

on onion bulbs can be checked by rapid curing of the onions as soon as they are dug.

Ref. (48).*

RHIZOPUS ROT.

OF BEAN, BEET, CABBAGE, CARROT, ONION, SQUASH, SWEET POTATO, TOMATO, AND OTHER VEGETABLES.

Cause: A fungus (*Rhizopus*).

In its early stages, *Rhizopus* rot consists of soft, water-soaked areas. These enlarge rapidly and often lead to a leaky condition of affected stock. Under proper moisture conditions, there is produced a coarse, white, stringy mycelium bearing white, glistening heads or sporangia, which later turn brown or black. This mycelium is often called "whiskers" because of the long, loose growth of hyphae.

Generally the rot is accompanied by a sour, acid odor, noticeable when the diseased tissue is freshly broken. In carrots, sweet potatoes, and other crops which are rich in starches and sugars, acetic acid is produced, giving rise to a vinegar-like odor. In cabbage lactic acid is produced and an odor of sauer kraut is the result.

This rot is distinguishable from watery soft rot by its sour odor and by the presence of the fluffy, stringy mycelium with its sporangia. The mycelium of *Sclerotinia* is white and cottony or matted. In advanced stages, the *Rhizopus* mycelium collapses, becomes matted, and appears gray or brown, but lacks the black sclerotia of *Sclerotinia*.

The sporangia, the color of the mycelium, and the sour odor of the rot distinguish it from gray mold rot, and the presence of the mycelium distinguishes it from slimy soft rot.

Rhizopus rot is favored by high temperatures. In this respect it differs from watery soft rot or gray mold rot, both of which develop best at moderate temperatures. It is common in overheated, humid cars, especially in the summer, or in refrigerated cars in which the ice was allowed to run low.

Usually the fungus gains entry through wounds. The original infections may take place in the field, in transit, or in storage since the spores of the fungus seem to be everywhere present. The rot develops and spreads very rapidly. Like *Sclerotinia*, *Rhizopus* spreads from affected to healthy stock by contact.

Sorting out of bruised stock, drying of moist surfaces, and proper ventilation and refrigeration will act as effective control measures in transit and storage.

SLIMY SOFT ROT.

OF BEAN, BEET, CABBAGE, CARROT, CELERY, LETTUCE, MUSTARD, ONION, POTATO, RADISH, RUTABAGA, SHALLOTS, SPINACH, TURNIP, AND OTHER VEGETABLES.

Cause: Bacteria (*Bacillus carotovorus* group and other bacteria).

*Reference is made by number to "Literature cited," pp. 13-17.

This disease is a soft rot characterized by a slimy, slippery condition of the affected tissues, by a foul odor, and by the absence of fungous growth. The last characteristic differentiates it from rots caused by fungi. Green, leafy tissues affected with slimy soft rot have a very dark green color at first much like hashed spinach but later become brown and black. Light colored tissues, such as cabbage and celery, become yellowish and finally brown.

Slimy soft rot is differentiated from gray mold rot by the absence of the powdery, gray growth of mold and of the gray to black sclerotia characteristic of gray mold rot. The offensive odor of slimy soft rot distinguishes it from the rot due to *Rhizopus*, which has an acid odor. Tissues affected with slimy soft rot do not leak as do tissues affected with watery soft rot, but become a mass of slime if kept in a moist place. Watery soft rot progresses even in a dry atmosphere after once well started, while the progress of slimy soft rot is arrested by drying of the tissues.

The rot proceeds from any part of the plant, and tissues are predisposed to it by bruising, chilling, freezing, tip-burn, sun-scald or aging. In the case of the southern bunch crops, only the leaves are affected. It is prevalent in root crops whenever tissues have been killed by exposure to extremely wet conditions, by extremely high temperatures, or by freezing. During rainy seasons, slimy soft rot may ruin southern potatoes if they are not dug at once and dried. Future investigations may show that the slimy soft rot of the leafy parts, and the soft mushy rot of the root or stem parts of plants, well marked in carrots and cabbage, are not caused by the same organism. The rot of the latter sometimes is called the "true soft rot." In carrots that have been in storage, soft rot is often found, the central tissue of the root being most readily attacked. In the north, soft rot is common in cabbage in the field late in the fall, especially where many plants have been weakened or killed by the soil disease known as cabbage yellows. Such rotted heads in the field are a source of danger to sound stock since infection may be carried on the knives used in cutting off the heads. This may account for the common occurrence of the so-called "stump rot" in cabbage in storage or transit.

Slimy soft rot occurs commonly in southern winter-grown bunch crops. It results from infection in the field, in transit or in storage, and develops and spreads under any of these conditions. It is favored by the moist atmosphere prevailing in iced barrels, crates, and hampers in which these crops are shipped, and in the cellars and pits in which northern stock, especially cabbage and celery and the root crops, are stored. High temperatures are of even greater importance, however, and greatly accelerate the progress of this rot, especially in potatoes.

Affected stock generally is not marketable, although affected tissues can be trimmed away, leaving the sound remainder salable.

The rot can be controlled by guarding against sun-scald, freezing injury, and excessive bruising, by drying the surfaces of vegetables, especially those cut and bruised, and by maintaining dry and cool conditions in transit and storage.

Ref. Carrot and other vegetables (33); cabbage (26); lettuce (4), (36); onion (67).

WATERY SOFT ROT.

OF ASPARAGUS, BEAN, BEET, CABBAGE, CARROT, CAULIFLOWER, CELERY, LETTUCE, KOHL-RABI, PEPPER, SALSIFY, SHALLOTS, SQUASH, TURNIP, AND RUTABAGA.

Cause: A fungus (*Sclerotinia*).

This disease is a watery soft rot of the affected tissue characterized by a white, cottony, at times matted, mycelium; by the presence of hard masses or knots (*sclerotia*) which are white at first and then become purplish black; and by the absence of any offensive odor.

The watery disintegration associated with watery soft rot is often so complete that water runs freely from crates, hampers, and even cars containing affected stock. Due to this loss of water, affected stock may shrink to but a small portion of its original volume.

Affected tissue may seem only slightly discolored and rotted, but upon application of pressure it is noted that the tissue is completely softened and that water escapes with the greatest ease. The slimy feeling noted in connection with the slimy soft rot of succulent tissues is entirely absent.

The affected plants, the containers, and even the car may be overrun by a heavy growth of loose, cottony or matted mycelium, which under proper conditions forms the *sclerotia* previously mentioned. The fungus often is responsible for the so-called "nesting" of beans.

Affected celery tissue often has a pinkish or rose color, especially in the early stages, and consequently the rot is sometimes known as "pink rot" of celery. At times purplish tints are noticeable. Generally, however, the diseased tissue is yellowish or brown.

In typical field attacks upon lettuce, which are known as lettuce "drop," or upon cabbage, the disease begins on the stalk near the ground, or on the leaves touching the ground, progressing from these to the stalk, which becomes softened and at times entirely destroyed. The result in either case is a collapse of the entire plant. In the field the disease is known as "foot rot" of celery because of the frequent initial attacks at the base of the plant. However, under storage and transit conditions, plants may be attacked at any point. Well headed cabbage or lettuce often remains intact because of the overlapping leaves. Often one finds, however, when an attempt is made to lift an affected head, that it

falls to pieces quite unlike plants affected with slimy soft rot which tend to slip out of one's hand.

On root crops the rot is not as watery as on leafy tissues. Under dry conditions, evaporation keeps pace with the liberation of water by the fungus and the affected tissue dries out and shrinks. Thus the watery nature of this rot in roots is not as evident as in the case of leafy or succulent tissues. However, under moist conditions, affected root crops exude water freely. The white cottony or matted mycelium and the black sclerotia serve to identify the disease on root crops when the watery characteristics are absent.

This rot can be differentiated from slimy soft rot by the fact that the latter is a slimy, slippery decay usually accompanied by a bad odor and lacks the white mycelium and large sclerotia of watery soft rot. The latter can be differentiated from gray mold rot by the typical powdery gray moldy outgrowth of the latter. Gray mold rot is generally found on overmature tissues, and is not as watery as the typical watery soft rot caused by *Sclerotinia*. Watery soft rot progresses at lower temperatures than does the soft rot induced by *Rhizopus*. *Rhizopus* rot is prevalent in the summer and in overheated cars in the winter, while watery soft rot develops in refrigerated cars.

✓ The disease is favored by high humidity and moderate temperatures. The original infection proceeds from the soil. Lettuce grown under glass, or plants grown in crowded quarters, are especially subject to infection. The disease develops and spreads very rapidly in transit and storage, the fungus passing from diseased to healthy tissue by contact.

Crop rotation and soil sanitation and aeration are control measures which can be applied in the field. Losses in transit and storage may be reduced by sorting out and discarding diseased plants and by thorough ventilation. After the diseased portions of affected plants are trimmed off, the remainder is edible. It is not advisable to store trimmed plants.

Ref.: Lettuce (5); (65); (66).

SUN-SCALD.

OF CUCUMBER, HONEY DEW MELON, ONION, PEPPER, POTATO, TOMATO,
AND WATERMELON.

Cause: Exposure to the hot sun.

Sun-scald is evidenced by the death and discoloration of a rather extensive area on the exposed surface of the vegetable. In most cases, the lesion is irregular in outline and at first resembles a water-soaked blister, but soon becomes slightly but sharply sunken and distinctly bleached in color. This bleaching is particularly noticeable in peppers, tomatoes, and cucumbers.

Sun-scald is particularly important from the market standpoint because the scalded areas are very subject to the

attacks of rot-producing bacteria and fungi. On onions, scald may be followed by slimy soft rot, and large losses were thus incurred in certain shipments from Stockton, Cal., in 1918. Tomato scald opens the way for fungous rots and was an important factor in the losses in Texas and Tennessee shipments in 1918. Sun-scalded muskmelons or watermelons are frequently invaded by saprophytic fungi such as black mold (*Sterigmatocystis*) and *Cladosporium* or *Alternaria*. Generally these are surface growths more or less confined to the dead tissue.

Potato scald is discussed elsewhere, as is also sun-scald of bean.

Because of its predisposition to rot during transit, scalded stock could profitably be culled out before shipment.

NEMATODE DISEASE.

OF BEET, CARROT, CELERY, PARSNIP, POTATO, SWEET POTATO, RADISH, RUTABAGA, AND TURNIP

Cause: A nematode or eelworm (*Heterodera radiculicola*).

This disease affects the underground parts of plants. It may be recognized on tubers by small, pimple-like swellings or by larger protuberances of the surface, which ordinarily becomes roughened at the infected places. These swellings when some distance apart are circular, but if occurring close together they take on various shapes and sizes. On roots the disease appears as definite galls or knots and consequently is commonly called root-knot.

Eelworm-infected tubers and roots sometimes are confused with those affected by other diseases (crown-gall of beets, club-root of crucifers, and "pimplly potatoes" due to flea-beetle injury), which also cause a swelling of the diseased tissues. The nematode disease, however, may usually be readily distinguished from other maladies by the presence of small, white, pear-shaped nematodes, the adult females, which may barely be seen with the unaided eye when well-infested tissues are broken or teased apart.

This disease occurs widely in most of the older trucking sections of the southern portions of the United States and in greenhouses everywhere.

Plants become infected in the field. The disease does not develop or spread in transit or storage. Diseased stock, however, is very subject to invasion by secondary organisms, particularly bacteria.

Affected stock may largely be eliminated by grading. It never should be shipped, not only because it is unsightly in appearance, of inferior quality, and impaired in market value, but also because it may carry the disease into uninfested regions.

Ref. (1a).

ARTICHOKE: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

ASPARAGUS: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

ASPARAGUS: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

BEAN: ANTHRACNOSE.

Cause: A fungus (*Colletotrichum lindemuthianum*).

Anthracnose is marked in the very early stages by minute oval or circular spots which are maroon to reddish in color. These spots are not water-soaked as in the bacterial blight. They form ulcer-like lesions, which increase rapidly in size and, though circular, often coalesce to form large irregular spots. Very soon, often in 24 hours, the spot becomes darker in color, the central tissue dries up and shrinks, and a depressed, considerably sunken spot results, with a black center and generally a reddish border. Under moist conditions the dark center of the spot becomes covered with orange-pink dots or spore heaps which may run together and form a slimy layer. These spore heaps are borne on cushions known as acervuli (singular, acervulus). The border of the lesion is sharply marked, especially on the wax pod varieties.

From pod lesions the fungus often enters the seeds. In case of light attack the seed shows only a yellowish spot. In severe cases the spots are yellowish, brownish, or black, and circular or irregular in shape, and are surrounded by a reddish zone. They may be sunken, but rarely show the pinkish slime noted on the pod spots. The spots on the seed are darker colored and more definitely marked than those of bacterial blight.

Anthracnose occurs in all varieties of wax pod, green pod, pole, navy, kidney, lima, and some scarlet runner beans. Its development is favored by moderately cool weather, and its spread, by wet weather. Since it is carried in the seed, the disease may occur in any bean-growing section, but the regions subject to cool, wet weather are most seriously affected. It is least prevalent in the Rocky Mountain and Pacific Coast States.

The original infection takes place in the field where both the vines and pods are affected. The disease develops on the pods in transit, and may spread under very moist conditions.

Crop rotation and the use of disease-free seed or disease-escaping varieties are the only known effective control measures. Seed grown in hot, dry regions is comparatively free from anthracnose.

Ref. (76); (14); (47).

BEAN: BACTERIAL BLIGHT.

Cause: Bacteria (*Pseudomonas phaseoli*).

In the early stages bacterial blight is marked by small,

water-soaked spots on the pods. These gradually enlarge and become irregular in shape, the green color fading as the affected tissue dries out. The lesions may have an elevated margin. Very soon the spots become reddish brown, first at the margins and then in the centers. Finally they turn a darker brown. In this stage the spot dries out and becomes sunken. Under moist conditions, a slime oozes out from the surface of the water-soaked spots. When dry, this exudate forms a translucent or yellow crust. The border between healthy and diseased tissue is not as sharply marked as in bean anthracnose, and is more irregular. The presence of the exudate and the irregular shape of the lesion usually serve to identify blight.

The bacteria from the pod lesions may penetrate and infect the seeds. Such seeds are marked by yellow spots or blotches of irregular shape or by surface crusts of a yellowish color. These spots may show dark red borders and at times a brown or black color. Seeds often are completely yellowed and shriveled.

Bacterial blight occurs in all bean districts east of the Rocky Mountains. Its spread is favored by moist warm weather, though it can develop in dry weather. It causes severe blighting of the foliage, and may kill plants outright. Pod infection takes place in the field, but the spots may develop or enlarge in transit, and may predispose the stock to slimy soft rot or watery soft rot.

Blight is a seed-borne infection, and may be controlled only by the use of disease-free seed and the practice of crop rotation.

Ref. (15); (75).

BEAN: SOIL ROT.

Cause: A fungus (*Rhizoctonia*).

Soil rot is characterized by large lesions generally near the end of the pod or at points where the pod has been in contact with the ground. The lesions are large and irregular in shape and light brown in color with soft, badly-rotted underlying tissues. At times the spots are concentrically marked. Anthracnose lesions are smaller and more regular in shape.

Soil rot occurs only in very moist seasons. The original infection takes place in the field. The fungus spreads from diseased to healthy pods in transit under moist conditions and often causes severe "nesting." The coarse, brown hyphae of the fungus and the sclerotia, if present, distinguish soil rot from the watery soft rot induced by *Sclerotinia* and from the rot caused by *Rhizopus*, both of which also cause "nesting."

Soil rot is controllable by careful sorting of the stock and by maintaining a low temperature and humidity during transit.

BEAN: RUSSET.

Cause: Unknown. Non-parasitic.

Russet is a rather prevalent chestnut-brown surface discoloration of green-pod and occasionally wax-pod beans. The discolored areas are of various shapes and sizes, sometimes involving nearly all of the pod. The affected tissue is firm and sound, and is not at all sunken. The discoloration is due to the death of the three or four outer layers of cells.

This disease seems to appear in beans that have been in transit or storage for some time, and is probably not of field origin. Russet is common in Florida beans in the northern markets. It is objectionable because of the injury to the appearance of affected stock.

No control is known.

BEAN: SUN-SCALD.

Cause: Exposure to the hot sun.

This injury first shows up on the exposed side of the pod as minute brown or reddish parallel streaks, which enlarge and merge to form brown or reddish areas of varying size. Sun-scald is not easily distinguished from bacterial spot, but is more likely to be limited to one side of the pod, and lacks the greasy exudate which often is present on blight lesions.

Sun-scald is due to exposure to the sun, and does not occur where the pods are shaded.

Ref. (41).

LIMA BEAN: POD BLIGHT.

Cause: A fungus (*Diaporthe phaseolorum*).

In the early stages, this disease is characterized by circular to semi-circular spots of darkened tissue. Later the affected tissue becomes studded with minute, gray elevations. These elevations soon break the skin of the pod, and emerge as black pustules, the fruiting bodies, or pycnidia, of the fungus. The pycnidia may be arranged concentrically or in chain-like fashion. In advanced stages, the entire pod may become diseased and covered with pycnidia.

Pod blight has been reported only in the Northern Atlantic States, and is not common. Infection takes place in the field, and the disease may develop and progress in transit.

Pod blight can be controlled by seed selection and disinfection and by spraying in the field. It is advisable to sort out and not market diseased pods.

Ref. (25).

BEAN: RHIZOPUS ROT.

(See Rhizopus Rot).

BEAN: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

BEAN: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

BEET: BLACK ROT.

Cause: A fungus (*Phoma betae*).

In this rot, the affected tissue is coal black, rather firm and extends deeply into the root. There is no bad odor. The surface becomes somewhat shrunken, and often bears the black pycnidia of the causal fungus.

This disease causes a leaf spot and a blight of the plants in the field. The fungus is carried with the seed.

Control consists of seed and seed-bed sanitation and removal of diseased leaves from roots previous to storage.

Ref. (17).

BEET: LEAF SPOT.

Cause: A fungus (*Cercospora beticola*).

This disease occurs only on the leaves where its lesions appear as distinctly circular spots with purple borders and tan to ashen gray centers. Leaves may be killed by the coalescence of numerous lesions. The death of the older leaves may cause the crown to elongate, thus affecting the shape of the root.

Beet leaf spot is widespread in its occurrence and is of considerable economic importance in the field. In the sugar beet crop, it lowers the efficiency of the leaves as sugar producers. In the market, leaf spot predisposes the leaves to the attack of slimy soft rot.

Spraying with Bordeaux mixture will control the disease.

Ref. (40).

BEET: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

BEET: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

BEET: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

BEET: NEMATODE DISEASE.

(See Nematode Disease).

BEET: MISCELLANEOUS DISEASES.**ROOT ROT.**

Cause: A fungus (*Rhizoctonia*).

This rot starts typically from the crown, and progresses downward from the leaf bases.

Ref. (17).

SCAB.

Cause: A fungus (*Actinomyces scabies*).

Beet scab closely resembles potato scab. Large corky brown excrescences are produced. Sugar beets and mangels are susceptible.

This disease is uncommon in the market.

BRUSSELS SPROUTS: BLACK LEAF SPOT.

(See Cabbage Black Leaf Spot).

BRUSSELS SPROUTS: RING-SPOT.

(See Cauliflower Ring-Spot).

CABBAGE: BLACK LEAF SPOT.

Cause: A fungus (*Alternaria brassicae*).

In the early stages, this disease is characterized by minute, circular, shiny, brown to black spots. Later these spots enlarge and lose their circular shape. The lesions generally are somewhat concentrically marked in target fashion and, under moist conditions, may be covered by a growth of brown to black mold. Generally, if affected heads are kept in a dry place, the centers of the spots fall out and leave holes.

The lesions are more pronounced and more common on the outer leaves, but under moist conditions, the fungus may penetrate from leaf to leaf and often may afford points of attack for the bacteria of slimy soft rot.

Infection occurs in the field, and the disease develops in transit and storage.

Disinfection of storage houses, care in handling, and a regulation of temperature and moisture conditions will control the disease in storage. The spots do not render cabbage unfit for transit and storage if the affected leaves are trimmed off.

Ref. (26).

CABBAGE: BLACK ROT.

Cause: Bacteria (*Pseudomonas campestris*).

Black rot is characterized by a black discoloration of the water-conducting tissue of the plant. Sometimes the discoloration occurs only as a ring in the stalk, but not infrequently it extends into the leaf midrib and veins. This discoloration in the stalk can be seen easily if a fresh section is cut, and its progress into the leaves can be noted by breaking the leaves from the stem, which will reveal a group of black dots in the leaf scars. Badly affected leaves have a steel gray or purplish hue, due to partial masking of the blackened veins by the white or yellowish leaf tissue.

If no secondary rots set in, the disease may show itself as a slowly progressing rot which does not necessarily proceed from the outside leaves as in slimy or watery soft rot, but may appear in the covered leaves, even in the heart of the head. Generally, however, a typical slimy soft rot sets

in, proceeding either from the outside or inside, and the head rots rapidly with a very offensive odor.

This disease occurs in all cabbage-growing regions. Infection takes place in the field. The bacteria enter the plant at the margins of the leaves, proceed downward through the veins to the main stalk, and then upward into the head.

The disease develops in transit and storage. Affected stock is very subject to secondary rots, and as a result is a menace to healthy stock.

Control of the disease in the field consists of seed disinfection, seedbed sanitation, and crop rotation. Severely affected stock is unfit for food. It is not advisable to store or ship slightly affected stock.

Ref. (61); (26).

CABBAGE: FREEZING INJURY.

Cause: Exposure to low temperatures.

Freezing injury is marked by a glassy yellowish appearance of the affected tissues.

Affected stock, if thawed rapidly in a warm place, or if kept in a moist atmosphere, is soon destroyed by slimy soft rot. Therefore it is not suitable for storage. It is generally assumed that cabbage can be frozen once or twice, if thawed out properly, without any injury except a slight shrinkage and flabbiness. The outer leaves can be frozen and thawed without injury, but if the freezing extends to the interior tender tissues, these are killed and, upon thawing, fall a ready prey to slimy soft rot.

Generally all frozen heads which do not show a glassy, yellowish ring in the tissue of the stalks upon thawing are fit for marketing for immediate consumption. This is not a positive test, however, since not all affected heads show a discoloration or decay of the stalk.

CABBAGE: LEAF SPECK.

Cause: Not known; probably non-parasitic.

Leaf speck consists of small, sharply-sunken, shiny, brown spots or black specks which may occur on all leaves of an affected head. Speck can be differentiated from black leaf spot by the smaller size of the spots, and by the absence of concentric rings, or any black fungous outgrowth.

Leaf speck occurs in cabbage from all sections, and is very severe in some Florida stock.

Nothing is known about the cause, point of origin, and conditions favoring the development of the disease.

No control measures are known. It is advisable to sort cabbage carefully because affected heads have a lower market value.

CABBAGE: SUN-SCALD.

(See Sun-Scald).

CABBAGE: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

CABBAGE: RHIZOPUS ROT.

(See Rhizopus Rot).

CABBAGE: SLIMY SOFT ROT; SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

CABBAGE; WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

CARROT: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot and Artichoke Gray Mold Rot).

CARROT: RHIZOPUS ROT.

(See Rhizopus Rot).

CARROT: SLIMY SOFT ROT; SOFT ROT.

(See Slimy Soft Rot).

CARROT: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

CARROT: NEMATODE DISEASE.

(See Nematode Disease).

CAULIFLOWER AND BRUSSELS SPROUTS: RING-SPOT.

Cause: A fungus (*Mycosphaerella brassicicola*).

The symptoms of this disease of cauliflower are small definitely circular spots on the leaves. They are visible on both sides of the leaf, have light brown or grayish, dry centers, and are surrounded by olive-green or blue-green borders which shade off into the natural color of the leaf. When the leaves turn yellow, the spots retain their green borders and become very conspicuous against the yellow background. The borders of the lesions are frequently raised, and may show concentric circles. Very minute, black dots, pycnidia, cover the outer edges of the spots on both surfaces of the leaves. They are densely crowded in the outer parts of the lesions, but are more scattered or absent in the centers of the spots.

Ring-spot is a field disease on the Pacific Coast. Infection takes place in the field, but the lesions may develop and enlarge during transit.

This disease causes premature yellowing and death of the leaves and thus affects the quality of the stock.

Ref. (52).

CELERY: BACTERIAL LEAF SPOT.

Cause: Bacteria.

This disease is characterized by shiny, dark-brown irregular, translucent or parchment-like spots on the leaves. Often these lesions extend along the veins and down the petioles causing a water-soaked condition. This disease causes a premature yellowing and death of the leaves. The lesions of this disease can be differentiated from the early blight lesions by the ashen gray, opaque centers of the latter and from late blight lesions by the absence of the black shiny dots or pycnidia.

Bacterial leaf spot occurs in Michigan and New York celery.

Infection takes place in the field and the disease may develop, but does not spread in transit or storage.

Severely affected stock is discriminated against because of its unsightliness and because the petiole lesions destroy the edible parts of the plant.

Spraying with Bordeaux will control the disease.

CELERY: EARLY BLIGHT.

Cause: A fungus (*Cercospora apii*).

Early blight is characterized by irregular, slate-colored spots on the leaves, with ashen-gray to tan centers. These can be distinguished from late blight lesions by their larger size and the absence of the conspicuous black dots or pycnidia. Generally early blight occurs only on the leaves, while late blight occurs very commonly on the petiole as well as on the leaf blade. At times early blight occurs on the petioles as elongated, tan, sunken areas, and causes a wilting and drying out of the leaf and petiole. This disease occurs in all celery districts, but is most common in Florida celery. It is favored by hot, dry weather.

Early blight starts as a field infection, and develops in transit or storage. Unlike late blight, it does not lead to a rot, but causes the tissues to dry out and shrivel.

Severely affected stock is unsightly and its market value is reduced.

Early blight can be controlled by spraying with Bordeaux mixture.

CELERY: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

CELERY: LATE BLIGHT.

Cause: A fungus (*Septoria petroselini*).

Late blight is characterized by small, irregular, brown spots on the leaf blade and petiole. Under transit conditions, the lesions may be dark green and water-soaked. The centers of the spots bear small, black, glistening dots, the fruiting bodies or pycnidia of the fungus. This disease

can be differentiated from early blight and bacterial leaf spot by the presence of the shiny pycnidia and the more common occurrence of spots on the petioles, where the pycnidia are even more conspicuous than on the leaf lesions.

This disease occurs generally in all celery districts, especially in California, Florida, Michigan, and New York. It is favored by cool, moist weather.

The fungus overwinters on the seed and in the soil. Late blight spreads in the field during the growing season, and although the original infection takes place in the field, the disease develops and spreads in transit and storage, leading to a soft rot of the leaf blades and petioles.

Severely affected stock is unfit for market and storage purposes.

Crop rotation, use of disease-free seed, and proper spraying will control the disease. It is advisable to discard severely diseased plants because of the continued development of the disease at low temperatures.

Ref. (11); (55).

CELERY: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

CELERY: WATERY SOFT ROT; SCLEROTINIA ROT; FOOT ROT; PINK ROT; RIB ROT.

(See Watery Soft Rot).

CELERY: BLACK HEART.

Cause: Unbalanced water relations.

In the earliest stages this disease is marked by shiny, light brown lesions on the blades of the youngest leaves. Very often these are not apparent until the heart of the plant is dissected, since the innermost, most completely protected leaves frequently are the only ones affected. Later the lesions enlarge so as to involve the entire leaf blade, and become very moist and turn black, the whole heart, even the entire stalk, becoming involved if sufficient time is allowed. The advanced symptoms of the disease are caused by bacteria of the *Bacillus caratovorus* type, which invade the tissue killed by the unbalanced water relations of the plant.

Black heart originates in the field. Its evil consequences are so well realized by shippers that most affected stock is culled out before shipping. Whether or not stock will develop black heart depends upon the plant itself, upon the nature of the soil on which the crop is grown, and upon the weather. In Florida the disease is most severe in late season plants of the Golden Heart variety.

Ref. (60).

CELERY: NEMATODE DISEASE.

(See Nematode Disease).

CUCUMBER AND MUSKMELON: ANTHRACNOSE.

Cause: A fungus (*Colletotrichum lagenarium*).

On cucumbers the anthracnose lesions are found in the market as circular, sunken, water-soaked spots which usually bear a slimy, orange-pink coating of spore masses. As the rotted tissue dries out, the surface may crack.

On Honeydew muskmelons the oval, light gray lesions with concentric rings of pink or black dots (acervuli) are very conspicuous. Large lesions become sunken, and the central tissue often cracks open, affording a port of entry for other fungi.

On netted cantaloupes, the lesions are sharply sunken but not very conspicuous until the salmon-colored spore masses appear.

The same fungus causes anthracnose of watermelon (discussed elsewhere).

Anthrachnose is a field disease occurring on leaves, stems, and fruits. Infection proceeds from infested seed or soil, and the disease spreads during wet weather. Fruit infection takes place in the field and the lesions enlarge in transit, causing serious blemishing. Frequently, secondary rots enter through the anthracnose spots.

Anthrachnose is of widespread occurrence. In the market it has been noted commonly on Louisiana cucumbers and Colorado Honeydew and Osage muskmelons.

Affected fruits are unsightly and rapidly become unsalable.

The disease can be controlled by crop rotation, use of disease-free seed, and spraying with Bordeaux mixture. It is advisable not to ship fruits showing any signs of the disease because of its rapid development in transit.

Ref. (21).

CUCUMBER: BACTERIAL SPOT.

Cause: Bacteria (*Bacterium lachrymans*).

The lesions of bacterial spot appear as small greasy or water-soaked spots, usually showing a small whitish dot in the center. The diseased spots may enlarge and lead to a secondary soft rot of rather large areas. Often a jelly-like mass of exudate is formed on these lesions. Under certain conditions, such rotted tissue may dry out, leaving cavities in the surface of the fruit.

Bacterial spot is a common and widespread disease of the vines causing what is known in the field as angular leaf spot. The disease is carried with the seed and spreads in the field in wet weather. Fruits become infected in the field, and the disease may progress but does not spread during transit.

The disease may be controlled by seed disinfection, crop rotation, and spraying with Bordeaux mixture.

Ref. (6); (62); (8).

CUCUMBER: MOSAIC.

Cause: Unknown.

Cucumbers affected with mosaic may be variously misshapen, often bearing large warts. They are often distinctly green mottled in color. A common type is a combination of green warts on a white or yellowish background.

Mosaic is a widespread and very destructive disease of the vines which greatly reduces the yield. It is extremely communicable and is spread by insects.

Usually the more severely affected fruits do not reach the market. So far as known, the edibility and keeping qualities of affected fruits are not impaired, but the market value is materially lowered.

No practical control is known.

Ref. (13); (22); (31).

CUCUMBER: SCAB.

Cause: A fungus (*Cladosporium cucumerinum*).

The symptoms of this disease on the fruits are small, circular, sunken lesions covered with a greenish velvety growth of mold. Drops of a red gummy exudate may be formed on these spots. The disease is not simply a scab as the name would imply, since the tissues are rather deeply involved.

The fungus attacks the vines, and fruit infection occurs in the field. Young growing parts are very subject to attack. The disease is very important in the pickle crop, where it becomes severe late in the season. It is favored by cool, moist weather.

Ref. (12).

CUCUMBER: SUN-SCALD.

(See Sun-Scald).

EGG PLANT: FRUIT ROT.

Cause: A fungus (*Phomopsis vexans*).

At first fruit rot consists of small, circular or oval spots. Usually the lesions are at first much lighter in color (tan or gray) than the surrounding tissue. Later the lesions become dark brown and sunken, and under favorable conditions increase very rapidly in size. By the coalescence of such lesions, much or all of the surface of a fruit may be involved. Small, brown to black, pimple-like pustules or pycnidia break through the surface and cover the inner and older zones of affected tissue. The affected regions are brown and softened. At times they are quite dry, suggesting a dry rot.

Fruit rot occurs in all egg plant growing regions, but is especially severe in the South.

The disease occurs on the plants in the field, where it attacks leaves, stems, and fruit, and is known as leaf spot,

stem blight, and fruit rot. The original infection of the fruit takes place in the field, but the disease develops and spreads in transit and often causes heavy losses.

It is not safe to ship spotted fruits.

Ref. (23).

EGG PLANT: RHIZOPUS ROT.

(See Rhizopus Rot).

LETTUCE: BLACK ROT.

Cause: Bacteria.

Black or brown spots or irregular patches on the outer leaves, increasing in size under proper conditions until entire leaves are involved, are the symptoms of this disease. Later the inner leaves may be attacked, and a soft dark brownish rot of the head may result.

This is a field disease reported from Florida affecting seedlings and older plants. It may develop during transit.

Other bacterial field diseases of lettuce have been reported from North Carolina and Louisiana.

The relation of these field diseases to the slimy soft rot prevalent in the market has not yet been determined.

Ref. (60).

LETTUCE: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot and Cauliflower Gray Mold Rot).

LETTUCE, ENDIVE, ESCAROLE, AND CHICORY: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

LETTUCE: WATERY SOFT ROT; SCLEROTINIA ROT; DROP; DAMP-OFF.

(See Watery Soft Rot).

LETTUCE: RUSSET.

Cause: Not known, probably non-parasitic.

This disease is characterized by small reddish to russet spots or streaks which may occur on practically all of the leaves of an affected head. The brown streaks usually occur along the large veins of the leaves. The vascular bundles in the stalk and in the petioles may also be discolored.

Russet occurs in lettuce from all regions.

Nothing is known about control of the disease.

It is advisable to sort carefully before shipping because the presence of russeted heads reduces the value of a shipment.

LETTUCE: TIP BURN.

Cause: Non-parasitic (irregular water supply).

This disease is characterized by the dead, brown borders

of the leaves throughout the head. It is found in head lettuce from all except the Boston district.

Tip burn is a field disease probably caused by an irregular water supply. The injury is most likely to occur when bright warm weather follows a period of cloudy or rainy weather, and is much worse on certain soils than on others. The disease seems to be least prevalent on a soil with a high sand and low clay content, combined with a high water-holding capacity. In such a soil, lettuce becomes deep rooted, and can apparently obtain water with sufficient rapidity to prevent injury from too rapid transpiration. In the Imperial Valley of California, this disease appears to be associated with an excess of alkali in the soil.

Tip burn often predisposes affected stock to slimy soft rot during transit and storage. The slimy soft rot starts in the dead tissues, especially in the interior of the head where moist conditions prevail. Sometimes the rot gets a foothold even in the growing plant.

No effective method of control has been established for tip burn. A well-regulated water supply and the use of varieties properly adapted to the soil and climatic conditions are important considerations.

Ref. (19); (20).

LETTUCE: MISCELLANEOUS DISEASES.

DOWNY MILDEW.

Cause: A fungus (*Bremia lactucae*).

The symptoms of this disease are sharply angular leaf spots, yellowish to brown when viewed from above, and bearing on the lower surfaces a white felt-like outgrowth of mold, the spores and spore-bearing mycelium of the fungus.

Downy mildew occurs on lettuce grown under glass and on the field crop where moist cool weather prevails, as in the Colma district in California. In head lettuce, the inner as well as outer leaves are attacked. This disease is of some importance because of its direct attack on the edible leaves and because of its tendency to predispose the tissues to slimy soft rot.

LEAF SPOT.

Cause: A fungus (*Septoria consimilis*).

This disease causes brown spots on the older leaves bearing black points, the pycnidia of the fungus. It occurs on garden varieties late in the season, and is of little importance.

LEAF SPOT.

Cause: A fungus (*Cercospora lactucae*).

Small tan-colored spots on the older leaves are the symptoms of this disease, which has been noted in the San Francisco market.

SHOT HOLE OR ANTHRACNOSE.

Cause: A fungus (*Marssonina panattoniana*).

Small brown spots on the leaves, from which the dry centers often crack and fall out, are characteristic of this disease, which occurs on greenhouse lettuce.

Ref. (2).

MUSKMELON: (HONEYDEW AND CASABA): FRUIT SPOT.

Cause: A fungus (*Alternaria*).

On Honeydew melons, this disease appears first as small water-soaked or chestnut-brown spots on the surface. As these enlarge, they become more oval in shape and may have wide water-soaked borders. Later the lesions become more or less sunken and usually develop black centers. Under humid conditions a velvety surface growth of mold is produced on the lesions. This mold is at first gray, but soon becomes dark greenish gray and later dark brown or almost black.

Continued development and coalescence of these lesions lead to an extensive shallow rot of the rind accompanied by a cracking of the surface. The affected tissue is at first very soft and watery, later becoming yellowish and very dry, tough, and leathery. The rot eventually penetrates through the rind into the edible pulp below.

The symptoms on the Casaba melon are essentially similar to those on the Honeydew melon. On the striped Christmas Casaba melon, the lesions are brown and less conspicuous and the surface growth of mold is not common.

Fruit spot lesions differ from those of anthracnose in the absence of acervuli and in the moldy outgrowth usually present.

This disease was found very prevalent during the fall of 1918 on Colorado Honeydew melons, and on both the Casaba and Christmas Casaba melons from Turlock, Cal.

The source of infection is not known. The lesions appear during transit and storage and become very conspicuous in the market, not only detracting from the appearance of the melons but also causing an objectionable surface rot. In addition, the lesions also serve as points of entry for other rot-producing organisms.

No control measures are known.

MUSKMELON: SUN-SCALD.

(See Sun-Scald).

MUSKMELON (CANTALOUPE): MISCELLANEOUS DISEASES.

BACTERIAL SOFT ROT.

Cause: Bacteria.

Wounds and fruit spot lesions may open the way for the entrance of bacteria which cause a soft rot of the tissues. This rot is very soft and mushy and the tissue appears water-soaked.

FUSARIUM ROT.

Cause: A fungus (*Fusarium*).

This is a rapidly progressing rot usually characterized externally by a dense and profuse outgrowth of pinkish white mold. The rotted tissue is rather dry, and the pink mycelium may cause the entire rotted area to appear pinkish in color. Other types of *Fusarium* rots may occur in the field. These rots progress more rapidly than the rot caused by *Alternaria*.

GREEN MOLD ROT.

Cause: A fungus (*Cladosporium*).

This disease occurs very frequently on cantaloupes after they have been in transit a long time. It is characterized by a green, velvety fungous growth, which causes a slowly progressing rot.

MUSKMELON (CANTALOUPE AND HONEYDEW): ANTHRACNOSE.

(See Cucumber Anthracnose).

MUSKMELON (HONEYDEW): BLACK MOLD ROT.

Cause: A fungus (*Alternaria*).

This is marked by a brown to black, velvety surface growth of the fungus. The fungus usually invades tissue killed by sun-scald and causes a rot of the underlying tissues. This rot is also very common on watermelons affected by sun-scald.

ONION: BLACK MOLD ROT.

Cause: A fungus (*Sterigmatocystis niger*).

Black mold is characterized by black, powdery masses on or between the scales. When these masses occur between the scales they have a tendency to follow the veins.

Affected stock may show no symptoms other than the presence of this black powder. At times sunken and discolored areas are found underlying the powdery black masses. Under dry conditions the affected tissue is papery and brittle and sometimes highly colored.

Although commonly called "smut," this disease should not be confused with true smut. The latter is rarely found on the market except on sets, and is marked by black, powdery masses within the scale tissues.

All varieties of onions are susceptible. The disease is very common on California and Texas onions. Infection occurs in the field, though the fungus continues to grow in storage.

As a rot, this disease under dry conditions usually is of minor importance, but as a blemish it causes very serious depreciation in value of the affected stock. Under moist conditions the fungus may cause severe rotting.

Since this disease does not progress very rapidly, affected bulbs can probably be held for some time if they are kept

in very dry storage at 32° to 35° F., preferably in slatted crates rather than in bags.

Ref. (74).

ONION: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

ONION: NECK ROT.

Cause: Fungi (Botrytis; Sclerotium).

This is a semi-watery rot followed by a shrinking and shriveling of the scales. The rot occurs typically as a neck rot in the field, while in storage other regions of the bulb also may be attacked. A gray moldy growth and hard, grayish to black masses of the fungus (sclerotia) develop on the outside of the scales. Neck rot is an example of the gray mold rot of vegetables elsewhere described.

Neck rot has a tendency to rot all scales uniformly downward rather than to follow certain scales as does slimy soft rot. Typical neck rot is especially conspicuous in white onions. In red and yellow varieties, the infection may be confined to the inner scales, and external neck rot symptoms may be lacking. In colored varieties, the soft rotted condition is often found, and the diseased flesh frequently becomes pinkish, but the gray mold growth and sclerotia are less commonly found. Unlike slimy soft rot, neck rot does not have a foul odor.

Infection takes place in the field, at or shortly after the harvest. The fungus enters the cut necks of bulbs, and gradually progresses downward. The rot develops in storage under moist conditions. Chilled or frozen onions and scallions are subject to neck rot. In chilled onions, the rot is more frequent in places other than the neck.

If onions show a high percentage of neck rot in storage, transit, or market, disposal for immediate consumption is advised.

The disease can be controlled by prompt curing of the crop and by storage in a dry place at 32° to 35° F. It is destructive in onion sets as well as in table stock.

Ref. (48).

ONION: SLIMY SOFT ROT.

Cause: Bacteria (*Bacillus carotovorus* group).

This is a very soft, mushy rot of the scales, which progresses downward from the neck and is accompanied by a very repulsive odor. Often it is confined to only one or two scales in the interior of the bulb. Lesions may occur anywhere, however, especially if the tissues have been killed by sun-scald or bruising.

Slimy soft rot progresses faster than neck rot, and is not accompanied by a gray mold and sclerotia as is neck rot. Neither does it progress uniformly downward destroying all

scales as does neck rot, but usually follows certain scales all the way around.

Slimy soft rot occurs in all onion-growing districts in the United States, and is most common in onions harvested during warm, rainy seasons, or in onions sun-scalded during the curing process. It is also very common in Spanish onions. It is claimed by growers that yellow varieties are more susceptible than red. Careful handling to avoid sun-scald and bruising during the harvesting operation is of primary importance as a preventive measure.

In badly affected shipments, immediate drying of the stock will check the progress of the rot. This may be done by dumping the sacks at once and spreading the contents.

Ref. (67).

ONION: SMUDGE.

Cause: A fungus (*Colletotrichum circinans*).

Smudge or anthracnose is a disease of white onions characterized by black blotches or aggregations of minute black dots on the outer scales. Each one of the minute dots is an acervulus (plural, acervuli): These acervuli are often arranged in concentric rings. In severe cases, the smudgy spots are so extensive that the side of the onion appears smoked. Generally the smudge is on the outside scales, though it may appear on the inner scales as well. It should not be confused with the black powder which is characteristic of black mold.

It is of widespread geographic distribution, but only white varieties are seriously affected. Infection takes place in the field from the fungus which overwinters in the soil. The disease appears shortly before the harvest, and develops rapidly in the crated onions after the harvest if the weather is moist. It causes very little shrinkage of affected bulbs, the chief damage being due to the appearance and reduced market value of affected bulbs.

Smudge can be prevented by rapid curing of the crop as soon as it is harvested. If the bulbs are stored in a dry place, the disease makes little progress. In a moist atmosphere, the fungus penetrates from scale to scale, and causes a softening of the tissues.

Ref. (72).

ONION: SMUT.

Cause: A fungus (*Urocystis cepulae*).

Onion smut appears as dark-colored, slightly raised streaks or blisters on the bulbs and leaves. The leaves are often recurved and distorted. Sets may be greatly shrunken with the whole exterior covered with the blisters. When cut open, these ridges or pustules are found to be filled with a greenish black powdery mass. Smut differs from black mold in that the black powder is within the tissues of the scale, not upon or between the scales.

Onion smut is common in northern growing regions, especially in Wisconsin, Illinois, New York, Ohio, Iowa, and Massachusetts.

The fungus persists in the soil, and infection occurs when the plants are seedlings. Badly infected plants are usually killed and large losses are thus caused in the field. There is no progress of the disease in storage or transit. Smut is uncommon in the market except on onion sets.

Control measures are planting in clean soil or the use of a formaldehyde drip on the seed drill.

Ref. (71).

ONION: MISCELLANEOUS DISEASES.

BLOTCH.

Cause: A fungus (*Macrosporium*).

The name "blotch" has been temporarily adopted for this blemish on the outer scales of red and yellow varieties of onions. The symptoms are large bleached or greenish discolored areas on the bulbs, bearing numerous fine black linear marks or ridges parallel to the veins.

BLUE MOLD.

Cause: A fungus (*Penicillium*).

Under certain conditions in storage or transit, blue mold occurs on the outside of the bulbs, and is usually associated with insufficient ventilation and some predisposing injury to the tissues, such as wounds, freezing, or sun-scald.

While most fruits are subject to attack by species of *Penicillium*, among vegetables, only onions, sweet potatoes and sweet corn are commonly attacked.

FUSARIUM ROT.

Cause: A fungus (*Fusarium*).

This rot usually but not always proceeds from the base of the bulb. The affected region is soft and flabby and becomes shrunken and shriveled in appearance. The surface may be studded with small white pads, the fruiting bodies of the causal fungus. There is no development of gray mold or black sclerotia as in neck rot.

GRAY MOLD SPOT.

Cause: A fungus (*Botrytis*).

This disease causes a bright red lesion on the neck at the ground line, which results in the death of the outer leaf. At times a slight outgrowth of gray mold may be formed in the lighter colored center of the lesion. Gray mold spot is found only on winter-grown green onions in the spring, and has been noted in the Kansas City market gardens.

SUN-SCALD.

(See Sun-Scald).

PARSNIP: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

PARSNIP: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

PEA: BACTERIAL SPOT.

Cause: Bacteria.

On the pods this disease appears as greasy water-soaked spots which may enlarge to irregularly circular, slightly sunken watersoaked blotches with gray centers.

This is a rather widespread disease of the vines, and pod infection occurs in the field. It seems likely that the spots enlarge during transit. Bacterial spot was found commonly in the Chicago market in the summer of 1918, and appeared to predispose the stock to secondary rots.

No control is known at present.

PEA: POD SPOT.

Cause: A fungus (*Ascochyta pisi*).

The symptoms of this disease are rather small, definitely circular and sharply depressed spots with tan-colored or pinkish centers often bearing small pimples, the pycnidia of the fungus.

This is a serious field disease affecting the leaves and stems as well as the pods. Pod infection occurs in the field, and there is probably little spread of the disease during transit.

Control involves crop rotation and the use of disease-free seed.

Ref. (73).

PEA: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

PEA: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

PEPPER: ANTHRACNOSE.

Cause: A fungus (*Gloeosporium*).

Slate-colored to charry black, sunken lesions are characteristic of this disease. The margins of the lesions either are darker colored or watersoaked but of the same color as the healthy tissue, and wrinkled. Under moist conditions salmon-colored dots or spore heaps occur, which may run together and form a slimy mass. These dots are the acervuli (sing. acervulus) of the fungus and furnish a positive diagnostic character for this disease.

This disease is found frequently on peppers. Very frequently, under market conditions, the lesions do not bear

acervuli but become covered with a black, velvety growth of *Alternaria* species.

PEPPER: RHIZOPUS ROT.

(See Rhizopus Rot).

PEPPER: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

PEPPER: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

PEPPER: SUN-SCALD.

(See Sun-Scald).

POTATO: BLACK SCURF AND RUSSET SCAB.

Cause: A fungus (*Rhizoctonia*).

Black scurf and russet scab are skin diseases. Affected potatoes are rough and dirty in appearance, or may have a cracked or corroded skin. Both are field diseases caused by the same fungus.

Black scurf is characterized by the presence of small brown or black masses or sclerotia on the tuber, often referred to as "dirt which will not rub off." These masses may be circular or irregular in outline and single or joined into series. Washing the tuber brings these into sharp relief.

Russet scab is a corrosion of the tuber skin. In some cases smooth-skinned tubers show local or general netting, either slight, or so extensive as to resemble the skin of netted varieties. In advanced stages the corrosion becomes channeled, and the intersecting channels may become so deep that the tuber surface suggests alligator hide.

In many cases, tubers are literally covered with black scurf without any apparent russetting, while in other cases the presence of the fungus seems to cause only a cracking or scabbing, without any sclerotia.

Generally neither black scurf nor russet scab are serious enough to affect the market value of potatoes. The former occasionally is severe enough to affect the appearance of the tuber. At times, in very moist cars or storage places, the sclerotia germinate, and the potatoes become covered with a luxuriant growth of mold which detracts from the good appearance of the lot. Russet scab, however, affects the appearance of the tuber more markedly, and often corrodes the skin sufficiently to necessitate deep paring, which is attended by a waste of food.

Ref. (50); (64).

POTATO: SILVER SCURF.

Cause: A fungus (*Spondylocladium atrovirens*).

Silver scurf is a skin disease which is purely superficial, and does not penetrate below the cork layer. The infected areas are marked by their silvery appearance, which is especially pronounced if the tubers are washed. In late stages of the disease, the affected skin becomes wrinkled and sloughs off.

Generally, affected areas occur near the stem end, but they may be found over the entire surface. Diseased areas vary from one-fourth inch to one inch or more in diameter.

In storage, especially under moist conditions, a sooty layer composed of the spores of the fungus may be found on the diseased areas.

Infected stock is fit for table use.

Ref. (43) ; (57).

POTATO: WART.

Cause: A fungus (*Chrysophlyctis endobiotica*).

In its early stages, wart is characterized by small, warty outgrowths which usually occur at the eyes of the tuber. These warts later become very large, and in severe cases, cover or involve the entire tuber. They are rough and black in color.

This disease is serious in certain parts of Europe, and has recently made its appearance in the United States (Pennsylvania). It is a very destructive field disease, and may be carried with diseased seed tubers. Once having gained a foothold, the fungus persists for long periods in the soil.

Infection takes place in the field while the tubers are growing. The disease does not progress in storage, and does not spread to healthy tubers. Diseased stock is very subject to secondary rots, and in this way it is a menace to healthy stock. Slightly affected tubers may be used for table purposes.

Precaution against the use of diseased tubers for seed purposes is of prime importance in the prevention of this disease. With this end in view a National quarantine has been established.

Ref. (35) ; (63).

POTATO: COMMON SCAB.

Cause: A fungus (*Actinomyces scabies*).

Scab is characterized by rough corky elevations, or by pits. If tubers are attacked early, the pits are deep; if late, they may be shallow and superficial. In the early stages, affected tubers are marked with minute, reddish or brownish surface lesions. Cork formation usually occurs underneath these lesions. Often the lesions coalesce and in severe cases the entire tuber may be covered by a rough incrustation. Scabby potatoes are little if any more disposed to decay than clean potatoes.

Deep scab is an advanced stage of common scab. Often insects or mites live in scab pockets. It is not known to just what extent these are responsible for the lesions.

Common scab occurs in all varieties of potatoes and is most common in potatoes grown in alkaline soil. Treatment of seed stock with corrosive sublimate, and planting in uninfected soil or acid soil are fair preventives of the trouble.

Wireworm or grub injury often are confused with scab lesions. It frequently is difficult to differentiate between them. Wireworm injury is generally marked by extensive channeling in the tuber skin, while grub injury is marked by deep, broad pits with protruding or overhanging rims. Wireworm and grub injuries often are points of entry for *Fusarium* species.

Scab and insect injuries depreciate the value of tubers for table use by making them unsightly, and by necessitating deep paring with attendant loss of food.

Ref. (10); (38); (39); (50).

POTATO: POWDERY SCAB.

Cause: A slime mold (*Spongospora subterranea*).

In the early stages powdery scab consists of pimple or blister-like eruptions, one-sixteenth to one-quarter inch in diameter, which are completely covered by the skin of the tuber. Later these coverings rupture and expose pits, which are single or joined, and fringed by the flaring, papery, torn, and more or less toothed remnants of the skin. The interior of these pits is at first filled with a brown powder or dust, which is usually absent when the tubers reach market. Often the tissues surrounding the pits become discolored and sunken, and each pit or group of pits appears as a crater in a sunken zone.

Powdery scab differs from common scab in the more nearly circular lesions, smaller pits, presence of brown dust, star-like rupture of the skin, and relative absence of cork formation.

This disease is not as serious a menace as it was once thought to be. It seems to be localized in a few sections of North America, and is causing little damage. It is favored by cool, moist weather.

The causal organism is introduced into the soil with diseased seed stock, and tuber infection occurs in the field.

Affected stock is fit for table use. Its market value, however, is decreased because of its unsightliness, and because of the waste due to deep paring.

Ref. (44); (46).

POTATO: LATE BLIGHT TUBER ROT.

Cause: A fungus (*Phytophthora infestans*).

Late blight tuber rot is characterized externally by depressed, discolored areas of irregular but definite outline. These may occur merely as spots or as very extensive lesions.

The affected areas often have a metallic or purplish tinge which is especially marked at the margin where the diseased tissue joins the healthy. At times, a pinkish color is apparent when the outer skin of affected areas is first removed. The diseased tissues underlying the sunken areas are browned, and unless secondary rots have set in, are quite dry and firm. The tissue is merely killed, not disintegrated. In cross section it is seen that the affected tissue tends to be limited to the outer parts of the tuber, and that the advancing edge is irregular and feathery.

Under storage conditions, especially if the air is very moist, secondary rots due to bacteria or fungi set in. At times, in advanced cases, it is difficult to ascertain whether the primary cause of rotting was freezing injury, with subsequent bacterial and fungous infection, or late blight. In such cases the deciding factors are the presence in the shipment of clear-cut cases of late blight or of freezing injury.

Potatoes affected with late blight do not show the hollow brittle regions found in potatoes affected with *Fusarium* tuber rot, nor the shriveled and wrinkled surfaces caused by *Fusarium* or freezing injury. The feathery edge of the tissue killed by the late blight fungus also serves to differentiate it from tissues killed by *Fusarium* or freezing.

Late blight tuber rot occurs most commonly in potatoes grown in the Eastern and North Central States, and in the coastal portions of the Pacific Northwest States. Under certain weather conditions, it also occurs in southern potatoes. During the rainy season of 1918, late blight was prevalent in Florida potatoes. It is not a hot weather disease but is favored by cool, wet weather.

Late blight is one of the most important field diseases of the potato, causing a severe blight of the vines in addition to its attack on the tubers.

The fungus overwinters in diseased tubers, and is introduced into the field with diseased seed stock. From diseased seedlings produced by such seed, the fungus spreads rapidly throughout the field when weather conditions are favorable. Tubers are infected in the field by spores washed down or spattered from the diseased vines. The disease also develops in transit and storage.

Late blight can be controlled in the field by spraying with Bordeaux mixture. Its development in transit and storage is checked by a dry atmosphere and a low temperature (34° to 36° F.; 45° F. is too warm). It is highly desirable that diseased tubers be sorted out as soon as they can be detected. In case of a late blight epidemic, digging should be postponed until the tops have dried up.

Affected tubers are not marketable for table use. They are also a menace to sound stock in transit and storage since secondary rots, such as *Fusarium* or slimy soft rot, very frequently follow late blight.

Ref. (9); (18); (34); (45).

POTATO: FUSARIUM TUBER ROT.

Cause: Fungi (*Fusarium* species).

Fusarium tuber rot is generally marked by sunken, shriveled, wrinkled, or broken areas on the tuber surface. These areas may be brown to black in color and on them may appear masses of whitish or brightly colored mold.

The diseased tissue underlying such discolored and shrunken areas may be dry and brittle, and may contain cavities lined with a white or bright colored growth of the fungus responsible for the rot; or this tissue may be watery but intact, depending upon the species of fungus responsible for the rot and the conditions under which the affected tuber was kept. Usually, if affected tubers are kept in a dry cool place, the dry, brittle, and hollow type of powdery dry rot results. If they are kept in a warm moist place the soft intact type of rot is usually the result. Affected tissue may be gray or brown to black in color.

The rot may proceed from the stem or seed end, from the eyes or from broken places in the skin, such as cuts and bruises. At times the infection is confined to a mere discoloration of the vascular ring proceeding from the stem end of the tuber. This is not visible until the tuber is cut. This discoloration cannot always be positively differentiated, without microscopic examination and cultural test, from discolorations due to freezing injury or to varietal characteristics. *Fusarium* discoloration penetrates much farther into the tuber, however, than other vascular discoloration. In southern potatoes, this discoloration in the ring can be told from a similar discoloration due to brown rot by the absence of the slimy bacterial masses or droplets which ooze out when tubers affected with brown rot are cut.

This stem-end infection and some lenticel and eye infections occur in the field. Stem-end invasion is very common in potatoes produced by plants affected with *Fusarium* wilt. Jelly-end is another type of stem-end infection which occurs in the field. However, most of the tubers rotted by *Fusarium* species are infected through breaks in the skin caused during and after the harvest.

Tubers with areas injured or killed by freezing are very subject to *Fusarium* rots. The rot usually starts in at some point which did not heal over completely with cork or was not entirely sealed with starch following the drying out of the frozen tissue. At times it is very difficult to distinguish between potatoes with frozen areas which were subsequently infected with *Fusarium* tuber rot, and potatoes which were originally affected with the rot and then kept under moist conditions. The end result in both cases is usually a wet brown rot. This wet type of rot is especially marked in the Burbank and Netted Gem potatoes from Idaho. It may be practically impossible at times to determine the cause of the decay by examination of a single tuber

without a microscopic examination or cultural test. An examination of the entire lot, however, will reveal the presence or absence of typical signs of freezing injury. The frozen tissue of a tuber usually is set off from the healthy tissue by a more or less straight purple or brown line which extends across all the tissues of the tuber, or by a gray, chalky, brittle layer of starch. On the other hand, most *Fusarium* species generally tend to rot the center of the tuber faster than the cortical tissue, leaving a shell of sound tissue enclosing a rotted center.

An examination of the surface of a tuber may not be sufficient to determine whether the tuber is affected with late blight or *Fusarium* tuber rot. Both produce sunken discolored areas. The late blight tuber rot, however, causes more of a metallic luster than the *Fusarium* tuber rot, and usually the sunken area is not shrunken and shriveled. In tubers affected with late blight, the diseased tissues underlying the discolored areas are solid and dry and have a feathery edge, while in the *Fusarium* tuber rot the diseased tissue, if dry, contains cavities or is watery and soft and in cross-section is set off from healthy tissue by a sharp, smooth edge.

Tubers affected with the soft, wet, brown type of *Fusarium* tuber rot can be differentiated from tubers affected with the slimy soft rot by the absence of the foul odor so characteristic of all bacterial soft rots. *Fusarium*-infected tissue is not slimy even though it is soft and disintegrated.

Deterioration of tubers due to *Fusarium* tuber rot is sometimes rapid and often complete. Infected tubers are a menace to healthy ones. Immature tubers, cut and bruised tubers, tubers with second growth knobs which are easily broken off, tubers affected with other diseases such as late blight tuber rot or blackleg, and tubers with frozen areas are an easy prey for the *Fusarium* species causing tuber rot.

One or more forms of *Fusarium* tuber rot occur in practically all potato districts. *Fusarium* tuber rot is much more common in northern than in southern potatoes. It does develop, however, in southern potatoes shipped north and causes a very soft, watery rot or a stem-end rot. The original infection may occur in the field, in transit or in storage, but in most cases the rot develops and spreads in transit and storage. In a few cases, such as jelly-end rot and black field rot, the disease develops in the field.

The *Fusarium* tuber rot in potatoes from the Central Western States, especially in the Early Ohio stock from Nebraska and Minnesota, and the Burbank and Netted Gem stock from Idaho, is called powdery dry rot. This term refers more to the appearance of the spore masses of the fungus than to the diseased tuber tissues. The *Fusarium* tuber rot in potatoes from the Eastern States is designated as tuber rot or dry rot and generally is of the wet type. The

Fusarium tuber rot appearing at the end of the tuber in the long, white varieties of the Northwest, such as the Burbank, is known as jelly-end, while that appearing in other parts of the tuber, especially marked in the round varieties such as the Rural, is known as "black field rot." These rots are caused by *Fusarium radicum*, which also causes a very soft leaky rot and a dry, black stem-end rot of potatoes grown in the southern half of the Mississippi Valley.

Wounded or bruised surfaces which have become sealed with starch or cork should not be confused with *Fusarium* infections. Generally tuber rot starts in bruises and cuts. During the early storage season, it is often impossible to determine whether or not a bruise or cut will develop tuber rot. Later in the season the presence of soft discolored tissue or cavities lined with mycelium makes a diagnosis much easier.

The safest and surest methods of control are careful handling of tubers to avoid cutting and bruising; sorting out of bruised, broken, cut, diseased, and frozen potatoes; and storing of tubers in a well-ventilated place at a temperature between 36° and 40° F.

Ref. (7): (37).

POTATO: JELLY END ROT; BLACK FIELD ROT.

Cause: A fungus (*Fusarium radicum*).

Both jelly end and black field rot are types of the *Fusarium* tuber rot previously described. In jelly end rot, the end of the tuber is shrunken and collapsed, while black field rot is characterized by shrunken, collapsed, black to brown areas occurring anywhere on the surface of the tuber.

The diseased tissue underlying the discolored or shrunken surface is soft, watery, and light to dark brown or black in color, or it may be rather firm and black. Unlike powdery dry rot, cavities generally do not occur in the affected tissues.

The moist condition of jelly end rot is not observed unless the potatoes have just been dug or have been removed recently from a car which sweated or became overheated in transit. Generally, under market conditions, the disintegrated tissue dries out and resembles typical dry rot. Very often stock affected initially with jelly end or black field rot subsequently becomes affected with powdery dry rot, which progresses rapidly in storage.

These rots occur most commonly in Nebraska, Idaho, and Pacific Coast potatoes. Jelly end rot is prevalent in the long varieties such as the Burbank type, while black field rot is most clearly marked in the round varieties such as the Rural. Without a cultural examination it is impossible to differentiate at times between jelly end rot and so-called dry rot of the end of the tuber caused by *Fusarium* species other than *Fusarium radicum*.

The original infection takes place in the field. Both diseases develop in the field. They probably do not develop or spread in transit and storage, but do open the way for other *Fusarium* tuber rots.

Fusarium radiclecola may cause in southern potatoes a very watery leaky rot which involves the entire tuber and resembles the watery soft rot of other vegetables caused by *Sclerotinia*. This rot differs from jelly end and black rot in that the whole tuber is affected and that the rot is watery rather than jelly-like or firm. The diseased tissue is only slightly discolored. Under certain conditions in transit, an extensive mycelial growth and numerous bodies which resemble small sclerotia develop on the surfaces of affected tubers.

In this leaky rot of southern-grown potatoes caused by *Fusarium radiclecola*, the original infection takes place in the field. This type of rot is favored by hot weather, and is very frequently associated with scald. It is probable that this disease, unlike jelly end or black field rot, develops and spreads rapidly in transit.

Ref. (7) ; (54).

POTATO: POWDERY DRY ROT.

Cause: A fungus (*Fusarium trichothecioides*).

Powdery dry rot is a type of the *Fusarium* tuber rot previously discussed. In the name powdery dry rot, the word powdery does not refer to the appearance of the diseased tissue but to the powdery masses of spores formed by this fungus.

In this disease the affected region contains cavities separated by dried brownish tissue and starch and lined with whitish fluffy mycelium or powdery pink masses of spores. In the early stages, the affected tissue is light brown to black in color and is sharply delimited from the healthy tissue by a layer of brown or black tissue. Affected tubers are much lighter in weight than healthy tubers because of the extensive hollow areas. This rot is most pronounced in the central portion of the tuber, and as a result, affected tubers often become more or less hollow shells.

If tubers infected with powdery dry rot are stored in a very moist atmosphere, or if they become infected with bacteria, the typical symptoms are partly masked by the presence of soft watery tissues of chocolate color, which generally are more extensive than the typical dry areas.

Potatoes just taken from a car which has been in transit a long time, or which was not cooled or ventilated properly, show the symptoms of wet *Fusarium* rot rather than the symptoms of powdery dry rot. However, upon exposure to the air the typical dry rot symptoms soon develop.

Powdery dry rot appears to progress more rapidly at lower temperatures than the other *Fusarium* tuber rots. It occurs mainly in Idaho and other Rocky Mountain districts,

and in western Nebraska, being especially severe in the Early Ohio variety. It is prevalent in bruised or frozen potatoes from Washington, Idaho, Nebraska and Colorado.

Infection takes place through breaks in the skin of the tuber. These may be brought about by cuts or bruises, by freezing and by attacks of other fungi. The spores of *Fusarium* are usually everywhere present in the soil, on the surfaces of tubers, and in storage places, so that any break in the tuber skin is very likely to become infected.

Control of this disease depends upon careful handling of potatoes to minimize injury as far as possible. Because of the rapid progress of the rot, it is advisable to dispose of affected stock at the earliest opportunity. It is not profitable to ship severely affected stock.

Ref. (37); (53).

POTATO: LEAK.

Cause: Fungi (*Pythium debaryanum*; sometimes *Rhizopus nigricans*).

Leak is marked at first by a brown discoloration around wounds or bruised areas. The discoloration later becomes extensive, and the entire interior of the potato may become soft, and buff or light brown in color. Under pressure, the affected tubers exude a brownish watery liquid, and diseased tubers often leak badly. Potatoes affected with leak are much more watery than those affected with the wet type of *Fusarium* tuber rot.

Leak has been reported only from the Delta lands of the San Joaquin Valley of California and from Idaho. It does not occur in cool weather, and is entirely checked by heavy frosts.

Infection takes place in the field, and tubers are infected only through wounds, such as those due to fork injury or the breaking off of second-growth knobs. Infection occurs only in hot weather, but once the disease has gained a foothold, it may develop at lower temperatures.

Leak can be controlled by careful digging and handling of the potatoes and sorting out of bruised and broken tubers. In addition it has been found advisable to hold suspected potatoes 4 days in the warehouse before shipment so that the disease, if present, may develop and be more readily detected. It is not likely to be a storage trouble after the warm season.

Ref. (28); (29).

POTATO: BROWN ROT (SOUTHERN BACTERIAL WILT).

Cause: Bacteria (*Bacillus solanacearum*).

Brown rot may be indicated externally by a slight depression at the point of attachment of the stolon to the tuber, or by gray discolored patches on the surface. Sometimes no external symptoms may be visible. Upon cutting infected tubers, a moist brown discoloration and slight softening of

the ring (vascular) tissue of the tuber is seen. White sticky globules of bacteria ooze out from the cut tissues. In this stage, the rot is odorless.

The rot begins in the vascular ring, causing cavities which are filled with a dirty white, slimy, bacterial mass. Brown rot may be followed by slimy soft rot to which the foul odor of affected tubers is due.

This rot should not be confused with the ring discoloration associated with *Fusarium* wilt. The latter is most common in northern potatoes, while brown rot occurs only in southern, especially Florida, stock. There is no bacterial exudate from the bundles when a *Fusarium* infected tuber is cut.

Infection takes place in the field and the disease is there known as bacterial wilt. The infection proceeds from the affected plant through the horizontal underground stems (stolons) into the tubers which are enlargements of the stolons. Potatoes grown in new soil are most severely affected.

It is advisable not to plant potatoes in new ground. Infected stock is subject to decay and should be rapidly disposed of.

Ref. (61).

POTATO: SLIMY SOFT ROT.

Cause: Bacteria (*Bacillus carotovorus* group).

This disease of the potato belongs to the class of slimy soft rots described elsewhere, but, since it presents itself as a special phase in southern potatoes, a separate discussion is warranted. It is a soft, soupy, exceedingly foul smelling rot. Unless affected tissue is discolored by other causes, there is little change in color, the disintegrated tissue being whitish to yellow. The boundary between the soft disintegrated and the firm sound tissue is very sharp. As the more or less watery tissue dries, it becomes slimy and finally, when completely dry, a mere chalky white crust. During shipment the skin of affected areas is broken easily, and the slime commonly is smeared over neighboring sound tubers. In car-lot shipments this very materially increases the difficulty of sorting.

Slimy soft rot very generally invades killed tissue. In a wet soil, tubers often become asphyxiated, and the bacteria enter the tuber through scab wounds, through lenticels (the breathing pores of the tuber), through broken places in the skin, or through diseased stolons. Infection also occurs very generally through bruises received during digging and handling. If infected tubers are dug while wet, and scalded by exposure to the hot sun, or if sacked with the wet soil adhering, a procedure which may lead to heating or sweating, the rot may progress rapidly from the surface inward through the whole tuber.

Brown rot may be followed by slimy soft rot as a result of which large cavities are formed in the interior of the tuber. The decayed tissue often is so viscous that it can be pulled like very soft taffy.

Slimy soft rot is prevalent as a surface rot in potatoes shipped from Florida, Louisiana and Texas. It is favored by wet, hot conditions in the field, in transit or in storage. After the rot has once started, the temperature is of less importance than the humidity. This rot occurs also in northern potatoes. Tubers affected with black-leg or with scald or freezing injury are predisposed to slimy soft rot. In fact, slimy soft rot commonly follows freezing injury if the temperature after thawing is high enough to permit the growth of the bacteria.

The control measures are careful sorting of stock which has been subjected to unfavorable conditions before it is put into transit or storage, and improvement of handling methods to avoid wounds and scald. The rot progresses and spreads very rapidly in transit and storage, but can be checked by rapid drying of affected stock.

POTATO: SURFACE MOLD.

Cause: Various fungi.

Under transit or storage conditions, potatoes often show a superficial development of mold growth which is absolutely non-injurious to the tubers except as it affects their appearance. Various fungi are concerned, including blue mold and *Rhizoctonia*, and their growth is favored by the presence of moisture, especially the juice from nearby frozen or rotted tubers. Wet, dirty tubers and cut or bruised areas are likely to become covered with surface molds.

Surface molds are objectionable in bulk shipments because of the bad appearance of the stock.

Proper ventilation to dry the surfaces of the potatoes will aid in preventing this trouble.

POTATO: HOLLOW HEART.

Cause: Rapid growth.

Hollow heart is a more or less irregular cavity in the center of the tuber, varying in size and often lined by tissue which is browned and glassy.

Hollow heart occurs most frequently in large, coarse, rapid-growing varieties, particularly when these are grown in very fertile soils. It also is common in red varieties grown in the sand hills region of Nebraska.

Hollow heart and black heart may be confused since cavities occasionally develop in tubers affected with black heart; but these differ from those of hollow heart by their association with discolored tissue.

Usually hollow heart cannot be detected until the affected potatoes are cut. It does not affect their keeping quality.

Affected stock is very undesirable for table use.

Ref. (49).

POTATO: GROWTH CRACKS; SECOND GROWTH.

Cause: Unfavorable growing conditions.

Second growth and misshapen tubers are marked by a development of knobs on the tuber's surface, and growth cracks by wide, deep fissures, which are usually covered with the normal protective cork layer of the tuber.

These malformations occur in all varieties and in all localities, but are most pronounced where there is considerable fluctuation in the water supply of the growing crop.

Second growth tissues are immature, and are subject to peeling, bruising, and freezing injury. These knobs are often broken off, opening a way for *Fusarium* infection.

Growth cracks ordinarily are not subject to tuber rot infection, whereas mechanical injury such as cuts, splits, and bruises, which are due to careless handling, do not develop the normal layer of cork, hence become infected very readily.

Tubers showing these deformities are of inferior grade, since a considerable waste in paring is necessitated.

POTATO: IMMATURITÝ.

Cause: Premature death of vines in the field, or premature digging of the crop.

Immaturity is characterized by excessive peeling of the skin, by a greenish tint under the skin, and by a tendency to excessive wilting and flabbiness during transit and storage.

This trouble occurs mainly in late varieties of the North and in southern potatoes dug for early marketing.

Immature tubers are subject to bruising, therefore to *Fusarium* tuber rot infection, to freezing injury, and to excessive shrinkage if they are shipped in overheated cars. They are not high grade stock and often are bitter in taste.

POTATO: NET NECROSIS AND INTERNAL BROWN SPOT.

Cause: Unfavorable field conditions (possibly mosaic disease).

Net necrosis is characterized by fine, extensive brown streaks which follow the vascular tissues and form a network. The discoloration begins at the stem end, where it is often localized; or, in later stages, it may extend throughout the whole tuber. The discolored tissues are dead.

It occurs in many varieties, and is favored by poor soil conditions and by hot weather.

Internal brown spot is marked by brown or yellow regions of dead tissues scattered throughout the tuber. The affected regions may be small or large, even one-half inch in diameter. They may be scarce or numerous, and are isolated and not connected as in net necrosis.

The disease occurs in all varieties, and is favored by an inadequate water supply during the growing period.

Neither net necrosis nor internal brown spot should be confused with freezing injury, which sometimes produces very similar symptoms, but usually is otherwise distinguishable.

These internal discolorations are not decay and do not impair the food value of the tuber, but affected portions usually are rejected in the preparation of potatoes for the table.

Ref. (49); (50).

POTATO: FREEZING INJURY.

Cause: Exposure to low temperatures in the soil or during harvest, transit, or storage.

The symptoms of freezing injury are varied and complex and may be general, appearing in all the tissues of a tuber, or local and restricted to sharply limited regions of the tuber. They depend upon the variety, maturity, and individuality of the potato affected, upon the varying predisposition of the several tissues of the tuber to freezing injury, upon the temperature to which the tuber was exposed, and upon the rate of fall of the temperature as well as the duration of the exposure. The symptoms also depend upon the interval between the time of injury and examination of the affected tuber, and may or may not depend upon the rate of thawing and the conditions under which this takes place.

Thoroughly frozen potato tissue no longer possesses the natural crispness or brittleness of the sound potato. Frozen tissue looks dull and does not cut readily nor with snap. This is due to the formation of ice in the tissues. In the freezing process, the cell water passes out of the cells into the intercellular spaces, the spaces between the cells, where it forms ice crystals. The extent to which the cell water leaves the cells depends upon the point to which the temperature is lowered, upon the rate at which this takes place, upon the duration of the exposure, and upon the nature of the potato.

The symptoms presented by thawed potato tissues are very perplexing. Sometimes in case of severe freezing, the tissues become turgid, blister-like, and swollen, and the skin may be discolored. At other times they are quite firm and remain intact and, contrasted with healthy tissue, merely appear dull and colorless. If only a portion of a tuber was severely frozen, usually a purplish band marks the border of the uninjured tissue. When such a tuber is cut, the border between healthy and injured tissue generally is marked by a sharp purplish to dark brown line. However, this line is not always present.

If no infection sets in, the more or less watery thawed tissue dries down to the consistency of a rather moist mealy mass or a shrunken, dull, grayish, very tough and leathery granular mass which is composed of shrunken cells and starch. Very frequently bluish to black colors develop. In

this stage the dried-out starchy remnants of the tissue are separated from the uninjured tissue by a dark brown corky layer.

Very frequently the starchy or corky layer is not formed before infection with *Fusarium* takes place, or it is subsequently broken and admits the fungus. Severe freezing injury generally affects all tissues straight across the tuber, while *Fusarium* rot develops most rapidly in the pith of the tuber, leaving a shell of cortical tissue. This characteristic sometimes is of service in differentiating freezing injury and *Fusarium* tuber rot.

Thawed tissue, especially when in a warm damp atmosphere, is predisposed to slimy soft rot. In fact, most severely frozen potato tissue is dead and sooner or later becomes infected with the bacteria which cause this rot. The bacteria get into the watery tissue, which is an excellent growing medium for them, through breaks in the skin, and cause a slimy, mushy, or soupy rot which has a very offensive odor. Frequently the much wrinkled skin of such tubers bursts, and the neighboring potatoes are smeared with the slimy disintegrated tissue which dries out grayish and chalky when exposed to the air.

It is very difficult at times to differentiate freezing injury from injury due to scalding. This is especially true when the first or last freezes of the season occur.

The severe general type of freezing injury is marked by a killing of all the cells of the potato. The less severe type, popularly known as chilling, is definitely localized and restricted to the most susceptible tissues. It is known as freezing necrosis. It appears first in the stem end, and at times only the conducting tissues are killed and appear yellowish-brown to black. Very often the conducting tissues in the pith also are killed and appear as a net resembling net necrosis. The storage tissues also may be affected, either in extensive regions, or in small pockets. Starchy tissues so affected are grayish to steel blue, even black in color, granular, and very tough and leathery.

Sometimes the thawing tissues are pinkish to red in color when freshly cut. These colors change quite rapidly in the air, becoming gray, brown, or dirty red. These colors should not be confused with the pinkish color of the vascular ring or pith tissue of some of the red varieties of potatoes. At times white potatoes which have not been exposed to low temperatures also have a pink color. Potato tissues when freshly bruised and exposed to the air often are reddish in color.

Potatoes subjected to freezing temperatures may show none of the symptoms enumerated above but only a tendency to excessive wilting and flabbiness. The cut tissue may appear glassy and abnormally moist. When cooked they may turn black in spots or may have a sweet taste. This

condition also is frequently referred to as chilling injury. When potatoes that have become sweet following exposure to low temperatures are kept at normal temperatures the sweet taste disappears. This is due to the fact that the increased respiration due to rise in temperature oxidizes the excess of sugars which accumulated at temperatures of 29° to 34° F. At these temperatures the rate of respiration is cut down more than the rate of digestion of starch into sugar and consequently the sugars accumulate.

It is very difficult at times to differentiate immature tubers from those showing the less severe types of freezing injury. In both cases the tissues may have a greenish yellow color, and may be flabby and bitter in taste. Immature tubers are also bruised easily by impact or pressure, which causes regions in the tuber to resemble chilled tissue so closely that a diagnosis is exceedingly difficult. It also is exceedingly difficult to differentiate between thawed, dried-out tissues and bruised tissues.

It seems in fact that the presence of ice in the tissues is the only positive proof of freezing injury. All other characteristics enumerated as symptoms of freezing and chilling injury can also be brought about by other causes.

Slightly affected stock without extensive, discolored, killed regions is suitable for food even though it is not as palatable as the uninjured. It shrinks excessively in transit and storage.

Guarding against the exposure of potatoes to temperatures under 32° F. will prevent this type of injury. Losses during transit may be avoided by careful sorting to eliminate field frozen tubers previous to shipment, and the use of all proper precautions to prevent freezing injury during hauling, loading, and rail shipment. This involves proper lining and preheating of the car, installation of false flooring and ends, and an adequate heating apparatus regulated during transit so as to avoid both freezing and black heart injury.

Ref. (33a).

POTATO: BLACK HEART.

Cause: High temperature or insufficient aeration.

The symptoms of black heart vary, depending upon whether the potatoes are exposed to high temperatures with an adequate air supply, or to high or normal temperatures with an insufficient air supply. In the former case, no external symptoms develop; in the latter, both external and internal symptoms appear.

The external symptoms of black heart are moist areas on the surface which may be purplish at first but turn brown and black in a short time. The internal symptoms are a dark gray to purplish discoloration which later becomes jet-black. The discolored areas are usually sharply set off from the healthy tissue.

Generally the discoloration is restricted to the heart of the tuber, but frequently it radiates to the exterior as well. It may also appear on one side of a potato if this was exposed to a stove in a railroad car or in the storage house. The discolored regions may appear in zones in the peripheral parts and may be absent or less evident in the center. In advanced stages, the affected tissue dries out and forms cavities. In case cavities develop, these may be differentiated from true hollow heart by their black lining of killed tissue.

Exposure of potatoes in the field or in storage or transit for a day to temperatures of 90° to 104° F. causes this injury. Potatoes lying in a very hot soil or lying on the soil after digging may show it. More generally the injury is caused in stove-heated cars or by storing potatoes at 45° to 50° F. in piles deeper than 6 feet. In stove-heated cars, the injury usually is most severe near the stove and at the top of the load.

Black heart injury predisposes affected tissues to slimy soft rot. The heated, killed and watery tissues offer excellent growing conditions for bacteria.

Black heart does not impair the food value of non-affected tissues.

Prevention of this injury in cars involves proper ventilation, protection of the load near the stove with tin sheeting, and careful attention to the fire. The temperature should never go over 60° or 70° F.

The injury can be prevented in storage places by providing proper ventilation and by storing tubers in piles not deeper than 6 feet.

Ref. (1); (68).

POTATO: SCALD.

Cause: Exposure to the hot sun.

Scald on potatoes may appear first as large blisters which soon become sharply depressed or as slightly bleached areas with a very irregular and lobed margin of a darker metallic color. Upon cutting such a tuber, it is found that a shallow surface layer of tissue has been killed. This killed tissue is dull gray in color, and is separated from the uninjured tissue by a brown and very irregular line. The killed tissue, if not infected, dries out and becomes tough and leathery.

In many cases, more of the tuber is involved, both in surface area and depth of the killed tissue. This type has been tentatively named "deep scald," and shows much more discoloration than the ordinary scald. Bluish black patches often surround the lenticels, and the killed tissue darkens very rapidly when cut surfaces are exposed to the air.

Scald differs from late blight tuber rot in that the affected tissue is not brown in color, and in the sharp line of demarcation between the dead and the living tissue. Scald differs from freezing injury in its much more irregular surface out-

line and in the more irregular plane of separation between killed and sound tissue. At certain seasons, it may be very difficult to make this differentiation positive without rather careful inspection of car lots. Freezing injury, however, should be accompanied by other unmistakable symptoms.

Scald of potato is probably caused by exposure of newly dug tubers to the hot sun in the field.

Scald is of the highest importance from the market point of view. The killed tissue is very subject to the attacks of rot-producing organisms such as *Fusarium* and especially the bacteria of slimy soft rot. In the Chicago market in the summer of 1918, there were enormous losses due to slimy soft rot following scald in shipments of potatoes from the South and the entire Mississippi Valley.

It is not advisable to ship potatoes showing scald because of their extreme predisposition to slimy soft rot during transit.

POTATO: SALT INJURY.

Cause: Contact of potatoes with the floor or walls of a car in which fertilizers, salt, or salted hides have been shipped.

Usually the side of the potato in contact with the floor or wall is flattened, and the injured tissue is soft and flabby and at times semi-watery. At times the injured tissue is firm and leathery. Before the air has free access to the tissue it usually is colorless. After a short exposure to the air if not infected the injured tissues become brown to black, and begin to dry out. The exterior of the injured region generally is black. Frequently the injured tissue has a distinct salty taste, and salt injury may be thus diagnosed.

Tubers affected with salt injury are predisposed to slimy soft rot, and generally become infected and soupy if not removed promptly from the car.

POTATO: SUNBURN.

Cause: Exposure of tubers to light, either in the field, or during harvest, shipment and storage.

Sunburn is marked by a green discoloration of the surface layers, and in severe cases, of the interior portions of the tuber.

Sunburn occurs in potatoes of all varieties upon prolonged exposure to the light. It is common in eastern stock shipped in midsummer.

Affected stock is bitter and unfit for food.

POTATO: BRUISES, CUTS, AND SPLITS.

Cause: Mechanical injury.

Bruises may be superficial or internal. The most common type of bruise occurs in immature potatoes whose tissues are very tender and crisp, and consequently are easily broken either by pressure or by friction. Often an immature

tuber is gouged by rubbing against the container or by being walked upon. In either case, the result is a loss of water by the injured cells, leaving a solid, grayish, starchy deposit. At times this deposit is black, and externally the bruised areas often appear black. These types of bruising are very common in potatoes grown under irrigation. The injury due to gouging is more severe than peeling or feathering though it usually is due to the same causes.

Often potatoes are bruised or crushed by impact or by the weight of the load. In addition to the injuries above described, impact or pressure may cause symptoms which may be wholly internal or may be indicated externally only by small, slightly sunken spots. At times these internal injuries resemble those due to freezing and consist of small pockets of grayish, starchy masses, the remnants of ruptured, dried-out cells.

Most of the cuts in potatoes are caused by digging implements, and by the pernicious custom of handling potatoes with steel shovels or forks. Cuts may be broad and shallow or very deep.

If the cut or bruised tissue has an opportunity to dry out rapidly the injury may be sealed by a grayish granular deposit of starch, and later by a brown layer of cork. Very often, however, especially under moist conditions, *Fusarium* gets a foothold before the bruised or cut tissue is healed. It may also enter through subsequent breaks in the dry starch layer. In very hot weather, especially in southern potatoes, slimy soft rot gets a start in bruises and cuts. Shallow, broad, or smooth cuts are less subject to infection than those which are deep and ragged.

Potatoes often are fractured by pressure or by impact. Careless throwing about of potatoes, heavy loading, walking upon potatoes, and similar rough treatment, are responsible for these fractures or so-called splits. These may be visible externally or they may be wholly internal. In either case the surfaces of the fracture are lined with a deposit of starch or a brown layer of corky tissue. At times freshly dug potatoes develop splits if they dry out too rapidly.

Bruised, cut, or split potatoes are suitable for food, though the injuries necessitate very appreciable losses in paring. Their presence, however, lowers the market value of a shipment.

These mechanical injuries generally are ports of entry for fungi and bacteria which cause tuber rots. In most cases of *Fusarium* tuber rot, the infection starts in such injuries. In the early part of the shipping or storage period, it is impossible without a cultural test to determine whether a mechanical injury will remain merely an injury or develop a tuber rot. However, as the season progresses, the rot symptoms become more pronounced.

This type of injury with its frequently disastrous consequences could be prevented if it were constantly borne in

mind that potatoes consist of living cells and should not be handled like stones.

POTATO: ENLARGED LENTICELS.

Cause: Excessive water supply during the growing season.

This is marked by flat, pimple-like swellings, which occur at the lenticels of tubers. These swellings are formed by proliferation of cells lining the cavity of lenticels. Normally these cells remain dormant but they may enlarge and divide if potatoes are grown in very wet soil or if the soil becomes waterlogged after the tubers are formed. If this process continues long enough a mass of cells protruding above the tuber surface is formed.

At first these cells are quite soft, whitish in color, and quite easily rubbed off. Later they harden and become brown and corky.

Potatoes showing enlarged lenticels are objectionable because of bad appearance of the stock. Their food value is not impaired, since the swellings are superficial.

POTATO: FLEA-BEETLE DISEASE.

Cause: The larva of the flea beetle (*Epitrix cucumeris* Harr.).

In the simplest cases flea-beetle injury is marked by corky pimples or raised places on the tuber. At times the surface of such pimples is slightly broken. In the more complex and advanced stages the surface of the tuber is a mass of pimples and of channels or furrows formed by breaking of the skin. The margins of such furrows and channels frequently are elevated and swollen so that the whole furrow stands above the surface of the tuber. This type of injury frequently is known as worm tracks.

By cutting into the pimples or furrows brown, tough splinters of corky tissue are found extending perpendicularly into the tuber tissue. Sometimes these splinters are a fourth of an inch in length. Their presence serves to differentiate the simple, pimple stage of flea-beetle injury from slight nematode injury, and the furrowed type from other tuber diseases such as scab, grub injury, or wire-worm injury.

Flea-beetle injury is most frequent in Colorado potatoes. It injures the appearance of potatoes and generally necessitates very deep paring.

POTATO: NEMATODE DISEASE.

(See Nematode Disease).

RADISH: BLACK ROOT.

Cause: A fungus (*Rheosporangium aphanidermatum*).

This disease of radish roots is characterized by blackened regions of varying size which may be rather superficial, or may extend deeply into the root. The darkened tissue is

firm and not disintegrated. The lesions are often cracked or fissured on the surface as a result of root growth, and may be accompanied by some disfiguration of the root due to checked growth.

This disease is of widespread occurrence. Both red and white varieties are susceptible. The fungus persists in the soil and often causes damping-off of seedlings.

Badly affected roots often are found in the market, but are not considered edible, and their presence lowers the value of the bunch.

Control involves crop rotation or soil sterilization. Badly affected roots should not be sent to market.

Ref. (16).

RADISH: MISCELLANEOUS DISEASES.

DOWNY MILDEW.

Cause: A fungus (*Peronospora parasitica*).

Downy mildew causes angular spots on the leaves, bounded by the larger veins. These spots are yellow in color when viewed from above, and the under surface is covered with a white felt-like outgrowth of mold.

This disease often is found in the market but is of no importance since the root is not attacked.

WHITE RUST.

Cause: A fungus (*Cystopus candidus*).

The symptoms of this disease on the leaves are small, irregular lesions bearing on the under surface smooth, pure white, porcelain-like cushions or blisters. These rupture, allowing the spores to escape, and the lesions are then recognizable by the torn edge of the empty pustule. Often these lesions cause marked distortion in the leaves and other affected parts.

Since this disease does not occur on the roots, it is of no importance in the market.

ROOT ROT.

Cause: A fungus (*Rhizoctonia*).

This disease is marked by a rather large brown circular lesion on the side of the root in which the tissue is rotted as in watery soft rot.

This has been found occasionally in the market.

NEMATODE DISEASE.

(See Nematode Disease).

RADISH; SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

RUTABAGA: DRY ROT.

(See Radish Root Rot).

RUTABAGA: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

RUTABAGA: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

RUTABAGA: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

RUTABAGA: NEMATODE DISEASE.

(See Nematode Disease).

SALSIFY: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot and Beet Watery Soft Rot).

SPINACH: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

SQUASH: RHIZOPUS ROT.

(See Rhizopus Rot).

SWEET POTATO: BLACK ROT.

Cause: A fungus (*Sphaeronema fimbriatum*).

Black rot is characterized by greenish to nearly black, circular, depressed spots, varying in size from $\frac{1}{2}$ to 2 inches in diameter. At times the spots are very irregular, occurring in bruises and injured places. The lesions are shallow, rarely penetrating to the heart of the root, and the affected tissue is quite firm and tough. Often at the center of the spots small, black bodies are developed, which appear at times as dense masses of small, black bristles. These are the fruiting bodies or pycnidia of the fungus. Wetting the surface of the lesion will bring out better these distinguishing characteristics.

Black rot is of common occurrence in all sweet potato regions. The roots become infected in the field, often through wounds. The diseased areas may enlarge in storage or transit, where overheating and lack of ventilation favor the progress of the disease. Generally, affected stock is culled out and is not sent to the markets.

Affected stock is unfit for table use since the diseased tissue is very bitter. When cooked, the entire root has a bitter flavor. Diseased sweet potatoes also are unsuitable for seed purposes, since black rot is spread by planting infected seed roots.

Ref. (24); (27); (70).

SWEET POTATO: SOIL ROT.

Cause: A slime mold (*Cystospora batata*).

The symptoms of this disease are small, circular, sunken spots about one-half inch in diameter in which the tissue

has dried out and cracked. The lesions may appear as circular pits or depressions from which the diseased tissue has fallen out. There may be some constriction of the root where such lesions are located.

Infection occurs in the field and the disease does not progress in storage.

The economic importance of soil rot is due to the fact that it reduces the yield and disfigures the roots.

Ref. (69).

SWEET POTATO: SCURF.

Cause: A fungus (*Monilochaetes infuscans*).

Scurf is marked by small, circular, dark clay-colored spots on the skin of the root, which may unite to form large blotches. The spots are only skin-deep. In advanced stages, the skin turns to a deep brown color and becomes wrinkled and rough.

Scurf occurs in practically all sweet potato growing regions.

Infection takes place in the field, but the spots may develop in storage. The disease does not spread from one root to another. The market value of diseased stock is slightly reduced because of its unsightliness.

Ref. (24); (27).

SWEET POTATO: SOFT ROT.

Cause: A fungus (*Rhizopus nigricans*).

This rot is one of those elsewhere described as *Rhizopus* rot. Soft rot develops very rapidly, and unless checked, soon involves the whole root. In the early stages, the affected tissue is soft, watery, and stringy, and yields a brownish-yellow liquid when compressed. The early stage is also attended by an odor of vinegar or acetic acid. Under warm, moist conditions, the characteristic mycelium and sporangia may appear on the outside of the root. The affected tissue eventually becomes dry, shrunken, and yellowish brown. At this stage an aromatic odor may be detected.

Infection, which takes place through wounds, occurs either in the field, in transit, or in storage and is favored by high temperature and humidity. The rot progresses and spreads very rapidly in transit and storage. Infection may set in at the end or the middle of the root. In the former case, it is called soft rot; in the latter, if encircling the root, ring rot.

Control consists of careful handling of the roots to avoid wounds, careful sorting, proper curing, and storage in a dry atmosphere at a temperature of 50° F. When wet weather prevails during harvest, the shipment of roots not properly dried often results in severe losses. An additional control measure is the disinfection of storage houses and cars with copper sulphate or formaldehyde.

Ref. (24); (27).

SWEET POTATO: STEM ROT.

Cause: Fungi (*Fusarium batatatis* and *F. hyperoxy-sporum*).

In stem rot the end of the root is light and shriveled, and the interior has a deep brown color and a peculiar cinnamon odor. Usually, however, stem rot is not plainly visible externally, and the disease manifests itself merely by a discolored ring, and at times by a secondary ring, visible in transverse sections of the root. If the outer part of the root is carefully removed, the discolored parts appear as a network of brown fibers surrounding the heart.

Stem rot is of widespread occurrence in all sweet potato districts.

This disease causes a wilt of vines in the field, and reduces the yield. Infection of the roots takes place in the field, and the disease progresses only slightly in storage. Stem rot is found infrequently in the market, and is described here principally to avoid confusion with freezing injury, which it somewhat resembles.

Ref. (24).

SWEET POTATO: MISCELLANEOUS DISEASES.**DRY ROT.**

Cause: Fungi (*Fusarium* species).

This disease is comparable to the *Fusarium* tuber rot of the Irish potato. It is marked by shrunken areas showing discolored flesh, and usually starts at one end of the root. Dry rot is not common or of much importance.

FOOT ROT.

Cause: A fungus (*Plenodomus destruens*).

This is a storage rot of some importance. It follows a blighting of plants in the field. It is a firm brown rot with pimple-like protuberances on the surface. Foot rot may enter roots through wounds and bruises and thus cause some loss in storage.

Ref. (27).

NEMATODE DISEASE.

(See Nematode Disease).

TOMATO: ANTHRACNOSE.

Cause: A fungus (*Colletotrichum phomoides*).

In its early stages, anthracnose is characterized by small, circular, sunken spots of the same color as the sound tissue. These lesions enlarge rapidly, become watersoaked and more sunken, and bear pinkish spore heaps which often coalesce to form a slimy layer. These spore heaps or acervuli turn black at times and resemble the pycnidia of *Phoma*, so that anthracnose may be mistaken for *Phoma* lesions. However, the spore heaps never are as definitely rounded as

the pycnidia, and the pycnidia never are pink. The centers of anthracnose lesions are at first light brown, and do not become as black and charry as those of *Phoma* rot.

Anthracnose is common on northern-grown tomatoes, especially New Jersey stock. Green fruit is rarely affected.

The original infection takes place in the field. The disease develops in the field, and may also spread and develop in transit and storage. Often this fungus destroys the entire fruit.

It is advisable not to pack or ship infected fruit because it rots rapidly and is a menace to healthy fruit.

Ref. (30).

TOMATO: BUCKEYE ROT.

Cause: A fungus (*Phytophthora terrestris*).

No better description of this disease can be given, so far as the appearance of the fruit on the vine or in transit is concerned, than is implied by the name "buckeye." In color and in its surface appearance, the diseased tissue resembles a horse chestnut very closely. The color is grayish to brown, and the surface is uneven, being sunken in places. The rot at first is firm and even hard. The border is irregular and not sharply defined, and often merges into water-soaked, slightly bleached, green tissue. At times there appear widely separated concentric rings of more or less irregular wavy outline. These rings, however, are by no means a constant accompaniment of the disease. Under moist conditions the lesions generally are covered by a white, fluffy growth of mold.

In case concentric rings are found in buckeye rot, they are farther apart and less regular in outline than in soil rot. In soil rot, the mycelium is brown and may produce sclerotia, while the mycelium of buckeye rot is white and produces no sclerotia. The absence of white, glistening or black sporangia differentiates buckeye rot from *Rhizopus* rot.

This disease occurs most commonly in southern tomatoes during rainy weather. The original infection takes place in the field, and occurs only on fruit which has been in contact with the soil or close enough to it to be spattered during rains.

As in the case of soil rot, buckeye rot can be controlled in the field by staking the vines to keep them off the ground. At harvest it is advisable to sort out diseased fruit because it is unsuitable for food and is a menace to healthy stock since the fungus passes through the wrappers from diseased to healthy fruit.

Ref. (56); (59).

TOMATO: NAIL-HEAD SPOT.

Cause: A fungus (*Alternaria solani*).

In the early as well as the later stages, nail-head spot is marked by small, dry, slightly though abruptly sunken scab-like spots which are brown to black in color.

These spots or lesions generally are mere blemishes, and cause little damage. The lesions may be circular or elliptical and at times may coalesce to form a large, irregularly shaped area. At times the spots on ripening fruits are surrounded by a green zone due to delayed ripening of the bordering tissue. The tissue underlying the spot is not affected, and the diseased tissue can easily be removed from the sound tissue underneath. As the lesion ages, it often cracks and opens the way for the entrance of secondary rots such as *Phoma*.

The small size and the slightly sunken, flat and scab-like nature of the lesions of nail-head differentiate these from the lesions of other tomato fruit diseases, most of which, in contrast, are large or water-soaked, often deeply sunken and involve more rotting of the tissue.

Nail-head spot is a warm-weather disease, and is prevalent on fruit from the Gulf States. It is a serious field disease, attacking leaves, stems and fruits, and is known by the growers as early blight. The fruit is infected before it leaves the field, and the spots are visible when the fruit is packed. The spots may enlarge slowly in transit and storage.

Spraying will control the disease in the field. In packing it is advisable to sort out spotted fruit because its presence lowers the market value of a shipment and affected fruits are predisposed to rots.

Ref. (56); (60).

TOMATO: PHOMA ROT.

Cause: A fungus (*Phoma destructiva*).

In its early stages, *Phoma* rot is marked on the green fruit by minute, slightly sunken circular spots with a brown or black border and a lighter center. Later the center of the spot becomes covered with black pimple-like bodies. These are known as pycnidia (singular pycnidium). In the early stages, spots on ripe fruit are water-soaked and concave. Later, as they enlarge, the centers become brown to charry black, leathery and firm, and covered with pycnidia. The borders are slightly sunken and water-soaked. The lesions increase indefinitely and quite rapidly in size but remain more or less circular and concave.

The lesions generally occur at the stem end of the tomato, and the disease is often known as stem-end rot. The spots, however, are not confined to this part of the fruit, as the fungus may attack the fruit wherever it is bruised or injured. The fungus may also enter through nailhead spots. Until the lesion becomes very large, the underlying brown or black tissue is quite firm.

Without a microscope it is quite difficult at times to differentiate the various rots of mature tomato fruits caused by *Phoma*, *Colletotrichum*, and other fungi. This is especially true when secondary rots have set in and covered up or confused the typical clear-cut symptoms.

It appears, however, that anthracnose and other fruit rots are more common on over-ripe fruit, and occur in southern stock later in the season than does the *Phoma* rot. The slimy, orange-pink spore masses of anthracnose aid in differentiating it from *Phoma* rot. In *Phoma* rot, slimy spore masses appear very frequently at the openings of the pycnidia, but these are whitish rather than pink in color.

This disease is most marked in southern winter-grown tomatoes; that is, the Cuba, Florida, and California crops. It was exceedingly destructive to Cuban tomatoes during the winter of 1917-18, and to Florida tomatoes during April and May of the same season.

The disease occurs on the vines, where it is known as *Phoma* blight or black spot. It is not known definitely whether the original infection of the fruit destroyed in transit occurs in the field or in the packing house. The disease develops in transit and in the ripening rooms, and is favored by a warm, moist atmosphere.

Affected fruit is subject to infection with bacteria and other fungi.

No satisfactory control of the disease in transit and in the ripening rooms has been worked out.

Ref. (32); (56); (60).

TOMATO: SOIL ROT.

Cause: A fungus (*Rhizoctonia*).

In its early stages, soil rot on green or ripe fruit is characterized by firm, brown spots which may be marked with concentric rings. Later they increase in size and generally a brown, firm mat of mold covers the older lesions. On mature fruit in the market, soil rot lesions are large, brown, water-soaked areas not sunken and not concentrically marked.

Soil rot progresses and spreads very rapidly in transit and storage, the fungus passing from one fruit to another through the wrappers. The most striking characteristic under these circumstances is the plentiful development of a coarse, white, and later, brown mycelium with small brown sclerotia. The growth of the hyphae is of a peculiar radiating type, resembling densely crowded spokes of a wheel, and is often further characterized by concentric zones. Very often the mycelium, the sclerotia, and the paper wrapper form a thick, dry and hard mat, or the hyphae may hold the wrapper quite firmly to the fruit so that shreds of it remain when the fruit is unwrapped. Examination of decayed fruit in the market often reveals the original soil rot lesion with its firm, brown and concentrically marked center surrounded

by water-soaked but firm tissue. Later there appears the coarse white mycelium which turns brown and forms sclerotia. The water-soaked areas are often referred to by the trade as "water blisters."

The fact that this rot is not limited to the blossom-end of the fruit, and is accompanied by a mycelium, differentiates it from blossom-end rot. It may follow blossom-end rot, however. Its solid mat of brown mycelium and its large lesions differentiate it from nail-head spot. The brown mat of fungus and the more regular and more closely arranged markings also differentiate soil rot from the buckeye rot. Furthermore, in the field, soil rot always shows these concentric rings while buckeye rot may not. Soil rot differs from *Phoma* rot by the absence of a black center with black pycnidia and from anthracnose by the absence of the orange-pink spore heaps.

Soil rot is especially severe on Florida tomatoes during rainy weather. It also occurs in early shipments of California tomatoes.

The original infection takes place from the soil in the field. Fruits touching the ground or hanging low enough to be spattered by the soil during rains are infected. The rot develops and spreads in the pack.

This disease can be controlled in the field by staking the plants. It is advisable to sort out diseased fruit in the packing house, since affected fruit is unmarketable, unfit for food, and a menace to sound fruit.

Ref. (56).

TOMATO: BLOSSOM-END ROT.

Cause: Non-parasitic (probably irregular water supply).

In the early stages this disease appears as a small spot at the blossom-end, or it may involve the whole blossom surface of the fruit. At this stage the spot looks like a bruise, is dark green in color and water-soaked. Later it becomes darker, and the affected tissue collapses and becomes firm and leathery. Sometimes the collapse is so sharply marked that the lesion appears as a distinct depression. Generally, however, it results only in a flattening of the end of the fruit. The lesions are very shallow and dry; this, together with the restriction of this injury to the blossom-end, serves to differentiate blossom-end rot from the buckeye rot of green tomatoes and the soil rot of both green and ripe fruits with which it might be confused.

Blossom-end rot is a field disease, and occurs in all tomato growing districts.

Affected tissue is predisposed to secondary rots, which develop in the field, in transit, and in storage. The non-parasitic injury is, in fact, commonly followed by fungous rots.

Blossom-end rot has not been successfully controlled. Regulation of the water supply, and the use of varieties

suiting to the soil and climate, are important points to consider.

It is not advisable to put badly affected fruit on the market. Slightly affected fruit, if not infected by fungi, can be used for food.

Ref. (3).

TOMATO: MOTTLING.

Cause: Unknown, probably mosaic.

This disease is characterized by a more or less extensive brown discoloration of the surface of the fruit in which the affected areas show great diversity of pattern, such as circular spots, blotches, loops, and streaks. The affected areas are somewhat sunken and on green fruits the brown color may be absent. The affected tissue is firm and hard. The entire thickness of the wall and even portions of the septa (partitions) are involved. It seems that portions at least of the affected tissues are dead. At any rate, diseased fruits will not ripen properly in the ripening rooms, and the browned or sunken areas commonly become infected with various fungi. The disease resembles in some ways the mosaic disease of cucumbers.

In shipments of California tomatoes received in Chicago during the fall of 1918, this disease was undoubtedly the most important loss-producing factor, the loss amounting in some cases to 50 per cent or more.

No control is known. Since the symptoms are evident on the green fruits, it is advisable to sort out affected stock before shipment, as it is a total loss at destination.

TOMATO: BLACK ROT.

Cause: A fungus (*Alternaria*).

The most striking symptom of this disease is the conspicuous, black velvety growth of the fungus on the lesions. The disease generally occurs on ripe fruit, where it causes a slowly progressing rot. The fungus probably gains entry through wounds and growth cracks.

TOMATO: CATFACE.

Cause: Unknown, probably varietal.

In this disease the blossom end of the fruit is badly misshapen or puckered, probably due to the imperfect development of an area which at maturity is represented by an irregular leathery scar. Occasionally secondary rots occur in the malformed areas. The presence of catface in shipments lowers their market value.

TOMATO: GROWTH CRACKS.

Cause: Probably arrested and resumed growth.

These are rather deep ruptures or cracks radiating from the stem end. Occasionally they are arranged concentric-

ally about the stem end. They seldom, if ever, occur in the blossom half of the fruit.

These cracks are actual wounds. Either before or after the fruit leaves the field, they are very likely to become infected with rot-producing organisms. From the market point of view, growth cracks assume a very great importance because rots so frequently start in them.

TOMATO: RING ROT.

Cause: A fungus (*Melanconium*).

A large, circular, flattened lesion, very conspicuous because of its broad, concentric rings, is characteristic of this disease. The background color is brown and the center and the margin may be slightly elevated. Under moist conditions acervuli (singular acervulus), the fruiting bodies of the fungus, appear. The spores which exude from them frequently germinate and the resultant white mycelial growth covers the center of the lesions. The rotted tissue is firm, but affected fruits are worthless. Ring rot has been found to a limited extent in Cuban tomatoes.

Ref. (72a).

TOMATO: FUSARIUM ROT.

Cause: A fungus (*Fusarium*).

The differentiating symptom of this disease is a heavy, compact, pinkish-white growth of the fungus on the lesions. The advancing edge of the lesions frequently is free from visible fungous growth and is water-soaked and shrunken. The fungus probably attacks the fruit through wounds.

TOMATO: RHIZOPUS ROT.

Cause: A fungus (*Rhizopus nigricans*).

The general features of this disease have been taken up under the discussion of *Rhizopus* rot. It is a very soft, leaky rot. Affected fruit has an acid odor and when ripe usually has a bleached appearance. This rot rarely is found on green fruit.

The fungus attacks the fruit through breaks in the skin. These may be due to careless handling and so minute as to be invisible to the unaided eye, or they may result from skin pricks, nail injury, or mashing in packing or transit. The disease spreads in transit, the fungus growing through the wrapper from diseased to healthy fruit.

Ref. (56).

TOMATO: FOUL-SMELLING ROT.

Cause: Bacteria and fungi.

The various tomato rots that are accompanied by foul odors have not been studied sufficiently to be differentiated and named.

TOMATO: SUN-SCALD.

(See Sun-scald).

TURNIP: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

TURNIP: SLIMY SOFT ROT; BACTERIAL ROT; SOFT ROT.

(See Slimy Soft Rot).

WATERMELON: ANTHRACNOSE.

Cause: A fungus (*Colletotrichum lagenarium*).

In its early stages, anthracnose is characterized by small circular raised welts on the rind, dark green in color. As these increase in size, the centers turn brown, become sunken, and under moist conditions the pink acervuli of the fungus are formed. As a lesion enlarges, the center becomes more sunken, often turns black, and eventually the surface cracks, exposing the rotted tissue or a cavity underneath. The fungus causes a rather slowly advancing dry rot of the rind tissue, and finally penetrates the edible pulp. Under extremely dry, hot conditions, such as occur in California during the melon harvest, the lesions may remain in the blister or nail-head stage.

Anthrachnose is a serious disease of the vines and fruits in the field. The fungus is spread with the seed, overwinters in the soil, and spreads in the field during rainy weather. This is the same fungus which causes cucumber and muskmelon anthracnose.

Many fruits are rendered worthless in the field. The disease is also present in almost every car of melons shipped north as a result of field infection which may or may not have been detectable at the time of loading. The lesions enlarge during transit and not only seriously impair the appearance of the fruit, but open the way for the entrance of other rot-producing organisms.

Anthrachnose is prevalent throughout the southern melon regions and the Ohio Valley. It is the source of great losses in the field, in transit, and in the market.

The disease can be controlled in the field by crop rotation, use of disease-free seed, and spraying with Bordeaux mixture.

Ref. (58).

WATERMELON: STEM-END ROT.

Cause: A fungus (*Diplodia*).

This is a rapidly progressing soft rot of the stem end of the melon, marked externally by a brown discoloration and shrivelling followed by the development of abundant black pycnidia or a dark, gray, moldy outgrowth of the fungus. Whitish spore masses are often discharged from the pycnidia. The disease usually affects the stem-end of fruit, but occurs also at wounds and, in the field, as a blossom-end rot.

While the rot is rather common in the field, diseased fruits are not often shipped, and it seems quite likely that

infection in the case of the stem-end rot found in the market takes place through the end of the stem after it is cut. The rot then develops with extreme rapidity during transit.

This is the most important rot of watermelons, and is rather general in southern grown stock, causing very heavy losses in some cars.

Stem-end rot as it occurs in transit can be prevented by proper treatment of the melons at the time of loading. After a tier of melons is in place, the ends of the stems are cut off and a paste made of starch and copper sulphate is applied to the freshly cut surfaces. This prevents infection by the fungus.

Ref. (42); (51).

WATERMELON: MISCELLANEOUS DISEASES.

BLOSSOM-END ROT.

Cause: Unknown (not parasitic).

This disease appears as a flattened, dry, leathery, blackened spot about as big as a silver dollar, at the blossom-end of the fruit. The tissues underneath are sound unless the killed area becomes infected with some fungus such as the *Diplodia* of stem-end rot, which is a common secondary invader.

While the cause of this trouble has not been demonstrated, it is quite likely that it is due to an unbalanced water supply as in the similar disease of tomatoes. Although of minor importance in the market, it may open the way to rot-producing fungi.

Ref. (51).

CHEMICAL INJURY AND ABRASION.

Cause: Contact with the walls or floors of cars.

This injury consists of large brown sunken areas or gouged channels in the rind. It is most common in cars previously used for chemicals such as fertilizers, especially where some shifting of the load has occurred. Sometimes fungi gain entrance through these injuries, and cause decay.

Compared with stem-end rot, this trouble is not of common occurrence.

Ref. (51).

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