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VIRUS AND VIRUSLIKE DISEASES
OF STONE FRUITS IN UTAH

A HANDBOOK FOR THEIR
IDENTIFICATION AND CONTROL

B. L. Richards
and
L. C. Cochran

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Acknowledgements

Research upon which this publication is based has involved the inoculation and critical examination and study of thousands of trees in experimental plots and study orchards throughout the state. Numerous orchard plant surveys have likewise contributed. Many workers and various agencies through their cooperation have enlarged greatly the scope of the research program.

Special recognition is here given to DeLore Nichols, extension agricultural agent, Davis County, Utah, and to E. L. Reeves, pathologist, U. S. Department of Agriculture, for their active participation in initiation of the virus project. The State Road and Davis County Commissions played vital roles in providing land, water, and labor. Also, through the efforts of former Commissioner T. R. Welling the Utah State Department of Agriculture made valuable contributions.

With added greenhouse facilities and with new and extensive equipment in a well-organized virus laboratory at the Utah State Agricultural College, the work will go forward under the direction of George W. Cochran, Bryce N. Wadley, and George Kaloostian. Through the efforts of these men, much of the earlier work has been confirmed and new phases of the work are being initiated.

The authors are especially indebted to President Emeritus Elmer G. Peterson, President Emeritus Franklin S. Harris, and to Dr. R. H. Walker, director of the Division of Agricultural Sciences, for their encouragement and material aid.


High cost of printing makes a charge of $1.50 necessary for this handbook.
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VIRUS AND VIRUSLIKE DISEASES
OF STONE FRUITS IN UTAH

a handbook for their identification and control

B. L. Richards and L. C. Cochran

Introduction

Intensive research over the last 20 years has shown that many of the previously unexplained and seemingly mysterious disorders of stone fruit trees in Utah are infectious diseases caused by viruses. Others were shown to be the result of noninfectious genetic abnormalities or of adverse chemical and physical factors in the environment of the plant. The purpose of this handbook is to help orchardists and others recognize these diseases and disorders and understand better their etiology or cause. The handbook will also bring together basic information which will aid in their control.

Fungus and bacterial diseases destructive to stone fruits in more humid areas, particularly in eastern United States, are in general of comparatively minor significance in Utah. This fact has simplified the problem of stone fruit production in the state. On the other hand, in recent years there has been a growing recognition that for unknown reasons many stone fruit orchards have become prematurely unproductive and short-lived. Peach trees in large numbers frequently turn yellow and become misshapen and scraggily; their fruits ripen early, are undersized, and are of poor quality. Sweet and sour cherry trees of all ages frequently wilt and die early in the summer. Many others lose their normal green color and produce undersized, off-colored fruits which fail to mature. Such trees remain permanently unproductive.

In the past these puzzling conditions have been attributed to such factors as drought, winter injury, poor pollination, unfavorable soil conditions, insect injury, and to bacterial and fungus diseases. Yet, after all of these factors had been carefully evaluated, a wide range of disorders for which there was no apparent explanation remained.

Research studies in Utah subsequent to 1935 have clearly demon-

1Formerly professor of plant pathology and head of the Department of Botany and Plant Pathology, Utah State Agricultural College, and principal plant pathologist, Horticultural Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture, respectively.

2These investigations were initiated in 1939 as a Utah Agricultural Experiment Station project under the direction of the senior author. In 1940 cooperative relations were established between the Utah Agricultural Experiment Station and the U. S. Department of Agriculture, Bureau of Plant
strated that many of these disorders are the result of virus infection. Among these newly identified virus diseases are some of the most destructive maladies known to fruit trees.

Prior to 1930, only five virus diseases of stone fruits were recognized in North America; all were diseases of peach and were restricted in their distribution to eastern North America. The discovery of peach mosaic in 1930, later found to be widely distributed in many sections of southwestern United States, started a new era in fruit tree virus investigations. The U. S. Department of Agriculture Handbook No. 10, published in 1951 and entitled "Virus diseases and other disorders with viruslike symptoms of stone fruits in North America," listed 48 virus diseases. Forty-three of these have been discovered in Colorado, Utah, California, Idaho, Oregon, Washington, and British Columbia; 22 of which affect stone fruits in Utah. Some of these affect more than one fruit crop—7 in peach, 12 in sweet cherry, 9 in sour cherry, 1 in chokecherry, and 2 in plum and prune. Other abnormalities, of unknown cause, found to affect commercial, ornamental, and wild species of stone fruits in Utah may yet prove to be of a virus nature.

In presenting and evaluating the known facts regarding these recently discovered virus and viruslike diseases in Utah orchards, the authors have in mind growers, nurserymen, agricultural agents, and inspectors concerned with diagnosis and control. They hope that the information and illustrations presented will be of interest also to students and to others concerned with biological and economic aspects of virus problems.
General Characteristics of Viruses

During the early studies of infectious diseases, emphasis was placed primarily on two groups of causal agents—bacteria and fungi. Members of these could be seen through the light microscope and in general could be isolated and grown in culture disassociated from the host in which they induced disease. Viruses, on the other hand, were overlooked because they are too small to be seen through the light microscope and do not reproduce except in the tissues of living hosts.

In 1892 a Russian scientist, Iwanowski, discovered that the substance responsible for tobacco mosaic was carried in the juice of diseased plants and could pass through porcelain filters, the pores of which were small enough to hold back the known bacteria. Iwanowski believed his filters were faulty. Four years later Beijerinck, a Dutch scientist, repeated the experiment, and because he could see no particles in the infectious liquid which came through the filters, he referred to the causal agent as a "contagious-living-fluid." More recently the term "virus" (literally meaning poison) has become generally accepted to designate this third big group of contagious-disease-producing agents.

In the early thirties of the present century, viruses were demonstrated to be made up of separate units or particles. In 1935, W. M. Stanley isolated from mosaic-diseased tobacco a protein which he crystallized and showed to be able to reproduce the disease in healthy tobacco plants. We know now that the crystals of this protein are the pure virus which causes tobacco mosaic; that they are composed of masses of rod-shaped particles which become cemented together as water is removed from between them. Subsequently, with the aid of the electron microscope, we have learned much about the size and shape of many viruses. Through chemical and other studies we have learned about their rate of growth and reproduction, their means of dissemination, and also more definitely about their true role as the cause of disease.

In the light of this more recent knowledge, and for all practical purposes, a virus may be regarded as an exceedingly minute obligate parasitic organism which grows and multiplies within, and at the expense of, living hosts, but which is too small to be seen through the ordinary light microscope. In many ways viruses resemble and react similarly to bacteria which induce infectious diseases. Kunkel (1947) described plant viruses as "the most efficient parasites we know anything about. While fungi clumsily bore from without in their attempt to anchor ill-fittinghaustoria, the viruses get themselves injected into cells by some of the finest and most efficient hypodermic syringes known to man, the proboscides of insects. If they get into trouble because some plant they try to invade is immune, they mutate to produce a new virus strain which can attack this plant. To obtain distribution most fungi have to depend on having their spores blown around. The plant viruses have wings to take them where
<table>
<thead>
<tr>
<th>Virus/organism</th>
<th>Diameter or width X length in μm</th>
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<tbody>
<tr>
<td>Red blood cells</td>
<td>7500</td>
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<tr>
<td>B. prodigiosus (Serratia marcescens)</td>
<td>750</td>
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<td>Rickettsia</td>
<td>475</td>
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<td>Psittacosis</td>
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<td>Canary pox</td>
<td>260 x 310</td>
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<td>Myxoma</td>
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<td>Vaccinia</td>
<td>210 x 260</td>
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<td>Pleuro-pneumonia organism</td>
<td>150</td>
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<td>Pseudo rabies</td>
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<td>Herpes simplex</td>
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<td>Rabies fixe</td>
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<td>Influenza</td>
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<td>Newcastle disease</td>
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<td>Vesicular stomatitis</td>
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<td>Staphylococcus bacteriophage</td>
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<td>Fowl Plague</td>
<td>90</td>
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<td>Mouse mammary carcinoma</td>
<td>80</td>
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<td>T2 coli bacteriophage</td>
<td>60 x 80</td>
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<td>Chicken tumor I</td>
<td>70</td>
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<td>Equine encephalomyelitis</td>
<td>50</td>
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<tr>
<td>T3 coli bacteriophage</td>
<td>45</td>
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<td>Rabbit papilloma (Shope)</td>
<td>44</td>
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<tr>
<td>Pneumonia virus of mice</td>
<td>40</td>
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<tr>
<td>Tobacco mosaic and strains</td>
<td>15 x 300</td>
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<tr>
<td>Cymbidium (orchid) mosaic</td>
<td>12 x 480</td>
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<tr>
<td>Gene (Muller’s est. of max. size)</td>
<td>20 x 125</td>
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<tr>
<td>Southern bean mosaic</td>
<td>31</td>
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<td>Rift valley fever</td>
<td>30</td>
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<tr>
<td>Tomato bushy stunt</td>
<td>30</td>
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<tr>
<td>Poliomyelitis (Lansing)</td>
<td>25</td>
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<tr>
<td>Hemocyanin molecule (Busycon)</td>
<td>22</td>
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<td>Yellow fever</td>
<td>22</td>
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<td>Turnip yellow mosaic</td>
<td>20</td>
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<td>Louping ill</td>
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<td>Tobacco ring spot</td>
<td>19</td>
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<td>Japanese B encephalitis</td>
<td>18</td>
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<td>Alfalfa mosaic</td>
<td>17</td>
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<td>Tobacco necrosis</td>
<td>16</td>
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<td>Foot-and-mouth disease</td>
<td>10</td>
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<tr>
<td>Hemoglobin molecule (Horse)</td>
<td>3 x 15</td>
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<tr>
<td>Egg albumin molecule</td>
<td>2.5 x 10</td>
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Fig. 1. Relative sizes and shapes of viruses as compared with red blood cells, bacterial cells, and one another. The micron (μ), the unit commonly used to measure bacteria, fungus spores, and other minute objects, is 1/1000 of a millimeter (mm). The millimeter equals approximately the diameter of an ordinary pin head or the width of the lead in a fine-line lead pencil. The millimicron (μμ), used to designate virus sizes, is 1/1000 of a micron, one-millionth of a millimeter, or 1/250,000,000 of an inch. It would take 100,000 particles of the poliomyelitis virus or of the virus producing foot-and-mouth disease of cattle placed side by side to equal a line long enough to reach across the head of an ordinary pin. (Revised in 1952 by W. M. Stanley from chart which appeared in Chemical studies on viruses, Chem. & Engin. News 25: 3786. 1947.) Courtesy the J. B. Lippincott Company, Publishers, and the National Foundation for Infantile Paralyses, Inc.
they wish to go, the wings that are
guided in their flight by the appetites
of insects.” A concept of the nature
of viruses is essential for the under­
standing of the diseases they induce.

Viruses as Disease-Producing
Agents

Viruses are among the oldest, most
contagious, and most deadly disease­
producing agents known to affect
man. They also induce diseases of
animals and plants and are even im­
portant as parasites of bacteria. A
recent textbook on virology lists 125
viruses affecting animals, 65 of which
affect man; approximately 300 or more
affect plants. In all, over 400 viruses
have been described and new ones
are continually being discovered and
reported.

Among the common virus diseases
induced in man are smallpox, yellow
fever, poliomyelitis, measles, mumps,
influenza, warts, cold sores, virus
pneumonia, and common colds; in
animals, hog cholera, foot-and-mouth
disease of cattle, rabies, Newcastle
disease of chickens, and certain malig­
nant tumors of mice and rabbits. In
plants, tobacco mosaic, aster yellows,
mosaic and leaf-roll of potatoes, curly
top of sugar beets, and western mo­
saic of celery provide familiar ex­
amples. These few examples indi­
cate the universal significance of
viruses in human economy.

Sizes and Shapes of Viruses

Virus bodies are truly the pygmies
among the organisms responsible for
contagious diseases. In actual meas­
urement they range in size downward
from 300 millimicrons (mμ³) to 10
millimicrons in diameter (fig. 1). The
larger viruses, such as the smallpox
virus (200 to 300 mμ), approach in
size the smaller bacteria, whereas the
smallest, for example those inducing
such diseases as alfalfa mosaic and
foot-and-mouth disease of cattle (10
mμ), are scarcely larger than the pro­
tein molecules comprising the living
substance of healthy plants (figs. 1, 2).
The numerous viruses are specific in
size and form a gradient between
these two extremes.

High magnification obtained by use
of the electron microscope reveals
that virus bodies vary not only in
size, but also in shape and structure
(figs. 1, 2). Some are cubical, some
oblong, some spherical, some hexa­
gonal rods and polyhedrons, and
others, especially those attacking and
producing disease in bacteria, are
tadpole-shaped, possessing head and
tail-like features as shown in figure 3.

Growth and Reproduction of
Virus Bodies

Virus growth and reproduction ap­
pear to be tied up with the con­
stituents of living host tissues. The
theory has been advanced that viruses
cannot utilize primary or elemental
materials from which plants or ani­
mals build their tissues, but must use
amino acids, proteins, or other com­
plex materials formed in living tis­
sues, diverting these from the normal
use of their hosts to build their own
tissues.

Multiplication and growth of most

\[ \text{One } \mu = \frac{1}{250,000} \text{ of an inch. A } m\mu \text{ is one thousandth of a } \mu, \text{ or } \frac{1}{250,000,000} \text{ of an inch.} \]
Fig. 2. Electron micrographs of virus bodies responsible for contagious diseases in man, animals, higher plants, and bacteria. All micrographs represent the same magnification, thus the differences indicated are actual. 1. **Vaccinia** virus, responsible for smallpox in man, one of the earliest virus diseases known. 2. Influenza virus (Lee strain), the greatest killer of humans of all times. 3. Tobacco mosaic virus, one of the first plant viruses to be studied and the first to be isolated, purified, measured, and weighed. 4. Potato X virus, universally present in all American potato varieties. Mixed with potato virus A, virus X causes crinkle or mild potato mosaic; mixed with potato virus Y, it causes the destructive rugose mosaic. 5. **T**₅ coliphage, or virus, which infects, produces, and finally destroys cells of bacteria of the *B. coli* type. 6. Shope rabbit papilloma virus, which causes warts that develop into typical cancer in domestic rabbits. 7. Southern bean mosaic virus. 8. Tomato bushy stunt virus. Courtesy of W. M. Stanley, the J. B. Lippincott Company, Publishers, and the National Foundation for Infantile Paralyses.
Fig. 3. Cells of *Streptococcus lactis*, the bacterium which causes milk to sour, infected with virus (bacteriophage), magnification approximately X 20,000: A, Three cells surrounded with virus particles recently introduced into the culture of living bacteria; B, a completely disrupted cell spilling out protoplasm and newly formed virus particles 5 hours after the introduction of the virus into the bacterial culture. Taken from the Journal of Bacteriology 57:393. 1949. Courtesy of Williams and Wilkins Company, Publishers.
viruses are exceedingly rapid, far surpassing those of the more complex disease-producing organisms, such as bacteria. In fact, bacteria themselves are attacked by viruses called "bacteriophages;" individual bacterial cells are destroyed within a few hours after they are attacked. It has been estimated, for example, that an individual virus particle entering a single bacterial cell may produce 100 to 300 new virus particles within 15 to 30 minutes (fig. 3). Every one of the millions of living cells of a 10-year-old Lambert cherry tree may become filled with millions of necrotic rusty mottle virus particles within 4 to 5 months after a comparatively few such virus particles are introduced into a twig of a healthy tree.

**Mutation in Viruses and the Possible Origin of New Diseases**

During reproduction and multiplication, viruses, like bacteria, fungi, and even more complex organisms, frequently change or mutate, and give rise to strains or variants different from the parent, with altered ability to produce disease. Such variants may either produce milder effects in their hosts than their parents, or they may even kill their host, and so become a threat to their own survival. A new virus strain may induce symptoms in host plants or animals so radically different from those caused by the parent as to result in what might be considered a new disease.

Human and higher animals recovering from virus infection do so by development of active immunity to that strain of the causal virus. By mutation new strains arise to which the host is not immune and thus apparent recurrence of the disease may result. Mutation is probably responsible for the new and virulent virus strains which have swept across cities, states, and nations, as did the influenza epidemic of 1918-19. Fortunately, some viruses like Vaccinia, causing smallpox, are stable and few if any strains arise. The common cold virus, affecting humans, and many viruses affecting plants appear to be unstable, existing in many strains.
Effects of Viruses on Plants

When viruses invade plant tissue, the normal processes of growth within the plant are modified, and visible external abnormalities, generally referred to as "symptoms," result. These are usually characteristic for the invading virus. Symptoms may develop in any tissues or parts of the plants, including roots and stems, but are most commonly seen in leaves, floral parts, and fruits.

Many factors may modify the time, intensity, and pattern of symptom expression, or even completely mask the most characteristic expressions. For example, symptoms may vary from season to season as an effect of varying temperature and moisture, also with the vigor of the host and with the length of time the host plant has been diseased. A single virus may produce very dissimilar symptoms in different hosts. A few diseases are known to be the expression of the combined effects of two or more viruses present at the same time in a host plant.

Responses of plants to viruses are numerous and varied and are difficult to characterize. These consist of color changes, necrosis, stunting, overgrowths, malformations, sterility, and even death of parts or of the entire plant.

Color Changes Produced by Altering Chlorophyll and Other Plant Pigments

Mosaics and yellows. Most plant viruses affect the chloroplasts—the green-pigment- or chlorophyll-bearing structures of the plant cell—resulting in a loss or lack of green color, a condition spoken of as "chlorosis." If chlorosis occurs in irregular patches and a type of mottling results, it is spoken of as "mosaic," but if the chlorophyll is uniformly reduced and a generalized chlorosis results, it is referred to as "yellows." Combinations of red and orange coloring are spoken of as "xanthosis."

Mosaics generally result from reduced formation of chlorophyll in tissues which are interspersed among others that have normal green color. Yellows generally result from destruction of green color once present in normal tissues. Leaves produced on plants in late stages of yellows-type diseases are reduced in size and pale in color from the beginning of their development because of interference with normal growth.

Vein clearing and vein banding. Some virus diseases are characterized by the absence or reduction of green color in tissues immediately adjacent to or in the veins of the leaves. This particular expression is spoken of as "vein clearing." If bands of chlorotic tissue develop along the veins and the adjacent tissue remains green, the condition is referred to as "vein banding." Vein clearing and vein banding may persist as essential diagnostic symptoms of diseases, or they may represent only an early or transitory stage and may disappear or be replaced by other more characteristic symptoms.

Ring spots and line patterns. Chlorotic spots and rings in leaves are characteristic of numerous virus diseases. These spots vary greatly in shape, size, and number, and frequently in color. In some cases the
rings of tissue surrounding living tissue may become necrotic. These are referred to as "necrotic ring spots," and if the necrosis completely surrounds the central area it will die and drop out. In others referred to as "necrotic spots," the central tissue dies and remains intact. The tissue surrounding the necrotic centers may develop concentric rings of varying colors. These may alternate with necrotic tissue with or without brown centers. Irregular chlorotic patterns composed of rings and streaks, often more or less parallel on opposite sides of the leaf midrib, are referred to as "line patterns."

**Necrosis**

Viruses may produce dead or necrotic areas in any part of the plant. In the leaf such areas may vary greatly in size and shape and in the degree to which they characterize the disease. The necrotic tissue may remain attached or may drop out, producing a shot-hole effect, or, if necrosis is severe, result in a general tattering of the leaf. Necrosis produced in the phloem or the food conducting tissue of the plant results in a variety of effects, such as stunting, malformation, general yellowing, and even death of entire plants. Some viruses produce necrosis only during the initial stages of infection or only in certain non-vital tissues. Viruses which cause necrosis in vital parts of the plants, resulting in death, are justifiably referred to as "killers."

**Stunting, Dwarfing, and Malformations**

Most virus-infected plants are smaller than normal in size. This reduction may be expressed in shorter internodes, smaller leaves or fruits, or smaller size of various other plant parts. In some diseases this dwarfing may be extreme, involving the entire plant. When growth is slowed up unevenly or occurs only in local tissues, a great variety of malformations may result. Malformed leaves, for example, are characteristic of virus diseases such as peach mosaic, prune dwarf, and cherry mottle leaf.

**Excessive Growth Symptoms**

Viruses frequently cause excessive growths such as witches'-brooms, excess twigging, galls, enlarged buds, enlarged stipules, enations, and swollen veins. These symptoms may provide the most reliable diagnostic features of a specific virus disease. In Utah, the bunchy growth, excess twigging, and enlarged and persistent stipules provide the only reliable diagnostic symptoms of western X little cherry during the major part of the growing season. Enations and related leaf distortions likewise characterize the virus disease of sweet cherry known as rasp leaf. Swollen or enlarged veins form part of the "symptomatological picture" of such virus diseases as western X decline in peach.
Dissemination of Plant Viruses

Plant viruses are spread in a variety of ways: Mechanically in juices of infected plants; by budding and grafting with buds or scions from infected trees; through seeds or other specialized plant parts used in propagation from infected plants; in a few cases from infested soil; but most commonly in the orchard by insects. The ease by which they can be passed from diseased to healthy host plants renders them difficult to control. Effective control procedures can be devised and practiced only after the various means of dissemination have been determined.

Transmission of Viruses Mechanically in Juice of Infected Plants

Some of the more highly infectious viruses, such as those producing mosaics of tobacco, tomato, and cucumber, are readily transmitted by rubbing juice from infected plants onto leaves of susceptible healthy ones. So infective are some of these viruses that the mere handling of diseased plants along with healthy ones during transportation, planting, and tillage provides a means of virus spread.

A large number of viruses, especially those in the yellows group, are seldom transmitted by such mechanical means. Likewise, viruses affecting woody plants have not been transmitted mechanically in juice from tree to tree. So far as known, stone fruit viruses, therefore, cannot be transmitted by pruning knives, saws, or cultivation equipment which bruises or cuts tissues of diseased trees, and later cuts those of healthy trees.

Virus Transmission by Grafting

Infected nursery stock is one of the most important means by which virus diseases have been spread. Most viruses affecting woody plants become so well distributed in diseased plants that cuttings, scions, buds, or other propagative tissues taken from them usually carry the causal virus with them. In fact, graft transmission is one of the important criteria by which the virus nature of diseases is determined.

Buds or scions from diseased plants, especially those from parts showing characteristic symptoms, impart the virus to plants on which they are budded or grafted. Some viruses can be transmitted with tissues other than those normally used in propagation, such as bark patches, pieces of fruit, or even pieces of leaf blades. A few viruses are limited to woody tissues and require inoculum containing woody tissue, which must remain alive until the virus has moved across the graft union.

Unless the presence of viruses is recognized and diseased plants avoided, orchardists or nurserymen may, unwittingly, widely disseminate virus diseases in their propagated stocks.

Transmission by Seeds or Other Specialized Plant Organs Used in Propagation

A few plant viruses are transmitted through seeds and pollen, and part of the world-wide occurrence of certain
viruses can be attributed to this method of distribution. Vegetative organs of perennial plants (such as tubers, bulbs, corms, cuttings, suckers, and roots) used for propagation provides one of the most effective means of virus dissemination. Once the virus enters into propagation organs, their use is nearly certain to result in disease in subsequent crops. Viruses, therefore, are more generally present in crops propagated from bulbs, tubers, and roots. Such perennial reproductive organs provide a common reservoir not only for over-wintering but for disseminating viruses.

**Transmission by Insects and Mites**

Insects and mites are the principal agents which spread viruses from diseased to healthy plants in nature. In feeding they acquire infective sap from diseased plants and migrate to healthy plants into which they introduce the virus. In most instances the insect or mite is the only natural means by which the virus can pass from one plant to another. Insect vectors with sucking mouth parts such as aphids and leafhoppers are the most common carriers of viruses. Rarely do chewing insects transmit plant viruses.

Not all sucking insects transmit viruses. Some species are not known to transmit any, whereas others may transmit one virus but not another, even to the same host plant. Some viruses are transmitted by a single insect species and by no other. Other viruses may be transmitted by a large number of species. Peach yellows is transmitted only by the leafhopper species *Macropsis trimaculata*, but Pierce’s disease of grape is transmitted by more than 20 species.

Viruses producing mosaic-type symptoms are most commonly transmitted by aphids; a few are transmitted by thrips, white flies, and mites. Viruses producing yellows-type symptoms are transmitted most commonly by leafhoppers; a few are transmitted by aphids. The insects transmitting the mosaic types carry the virus on their mouth parts and usually retain the viruses for only a few hours. Those transmitting the yellows types generally retain the virus within their bodies and may remain infective for long periods, even throughout the remainder of their lives. Some evidence indicates that such viruses may continue to reproduce within the insect’s body.

**Infection from Soil**

Although most viruses infecting plants die when their host plants die, a few, including two which affect peach, are able to persist in the soil after a diseased tree is removed and can infect a new tree planted where the diseased one grew. Neither of these diseases is known to occur in Utah.
Virus and Viruslike Diseases of Stone Fruit Trees in Utah

Viruses cause myriads of variable effects and symptoms on stone fruit trees in Utah which are likely to be confusing until the specific diseases they produce are understood.

Fruit men are generally accustomed to thinking of diseases and disorders in relation to the crop plants affected rather than from the standpoint of the causal agents. In view of this, most of the information in this bulletin has been organized around crop plants. Because the western X and ring spot viruses are already widely distributed and affect most of the major stone fruits grown in Utah, their more general features and the diseases they produce are treated in the following sections apart from the hosts. The diseases they produce are more thoroughly treated in sections under hosts.

In the sections on nutrient deficiencies and excesses, genetic and heritable disorders and chemical injuries are treated from the standpoint of causes and the descriptions apply to all hosts unless specifically noted.

WESTERN X VIRUS AND THE DISEASES IT PRODUCES IN STONE FRUITS IN UTAH

Reports indicate that the stone fruit western X virus, or its similar relative X virus, is geographically widely distributed in North America, occupying a belt from the Atlantic to the Pacific Ocean and ranging north and south approximately coextensive with its natural bridging host, the common chokecherry (Prunus virginiana) or its variants.

Diseases on stone fruit trees caused by these viruses have been discovered and described at different times by workers in diverse areas. As a result, a variety of names have been applied to the causal viruses, as well as to the diseases they cause. The character "X", representing a mathematically unknown quantity, was originally applied in Connecticut to the X-disease of peach because of its obscure nature and non-conformity to the symptoms of known virus diseases of fruit trees. Use has now firmly fixed this character in the name "X-disease." "Western" was added to the name to distinguish the virus occurring in the West from that in the East. The distinction was made, essentially, on the basis of differential patterns of spread and host relations rather than upon fundamental differences between the viruses. More recent investigations indicate that what were thought to be differences between X virus and western X virus can be explained on the basis of different vectors, climate, and other environmental factors.

The western X virus probably has been present for 50 years or longer in Utah, where it has become generally distributed and firmly established in a variety of hosts in the fruit-growing areas in the northern part of the state. It has been critically studied and has been referred to locally as
western X since 1940, when it was discovered in Utah. In this handbook the term “western X” is employed as a name for the virus and as a prefix to designate the names of diseases it induces.

Diseases Induced by Western X Virus in Utah

Western X red leaf in chokecherry (P. virginiana var. demissa) and western X decline in peach were observed in the Columbia River area in northeastern Washington in 1935 in connection with smelter injury investigations. These same disease symptoms in both chokecherry and peach were observed in Utah in 1937, at which date investigations were begun. The virus nature of the two diseases and the fact that both are induced by the same virus in Utah were conclusively established during the period from 1937 to 1944. Continued research studies in Utah and neighboring states from 1940 to 1946 further demonstrated that the same western X virus not only caused red leaf in chokecherry and decline in peach, but likewise induced wilt and decline in sweet and sour cherries growing on mahaleb rootstock. The virus was also found to be the cause of failure of fruit to develop and mature on affected sweet and sour cherries growing on mazzard rootstock. It is thus firmly established that the western X virus in Utah induces six distinct diseases in stone fruits:

1. Western X red leaf in chokecherry
2. Western X decline in peach
3, 4. Western X wilt and decline in sweet and sour cherries on mahaleb rootstock
5, 6. Western X little cherry in sweet and sour cherries on mazzard rootstock

Host Range of Western X Virus

Research studies in Utah and other states indicate that the western X virus attacks and produces decline symptoms in all important American varieties of peach (Prunus persica), including the nectarine; sweet cherry (P. avium), including mazzard seedlings; and sour cherry (P. cerasus). The virus also occurs naturally in western chokecherry (P. virginiana var. demissa) and has been transmitted experimentally to eastern chokecherry (P. virginiana). In Utah, the virus has been transmitted to Manchu cherry (P. tomentosa) and to Hansen bush or Bessey cherry (P. besseyi), producing rolling and yellow to red coloring of the leaves and frequently dwarfing and killing the plants. The virus has not been found naturally in apricot in Utah, but has passed from the top of an inoculated apricot tree to the peach rootstock without inducing symptoms in the apricot. So far no native vegetation other than chokecherry has been found naturally infected.

Insect Vectors of Western X Virus

Entomologists of the U. S. Department of Agriculture and state agricultural experiment stations working in several areas have found that the geminate leafhopper (Colladonus
*geminatus* [Van D.]) is the major insect vector responsible for the natural distribution of the western X virus in western United States. This leafhopper is commonly present in Utah orchards and has transmitted the western X virus experimentally between hosts in all combinations. Three other leafhoppers (*Scaphytopius acutus* [Say], *Fieberiella florii* [Stal], and *Keonolla confluens* [Uhl]) have been detected as vectors of lesser importance in Washington. Because of the low population density of these insects in Utah, they appear unimportant in the spread of western X virus.

Extensive research has demonstrated that the geminate leafhopper feeds and reproduces naturally on chokecherry, on all common varieties of peach, on sweet and sour cherries, and on a wide range of other hosts. It has also been determined that the insect will continue to feed when moved from one species of stone fruit to another. By feeding insects on western-X-infected trees of any one species and then transferring them to healthy trees of another species, typical expression of western X decline as it occurs naturally—red leaf in chokecherry, decline in peach, and wilt and decline in sweet and sour cherries—has been produced, thus duplicating results obtained earlier by experimental infection through budding and grafting procedures.

The geminate leafhopper feeds and develops naturally on alfalfa and clover and related herbaceous plants during spring and early summer, and only occasionally does it visit stone fruits and other orchard trees. In late summer and autumn, when the herbaceous plants are drying up or for other reasons are less attractive, the hoppers migrate to the greener foliage of the orchard trees. During September and October, large numbers of the migrants have been found in Utah on the various species of stone fruit trees. Here they feed until the leaves fall in late October or November, or until they are killed by frost. Early and periodic droughts during hot seasons may hasten migration and may even result in unusually large populations feeding on stone fruit trees. During such seasons, the spread of western X virus no doubt would be accelerated.

Kaloostian and his co-workers in Utah have shown that after the insect picks up the virus from a diseased plant, a period of approximately 30 days must elapse before it can transmit the virus to a healthy plant. This long latent period of the virus in the insect reduces its efficiency as a vector. Much of this inefficiency, however, is offset by the fact that a single insect acquires enough of the virus to infect a large orchard tree and may continue to spread the disease as it moves successively from tree to tree.

**Western Chokecherry the Native Bridging Host for Western X Virus**

The western X virus spreads rapidly from plant to plant in native stands of western chokecherry. In Utah, and generally throughout the Great Plains and the Rocky Mountain areas, chokecherries exist in thickets, in fence rows, and along stream beds, serving somewhat as a chain along which the virus has spread or may spread to isolated orchards. In fact, every chokecherry plant and every chokecherry thicket is a reservoir, or a potential reservoir that provides the
bridging-host responsible for the wide
distribution of the western X virus.
The eastern chokecherry (P. vir-
giniana) functions in the same manner
for the X virus in the East, the insect
vector for which is Colladonus clitel-
larius (Say). This insect appears to
prefer chokecherry to cultivated stone
fruits and moves to the latter only
where they are near chokecherries.
Correspondingly, X-disease has caused
losses in fruit orchards only where
they adjoin diseased chokecherries.
The disease can be prevented or
stopped in orchards by removing the
diseased chokecherries around the
borders. Surveys indicate that the
geminate leafhopper which spreads
the western X virus does not occur
or is rare in eastern North America.

RING SPOT VIRUS AND THE DISEASE IT PRODUCES IN STONE FRUITS IN UTAH

THE NAME “ring spot” was applied
to the virus and the disease it
produces because chlorotic and necro-
tic ring and spot patterns are the most
characteristic symptoms consistently
produced on the leaves of a number
of stone fruit trees. The disease was
originally described on peach, but is
now known to affect nearly all stone
fruit species. The ring spot virus is
probably the most prevalent and
widely occurring virus affecting stone
fruits. It occurs in most of the major
stone fruit growing areas in the
world. It is more prevalent in cher-
ries than in other stone fruits. In
some Utah cherry orchards scarcely
a tree free of the virus can be found.
It is now recognized that there are
numerous strains of the ring spot virus
and that each may produce somewhat
different effects on the host plant.

General Expression in
Stone Fruits

The presence of the ring spot virus
has not been generally recognized by
growers and others connected with
the fruit industry because most trees
have been infected for some time and
do not show striking symptoms.
When symptoms are present, particu-
larly the necrotic lesions produced on
some plants, they have been confused
with symptoms of fungus and bacte-
terial diseases, especially those of the
leaf-spot and shot-hole types. Most
species show symptoms only during
a portion of the growing season fol-
lowing infection and then become
symptomless carriers. Some peach
varieties like Rio Oso Gem and J. H.
Hale show ring and necrotic patterns,
bud killing, and twig dieback at the
beginning of growth but soon re-
cover, subsequently producing nor-
mal-appearing growth. Sour cherry,
like peach, suffers striking shock dur-
ing the first stages of the disease;
though it may retain some of the shot-
holed and tattered leaves during the
remainder of the season, new growth
on the previously affected arms is
without leaf patterns the following
year. Often large trees may show
symptoms on one side one year and
on the remainder the following year.
Some sour cherry and peach trees
which are severely affected may show
pale color and general lack of vigor
for a year or two following infection.
Sweet cherry varieties are variable
in their expression of ring spot. In
general, Black Tartarian shows more severe symptoms than does Bing. Some strains of the virus produce more striking expressions than others. Sweet cherry differs from sour cherry and peach in that trees infected with some strains of the virus show symptoms annually. Symptoms on sweet cherry vary from a few obscure, chlorotic oak-leaf and ring patterns to brilliant ring and necrotic patterns the centers of which may fall out, producing a tattered and lace-leaf effect.

Economic Importance in Utah

The loss caused by ring spot is difficult to determine. Sweet and sour cherry orchards, known to be nearly 100 percent infected, continue to produce economically profitable crops. Budwood from ring-spot-free sweet and sour cherries results in much more vigorous nursery stock and a better stand of trees. If orchards could be grown from such nursery stocks without becoming infected, no doubt they would be more productive than those from infected stocks. Ring spot causes more apparent damage on peach than on cherries. During the first year of infection, trees may be severely retarded and suffer extensive dieback. Such weakened trees are subject to sunburn and borers and may be killed before they recover.

Natural Spread and Prevention

The ring spot virus spreads from tree to tree in orchards and probably also spreads from one species of stone fruit to another. The virus is undoubtedly spread by an insect or mite, but the natural vector is unknown. Most of the disease in orchards results from use of infected nursery stocks. Infection in nursery stocks stems from two main sources, use of infected variety trees as a source of budwood and use of seedling rootstocks infected as a result of seed transmission. Virus-free budwood sources can be found by indexing procedure, placing buds from the desired variety on a virus-free sensitive test plant, such as the Shiro-fugen variety of flowering cherry; if no reaction occurs, the budwood source is considered not infected. Ring-spot-free seedling rootstocks likewise can be obtained by use of seed from trees known to be free of the virus.

Ring Spot Virus Interferes with Diagnosis and Study of Other Fruit Tree Viruses

The ring spot virus is so prevalent that it often occurs in trees along with other viruses. In sweet cherry, ring spot usually produces symptoms annually and thus may confuse the symptoms of other viruses which may be present. In most hosts, the ring spot virus is latent when occurring with other viruses but in transmission tests it becomes expressed and confuses symptoms of the other virus. In a few cases, particularly in graft transmission tests on peach, the ring spot virus may cause sufficient necrosis on the peach to prevent infection by other viruses in the same inoculum. The ring spot virus must be regarded as a common contaminant and tests for its presence should be part of the investigational procedure in the study of any new virus.
THE COMMON chokecherry (*Prunus virginiana* var. *demissa*) is widely distributed in Utah, but is particularly abundant in the mountains, along the foothills, and in the valleys of northern Utah. It has a limited use as an ornamental shrub and has been used as a conservation plant by the Soil Conservation and Wild Life Preservation Services for erosion control and to provide food and cover for wildlife. Chokecherry fruits are also prized for jelly.

**Chokecherry as a Reservoir of Western X Virus**

The useful values of chokecherry are greatly outweighed by the fact that it is a host of the western X virus. Every chokecherry plant is a potential reservoir of the virus from which spread can take place to orchard trees. Western-X-red-leaf-diseased plants are abundant in Box Elder, Cache, Davis, Morgan, Salt Lake, Summit, Wasatch, and Weber Counties, but have not yet been found in Kane and Washington Counties. In Box Elder, Cache, Davis, Salt Lake, and Weber Counties, diseased chokecherries are particularly abundant in the foothills near stone fruit orchards. Western-X-affected peach and cherry trees are more prevalent in orchards at the mouth of Provo Canyon, where diseased chokecherries are also prevalent and nearer to orchards.

**How to Recognize Western X Red Leaf**

**Leaf symptoms.** The first symptoms of western X red leaf in chokecherry appear in late May to July as a greenish-yellow color in the leaves. As the season progresses, affected leaves acquire various shades of red. On many of the plants all the leaves become brilliantly red-colored as contrasted to the green color of normal plants in the same clumps, thereby providing the basis for the name "red leaf" (pl. 2, D). The red color develops first around the leaf margin and between the veins; frequently the veins remain green until late in the season. In the second and subsequent years, diseased plants are usually stunted, the leaves are less brilliantly colored, smaller in size, fewer in number, and on some plants downwardly cupped.

**Fruit symptoms.** Diseased plants at higher elevations generally fail to set fruit, but in the lower valleys and under cultivation they may set good-sized fruit clusters. Fruits on diseased plants are small and pointed and remain salmon pink to dull red as contrasted to the dark red or black normal fruits (pl. 2, C). Seeds seldom form in affected fruits. The pointed shape, reduced size, and failure of normal color development of fruits are symptoms which resemble those occurring on western-X-little-cherry-affected cultivated cherries.
**Plant symptoms.** Effects of the western X virus on chokecherry plants are varied. Many plants, especially at the higher altitudes, die during the summer following the initial onset of the disease, or they may fail to survive the ensuing winter. At lower elevations diseased plants live on in a declining condition for several years; stunting always results. After the first year, diseased plants usually develop excessive twigging and shortened inflorescences and are inclined to off-season blooming.

**Control**

The chokecherry is a potential reservoir of the western X virus and therefore should be regarded as a "noxious weed" in or near fruit-growing districts. Eradication of diseased chokecherries amounts to eradication of the chokecherry itself and presents a problem of such size that it cannot be generally recommended. Eradication near fruit orchards seems feasible and with use of newly developed chemical brush killers could be efficiently accomplished. The chokecherry should be brought under the noxious weed law and its use as an ornamental or as a conservation plant in or near fruit-growing areas should be judiciously restricted.
WESTERN X decline of peach has been reported in British Columbia, Washington, Oregon, Idaho, Utah, California, Arizona, and Colorado. In all of these areas it is considered to be a very destructive virus disease. The disease is generally distributed in northern Utah in the counties of Cache, Box Elder, Weber, Davis, and Salt Lake. In these counties the diseased trees in orchards are so numerous that western X decline has become a major economic factor in peach production. The disease is much less prevalent in Utah County and has not been reported in Washington or San Juan Counties. Western X decline was destructive in Utah peach orchards long before its virus nature was determined in 1940.

How to Recognize Western X Decline

Diseased trees appear normal until late June or early July, after which symptoms may appear any time during the remainder of the growing season. In orchard trees initial symptoms are usually limited to a single twig or branch on one arm. More than one infection can take place in a tree; hence two arms could be infected separately in the same year. Infection commonly progresses from the point where the tree is inoculated by the insect to other parts of the arm or to other arms before it is expressed or noticed; therefore symptoms may be expressed in the foliage of a considerable portion of a main

Fig. 4. Elberta peach leaves showing early-season symptoms of western X decline: Normal at left and four leaves at right showing a sequence (from left to right) of irregular pale-green areas of varying sizes and shapes to necrosis and falling out of necrotic tissue.
arm when first seen. Often the first symptoms may appear in the sucker or water shoots. In this case, the infection probably took place at some other point in the tree and as the virus began to multiply, it was translocated along with food formed in the leaves to new growing points. Since sucker shoots grow rapidly, requiring more food than other tissue, they naturally become infected before other parts of the tree.

Symptoms vary in severity and type of patterns in trees of different peach varieties, under different weather conditions, in different portions of the affected tree, and at different seasons of the year. The severity of symptoms is also directly affected by the virus strain present; some strains produce only mild damage, whereas other kill trees within one or two years.

Affected twigs and often entire branches commonly die during the winter following expression, and growth during subsequent years tends to cause trees to develop a one-sided scraggly appearance (pl. 1, C, and 2, B). Peach orchard trees are seldom killed by the western X virus; instead, affected trees lose arms and decline slowly over a period of years (pl. 1). Trees severely weakened by the disease may be killed by pests such as the shot-hole borer.

**Early-season leaf symptoms.** In general the initial leaf symptoms in the early summer, and those occurring in leaves on rapidly growing shoots later during the summer, consist of irregular pale-green areas of varying sizes and shapes that may occur in any portion of the leaf blade (pl. 1, A, and figs. 4, 5). These affected areas may be few or many, or they may join and involve large portions of the leaf blade (fig. 4). Usually they separate from the normal por-

![Image](image-url)

*Fig. 5. Elberta peach leaves showing late-season symptoms of western X decline: Normal leaf at left and four leaves at right showing water-soaked areas, which become necrotic, producing tan-colored spots of varying sizes and shapes, usually surrounded by deep purplish borders.*
tions of the leaf and fall away, resulting in a tattered condition (fig. 6). Leaves affected early in the season drop, resulting in bare twigs, often with a tuft of yellowish green leaves at the tip. Affected leaves which remain attached become pale-green to yellow and seldom develop the striking red or yellow characteristic of the disease later in the season (pl. 1, B).

**Late-season leaf symptoms.** In late summer or early autumn, leaf symptoms appear first as water-soaked areas, which become progressively necrotic, forming spots of varying sizes and shapes. These necrotic areas do not drop out, but become tan to brown, usually surrounded by a reddish-brown or purplish border (figs. 5, 7.) The remaining portion of the affected leaf blade turns greenish-yellow with irregular-shaped spots, streaks, or splashes of red. Late in the season the reddish color is predominant along the veins. Leaf fall is a far less prominent feature than with early-season expression of the disease and the retention of the colored leaves gives the tree a characteristic yellowish to yellow-red appearance (pl. 1, C). Young, actively growing trees may show both early-season and late-season symptoms in the fall, with more of a tendency for affected leaves to be rolled upward toward the midrib (fig. 7).

In warm dry seasons, the necrotic spotting and red coloring, characteristic in the late summer, are less conspicuous. Instead, many leaves, both those showing spotting and others, progressively develop a golden-yellow color (pl. 1, B). Usually these leaves remain attached until late autumn, giving the diseased trees a striking yellow appearance.

**Fruit symptoms.** On severely affected branches fruits usually shrivel and drop shortly after the appearance
of the first leaf symptoms. Fruits on branches less severely affected, or on branches on which symptoms develop after the fruits are good-sized, usually remain attached but ripen early and fall prior to the time of normal harvest. Such affected fruits are undersized, somewhat conical in shape, withered at the apical end (pl. 2, A), and insipid with a bitter flavor. Abnormal red streaking and splashing of the fruit surface is frequently prominent in certain peach varieties, such as Elberta (pl. 2, A). Seeds fail to develop in affected fruits and the pits fail to become as hard as in normal fruits.

**Distribution and Economic Importance**

The distribution of X decline in peach orchards is indicated by data obtained in surveys made over a 12-year period in various parts of the state. Although the surveys in the different areas are not entirely comparable, they do indicate the general relative prevalence of disease within and between areas.

**In three northern counties—Davis, Weber, and Box Elder.** In a preliminary survey of 14 peach orchards in Box Elder and Davis Counties during late July 1939, none was found free from western X decline. Five of these orchards contained 10 to 25 percent infected trees, five from 26 to 50 percent infected trees, and infected trees in the other orchards ranged as follows: 51, 62, 63.8, and 74.8 percent. The last four orchards had been rendered economically unprofitable. The orchards surveyed varied in age from 6 to 20 years, and all were taken at random without any previous knowledge of their disease content.

During late August 1939, a more detailed study was made of 26 peach

![Fig. 7. Elberta peach leaves showing upward rolling and purple-bordered necrotic areas characteristic of late-season effects of western X decline.](image)
orchards in Davis County. These also were selected without regard to age or the percentage of disease present. Only 2 of the 26 orchards were found free from western X decline; six showed under 5 percent diseased trees; four, 5 to 10 percent; four, 10 to 25 percent; and ten, above 25 percent. In five of these orchards the percentage of diseased trees ranged above 40 percent, with two above 50 percent. An average of 22 percent of all the trees in the 26 orchards showed decline.

In a further detailed study during 1944 of 29 peach orchards in Davis County, including some of those previously studied, none was found free from western X decline. Only two orchards showed below 10 percent diseased trees; five, between 10 and 25 percent; 13, between 26 and 50 percent; seven, above 50 percent; and two, above 80 percent. Of the 5,408 trees studied, 1,775 (32.8 percent) were diseased. This increase in occurrence of western X decline in the peach orchards studied was generally representative of most of the orchards in Davis, Weber, and Box Elder Counties.

The general distribution and high incidence of diseased trees in the older orchards found in 1939-44 indicate definitely that western X decline is an old disease in Utah and was probably present in the state many years prior to its recognition in 1938. This conclusion is further supported by the earlier reported occurrence of wilt and decline in cherries, now known to be caused by the same virus and to exist in close association with peach X decline. The more spectacular symptoms of cherry wilt and decline no doubt caused it to be noticed, where- as peach X decline was also present but was overlooked.

**In Utah County.** Surveys were made in 1942, 1943, and 1944 to determine the distribution and incidence of western X decline in peach orchards in Utah County. In the 1942 survey, 22 young orchards, varying in age from two to five years, were examined without finding a single western-X-decline-affected tree. In 1943, 10,040 trees of all ages were examined in 32 orchards in ten peach-growing localities in the county. In these 122 trees (1.2 percent) showed western X decline. In 1944, 24 orchards, including some of those surveyed in 1943, were resurveyed; 355 trees (3.3 percent) were found diseased. Orchards in the 1944 survey were all 8 years old or older, and thus the higher percentage of disease would be expected, because of longer exposure. This last survey then would represent a maximum rather than the average of decline incidence for the county. Both the 1943 and 1944 surveys were made late in the growing season, and diseased trees which would not have been expressing symptoms earlier in the season probably were found.

Two more extensive surveys, in mid-August 1950 and 1951, were made in Utah County with the intent of exploring the possibility of control by removal of diseased trees. In the first survey, 167,397 trees were examined and only 301 trees (0.18 percent) were found showing symptoms of western X decline. In the 1951 survey, 559 (0.89 percent) of the 62,810 trees examined were diseased. Both 1950 and 1951 were cool seasons and it seems likely that the lower incidence of diseased trees
found, as compared with 1944, might have been a result of failure of symptom development.

The surveys showed that the incidence of disease was relatively low in Utah County and that affected trees were widely scattered. However, a larger number of diseased trees occurred in orchards near the mouth of Provo Canyon and the foothills along the east side of Utah Valley than in orchards farther west. These orchards in the higher elevations are closer to rather sparsely distributed chokecherries.

**Rate of Natural Spread**

The increase in number of diseased trees as orchards become older is a common feature of the peach western X decline problem. The rate of increase varies greatly with seasons and in proportion to the proximity of new orchards to infected orchards or chokecherry thickets, and possibly other factors. The rate of spread in Utah County has been much slower than in Davis and adjacent counties.

In 1939, 10 peach orchards of varying ages and with varying percentages of infected trees in Davis County were selected for the purpose of investigating the rate at which the western X virus spreads. Each orchard was studied and mapped to show the exact location of both diseased and healthy trees. In successive years, 1939 through 1944, all new cases of the disease appearing in the orchards were plotted and recorded. A summary of the results of this study is shown in table 1. It will be noted that new cases of the disease appeared each year in each of the 10 orchards during the 6 years studied, and, in general, the rate of spread appears to increase in proportion to the number of diseased trees in the orchards. The data show that on the average the percentage of diseased trees in the 10 orchards more than doubled during the 5-year period.

Spread of western X decline was consistently high in Davis County orchards during the 6 years 1939-44. The high percentages of diseased trees in older orchards, as compared with those in younger orchards in the Davis County surveys, provide a fair index of the rate of spread. The fact that many orchards (6-, 8-, and 10-year-old) showed as high as 50 percent infected trees further indicates a rapid rate of spread. One orchard in the 1944 survey was 37.5 percent diseased at 8 years; one, 81.8 percent at 13 years; and a third, 83.4 percent at 25 years.

**Factors Which Influence Rate of Spread**

**Influence of seasonal factors.** Seasonal factors greatly modify the type and degree of expression and the rate of spread in peach orchards. In Utah from 1939 to 1944 the spring seasons were early and relatively warm and dry. Long dry summers also characterized this period. Since 1946, the reverse conditions in general have prevailed; most spring seasons have been late with cold weather extending into late May and June. The summers were also more moderate and with fewer periods of relatively high temperatures.

During the period of 1939 to 1944, the rate of spread in the orchard was rapid (table 1) and the effects on the trees were severe. Leaf symptoms developed earlier and were more gen-
Table 1. Incidence of western X decline in 10 peach orchards in Davis County, showing number and percentage of increase of diseased trees from year to year, 1939 to 1944

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<td>50.0</td>
<td>63</td>
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<td>9</td>
<td>107</td>
<td>46</td>
<td>43.0</td>
<td>49</td>
<td>45.8</td>
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<td>11</td>
<td>466</td>
<td>135</td>
<td>29.0</td>
<td>202</td>
<td>43.3</td>
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<td>13</td>
<td>116</td>
<td>60</td>
<td>51.7</td>
<td>74</td>
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<td>123</td>
<td>49.8</td>
<td>153</td>
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<td>Total observed:</td>
<td>413</td>
<td>602</td>
<td>684</td>
<td>739</td>
<td>807</td>
<td>622</td>
</tr>
<tr>
<td>Average percentage for 1939 to 1944:</td>
<td>22.9</td>
<td>33.4</td>
<td>37.9</td>
<td>40.9</td>
<td>44.7</td>
<td>50.9</td>
</tr>
</tbody>
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erally distributed through the trees. The golden-yellow rather than the reddish-green color of the affected foliage dominated the symptom picture in all areas of the state where the disease occurred.

From 1946 to 1953, the reverse of all the foregoing symptom expressions prevailed. In most of these years the initial symptoms in early summer were greatly delayed, the affected portions of the trees were definitely smaller, injury to trees was far less pronounced, and the rate of spread was greatly decreased. Many trees recorded as diseased in previous years failed to show symptoms. The reddish-yellow or reddish-green, characteristic of the late-season expression of the disease, was universally dominant.

The long hot summers with occasional drought periods, which characterized 1939 to 1944, may have increased both the total population of insect vectors and the frequency of their migration from the drought-damaged herbaceous vegetation to the greener and more inviting orchard trees. A larger number of insect vectors feeding on trees would naturally increase the possibility of their feeding on diseased trees before migrating to healthy trees, thereby increasing the rate of spread. The increased severity, earlier development of symptoms, and golden-yellow color in the trees during 1939 to 1944 were direct effects of temperature and probably not related to rate of spread.

**Influence of chokecherry and other hosts.** No exact quantitative data are available to show the relation of chokecherry to the spread of western X virus in Utah peach orchards. It is significant, however, that the highest incidence of western X virus in peach, as well as in sweet and sour cherries, is found in orchards adjacent to the foothills of the Wasatch Mountains in Box Elder, Weber, and Davis Counties. These orchards are located immediately adjacent and parallel to almost continuous thickets of chokecherry, a high percentage of which show typical western X red leaf symptoms. The chokecherries also grow in mountain ravines which frequently extend well into orchard areas below and eastward to elevations above 9,000 feet. In Utah County the more frequent occurrence of western X decline in orchards near chokecherries at the mouths of canyons is even more striking. The significant relations of western-X-infected cherries on mazzard rootstock to spread of the western X virus in peach orchards and to cherries on mahaleb rootstock is discussed under western X wilt and decline on page 51.

**The role of nurseries in distributing western X virus.** The possible role of nurseries in distributing the western X virus has been given critical attention. In 1940, 1941, and 1942, all nurseries propagating peach trees in Salt Lake, Weber, and Davis Counties were inspected. A number of decline-affected trees were found in the nursery rows. All affected trees found were so small that they probably would not have become merchantable.

Nursery trees were propagated from X-decline-affected peach trees and grown in the plots at Bountiful, Utah, in 1942 and 1943 to determine whether diseased trees would attain suitable size for sale. In both years the buds used for propagation were
obtained from budsticks bearing leaves with unmistakable western X decline symptoms. Standard nursery propagation procedures were followed throughout, and all the buds on each stick were used regardless of whether they were subtended by a symptom-bearing leaf. Lovell peach seedlings were used as rootstocks. Of 487 trees grown in the two years, 211 (43.3 percent) were western-X-decline-diseased; the remainder developed into healthy trees, even though they grew from buds taken from budsticks which had shown unmistakable symptoms. None of the trees showing X decline symptoms exceeded half the height of the healthy ones (fig. 8). Most of the diseased trees survived winter storage, but it was obvious that such trees were third- or fourth-class nursery stock.

A further check on possible spread through nurseries was made by inspecting 22 young (2- to 5-year-old) orchards planted in Utah County from stock known to be propagated in Davis County from buds collected in western-X-infected orchards. None of these young orchards showed disease; therefore if any of the Davis County budwood carried the virus, the resulting nursery stock was not sold.

**Experimental Transmission**

Western X decline has been induced repeatedly in peach trees by grafting them with infected buds from
peach, chokecherry, or sweet and sour cherry trees growing on mazzard rootstock. This interspecific transmission has been duplicated by Kaloostian and his co-workers with the geminate leafhopper vector. It follows, therefore, that any infected tree of the forenamed varieties provides a source of inoculum of the causal virus, and that the virus can be spread in a peach orchard by grafting procedures or by the natural insect vector, *Colladonus geminatus* (fig. 9).

**Control**

When new peach orchards are planted, they should be as far removed as possible from other stone fruit orchards or from chokecherries. If adequate isolation cannot be obtained, efforts should be made to eliminate all infected trees in nearby plantings of peach and sweet and sour cherries. Chokecherries in nearby fence rows, washes, and brushland should be eliminated regardless of whether they show the characteristic red leaves. Young peach orchards should be systematically inspected during July to October and any infected trees promptly removed and replaced.

**Fig. 9.** *Colladonus geminatus* (Van D.), geminate leafhopper, the principal vector of the western X virus in Utah.

**Peach Mosaic**

Peach mosaic is a highly contagious virus disease of peach and certain other stone fruits. The disease invariably has resulted in extensive crop losses in all commercial peach-growing areas where it has occurred. Its presence in a small area of Utah has constituted a serious threat to peach growing in other parts of the state. The origin of the disease is unknown, but its widespread distribution in southwestern North America provides evidence that it has been present in certain sections for many years.

**How to Recognize Peach Mosaic**

The name "mosaic" means mottle. The most characteristic and consistent symptom of peach mosaic on peach is an irregular yellow and green mottling of the leaves, usually accompanied by distortion and reduction in size. Other common symptoms are breaking or white streaking of the pink color of flowers (pl. 3, A), retarded foliation in the spring (fig. 10, and pl. 3, D), reduced growth, and bumpy fruit (pl. 3, B). These symptoms are extremely variable on different varieties of peach or even on a single variety affected by different forms of the causal virus.

**Leaf symptoms.** Leaves on severely affected trees are distorted and small, and many show the characteristic green and yellow mottled patterns (pl. 3, C). In a single leaf or in
different leaves, the yellowish-discolored areas may vary in size from small flecks to variously shaped spots or blotches, and more or less irregular streaks, involving large parts or are more prevalent on one half of the leaf. Leaves extensively affected turn yellow and drop. Leaves formed subsequent to those showing veinlet clearing remain firmly attached and

often extending over the entire leaf (fig. 11). Although the mottling is generally irregular, oak-leaf patterns and marginal streaks are not uncommon (pl. 3, C).

On trees or parts of trees showing symptoms for the first time, retardation of growth in the spring is more pronounced than on trees in the second year of the disease and the first leaves formed show small yellow flecks adjacent to veins (veinlet clearing) (fig. 11, A, B). These flecks may be few or extensive and often develop the characteristic irregular-yellow-green mottling. As the season advances, the foliage as a whole becomes darker green in color and the mosaic patterns in leaves generally become less distinct.

Mottled leaves are sufficiently characteristic of peach mosaic that within an infected area the disease generally can be diagnosed on this symptom alone. Several other viruses that produce mottled patterns which can be confused with those of peach mosaic in peach leaves are known.
If other tree symptoms of peach mosaic are present, especially on non-tolerant varieties like J. H. Hale or Elberta, the combination of symptoms is usually sufficient for diagnosis.

**Flower symptoms.** On peach varieties with large pink flowers, mosaic causes white lens-shaped spots. These vary in size; the larger ones have an island of pink surrounded by a white border. Often several spots run together or large spots interrupted at the edge of the petal form streaks and lines of various design (pl. 3, A). Patterns are more pronounced on varieties having dark-pink flowers. In cool seasons, blossoms of most varieties remain on trees longer and develop darker color, causing symptoms to be more pronounced. Some forms of the virus cause crinkling of flower petals in addition to breaking of the color pattern. Color-breaking patterns have not been seen in any of the small-flowered peach varieties.

Flower symptoms occasionally occur in peach trees affected with the ring spot virus. On individual petals such symptoms are not distinct from those of mosaic, but usually only a few flowers on diseased trees are affected; whereas on mosaic-affected trees the majority of the flowers show symptoms.

**Twig symptoms.** Severely affected trees tend to produce short twig growth, particularly early in the season (fig. 12A). On newly infected trees of nontolerant varieties, lateral branches may be so shortened that the nodes are joined. Leaves on such twigs are narrow and rosetted (fig. 12, B). New growth on severely affected trees rarely attains more than 4 to 8 inches in length and usually has a tendency to excessive branching. Secondary whorls of twigs sometimes grow from the bases of short shoots produced the previous year. Shoot growth is not noticeably reduced on tolerant or mildly affected varieties.

**Fruit symptoms.** On severely affected trees of nontolerant varieties, such as J. H. Hale, Elberta, and Rio Oso Gem, the fruits are few in number, small, late-maturing, variously misshapen, and bumpy (pl. 3, B). The bumpiness begins to develop on the green half-grown fruit at about the time the pit begins to harden. The bumps appear as raised areas of normal tissue of various sizes and shapes, surrounded by depressions caused by failure of tissue to develop. The bumpiness becomes most pronounced as the fruit swells just prior to harvest. Bumpy fruits are nearly normal in flavor when mature but because of size and shape are substandard for market. Fruits on tolerant or little damaged varieties may be unaffected.

**Other Hosts of Peach Mosaic in Utah**

Peach mosaic virus occurs naturally in wild plums, such as the pottawatomie or wildgoose plum (*Prunus munsoniana*), and the American plum (*P. americana*). A high percentage of the pottawatomie plums growing on ditchbanks and hedges in the Moab area were found infected but showed no symptoms. This plum, prized for jelly, has been widely planted. It is generally propagated by transplanting...
suckers which grow from the roots of older trees. It seems probable that the peach mosaic virus may have been brought to the Moab area in pottawatomie plums from areas to the south along the Rio Grande River, where most of the plum trees are known to be infected. Rapid natural spread from pottawatomie plum to peach was demonstrated in experimental plots in the Moab district, indicating the importance of the pottawatomie plum as a natural host of the peach mosaic virus.

Apricots and almonds have been found naturally infected in the Moab area. As in peach, the types and degree of expression vary with the form of the virus present (fig. 13). Although several horticultural varieties of the Japanese plums have been experimentally infected, graft indexing of trees in commercial plum orchards near diseased peach indicates that they do not become naturally infected under field conditions. All attempts to infect cherries and cherry-like species, chokecherry, desert apricot (P. fremontii), and desert almond (P. fasciculata), have failed. These species are therefore considered immune.

Distribution and Economic Importance

At present peach mosaic in Utah is limited to a small area around Moab in Grand County, where it was discovered in 1935. It was found in Washington County in 1937 and in San Juan, Utah, and Salt Lake Counties in 1938, but so far as is known all diseased trees in these counties have been removed. Eradication from Grand County has not been affected principally because of existence of the virus in pottawatomie plums and other naturalized Prunus host plants in these areas.

Although peach mosaic in Utah has been confined to a relatively small portion of the peach-growing area of the state, the loss from diseased trees removed, the expense of annual surveys, and the expense of tree removal since 1935 have been costly. During this period 3,473 diseased trees were destroyed. As a result of the removal program, only 19 diseased trees were found in 1954 among more than 21,000 trees inspected, thus the loss is minor as compared to what it would have been had peach mosaic escaped and become established in the larger peach-growing areas of the state. This assumption is further emphasized by the fact that 40,000 peach-mosaic-affected trees were removed in one year in a neighboring area in Grand County, Colorado.

Transmission and Spread

The peach mosaic virus is easily transmitted by grafting tissue from any part of a thoroughly diseased tree onto a healthy tree. If scions from diseased trees are grafted on healthy trees at the time they are breaking dormancy, symptoms evidencing transmission appear in 2 to 6 weeks. Grafts made after growth has started require longer incubation. Trees graft-inoculated in the fall are invariably diseased the following spring; large trees may require more than one growing season to become universally affected. Mechanical transmission has been effected only where some tissue from the diseased tree was grafted on the healthy one; all attempts with juice from diseased trees have failed. There is no evidence to indicate that peach mosaic is spread
by mechanical contact, in irrigation water, in soil, in seeds, or by other cultural practices.

A microscopic eriophyid mite (*Eriophyes insidiosus* Keifer & Wilson) has been shown to be the natural vector which spreads the peach mosaic virus in orchards. The mite feeds and breeds under bud scales on peach and wild plums and is apparently dispersed from diseased to healthy trees by air currents as the bud scales are shed in the spring and early summer. The mite is efficient; a single mite taken from a mosaic-affected tree and allowed to feed on a healthy tree for 48 hours is sufficient to effect transmission.

Natural spread of peach mosaic from diseased to healthy trees occurs
in most areas and may be rapid in some. Rapid spread is often restricted to localized areas in which many trees in close proximity may become infected in a single year. Inspection records have shown numerous cases where individual orchards were 100 percent diseased 2 to 3 years after the first diseased tree appeared. Occasional diseased trees have been found up to 1½ miles from the closest previously diseased tree; such trees serve to start new centers of infection.

Control

The destructive nature of peach mosaic is such that many of the better freestone varieties cannot be profitably grown in the presence of the disease. Further, the rapid rate of natural spread emphasizes the importance of regulatory and quarantine procedures in preventing introduction of the disease into new areas and in eradicating it from areas where it is not well established. In areas where the disease is well established, further spread can be reduced by removal of diseased trees, thus reducing the virus reservoir from which spread takes place. In these areas the efficiency of control is increased by intensive surveys, followed by prompt removal of diseased peach trees and by removal of wild hosts. Such control is best operated as a regulatory operation because it must be on an area-wide basis.

The recent discovery of the mite vector of peach mosaic opens a new approach to control. It may be possible to increase the efficiency of control by reducing spread through control of the mite. Since there is no known cure for trees once diseased, diseased trees should still be removed.

Peach Ring Spot

The ring spot virus is of wide natural occurrence in many Prunus species in western United States. It is more prevalent in cherry than in peach, but since more is known about it as the cause of a disease of peach it will be described under the section on peach diseases.

The ring spot virus has apparently been present in stone fruit orchards for a long time, but it has not been generally recognized because of its latent nature in most fruit tree species; and because, even when symptoms are expressed, they are usually somewhat obscure. Probably the first record of occurrence of the ring spot virus was by Valleau in Kentucky in 1932 in relation to some plum virus diseases. A disease of mazzard cherry seedlings, which appears identical with ring spot, was described by Thomas and Rawlins in California in 1939. Ring spot was first described as a disease of peach in California in 1941 by Cochran and Hutchins. Subsequently, many workers found the ring spot virus widespread in stone fruit orchards in western and northeastern United States.

The discovery of the ring spot virus in peach was incidental to host determination studies with the peach mosaic virus. An attempt was made to inoculate sweet cherry trees by grafting them with scions from a peach tree supposed to be affected with a pure culture of the peach mo-
saic virus. Chlorotic ring and spotted patterns developed on the cherry trees, but when buds from the affected cherry trees were budded onto healthy peach trees, the peach trees developed ring spot instead of peach mosaic. This experiment showed that the original peach mosaic virus culture contained the ring spot virus as a contaminant and that sweet cherry was immune to the peach mosaic virus but susceptible to the ring spot virus. The cultures obtained in the sweet cherry represented pure cultures of ring spot virus separated from the peach mosaic virus because the peach mosaic virus was unable to infect the sweet cherry. The ring spot virus has now been found so prevalent in sweet and sour cherry orchard trees that studies on any virus affecting cherries must take into account the probability that the ring spot virus is also present as a contaminant in the culture.

How to Recognize Ring Spot

On peach the amount and severity of symptoms are dependent upon two factors—the differential reaction of peach varieties and the form of the virus present. Forms producing severe effects often cause severe die-back on varieties like Rio Oso Gem, J. H. Hale, Fay Elberta, and certain other freestone varieties, but may cause little damage to Lovell peach seedlings or to some of the clingstone varieties. Severely affected trees in the acute or initial stages of infection are retarded in the spring, many buds (both leaf and flower) die when partially open, and past season’s terminal growth may be killed, producing twig blight similar to that caused by the brown rot fungus. On some twigs necrotic cankers form, ranging in size from small brown spots in the bark to larger areas. Many of these cankers form around the bases of buds and envelop them.

Leaf symptoms. Symptoms show only in the first growth in the spring. Leaves arising from buds not entirely killed show all grades of patterns, from chlorosis to crowded ring patterns and shot holes (fig. 14). On varieties having red fruits, like Rio Oso Gem and J. H. Hale, the rings and necrotic spots develop red margins. Leaves developing necrotic rings and spots and also those with general chlorosis are usually shed during the first two or three weeks of growth. Leaves less severely affected remain on the tree and become green and their symptom patterns disappear. New leaves formed after the first flush of growth on diseased trees usually show no symptoms.

Fruit symptoms. Because most of the buds on severely affected peach trees or affected arms die, they set few fruits. Fruits which do set are undersized, tend to be flattened, and are usually sunburned because the sparse foliage provides insufficient shade. Mildly affected trees bear nearly normal crops and the fruits show no symptoms. Recovered trees also bear normal crops.

Distribution and Economic Importance

The ring spot virus is world-wide in its distribution and much more prevalent in cherry trees than in other stone fruits. If trees from most any cherry orchard in western United States are indexed by placing buds from them onto peach, the high percentage of
Fig. 14. A, Ring-spot-affected leaves of Lovell peach, showing rings, spots, and chlorotic patterns. B, Affected leaves of July Elberta peach on which the symptoms are predominantly of chlorosis but on which rings and shot hole occasionally occur; leaves like fourth to sixth from the left are usually shed during the acute stages, and ones like the others become green as the season progresses.
reaction on peach indicates that few cherry trees free of the ring spot virus will be found. Correspondingly, a high percentage of other stone fruit trees growing near cherry trees or produced from scion wood taken from trees near cherry orchards are carrying the ring spot virus. Surveys of peach orchards in southeastern United States, where cherries are not grown and where the peach trees have been propagated from local sources, have failed to indicate any ring spot virus in peaches. It is rare in peaches in eastern United States, even though a high percentage of the cherries grown there are carrying ring spot virus. Cherries and peaches, however, are not grown in as close association in eastern United States as they are in western areas, and most of the peach nursery stock planted in eastern United States is grown in southern areas where cherries are not grown. The wide occurrence of ring spot virus in cherries is primarily due to distribution of the virus in mahaleb and mazzard rootstocks grown from seeds from diseased trees; the virus passes through seeds of both species.

Ring spot virus causes an insidious type of disease. Symptoms are expressed during the early stages of infection and on some plants in this stage extensive damage is evident. Later these same plants appear to recover, and it is not known how much such plants are reduced in vigor or whether their yield or the quality of their fruit is much reduced. Many trees are so seriously injured during the shock stage that they are subject to sunburn, borers, and other orchard hazards, and never fully recover; some die. In such varieties as Rio Oso Gem, the ring spot virus is a limiting factor in production.

Fig. 15. Peach nursery tree showing severe shock and dieback symptoms of ring spot virus, typical on trees in the spring which were inoculated the previous fall. Healthy tree at right.
Fig. 16. Young peach tree showing necrosis and splitting of bark near the inoculation point caused by the ring spot virus. Inoculated arm on left was killed.

Ring spot virus causes serious loss to the nursery trade. When peach variety buds containing the virus are placed on healthy peach seedlings, the buds commonly die without making any perceptible union, resulting in poor stands. Buds which do grow produce smaller trees than do those which are free of the virus. Likewise, budwood from infected cherry trees produces poor stands of inferior trees.

**Hosts Affected**

All species of *Prunus* appear to be susceptible to the ring spot virus, but vary in the degree to which they are injured. Peach and sour cherry suffer severe shock during the early stages of infection, but tend to recover. Sweet cherry, on the other hand, suffers less initial shock but many trees show symptoms annually. Plums and apricots show various amounts of ring and mottled patterns, but appear to suffer little damage. Large numbers of wild *Prunus* species have been experimentally infected and some of these have been found naturally infected. Several species outside of the *Prunus* genus, including apple, rose, cucumber, and tomato, have been experimentally infected but have never been found naturally infected.

**Transmission, Incubation Period, and Rate of Natural Spread**

The ring spot virus spreads from tree to tree in orchards, but the natural vector is unknown. Spread is more rapid in some localities than in others and is more rapid where a large amount of inoculum is present; for example, where young orchards of virus-free trees are planted near old orchards.

The length of the incubation period appears to depend on the stage of growth of the tree at the time of infection. If young peach trees are graft-inoculated when breaking dormancy, symptoms may develop within two to three weeks. If trees in full leaf are inoculated, they may not show symptoms for six to eight weeks, or until the next growing season. If young trees are bud-inoculated in the fall, they usually show symptoms in the spring (fig. 15). Some forms of the virus cause severe necrosis in the bark around and below the inoculation points, and often kill the entire arm on which the inoculation is made.
Arms suffering severe necrosis develop rough cracked bark (fig. 16). In orchard trees symptoms usually are evident in the first growth of the spring and presumably infection took place sometime during the previous season.

The wide occurrence of the ring spot virus is a result of distribution in infected seeds and nursery stocks. The virus is readily carried in mahaleb and mazzard cherry seeds and occasionally in peach seeds. Seed used for growing rootstocks has been collected indiscriminately and much of it comes from infected trees. These seeds have been exchanged freely in the channels of world trade and have carried the virus with them. When
commercial fruit varieties are propagated on infected rootstocks they become infected but usually show no symptoms, since by the time growth from the variety buds has started the virus moving into them has already become latent. Such infected nursery trees carry the virus to orchards and serve as sources for orchard spread. The practice of indiscriminate collection of budwood from orchard trees is sure to increase further the amount of ring spot in the resulting nursery stock.

**Control**

The high prevalence of ring spot virus in stone fruits, particularly in species in which it is latent, makes eradication or even reduction by roguing orchards impractical. Some growers have questioned the advisability of using ring-spot-free nursery stock because such trees may be damaged when they become infected in the orchard. However, orchard spread in many areas is slow and healthy trees may grow for a long time without becoming infected, especially if they are isolated from old or infected orchards. The better yield and increased vigor of stocks produced from ring-spot-free budwood source trees make the use of such virus-free bud sources a good nursery practice.

The Shiro-fugen variety of flowering cherry is a good indicator host for determining the presence of ring spot. When shoots of the past season’s growth are budded with buds containing ring spot virus, necrotic lesions form around the bud insertion point (fig. 17). This species has generally been adopted as a test plant in the search for ring-spot-virus-free Prunus material.

**Peach Asteroid Spot**

The name “asteroid spot” was chosen because of the similarity of the symptoms on peach leaves to star-like spots. The disease was seen first in California in 1937 on peach shoots growing from plum understock. At about the same time it was discovered in peach orchards in central Texas. Subsequent surveys have shown asteroid spot to be widespread in southwestern United States and adjoining areas of Mexico, with occasional affected trees in Washington, Oregon, and Utah. Two affected trees reported in North Carolina were traced directly to infected nursery stock originating in Texas.

**How to Recognize Asteroid Spot**

Leaves on affected peach trees are normal in early spring. About 6 weeks after growth starts, small translucent light-green flecks form in the darker green of the fully expanded leaves. These are usually well distributed and become progressively chlorotic. The size of the spots is inversely proportional to the number present (fig. 18). In midsummer, scattered leaves turn yellow, but the fully formed spots on them remain yellowish-green, thus reversing the color pattern. Affected yellow leaves usually drop from the tree. The size and character of spots vary with different virus sources, indicating the
existence of forms of the virus. White-fleshed peach and nectarine varieties develop more pronounced symptoms than do yellow-fleshed ones.

**Economic Importance**

No controlled experiments have been made to determine whether asteroid spot causes reduction in yield or quality of peaches. Affected apricot trees drop a larger percentage of their leaves than do peaches, and trees which have been affected for several years appear to be perceptibly weakened.

**Hosts Affected**

Asteroid spot occurs naturally on
Fig. 19. Leaves of A, Late Santa Rosa plum and B Royal apricot affected with asteroid spot; normal leaf at center of bottom row.
peach, apricot, Japanese plum (fig. 19), domestica plum, Japanese apricot, and almond. No symptoms have been seen on sweet or sour cherry, but peaches have commonly become infected when budded from sweet cherry.

Transmission and Incubation Period

Transmission has been readily effected through grafts. Trees inoculated when just breaking dormancy developed symptoms about 8 weeks after leafing. Trees inoculated in the fall developed symptoms the following growing season. Limited observations indicate that asteroid spot does spread slowly in orchards, but most of the wide distribution is probably from use of infected budwood.

Control

Although the evidence indicates that asteroid spot causes only minor damage to peaches, it should be avoided in the propagation of nursery stock.

Peach Necrotic Leaf Spot

Necrotic leaf spot was first seen in 1940 in Michigan on peach trees which had been infected from buds placed on it from a Windsor sweet cherry tree. Since the original description was published, the disease has been found in many varieties of peach and is of wide occurrence in cherries, particularly in western United States. Necrotic leaf spot has been confused with other virus diseases, particularly ring spot and the early stages of western X decline.

How to Recognize Necrotic Leaf Spot

Infected peach trees produce normal foliage in the spring. The first symptoms appear as light-brown, membranous areas in the young unfolding leaves in midsummer (fig. 20). These die and turn brown rapidly, and the centers of most of the spots drop out, leaving clean-edged holes. The spots may occur on any portion of the leaves, sometimes in bands across the middle and sometimes in groups at the bases or tips of leaves. Symptoms develop only in unfolding leaves but continue to show after these leaves have matured. No symptoms have been seen on fruits. Necrotic leaf spot is easily distinguished from ring spot by the fact that symptoms of ring spot occur chiefly on the first leaves produced in the spring, whereas those of necrotic leaf spot occur only on leaves produced after midsummer. Also, symptoms of ring spot are limited to the first year after infection, whereas those of necrotic leaf spot occur annually on infected trees.

Distribution and Economic Importance

The harmful effects of necrotic leaf spot on peach and cherry orchard trees have not been precisely measured, but the amount of damage appears to be small. The disease does cause dwarfing in June-budded peach nursery stock. When infected buds
are forced, symptoms appear to be more severe in the young unfolding leaves, most of which drop. Loss of leaves in the early stage of growth inhibits growth and results in inferior small-sized trees.

**Hosts Affected**

The necrotic leaf spot virus has been seen on many varieties of peach and nectarine and appears to affect all in about the same degree. The virus has been recovered from cherry, but produces no symptoms on either sweet or sour varieties.

**Transmission, Incubation Period, and Rate of Natural Spread**

Peach nursery trees inoculated in August usually show symptoms in midsummer growth the following year.

The wide occurrence of necrotic leaf spot is believed to be chiefly the result of distribution of infected nursery stocks. No natural spread has been observed.

**Control**

Peach trees affected with necrotic leaf spot should be avoided as sources of propagating material.

PRUNE DWARF in peach (See description under prunes and plums, p. 88).

LINE PATTERN in peach (See description under prunes and plums, p. 89).
VIRUS DISEASES OF SWEET CHERRY

Cherry Western X Wilt and Decline

Western X wilt and decline, formerly referred to as common or quick wilt of sweet cherry and dieback of sour cherry, is an old disease in Utah. Growers have pointed out locations where sweet cherry orchards have gone out and have been replanted, and the new orchards are again rapidly wilting and dying. The fact that no other disorder of cherries which kills out entire orchards is known in Utah makes it reasonable to assume that death of the first orchards was the result of wilt and decline. The present distribution and general symptomatology of wilt and decline are nearly parallel in sweet cherry and in sour cherry. Conservative estimates indicate that wilt and decline may have been present in cherry orchards in Utah for half a century or longer.

The cause of wilt and decline remained unknown until 1942, when its virus nature was demonstrated by experimental transmission to young cherry trees. The disease in both sweet and sour cherries is now known to be caused by the western X virus and to occur only in cherry trees grown on mahaleb rootstock. It has been demonstrated experimentally that the western X virus is also the cause of western X little cherry occurring in trees on mazzard rootstock and of western X decline in peach, both of which are closely asso-

Fig. 21. Bing trees shown in plate 4, C, photographed 1 year later. Tree on left wilted in 1947, was dead in 1948; tree on right wilted in 1947, lived through winter, and showed advanced decline in 1948. Center tree is healthy. Apricot replant in left foreground. (Photographed in 1948)
associated with wilt-and-decline-affected trees in orchards.

**How to Recognize Western X Wilt and Decline**

**Tree symptoms.** In the orchard the wilt phase of the disease follows essentially the same pattern of development on either sweet or sour cherry and may be expressed at any time during the growing season. The first symptom is a light-green or yellowish color associated with a general flacid appearance of the leaves (pl. 4, C, and fig. 21). Severely affected trees sometimes die so rapidly that the leaves wilt and dry while still green in color. Such leaves gradually turn reddish-brown, making the diseased trees easily seen from some distance. These trees retain their dry leaves throughout the remainder of the summer and ensuing winter, and occasionally well into the following spring (pl. 4, D).

Most of the trees wilt less rapidly and their leaves turn light to yellowish-green; such leaves finally turn yellow and drop 2 to 3 weeks before normal leaf fall. Many such trees die during the ensuing winter but those less severely affected may continue to live indefinitely in a declined condition (fig. 22), or until killed by overproduction or by pests which attack weakened trees. Dieback and winter killing may be severe on declining trees. Decline varies from scarcely perceptible symptoms to severe stunting with successive death of individual limbs, and finally death of the entire tree.

Terminal growth of declined sour cherry trees is always shorter than

![Fig. 22. Young Napoleon cherry trees on mahaleb rootstock showing wilt experimentally induced following inoculations with buds from western-X-red-leaf-affected chokecherry (1944).](image-url)
normal and frequently forms a maze of small slender twigs, many of which may dry up during the summer or are winter-killed. The name “die-back” used to designate the disease in sour cherry, suggests the prominence of this feature. Portions of arms or whole arms on declining trees commonly die between growing seasons, resulting in ragged, off-shaped, brushy trees (pl. 5, D).

Leaf symptoms. Leaf symptoms are extremely variable and nondescript; on declining trees leaves emerge late in the spring and are small in size. In the early phases of decline, sour cherry leaves are only slightly reduced in size and develop yellow color between the veins, with a more intense band around the leaf margin (fig. 23). In advanced phases of decline, leaves are uniformly light green in color but are much reduced in size, attaining only one-fourth that of normal leaves. In still later phases, foliage becomes progressively sparse and may be entirely absent on many of the fruiting branches (figs. 24 and 25).

Fruit symptoms. Diseased trees of both sweet and sour cherries, especially in the early phases of decline, usually bloom later and more excessively than normal, and a correspondingly heavy fruit set results (pl. 5, E). Fruits on declined trees have no features that distinguish them from those on trees dying because of girdling from any cause. They are generally smaller than normal, are somewhat elongated, and ripen prematurely, but seldom if ever develop

Fig. 23. English Morello cherry leaves from western-X-affected tree, showing interveinal and marginal chlorosis. This type of leaf chlorosis is characteristic of western X decline on all varieties of sour cherry on mahaleb rootstock.
the normal flavor or color characteristic of the variety. Declined trees bearing heavy crops may wilt abruptly and die at any time during the growing season.

**Root symptoms.** The mahaleb understock roots of severely affected trees of either sweet or sour cherry are always damaged. The small rootlets and the bark of larger roots may be dead by the time symptoms appear in the top. The injury appears to start in the phloem, or food-conducting tissue, in the bark of the rootstock immediately below the graft union; often the bark below this point is extensively killed (fig. 26). Such injured phloem is largely nonfunctional, cutting off the food supply from the leaves, resulting in starvation and death of the feeder roots; roots so injured in turn fail to absorb soil water, thus causing the top of the tree to wilt. The same toxic material which kills tissue below the bud union may also account for part of the killing of rootlets. Affected roots die progressively from their tips toward the trunk. The degree of wilting and the rapidity of death are proportional to the amount of root injury. The woody cylinder of the trunk below the bud union and that of the large roots become dark in color compared with healthy roots (fig. 26). Even on dying trees, stem tissues above the bud union may retain their normal color for a considerable time after the roots are dead.

**Diagnosis and sequence of symptoms.** Wilt and decline symptoms are restricted to trees on mahaleb rootstock. Similar symptoms could result from girdling of the tree trunks by gophers or crown borers, or as a result of winter injury, me-

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*Fig. 24.* Montmorency cherry trees on mahaleb rootstock (right and left), showing wilt and decline. Center tree not inoculated. The diseased trees were inoculated in 1941 with buds from western-X-red-leaf-affected chokecherry; photographed October 1, 1942. Leaves at that date exhibited rusty-brown color characteristic of the late-autumn stage of decline; left tree was also severely defoliated.
chanical injury, crown rot, or other causes; milder forms of decline could be confused with the effects of crown gall, nutritional deficiencies, water deficit, continued poor culture, competition from cover crops, and other cultural hazards. Diagnosis is further complicated by the fact that the casual virus usually cannot be transmitted from the wilting tops of declined trees.

A knowledge of the sequence of the symptoms is helpful to reliable diagnosis. This is effectively illustrated by the response of 33 two-year-old sour cherry trees on mahaleb rootstock (9 English Morello, 12 Early Richmond, and 12 Montmorency) experimentally inoculated in 1941 with the western X virus from red-leaf-affected chokecherry. Of the 33 trees inoculated, 28 became diseased. Five (2 Early Richmond and 3 Montmorency) escaped infection. Four (3 English Morello and 1 Early Richmond) died the first season following inoculation. The remaining 24 survived the wilt phase, which developed during the first year period, and during subsequent years continued to exhibit varying degrees of decline. Six of the 24 succumbed prior to 1946 and 18 (1 English Morello, 8 Early Richmond, and 9 Montmorency) were alive at the end of the growing season of 1948.

Of the four trees which died the first season following inoculation, three collapsed suddenly and were dead by late July. The fourth tree wilted less rapidly, but gradually lost all of its leaves and was dead by late autumn. Wilting of all four trees was far too rapid to permit the development of any chlorotic leaf patterns.

Of the six trees which survived the first year but succumbed during the decline phase prior to 1946, two English Morellos died the second year following inoculation, two more and one

Fig. 25. Montmorency cherry tree on mahaleb rootstock inoculated from western-X-red-leaf-affected chokecherry in 1941, showing advanced decline; photographed in August 1944. The tree succumbed late in 1945. Trees at left and right are healthy controls that have not been inoculated.
Fig. 26. Trunk of a 4-year-old Montmorency cherry tree affected with western X wilt and decline, with outer bark removed to show dark color of the injured mahaleb rootstock below the union and the apparently normal light-colored tissue of the Montmorency trunk.

Early Richmond the third year, and one English Morello survived in a declined state until 1946, 5 years after contracting the disease. The 18 diseased trees surviving in 1948, when the experiment was terminated, exhibited widely varying degrees of decline. None showed any indications of recovery. The higher percentage of infection and more severe effects on the English Morello trees indicate less tolerance of that variety.

**Experimental Transmission and Natural Spread**

Wilt and decline was first produced experimentally in Utah in 1942 in young cherry trees growing on mahaleb rootstock. These were inoculated the previous fall with the western X virus in buds from naturally occurring red-leaf-affected chokecherry. Wilt and decline has also been produced on sweet and sour cherries with inoculum from western-X-decline-affected peach, and from sweet and sour cherries showing western X little cherry. In no case has wilt and decline been produced in cherry trees growing on mazzard rootstock; when trees on mazzard rootstock are infected they produce the western X little cherry, a disease which is described under a separate section, p. 60.

When 2- to 3-year-old cherry trees are inoculated in early summer, they may show wilt in late autumn; those inoculated in the fall usually develop symptoms shortly after the start of growth the following summer (pl. 4, D). Different sources of inoculum have caused differences in degree of severity of symptoms on inoculated trees, indicating the existence of strains of the western X virus.

Kaloostian and co-workers in Utah also produced wilt and decline in sweet cherry trees on mahaleb rootstock with the geminate leafhopper which had previously fed on cherry trees showing western X little cherry symptoms, western-X-decline-affected peach, or red-leaf-affected chokecherry.

**Distribution and Economic Importance**

Wilt and decline of sweet and sour cherries in Utah occurs in the same general areas as western X decline in peach and western X red leaf in western chokecherry. It also closely parallels the distribution of western X little cherry, which occurs on cherry
Fig. 27. Map showing high incidence and distribution of western X wilt and decline, A, D, and little cherry, B, in adjacent orchards and similar high incidence of X decline in adjoining peach orchard, C. The higher incidence of disease in Lambert and the large number of missing trees in the Bing orchard adjacent to the Lambert points to the Lambert as the major source of the western X virus.
trees on mazzard rootstock. It is most abundant in areas where diseased chokecherries are nearby, indicating spread from the chokecherries. Chokecherries are more abundant and closer to cherry orchards in Davis, Weber, and Box Elder Counties than in Utah County, and, correspondingly, more wilt and decline occurs in the cherry orchards in those counties.

The devastating effect of the disease in certain localities and certain orchards is well illustrated by the sweet cherry orchards shown in the map in figure 27. In 1947, when the data were taken, orchard A (shown on map in figure 27) was primarily Bing on mahaleb rootstock, approximately 30 years old, originally containing 315 trees, of which 59 percent were dead or missing, 9 percent of the remaining 129 trees showed wilt and decline, and only 32 percent of the original planting was still healthy. All Bing trees at the end of the orchard adjacent to the Lamberts had been killed and removed—their location is represented by the vacant space growing weeds as shown in the foreground of figure 28. Orchard B was Lambert on mazzard rootstock, originally containing 84 trees, of which 85 percent were still standing, but all showed western X little cherry (fig. 29). Orchard C was 12-year-old Elberta peach, showing a high percentage of western X decline. Orchard D originally contained 84 Lambert trees, 72 on mahaleb rootstock and 6 at each end on mazzard rootstock. All trees in this orchard had become diseased. The few
living trees on mahaleb rootstock showed wilt and decline and those on mazzard, while all still living, showed western X little cherry. Facts demonstrated in these orchards are, first, an example of the devastating effects of the western X virus; second, the high percentage of disease in different stone fruits where they are grown in close association; and, third, the differential expression of the western X virus in sweet cherries on different rootstocks. This incidence of disease in orchard A indicates a source of the virus at the south end, but does not point to any one host as the reservoir of the virus. Peach could well be maintaining the virus because affected trees are not killed. Sweet cherry on mahaleb rootstock would appear to be a poor source of the virus because infected trees are short-lived and also the virus can only rarely be graft transmitted from wilted trees. On the other hand, sweet cherry on mazzard appears to be long-lived and an excellent source of the virus, both for graft and insect transmission.

Control

The first essential in the control of western X wilt and decline in cherries is the choice of an orchard site. Preferably, new orchards should not be planted adjacent to old orchards, particularly orchards of peach or cherry where western X virus is known to be present. If new orchards are to be planted near wild brushland, the native brush should be killed in a border of 500 feet or more adjacent to the edge of the orchard prior to planting. If chokecherries, particularly diseased ones, are prevalent in the brushland, they are a serious locational hazard.

Wilt-and-decline-affected trees are worthless and should be removed, even though there is no evidence to indicate that they serve as reservoirs of virus from which further spread can take place.

A second means of control, which can be used in orchards where isolation is not practical, is the use of trees composed of mahaleb root, trunk, and scaffold arms with the desired cherry top-worked separately on a large number of the mahaleb arms. Experimental evidence indicates that although mahaleb cherry tissue is killed below the bud or graft union when sweet or sour cherry top-worked on it becomes diseased, the mahaleb tissue is immune to the virus to the extent that the virus will not pass from diseased cherry through it to other top-worked arms (pl. 4, A). When sweet or sour cherry arms become diseased on mahaleb framework trees, they can be removed and the arm either re-top-worked or the other arms allowed to fill in the gap. The advantage of the mahaleb frameworked tree over that of the trees grown from a single top variety budded low on mahaleb is that should a tree contract the western X virus, only a small portion is lost and this can be removed, the unaffected arms maintaining the tree. Whereas, when trees grown from a single bud become diseased, there are no unaffected arms to maintain the rootstock and the whole tree wilts and usually dies, or, what is more serious, lives on in an unproductive state. The mahaleb framework tree was originally developed in California for control of the buckskin disease of sweet cherry, a disease thought to be caused by a virus closely related to western X virus. In California some graft in-
compatibility has been experienced with certain varieties of sweet cherry (particularly Black Tartarian) on certain mahaleb seedlings. To avoid this, Black Tartarian can be grown on double-worked trees with a mazzard sandwich between the Black Tartarian and mahaleb.

**Western X Little Cherry**

Western X little cherry, like western X wilt and decline, has without doubt been present for a long time in cherry orchards in Utah, but it was not until 1946 that the disorder was recognized as a specific disease. Prior to 1946, a small-fruit condition had been reported from British Columbia, and early in 1946 from Washington State; however, as in Utah, the cause was unknown. Transmission studies from 1946 through 1948 proved that the disorder in Utah was caused by a virus, and finally established the disease as an additional member of the western X virus disease group. Western X little cherry has been found only in orchards of sweet and sour cherries growing on their own or on mazzard rootstocks.

**How to Recognize Western X Little Cherry**

**Fruit symptoms.** Fruits on individual arms or larger portions of trees which remain abnormally small and fail to mature and color at normal maturity time are the most reliable diagnostic symptoms of western X little cherry. The affected fruits are striking in contrast to normal ones and frequently are little more than one-half normal size, are inclined to be pointed, and retain the color of immature cherries (pls. 4, B, and 5, A-C). The color of affected fruits varies with varieties, those of Napoleon remaining dull white, those of Montmorency pink to light red, and those of dark-colored varieties, like Bing and Lambert, becoming darker red. In all cases the color of the affected fruits is much lighter than that of mature normal fruits. Affected fruits of all varieties, particularly at the normal time of maturity and later, generally exhibit a dull luster in place of the normal glossy appearance (fig. 30, pl. 5, A-C). They also lack the flavor and sweetness of normal cherries. When not harvested, affected fruits remain firmly attached to the tree, yet they never mature or acquire the color normal for the variety.

The distribution of affected fruits in diseased trees is variable. It is not unusual for them to be restricted to one or two branches the first year a tree is diseased. Even on trees diseased for several years, all the fruits are rarely affected. On some trees, affected and normal cherries occur on different spurs interspersed on the same arm, and in some cases even on the same spur. The reduction in size and other effects on fruits on different diseased trees varies, indicating that individual trees may be infected by different strains of the virus. Cultural practices, nutritional level, size of fruit load, and variety of cherry also affect the reduction in fruit size on diseased trees. These same factors can delay maturity and reduce fruit size to some degree on trees which are not virus infected and
thus complicate diagnosis. Diagnosis is much easier in well-cared-for orchards and on trees where some arms bear affected fruits and others normal fruits. Diagnosis is generally difficult in poorly cared for orchards.

**Other symptoms.** Symptoms other than small fruits are usually present and aid in diagnosis. Stipules may become much enlarged and appear as small leaves beside the bases of the leaf petioles where they are attached to the twigs (fig 31). These are pronounced on some trees and persist much longer than the small stipules on normal trees. Bud scales remain attached around the basal portion of new growth for several weeks after they would normally have been shed. Shortened internodes, as a result of suppressed growth and excessive branching, provide a distinct type of rosetting which in severe cases is striking (fig. 32). The margins of leaves in such rosettes become undulated and wavy.

Following harvest, the leaves on diseased trees are usually lighter green than on normal trees of the same variety. Late in the autumn terminal buds frequently break dormancy, producing short secondary, branched shoots (fig. 33). Dieback and winter killing are pronounced on severely affected trees. Older diseased trees show indications of general decline.

**Distribution and Economic Importance**

Western X little cherry occurs widely in western North America. The disease has been reported on both sweet and sour cherries from Washington, Oregon, Idaho, and Utah, and on sweet cherries from Okanagan Valley in British Columbia, Canada. The buckskin disease of sweet cherry, occurring in California, is essentially identical with western X little cherry.

Surveys in Utah have shown west-
ern X little cherry to be most prevalent and severe in old orchards, ranging from 20 to 50 years in age, although it is frequently found in much younger plantings. The disease is more prevalent in the northern fruit-growing counties of Box Elder, Davis, Salt Lake, and Weber than in Utah County. It has not been found in the cherry orchards of Washington County.

Detailed studies made in one block of four orchards of cherry and peach in Weber County in 1947 showed that 85 percent of the trees in a 30-year-old Lambert orchard were affected. Approximately the same percentage of trees in an immediately adjoining orchard of Bing cherry on mahaleb rootstock was either showing wilt and decline or had died and been removed. A similar high percentage of western X decline occurred in an adjoining orchard of Elberta peach. More extensive surveys, particularly in Weber and Davis Counties, would no doubt show other locations of similar high incidence and close relation of infection in peach and cherry.

The apparent lower incidence of the western X virus in cherries and peaches in Utah County prompted extensive studies in order to determine the feasibility of control by diseased-tree removal. In 1951 approximately 25,000 trees (mostly sweet cherry) on 75 properties were exam-
ined for fruit symptoms and 181 infected trees were found on 13 properties. In one of these orchards, 75 of the 150 trees were diseased. In two others, 27 and 37 of 300 trees were diseased. No data are available on the rootstocks of the 25,000 trees and it is possible that many of the 62 orchards in which no fruit symptoms were found were on mahaleb rootstock and would not have shown fruit symptoms.

The loss from western X little cherry results chiefly from unsalability of fruit borne on diseased portions of trees. Fruit on the unaffected parts appears to be normal in size and quality. The diseased portion may be restricted to a small part of the tree for several years but eventually enough of the tree will be involved to prevent it from producing an economic crop. Growers are inclined to keep diseased trees until they become unproductive. In the meantime, spread may have taken place from them to other orchard trees. Once well established, the disease continues to spread, making orchards unprofitable, in some cases at a relatively early age.

Sweet cherry orchards are slow to fruit and hence expensive to establish, but once they have reached fruiting age should have a relatively long productive life. When such orchards are ruined by western X little cherry, the monetary loss is greater than for peaches, which fruit at an earlier age.

Transmission and Natural Spread

Experimental transmission of western X little cherry is readily effected by grafting buds or scions from diseased trees on healthy trees. Cherry nursery trees on mahaleb rootstock, inoculated in late fall from trees showing typical western X little cher-

Fig. 32. Branches from Napoleon trees on mazzard rootstock: Three branches at left from tree inoculated with the western X virus, showing rosetting, shortened internodes, excessive branching, and resultant clumping of leaves. Branch at right is from healthy tree.
Fig. 33. Comparable branches to those in fig. 32 with leaves removed; normal branch at right. Affected branches show proliferation of stipules and tendency to out-of-season growth of buds.

...show wilt and decline soon after the beginning of growth the following spring. Trees on mazzard rootstock, or mazzard seedlings, similarly inoculated show stunting and rosetting and other symptoms characteristic of the disease. Similarly, peach or chokecherry inoculated from western-X-little-cherry-affected trees develops typical X decline and red leaf disease, respectively. Experimental transmission has also been effected with the geminate leafhopper. See more detailed account under section on western X diseases of stone fruits, p. 17.

The rate of natural spread through orchards is variable, and appears to be proportional to the number of diseased trees in the vicinity. The higher incidence of disease in orchards near diseased chokecherries or near orchards of western-X-decline-affected peach, as compared with isolated orchards, is indicative that spread takes place from these to the sweet or sour cherry. Correspondingly, the high incidence of wilt and decline in cherry orchards on mahaleb rootstock around cherries affected with western X little cherry indicates spread from the affected cherries on mazzard rootstock to those on mahaleb rootstock. Wilt-and-decline-affected trees on mahaleb rootstock, even though they are more strikingly affected, are less hazardous because they usually die shortly after they are infected; even if they remain alive in a declined condition, transmission studies indicate that they provide only a very limited source of the virus.

**Control**

The control of western X little cherry is similar to that for wilt and decline and consists, first, in the choice of an orchard site isolated from other stone fruit plantings. If the new orchard is planted near other orchards, any diseased trees in such orchards should be removed, preferably before the new orchard is planted. If the new orchard is planted near wild brushland, the native brush should be killed in a border of 500 feet or more adjacent to the new orchard.

The second essential of control is prompt elimination of diseased trees from the young orchard, should they appear. By such prompt removal, buildup of the virus can be retarded.

The third means of control is the use of trees composed of mahaleb root, trunk, and scaffold arms, as outlined under the section of “Control” described for western X wilt and decline in sweet cherry, p. 59.
Prior to 1944, necrotic rusty mottle\(^5\) of sweet cherry, referred to commonly as “cherry leaf spot,” “cherry leaf drop,” or “cherry leaf rust,” had become widespread in Utah and was reported as especially destructive in orchards in Utah and Davis Counties. In these counties, a large number of cherry orchards were commercially unprofitable; the yields of others were seriously reduced. In 1944 the virus nature of this disease was proved. As a result, the disease immediately took on added significance as a threatening factor in cherry production. However, research since 1944 has shown that the necrotic rusty mottle virus, perhaps because of the inefficiency of its natural vector, spreads slowly in Utah orchards. Also, it is well established that the virus has been spread chiefly in nursery stock and that further spread is unnecessary.

How to Recognize Necrotic Rusty Mottle

Leaf symptoms. Trees affected with necrotic rusty mottle virus show a marked delayed development of leaves and blossoms in the spring (fig. 34). Leaves on diseased trees during the first 3 to 5 weeks after petal fall appear healthy. At this stage they are dark green and have reached normal size. Brown necrotic spots of varying size, shape, and distribution develop rather abruptly in the more mature leaves (fig. 35). The amount of leaf necrosis varies; some cherry varieties are more affected than others and symptoms on a single variety may be more severe in one season than in another. In mazzard cherry seedlings and in the Lambert variety this initial necrosis, especially in cool wet weather, may be extensive. Severe necrosis usually results in partial defoliation.

Two to three weeks after the onset of the initial leaf symptoms, a second partial defoliation usually occurs. Many of the remaining leaves, with or without necrotic spots, become prematurely senescent (fig. 36) and fall. This second partial defoliation reaches peak expression some 2 or 3 weeks before harvest and on severely affected trees may result in a loss of up to 85 percent of the leaves.

About the time the fruits ripen, yellowish to rust-colored chlorotic areas appear in the older surviving leaves. Affected leaves may be generally distributed in the tree, and the chlorotic areas on them closely resemble those described for the mild rusty mottle disease of cherry occurring in Oregon and the rusty mottle disease occurring in Washington. Late in the season, usually after harvest the necrotic areas in the affected leaves frequently fall out, producing a conspicuous shot-hole effect. The rusty chlorotic areas may occur on the same leaves with the brown necrotic spots and the shot-holes during the latter part of the season. Apical leaves on current-year branches and on water sprouts (suckers) seldom show any symptoms.

\(^5\)The similarity of leaf symptoms to those of the rusty mottle disease, named by Reeves in Washington, suggested the name “rusty mottle.” The necrotic phase, however, was not a feature of rusty mottle, thus “necrotic” was incorporated into the name to distinguish this disease from rusty mottle.
General autumnal senescence and defoliation on diseased trees frequently occur 2 to 6 weeks earlier than on healthy trees. Senescent leaves from diseased trees often develop prominent rings and line patterns of dark green on a background of yellow, brown, or brilliant red (fig. 37).

Tree symptoms. On severely affected trees, part of the buds and leaf spurs on the older and lower portions of the branches are killed. This killing results in bare, rangy branches with terminal tufts of foliage (fig. 38). In the more advanced stages of the disease, the older branches of the tree succumb and numerous water sprouts may develop from the trunk or from the lower portions of the main branches. Late in the season leaves on these basal water sprouts usually show typical necrotic spotting characteristic of earlier symptoms on other parts of the tree.

On young branches of certain varieties, especially Lambert, numerous brown cankers occur in the cortex, which appear externally as blister-like lesions. New growth forces these lesions open, thus causing pronounced roughening of the bark. Frequently this feature is prominent.

Distribution and Economic Importance

A survey to determine the prevalence of necrotic rusty mottle was made of 129 sweet cherry orchards in
Fig. 35. Lambert cherry leaves affected with the necrotic rusty mottle virus, showing the general distribution of variously sized and shaped lesions. The lesions appear first as water-soaked areas, which turn reddish-brown and later into lighter brown necrotic spots. Necrotic tissue is usually surrounded by a dark reddish-brown border.

Weber, Davis, Salt Lake, and Utah Counties in 1944. Twenty-seven (20.9 percent) of the 129 orchards surveyed showed the disease. These were distributed in the counties as follows: Davis, 9 of 27 (33.3 percent); Utah, 14 of 31 (45.0 percent); Salt Lake, 2 of 9 (22.2 percent); and Weber, 2 of 62 (3.2 percent). Of 24,183 trees examined in the 4 counties, 576 (2.4 percent) showed necrotic rusty mottle. In 5 of the 27 infected orchards the percent of diseased trees ranged from 20.7 to 44.4; in 5 others, above 10 percent. In Utah County nearly 8 percent of all the trees examined were diseased; in 7 orchards containing

1,222 trees, 259 (21.2 percent) were diseased.

Subsequent to the 1944 survey, necrotic rusty mottle has been found in many additional orchards in the 4 counties. The disease has also been
found in Box Elder and Cache Counties. A few trees showing what appears to be necrotic rusty mottle have been located in Washington County.

Necrotic rusty mottle results in a weakened tree, which produces a correspondingly reduced yield of inferior fruit. The maintenance cost of such trees is equivalent to that of healthy trees, and thus represents reduced profits.

**Hosts Affected**

Necrotic rusty mottle occurs principally in orchards of Lambert, Bing, and Napoleon. The disease has been found occasionally in Windsor and Black Republican. Infected Black Tartarian trees showed no symptoms; hence this variety can become a symptomless carrier of the virus. The necrotic rusty mottle virus has been transmitted experimentally to choke-cherry, to peach, to Montmorency, Early Richmond, and English Morello sour cherries, to May Duke cherry, and to mazzard cherry seedlings. Symptoms were produced on mazzard cherry seedlings, but no recognizable symptoms were expressed on any of the other hosts. Infection in them was determined by taking buds from the inoculated trees and placing them on healthy Lambert cherry, resulting in the production of typical symptoms in the Lambert. Lambert is the most severely affected variety of sweet cherry tested in Utah and is considered the most reliable test plant. Mazzard cherry seedlings react variably and some have shown pronounced symptoms.

![Fig. 37. Lambert cherry leaves taken from tree inoculated with necrotic rusty mottle virus, showing irregular ring and line patterns characteristic of the disease in senescent leaves in late fall.](image-url)
Transmission and Rate of Natural Spread

Transmission studies have shown that the necrotic rusty mottle virus is infectious and easily transmitted when buds or scions from diseased trees are grafted on healthy trees or rootstocks. If young Lambert nursery trees or mazzard cherry seedlings in the greenhouse are inoculated when just breaking dormancy, symptoms develop in as few as 30 days; nursery trees growing out-of-doors inoculated in May show symptoms in 6 to 8 weeks; nursery trees inoculated in August or early September are uniformly infected and develop symptoms the following spring.

Evidence of virus spread by propagation practices is impressive. Orchardists in their attempts to establish branches of a pollinator variety in trees inadvertently have transmitted the necrotic rusty mottle virus. One grower in Davis County is known to have introduced the virus (by use of buds) from a single infected but symptomless Black Tartarian tree into every third tree in every third row of his rather extensive Lambert planting. In this operation all budded Lambert trees in the orchard developed necrotic rusty mottle and showed severe dieback, while the pollinator branches of Black Tartarian grown from the bud insert in every case remained symptomless (fig. 38).

Studies further indicate that nurserymen have unwittingly played a major role in the distribution of the necrotic rusty mottle virus in Utah. In one old orchard on the Mapleton Bench, Utah County, all trees were removed except 8 large Lambert trees and the area was replanted. Of the replants, 105 were Lambert; all of these were in advanced stages of necrotic rusty mottle when observed at 6 years of age in 1944. None of the older trees of the same variety between which the diseased trees were planted had contracted the disease.

In a 15-year-old block of Bing near Provo, 59 of 89 of the original trees planted showed typical necrotic rusty mottle. A block of Lambert trees of the same age and immediately adjacent to the 59 infected trees at the north was found entirely free. The fact that none of the Lambert trees was affected indicates that natural spread had not taken place in the orchard. The high incidence in the Bing indicates that they were in-
fected when planted. In another case, a 4-year-old block of Bing cherry trees in South Ogden, 25 of 69 trees were found showing symptoms of necrotic rusty mottle, with no evidence of spread into an adjoining 6-year-old block of Lambert trees on the same property. Infection in young orchards has been traced back repeatedly through the nursery to infected mother trees from which buds were taken.

Although most of the evidence indicates no natural spread of the necrotic rusty mottle virus from tree to tree in the orchard, a few cases on record cannot be accounted for on any other basis. It may be that in some locations natural spread does take place, but, if so, the rate is exceedingly slow.

**Control**

The first essential of control of necrotic rusty mottle is prevention of further spread in nursery stock. Nurserymen should cut buds only from source trees known to be free of the virus. The use of progeny-tested source trees maintained in budwood mother blocks is encouraged over cutting of buds indiscriminately from orchards. Growers grafting pollinator arms in trees should likewise obtain bud and scion wood only from trees known not to be infected.

In view of the destructiveness of necrotic rusty mottle and its exceedingly slow rate of spread, attention should be given to the possibility of eradication. Certainly diseased trees should be removed as soon as they become unprofitable.

**Dixie Rusty Mottle**

Dixie rusty mottle is one of three rusty mottle virus diseases of sweet cherry in Utah. The other two, necrotic rusty mottle and mild necrotic rusty mottle, so far as known are confined to the northern and central parts of the state. Dixie rusty mottle, on the other hand, occurs in Washington County, Utah's "Dixie." There it is established as a destructive disease of sweet cherry and is also generally distributed in peach. After its discovery in 1944 Dixie rusty mottle was shown to be caused by a virus readily transmitted from diseased to healthy cherry and to peach, both in the greenhouse and naturally in the orchard. Because of the destructive nature of the disease and also because of apparent natural spread in the orchard, Dixie rusty mottle must be considered a definite threat to the cherry industry in Utah.

**How to Recognize Dixie Rusty Mottle**

Dixie rusty mottle, like necrotic rusty mottle, is characterized by necrotic spots in the leaves, leaf fall, and dieback in sweet cherry. It is distinguished from necrotic rusty mottle by a more rapid killing of trees, particularly the Lambert variety, by the production of leaf symptoms on Black Tartarian, and by the fact that the necrotic leaf spots, at least in their senescent stage, are surrounded by a halo of yellow or chlorotic tissue with a dark-green to brown border. In cherry varieties, especially Lambert, blossom and leaf development on diseased trees are retarded. Leaves are normal
Fig. 39. Lambert cherry leaves showing bordered irregular rings characteristic of late-season symptoms of Dixie rusty mottle.

in appearance, size, shape, and color for 4 to 6 weeks after growth starts, but then the older leaves develop small necrotic areas of varying sizes and shapes. Affected leaves rapidly take on the appearance of premature senescence, turning yellow, orange, and reddish, with the chlorophyll degenerating in varying patterns of artistic design (fig. 39). On severely affected trees most of the leaves are shed, resulting in partial to complete defoliation by mid- to late-summer. The disease also results in death of auxiliary buds and in varying amounts of dieback. On Lambert large limbs may be killed (fig. 40) and often whole trees die.

Fruits on affected trees may be variously misshapen and reduced in size. On experimentally infected Bing trees fruits were decidedly bumpy (fig. 41). The raised areas are apparently formed from normal tissue and the depressions are a result of restricted growth caused by necrotic tissue in the flesh immediately below the depressions.

Dixie rusty mottle virus must be classed as a “killer.” In mixed plantings, the Lambert variety is strikingly affected and many trees may be killed. Trees of Bing, Black Tartarian, and Napoleon, while visibly affected, are not usually killed and serve as reservoirs of virus from which further spread may take place. The severity of symptoms, even on Lambert, appears to vary with seasons and cultural conditions.

**Distribution and Economic Importance**

No comprehensive orchard survey has been made to determine the distribution or amount of the disease in Washington County; however, the
disease has been observed in orchards near Leeds, Hurricane, Toquerville, and La Verkin. Two cherry orchards, one in Leeds and the other in Toquerville, have been studied, the incidence of the disease in them mapped, and the degree of its destruction recorded. Data from these two orchards indicate the destructiveness of the disease as follows: One hundred thirty-eight trees were originally planted in the Leeds orchard; these were about 35 years old at the time the data were taken in 1949; 53 (38.4 percent) of the original plants were missing or dead, and it appears logical to assume that these had succumbed as a result of the disease. Many of the remaining trees in this orchard showed leaf symptoms. In the orchard at Toquerville 32 percent of the trees were infected. The apparent natural occurrence in peach (fig. 42), adjoining diseased cherries, adds to the economic importance of the virus in Washington County.

**Transmission and Rate of Spread**

Dixie rusty mottle is readily transmissible to cherry trees with buds or bark shields from diseased sweet cherry or peach trees. The high percentage of infection obtained indicates that the causal virus is well distributed in the tissues of diseased trees. In the greenhouse, cherry trees inoculated when just breaking dormancy developed symptoms in 4 to 6 weeks. In the nursery row, cherry trees inoculated in the fall showed disease the following summer. Bing cherry nursery trees developed from diseased buds become large enough

Fig. 40. Lambert cherry tree showing advanced stage of Dixie rusty mottle. The virus kills terminal and lateral buds; in a relatively short time entire branches succumb. (This virus is a more rapid killer than the necrotic rusty mottle virus.)
to be salable and therefore can be an important means of spread.

The occurrence of Dixie rusty mottle in all three varieties in the orchard at Leeds indicates that natural spread from tree to tree has taken place within the orchard. The high percentage of disease in a peach orchard adjoining the affected cherry orchard at Leeds is further evidence of natural spread.

**Control**

Since the disease appears to be limited to a relatively small isolated section of the fruit-growing area, measures should be taken to prevent its escape to the major areas. No nursery stock grown in the infected area should be transported outside. Surveys should be made to determine the feasibility of eradication.

Within the infected area, growers would be more sure of avoiding the disease if they used nursery stock grown in other areas to start new orchards. New orchards should not be planted near infected orchards. Infected trees appearing in orchards, especially young orchards, should be removed promptly.
Fig. 42. Symptom pattern in Elberta peach leaves from trees inoculated from Lambert cherry trees showing Dixie rusty mottle.

Ring Spot
(See Peach Ring Spot)

Ring spot of stone fruits is described in detail under the section on peach diseases, p. 40. Because the causal virus occurs so generally in sweet cherry in Utah, a brief additional section concerning its effects is included here.

Surveys in sweet cherry orchards have shown many trees with symptoms in varying degrees. Indexing of symptomless orchard trees has shown that many are infected. In fact, search for variety trees free of the ring spot virus has shown it nearly universal in sweet cherry and so far no virus-free trees of some varieties have been found.

The wide occurrence of ring spot
virus in cherries is traceable to distribution in seeds, particularly those of mazzard and mahaleb rootstocks. The virus has been further distributed through use of buds and scions of trees grown on these infected rootstocks. Natural spread has been observed in peach orchards and is presumed to take place also in cherry orchards, but no natural vector is known.

How to Recognize Ring Spot

Symptoms on sweet cherry are extremely variable. The most common are chlorotic rings and irregular patterns often composed of overlapping rings. The rings and patterns often become necrotic and the necrotic portions fall out, leaving a skeletonized or lace-leaf condition (fig. 43). Trees showing one type of symptom will usually show the same type annually, but the severity of expression may vary. Experimental transmission results indicate that nursery trees usually express symptoms similar to trees from which they were infected; thus the wide variation in expression is a result of the existence of many forms of the virus. Cherry varieties react variably to a single form. Black Tartarian in general is more severely affected than is Napoleon or Lambert.

Economic Importance

Because of the extreme variability of reaction, the amount of damage caused by ring spot virus in sweet cherry is hard to determine. Preliminary results indicate that vigor of infected trees is reduced. Nursery stock propagated from diseased trees

Fig. 43. Necrotic ring spot symptoms on sweet cherry leaves. The tattered condition results from the dropping out of necrotic portions. The leaf on the right is normal.
is much reduced in vigor as compared with that from ring-spot-virus-free trees. Buds from virus-free trees produce a better stand of nursery stock as well as a much higher percentage of marketable trees.

**Control**

The wide occurrence of ring spot virus in orchards and the relatively small amount of damage it causes make any measures for treatment of orchards already planted impractical. The increased vigor of young orchards planted with ring-spot-virus-free nursery stock makes such planting seem worthwhile. Nurserymen will find the use of ring-spot-virus-free budwood sources an economical nursery practice.

**Rasp Leaf**

The name “rasp leaf” was applied to this disease because of the similarity of the abnormal outgrowths on the lower sides of affected leaves to the corrugated cutting surfaces of a wood rasp. Rasp leaf occurs in a number of sweet cherry orchards in Utah and has been seen in various localities of northwestern United States and adjoining areas of Canada. Outgrowths (enations) from the lower side of cherry leaves are fairly common, particularly in fast-growing shoots of orchard trees and in nursery stock. Not all such enations are indicative of the rasp leaf disease. Some are known to be caused by other viruses and some may be produced as a response to chemicals or injuries.

**How to Recognize Rasp Leaf**

The characteristic symptom of rasp leaf is outgrowths from the lower surface of leaves. The size, shape, and number of such outgrowths vary (fig. 44), but on severely affected trees the effects are striking. The outgrowths, which extend from the interveinal tissue, often resemble the edge of a leaf complete with serrations or may be merely rows of papillary tissue with gland-like structures at their apices. On severely affected trees leaves may be much distorted and reduced in size; hence the enations are crowded, giving the leaves a lacerated appearance. Such affected leaves tend to roll upward. Trees may be completely or more commonly only partially affected; where only part of a tree is affected there is a tendency for the affected parts to be the lower arms.

**Economic Importance**

The effects of rasp leaf on trees are variable. Some trees which were completely affected have been observed. Such trees produce no marketable fruit and often are dead at the end of 1 or 2 years. Other trees only partially affected have been observed to exist for several years without much spread to other arms of the tree.

The loss from rasp leaf has been small because of its limited distribution. In certain locations it has been observed to spread and cause considerable losses.

**Hosts Affected**

Rasp leaf affects all varieties of sweet cherry but appears to damage
Napoleon more than other varieties. In one locality in Colorado it was observed in Montmorency sour cherry.

Transmission and Spread

Transmission has been effected through bud and graft inoculations, but the causal virus moves slowly through trees. In spring symptoms on trees inoculated the previous fall are generally restricted to a few leaves within a few inches of the inoculation point. Severe pruning or heading back of infected trees causes rapid growth, which results in rapid distribution of the virus through the trees.

Control

Rasp leaf no doubt has occasionally been spread in nursery stock propagated from trees with only a small...
affected portion. Care should be taken in the selection of budwood sources or better, only progeny-tested sources should be used. Severely affected trees are worthless and should be removed.

**Mottle Leaf**

Mottle leaf occurs in widely scattered localities in northwestern United States and adjoining areas of Canada. In general there are two forms of the causal virus, one causing mild symptoms and the other severe. Severe mottle leaf causes extensive losses in sweet cherry orchards near the foothills and in canyon districts of central Washington. Mild mottle leaf is a limiting factor in cherry production in some parts of the Willamette Valley of western Oregon. Both forms of the virus are present in Utah, but neither is sufficiently prevalent to cause serious losses.

**How to Recognize Mottle Leaf**

Mottle leaf is a typical mosaic-type disease, characterized by irregular, chlorotic mottled, and distorted leaves early in the season (fig. 45), and puckering with less mottling later in the season. On severely affected trees, leaves are reduced in size and show variable amounts of lacerations, but there is no leaf fall. Fruits on severely affected trees are abnormally small, late in maturity, and insipid in flavor, but not misshapen. Affected trees may be stunted, giving the tree a rosetted appearance.

Bing and Napoleon are the most severely damaged of the sweet cherry varieties. Moderate symptoms occur on Black Tartarian, Black Republican, Centennial, and Governor Wood. Lambert shows only slight symptoms, even when it is infected with the strain of the virus which produces severe symptoms on Bing and Napoleon.

On trees affected with mild mottle leaf, leaves show mottled patterns but are not materially misshapen or lacerated (fig. 46). In Oregon loss of tree vigor and varying amounts of dieback have been associated with mild mottle leaf.

**Hosts Affected**

All varieties of sweet and sour cherries are susceptible to the mottle leaf virus, but Bing and Napoleon sweet cherries are the only varieties seriously damaged. Peach has been experimentally infected but has developed no symptoms. Wild bitter cherry (Prunus emarginata) is naturally infected in Washington and acts as a virus reservoir from which spread takes place to cultivated cherries.

**Distribution and Economic Importance**

Mottle leaf has been seen in only a few orchards in Utah, but care should be taken to avoid further distribution.

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Fig. 45. Cherry leaves affected with severe mottle leaf: A, Napoleon; B, Bing. C, branch of Bing showing symptoms in the new tip growth.
Experimental Transmission and Natural Spread

The mottle leaf virus is easily transmitted by grafting tissue from diseased trees on healthy trees and will be carried in all buds from diseased trees. Trees inoculated in the fall are usually thoroughly diseased when growth begins the following spring.

The natural spread of mottle leaf in orchards is erratic, more rapid in certain localities than in others and in certain seasons than others. The natural vector is not known.

Control

Trees affected with severe mottle leaf are unproductive and should be replaced with healthy trees. In young orchards, trees affected with mottle leaf should be removed. Nursery stock should be propagated from progeny-tested source trees known to be free of mottle leaf virus.

Rugose Mosaic

Rugose mosaic occurs in most cherry-growing districts of western North America, commonly on single infected trees scattered through orchards. Occasional trees have been seen in orchards in several counties in Utah, but the damage from this disease is small.

How to Recognize Rugose Mosaic

Leaves on affected trees commonly show general chlorosis midway between the midrib and edge, but they may also develop small chlorotic areas along the lateral veins. Re-
tardation of normal expansion in these areas results in various amounts of distortion (fig. 47). Rugose mosaic is sometimes confused with mild mottle leaf. The chlorosis caused by mild mottle leaf virus is more diffuse, is between the lateral veins, and is not associated with appreciable leaf distortion. Fruits on rugose-mosaic-affected trees may be variously misshapen and are often flattened on each side of the suture.

Host Range, Transmission, and Spread

Rugose mosaic occurs on most varieties of sweet cherry, but most commonly on Napoleon. It occurs naturally in sour cherry, and the virus has been transmitted experimentally to several other stone fruits.

Cherry trees inoculated in the fall develop symptoms when growth begins the following spring. There is

Fig. 47. Napoleon cherry leaves from tree naturally infected with rugose mosaic.
some evidence of distribution in nursery stock but little evidence of natural spread from tree to tree in the orchard.

**Control**

Severely affected trees in orchards should be replaced with progeny-tested, virus-free nursery stock.

**SOUR CHERRY YELLOWS** in sweet cherry (See description under sour cherry, p. 83.)

**PEACH NECROTIC LEAF SPOT** in sweet cherry (See description under peach, p. 49.)

**PRUNE DWARF** in sweet cherry (See description under prunes and plums, p. 88.)

**LINE PATTERN** in sweet cherry (See description under prunes and plums, p. 89.)
THE NAME “yellows” has been applied to this disease in recognition of the striking yellow mottling which occurs annually on leaves of affected sour cherry trees. The disease is also known to certain growers as leaf drop, yellow leaf drop, physiological yellow leaf, and by other names.

Sour cherry yellows apparently has been present in Utah orchards for a long time. It was recognized as a disease in New York in 1919, and since that time has been reported from every major sour-cherry-growing area of North America. Sour cherry yellows is known also in several countries of Europe, but it is not as generally recognized there as in North America. The wide distribution of sour cherry yellows in the world is without doubt traceable to distribution in the seeds of rootstocks used for cherries.

The virus nature of sour cherry yellows was demonstrated in 1939 by Keitt and Clayton in Wisconsin. Previously it was thought to be the result of unsuitable growing conditions, a natural weakness of certain clones of sour cherry, chemical injury from fungicides or insecticides, or other causes. One reason for not suspecting that the disease was caused by an infectious agent was the fact that trees appeared normal in the spring and no symptoms developed until 3 to 5 weeks after the beginning of growth; such reaction could usually be traced directly to weather or specific treatments.

How to Recognize Sour Cherry Yellows

The most striking symptom on sour cherry is the yellow and green mottling of some of the leaves (pl. 6, A, B), which fall from the tree. Leaves on affected trees appear normal until 3 to 5 weeks after petal fall, when irregular pale-green or yellow areas begin to develop in the older leaves. These areas may start anywhere on the leaves but progress until the whole leaf is yellow. Some leaves remain green longest along the larger veins (pl. 6, B). Leaf fall usually begins shortly after the first appearance of yellow and all the leaves showing any appreciable amount of yellow drop. Some leaves may drop which show no yellow color.

Development of sour cherry yellows symptoms is sharply affected by temperature. Affected trees will not develop leaf symptoms in areas where the temperatures are relatively high during the growing season. In the greenhouse symptoms were produced on affected trees maintained at 60° - 68°F. Symptoms developed on trees grown where temperatures were as high as 82° in the daytime and night temperatures 60° or lower. Trees in the orchard usually show symptoms in the oldest leaves first, often on the scale leaves, and under favorable conditions symptoms develop in younger leaves progressively toward the growing points of shoots. If a wave of yellows is interrupted by weather unfavorable for symptom development, there may be a cycle of leaves on shoots that remain appar-
ently healthy; yet if favorable weather is resumed leaves farther up the shoot may develop typical symptoms.

In the orchard the first wave of yellows symptoms is usually the most severe, but other waves of varying severity may occur until fall. The amount of defoliation caused by yellows is variable, ranging from a trace to more than 50 percent of the leaves. This variability, while it is most commonly a result of fluctuating temperatures, may also be related to the variable effects of different strains of the causal virus.

In climates favorable for expression, trees which have had yellows for several years show a weeping type of growth and a much reduced spur system (pl. 6, C, D). The short spurs bearing the flower buds, which are usually borne laterally along branches, lose their determinate type of growth and grow into lateral branches. Such branches develop few fruit buds. Yields on severely affected trees may be reduced 50 percent or even more. Fruits on yellows-affected trees are larger than normal and are of good quality.

**Distribution and Economic Importance**

Sour cherry yellows is the cause of serious economic losses. Surveys indicate that one-third of all the sour cherry trees grown in North America are affected. Based on a 50 percent crop reduction on diseased trees, the loss on a $26,000,000 crop in the United States in 1954 would approximate $4,000,000.

The prevalence of sour cherry yellows in Utah orchards is above the national average and infection in some orchards is nearly 100 percent. Surveys indicate that upwards of 75 percent of all the sour cherry trees in the state are affected. It seems likely that yellows is the most important limiting factor to sour cherry production in Utah.

**Hosts Affected**

The sour cherry yellows virus appears to be able to infect a wide variety of stone fruit species, but produces symptoms only on sour cherry. As shown by experimental transmission tests to sour cherry, the causal virus appears to be widely present in normal-appearing sweet cherry orchard trees. Similar tests have shown occasional plum trees to be naturally infected. Seedling rootstocks, both mazzard and mahaleb, obtained directly from nurseries where they were grown from seed have been found naturally infected. They are among the chief factors responsible for the high prevalence of the disease; any susceptible species grown on them would become infected. Various other *Prunus* species including peach, plum, chokecherry, and wild black cherry have been experimentally infected.

**Transmission, Incubation Period, and Rate of Spread**

Sour cherry yellows is easily transmitted experimentally by budding or grafting of tissue from diseased to healthy trees. In the greenhouse, with suitable materials and environment, symptoms can be produced in 6 weeks. In the field symptoms may appear during the season after inoculation but may be delayed until the second season after infection.

Natural spread of sour cherry yel-
lows from tree to tree in orchards appears to occur at a variable rate, depending on the numbers of diseased and healthy trees present as well as other factors. In some areas 10 percent of the trees in the orchard have become diseased in a single year, but more commonly spread averages about 3 percent per year. The natural vector is not known.

Control

The first essential of control is the use of yellows-free nursery stock in planting new orchards. To produce yellows-free nursery stock the nurseryman must use rootstocks produced from seed trees free of the yellows virus and must obtain his sour cherry budwood from sources known to be free of the virus. Such virus-free material cannot be judged on the basis of visual appearance. Freedom from virus can only be established by indexing procedure and progeny performance. Yellows-free stocks are now available to nurserymen and should be demanded by growers.

The second essential for control of sour cherry yellows in orchards is isolation of new plantings from old plantings of sweet or sour cherry. No definite data are available to indicate the distance needed for safety, but it stands to reason that the shorter the distance the greater the risk of spread. If old orchards are to be removed, they should be removed well in advance of new plantings, allowing sufficient time for any long-lived vector insect suspects to die.

Should yellows appear in young orchards, diseased trees should be removed as soon as they are found and replaced with healthy nursery trees. It seems doubtful whether removal of diseased trees from older orchards is practical, especially if an appreciable number are present. Such orchards should be removed as entire blocks when they become unprofitable.

Ring Spot

RING SPOT, also referred to as necrotic ring spot, is prevalent in sour cherry. The ring spot virus, originally discovered as causing a disease of peach, is discussed under “peach ring spot,” p. 40. Since the symptoms on sour cherry are somewhat different from those on other hosts, a brief description is included.

How to Recognize Ring Spot on Sour Cherry

The initial effect of ring spot on sour cherry is a pronounced delay of foliation on affected limbs or whole trees at the beginning of growth in the spring. New unfolding leaves are small and show dark rings of varying diameter, 1/16 to 1/4 inch. At first these rings have a water-soaked appearance (fig. 48, B, C), but later the darkened tissue becomes necrotic and sunken. The sunken ring symptoms may persist on some leaves well into the growing season but more commonly the tissue surrounded by the ring dies and drops out, leaving a hole. Affected leaves become roughened and undulated and late in the season become shredded and tattered (fig. 48, A). Symptoms are usually restricted to the first leaves formed...
in the spring; leaves formed later do not show symptoms. Affected trees or parts of trees usually show symptoms during the initial year of infection but not in subsequent years. Trees having symptoms only in one portion usually show symptoms in other portions the following year. Severely affected trees may bear small leaves and appear thin in foliage the year after they first showed the disease, but usually they show no ring symptoms in their leaves. Infected trees appear to recover gradually in succeeding years, but it is not known if they ever regain their original vigor. Such trees become symptomless carriers and all buds taken from them carry the virus.

**Distribution and Economic Importance**

Few orchard trees in Utah show symptoms of ring spot because most trees are already infected and have passed the stage where symptoms are expressed. The high incidence in sour cherry orchards is probably a result of distribution of the virus in mazzard and mahaleb cherry seedlings used for rootstocks. Buds from trees grown on infected rootstocks would, of course, carry the virus and would serve as a further means of distribution. Some spread takes place from tree to tree in the orchard; such newly infected trees are the only ones that express symptoms. The natural vector of ring spot virus is not known.

The economic importance of ring spot to sour cherry production is not clear. Trees in the first year of the disease are severely retarded and reduced in yield. Such retarded trees are more susceptible to insect pests and general orchard ills, but once they recover, production approaching that of normal trees is resumed. There is some evidence that trees infected with the ring spot virus are less vigorous than comparable trees free of it. Buds from ring-spot-infected trees give a poorer stand of nursery stock and the buds which do grow produce trees of less vigor than do buds from virus-free trees. The use of ring-spot-free sour cherry budwood and ring-spot-free seedling rootstocks results in sufficiently better nursery stock to make it an economically profitable procedure.

**CHERRY WESTERN X WILT AND DECLINE** in sour cherry (See description under sweet cherry, p. 51.)

**WESTERN X LITTLE CHERRY** in sour cherry (See description under sweet cherry, p. 60.)

**NECROTIC RUSTY MOTTLE** in sour cherry (See description under sweet cherry, p. 65.)

**RASP LEAF** in sour cherry (See description under sweet cherry, p. 76.)

**MOTTLE LEAF** in sour cherry (See description under sweet cherry, p. 79.)

**RUGOSE MOSAIC** in sour cherry (See description under sweet cherry, p. 80.)

**LINE PATTERN** in sour cherry (See description under prunes and plums, p. 87.)

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**Fig. 48.** Montmorency cherry leaves affected with necrotic ring spot, showing A, severe necrotic shock symptoms and B and C, water-soaked pattern consisting of rings and arcs.
Fortunately, no virus diseases causing serious economic losses to plums and prunes are prevalent in Utah. Only two diseases which cause symptoms on these hosts are present.

Prune Dwarf

The virus causing prune dwarf is far more prevalent in sweet and sour cherries than it is in prune. The reason for such prevalence is that it produces no symptoms in cherries and it is therefore propagated unwittingly in them, whereas symptoms in Italian Prune are so striking that diseased trees would not be used as a budwood source.

How to Recognize Prune Dwarf

The prune dwarf virus causes symptoms on the domestica-type plums, commonly called prunes, but can infect other type plums without symptoms. Italian Prune comprises most of the plum acreage in Utah but is seldom found naturally infected with the prune dwarf virus.

Leaves on prune-dwarf-affected trees are small, narrow, irregularly strap-like, rugose, thickened, and variously misshapen (fig. 49). The upper surface of affected leaves is usually shiny, as if lightly varnished. Terminal growth is reduced and nodes are shortened. In the first year following infection, diseased shoots may be scattered among branches that

Fig. 49. Four small, narrow, strap-like Italian Prune leaves from prune-dwarf-affected tree at left; normal leaf at right.
appear normal. Even on trees in advanced stages of disease, occasional healthy shoots may occur. Diseased trees bloom well but usually set few fruits. Occasionally these may be somewhat distorted but usually they are normal in shape and quality.

There are several strains of the prune dwarf. These differ chiefly in the severity of symptoms they produce and in the rate at which they move through infected trees.

**Hosts Affected**

The prune dwarf virus can infect several stone fruit species but produces symptoms on only domestic-type prunes and peach.

On peach the symptoms are a general stunting of growth with correspondingly shortened internodes and rosetting with narrow rigid leaves. Occasionally peach is found naturally infected in Utah.

**Transmission and Incubation Period**

The prune dwarf virus is easily transmitted by budding and grafting. Italian Prune trees usually show symptoms only in growth on the inoculated arm or near the point of inoculation the first year after infection. On some trees symptoms do not develop until the second year after inoculation. The differential in symptom development is probably a result of the different effect of strains of the causal virus.

**Control**

Prune dwarf virus can apparently be easily eliminated from nursery stocks by using stocks known to be virus-free as sources of propagating material.

**Line Pattern**

Reports from many widely separated areas indicate that the line pattern virus, originally described from Michigan, occurs world-wide. It was given the name “line pattern” because of the variously shaped patterns formed by chlorotic bands or lines in plum leaves (fig. 50, A). The line pattern virus is not recognized as the cause of serious economic loss to any of its many hosts, but its effects in combination with other viruses are unknown. The line pattern virus has occasionally been encountered in sweet cherry and plum in Utah.

**How to Recognize Line Pattern**

The symptoms of line pattern are generally similar on many hosts but differ chiefly in intensity of expression. Patterns on leaves consist of chlorotic rings and streaks, often more or less parallel on each side of the leaf midrib. The perimeter of the patterns is usually bordered by a chlorotic band of uniform width but extremely irregular in outline (figs. 50 and 51). Chlorotic bands along all the small veins of leaves of some plum varieties produce a netted pattern which may occur independently of or in combination with the line patterns.

The symptoms of line pattern are sometimes obscured or confused with those of other viruses. The Shiro variety of plum is considered to be a good diagnostic host.
Fig. 51. A, Leaf of Amanogawa oriental flowering cherry affected with line pattern (banded chlorosis). B - D, Leaves of various species showing symptoms of the same disease: B, Montmorency cherry; C, mahaleb cherry; D, myrobalan plum.

Fig. 50. A, Shiro plum leaves affected with line pattern; B, peach leaves showing symptoms of the same disease.
Distribution, Transmission, and Incubation Period

The line pattern virus has been found in many stone fruit species and appears to have been spread widely in nursery stocks. Little is known about spread from tree to tree in orchards. Trees inoculated in early spring often develop symptoms during the same growing season. Trees inoculated in the fall usually show symptoms in the new growth of the following season.

Control

Even though line pattern does not appear to cause measurable economic loss to affected plants, it should be avoided in the propagation of nursery stocks.
Control of Virus Diseases of Stone Fruits

Once an orchard tree becomes infected with a virus, there is no practical means of eliminating the virus and thereby curing the tree. General recommendations have been to destroy the infected tree and replace it with a healthy one. Control then becomes either a procedure of prevention or development of varieties which can thrive and produce even though they may become infected (tolerant varieties).

Prevention then can generally be divided into two classes: (1) Prevention by exclusion from the area, and (2) prevention by reducing the virus reservoir from which spread takes place in the orchard.

All evidence indicates that viruses, like other forms of life, do not arise out of nowhere but are direct descendants of pre-existing forms; therefore, procedures to exclude them from the area are the most effective means and should be used wherever practical. Federal quarantines have been promulgated to prevent entrance of viruses and other pests from foreign sources. State quarantines serve to prevent movement from one area to another. Barriers, such as bodies of water, deserts, and mountain ranges, increase the efficiency of quarantines.

The control of virus diseases already present within an area may be complex because of variable features of the diseases. Measures applicable and sufficient for one disease may not suffice for another or even for the same disease in two different areas. Before control procedures can be recommended, the nature of the disease, its distribution, host range, rate and manner of spread, effect on yield, and other facts should be known.

The question of whether roguing of diseased trees from orchards is practical depends on the rate of natural spread, whether spread stems from sources in the orchard or from outside, the amount of damage caused by the disease, and whether there are tolerant or immune varieties which can be grown. Roguing, coupled with orchard isolation, use of disease-free nursery trees, and wild host-removal programs, has been the only practical means of control for diseases that spread rapidly and severely damage all varieties of a given fruit. Isolation may be hard to achieve in intensively cultivated areas and may not be necessary for diseases that spread slowly.

For diseases which are generally distributed, have a rapid rate of spread, have symptomless hosts or other features that make it impractical to rogue diseased trees, other procedures of control have to be developed. The use of immune or tolerant varieties, tolerant top and rootstock combinations, and control of vectors are approaches that offer promise. Chemotherapy would be a welcome measure, but as yet there are no results which offer much promise. No treatment with chemicals, spray materials, fertilizers, or other materials has effected the cure of virus-diseased trees in the orchard.

There is ample evidence that stone fruit virus diseases and certain virus-like disorders have been disseminated in infected nursery stocks. No control procedures are effective unless such dissemination is stopped. Nurserymen should become acquainted
with the diseases and disorders occurring or likely to occur within the areas they serve and should use only approved budwood sources for propagating their stocks. Growers need the advantages of starting orchards with better stock and should demand that the stocks they purchase be from approved sources. There is need for further nursery improvement work. The problem can be materially assisted by the cooperation of nurserymen, growers, extension, regulatory, and research men.
Disorders With Viruslike Symptoms Not Proved Transmissible

In addition to the known virus diseases affecting stone fruits, there are a number of disorders which produce virus-like symptoms but which have been shown to result from other causes. Some of these appear to be of genetic origin, others are caused by insect or mite injury.

Virus diseases can be distinguished from these by the fact that viruses introduced into a healthy plant like other infectious agents grow and reproduce and thereby cause characteristic symptoms. Viruses affecting woody plants are generally not transmissible in juice but are usually transmitted when tissue from the diseased plant is grafted on the healthy plant. Transmissibility, then, has become one of the standard tests to distinguish a virus disease from other disorders which produce similar symptoms. Genetic disorders are perpetuated in shoot growth of scions or buds or scions are grafted on healthy trees, but do not develop in tissue of the healthy trees. Symptoms of insect or mite injury are neither transmitted nor perpetuated on graft-inoculated trees provided the insects or mites causing the symptoms are not allowed to feed on the trees.

Bud-perpetuated genetic disorders carried in nursery stock are the source of serious losses to Utah fruit growers. The loss includes crop loss and the cost of producing trees before they are recognized as worthless. Such losses can be avoided by the use of progeny-tested trees as budwood sources for the production of nursery stock.

Sweet Cherry Crinkle Leaf

Crinkle leaf is one of the most widely distributed disorders of sweet cherry. It has been reported from California, from the cherry-growing areas of the Pacific Northwest, from the Intermountain States, and from British Columbia. It has become generally distributed in Utah, where it causes serious losses in certain varieties of sweet cherry. It appears to be on the increase in many Utah cherry-growing areas.

Our far-from-complete knowledge regarding cherry crinkle leaf indicates that the disorder probably is not contagious; therefore, its wide distribution is a result of use of nursery stock propagated from affected trees.

Future losses and further distribution of the disorder are unnecessary and should be prevented.

How to Recognize Crinkle Leaf

Leaf Symptoms. Affected leaves are variously misshapen and mottled; margins are often indented and abnormally serrated and irregular in outline (fig. 52). Some leaves are so severely distorted that if removed from the tree they would not be recognized as cherry leaves. Irregular, often streak-like chlorotic areas with distorted venation, extending in the same general direction as the lateral veins, develop on either side of the
Fig. 52. **Upper**, Bing cherry leaves: Healthy at left, 3 leaves at right showing mottle and distortion typical of crinkle leaf. **Lower**, Black Tartarian cherry leaves and fruits: Three leaves at left showing crinkle leaf effects (mottling less pronounced than in Bing); healthy leaf at right. Fruits show the conical shape and the characteristic angular attachment to stems.
midrib. Distortion of leaf shape is caused by the inhibition of growth in these chlorotic areas and greater expansion of tissue in the greener areas. Occasionally one side of a leaf may be generally affected and distorted while the other half may appear nearly normal. In milder cases there is less distortion; yet affected leaves are sufficiently characteristic that they are easily recognized from normal.

**General tree symptoms.** The extent to which trees are affected by crinkle leaf varies greatly. Trees propagated from affected trees are usually affected throughout; on such trees nearly every leaf may be crinkled. On the other hand, it is common for orchard trees to have several arms with severely crinkled foliage and other arms with normal foliage. Affected trees, while distinctive in appearance, may not be greatly reduced in size.

**Fruit symptoms.** Affected trees or affected portions are often nearly sterile and produce little fruit. Fruits that do develop are characteristically flattened on the sides and ridged on the sutures, are generally more pointed than normal, and are attached at an angle to the stems (fig. 52). Affected fruits ripen unevenly, and in certain varieties color motting and splashing are prominent. Malformations in the fruits are frequently more pronounced in the early stages of development than in the mature state.

**Distribution and Economic Importance**

Cherry crinkle leaf was first recognized in Utah in 1938. Subsequent surveys showed the disorder present in every cherry-producing area in the state. In a survey of 123 orchards in 1944, 46 in Weber, 27 in Davis, 9 in Salt Lake, 18 in Utah, and 23 in Washington, involving 27,146 trees, 612 (2.25 percent) were crinkle-leaf-affected. Many of the trees in these orchards were Lambert or Napoleon, varieties which are not affected. Had the surveys been restricted to Bing and Black Tartarian, the percentage would have been much higher.

The percentage of crinkle leaf in some individual orchards, particularly in Washington County, approached 100 percent indicating that they were planted with stock propagated from affected trees.

Although severely affected trees live as long as normal ones, they produce relatively few fruits. If crinkle-leaf-affected fruits are harvested and mixed with the normal fruits, they are so abnormal that they lower the grade and price of the entire lot. All too frequently in Utah the high percentage of crinkle-leaf-affected trees in orchards has seriously reduced profits and in many cases has rendered entire plantings commercially unprofitable.

**Varieties Affected**

In Utah crinkle leaf symptoms have been observed on Bing, Black Tartarian, and Black Republican varieties, and on mazzard seedlings. The disorder, however, is most common on Bing and Black Tartarian. So far crinkle leaf has not been found in Utah on either Lambert or Napoleon. Kinman et al., in the U. S. Department of Agriculture Handbook No. 10, listed the following varieties on which crinkle leaf has been observed in California: Eagle, Burbank, Dr. Flynn, Ox Heart, Sheldon, and Waterloo. Crinkle-leaf-like symptoms have
been observed on Italian Prune, but no relation with those occurring on cherry has been established.

**Transmission Studies**

So far as known crinkle leaf is not transmissible and therefore is probably not of a virus nature. Although its symptoms resemble in many respects those of some virus diseases common on stone fruits, it is thought to be a frequently occurring mutation or bud sport. In many instances crinkle leaf has developed on one or more branches of Bing trees known to be free of any previous expression. Thus, it appears that crinkle leaf symptoms may develop spontaneously at most any time during the life of the tree.

Although crinkle leaf is not transmitted, it has been demonstrated repeatedly that buds from crinkle-leaf-affected trees will invariably produce affected trees. In one experiment in Utah, for example, 100 buds from a single Bing tree showing crinkle leaf were budded on mahaleb seedlings. Eighty-seven of the inserted buds survived the winter; all produced crinkle-leaf-affected trees. Scion wood or buds from normal trees grafted into crinkle-leaf-affected branches appear to produce normal growth indefinitely. Likewise, crinkle-leaf-affected scions or buds grafted into normal tissues have never, in our experience, caused healthy parts of the tree below the graft union to develop the disorder.

**Control**

The fact that buds from crinkle-leaf-affected trees invariably produce crinkle-leaf-affected trees indicates the need for rigid selection of the bud-wood and scions used for propagation and orchard rehabilitation purposes. Crinkle leaf can be avoided with greater surety if budwood from sources which have progeny performance records showing no trace of the disorder is used. All growing nursery stock should be inspected and rogued at a period most favorable for detecting crinkle leaf.

Trees on which any considerable portion of the leaves show crinkle leaf are invariably unprofitable. These either should be rogued or top-worked to crinkle-free sources of the same or more desirable varieties. All affected branches, regardless of how few, should be pruned out and the trees converted into healthy fruit-producing structures.

**Sweet Cherry Deep Suture**

The name “deep suture” was given to this disorder of sweet cherry in recognition of the fruit symptom. Affected fruits are grooved in varying degree on the suture line. Deep-suture-affected trees are common in orchards in California, Oregon, Washington, Idaho, Montana, and Utah.
The disorder is generally distributed in Utah orchards of the Bing variety and causes considerable losses, not only in production but also in grading and marketing. Like crinkle leaf, deep suture occurs on a number of sweet cherry varieties; however it is particularly prevalent on Bing. Deep suture is not graft-transmissible, but it is generally bud-perpetuated. Its distribution and ultimate prevention are directly related to dissemination in nursery stocks.

**How to Recognize Deep Suture**

Deep suture is characterized by deformed fruits and leaves, both of which commonly occur on affected trees.

**Leaf symptoms.** Affected leaves are conspicuously and abnormally narrow and elongated (pl. 7, top). On the Bing variety they are leathery in texture and have rough surfaces. The marginal serrations are irregular, reduced in number, and sometimes missing along portions of the leaf. These leaf abnormalities are frequently so pronounced that the disorder has been referred to as “long leaf” and “rough leaf” by growers and nurserymen.

**Fruit symptoms.** Deep-suture-affected fruits have a characteristic depression or groove on the suture side (pl. 7, top) and generally are more rounded than normal at the apex. The suture depressions vary in length and depth, even in fruits on the same tree. Sometimes they are so deep that the stone of the fruit is exposed. Such fruits have no commercial value even though the flavor and general edibility may not be impaired.

**General tree symptoms.** The portion of a tree which exhibits fruit and leaf symptoms varies greatly. Entire trees may be affected, or only an occasional branch may show symptoms. Also, the severity of symptom expression varies on different affected portions of trees and even on the same portion in different seasons. Mildly affected trees may show typical deep-suture-type leaves in the spring, whereas those produced later in the season may appear essentially normal. Severely affected trees often have a drooping appearance, much as if they were suffering from drought. Affected nursery trees are generally dwarfed in height and when transplanted they grow slowly and exhibit typically malformed foliage.

**Distribution and Economic Importance**

Deep suture was observed in California prior to 1930 and has now been recognized in all the important cherry growing areas of western North America. In 1937, Kinman reported 13 percent of the trees affected in three orchards in Sacramento Valley, and 50 percent of a large number of other orchards examined contained affected trees. Surveys in Washington in 1940-42 showed that less than 10 percent of the orchards inspected were free from deep suture. In Oregon deep suture is so prevalent that it is difficult to find Bing trees free from it. In some districts fruit on affected trees is not picked because packers refuse to handle the product.

The disorder was brought to the attention of Utah workers in 1939. Subsequent surveys from 1939 to 1944 showed it to be generally distributed in the major cherry-growing areas. In a late-season survey in 1944 based on leaf symptoms, deep suture was found
in various counties as follows: Weber, 25 of 46 orchards; Davis, 12 of 27 orchards; Salt Lake, 3 of 9 orchards; Utah, 8 of 18 orchards; and Washington, 17 of 23. The incidence of affected trees in the five counties averaged less than 1 percent of those examined. Subsequent observations showed that trees which produce deep suture fruit may not show sufficient leaf symptoms late in the season to be recognized. Had the survey been made before the fruit was harvested, a much higher percentage would no doubt have been found. In one orchard in Box Elder County, 21 percent of the trees showed affected fruits.

Annual crop losses are difficult to estimate because the degree of fruit malformation varies with the season. The loss from use of affected nursery stock involves not only the original cost of the tree, but, what is far more serious, the cost of maintenance until its worthless nature is discovered, which may take 5 to 7 years.

**Varieties Affected**

In Utah Bing is the principal variety affected. Deep suture is also frequently found on Black Tartarian and on mazzard seedlings.

**Perpetuation and Rate of Natural Distribution**

Numerous attempts in Oregon, Washington, and Utah to transmit deep suture from affected to healthy trees by grafting have failed. Repeated experiments have shown that scions and buds taken from normal trees produce normal growth when grafted into trees showing deep suture. Likewise, scions and buds from deep-suture-affected trees produce growth with deep-suture-affected leaves and fruits when grafted onto normal trees.

**Control**

In the main, reliance must be placed upon prevention by the use of deep-suture-free nursery stock. Buddwood source trees, in any event, should be selected before the fruit is harvested and, where possible, certified budwood source trees should be maintained in mother blocks so that progeny records as to performance can be used as proof of freedom from the disorder.

**Sweet Cherry**

A disorder of sweet cherry, characterized by creamy-white chlorotic patterns strongly resembling those of certain mosaic-type virus diseases, occurs sporadically in cherry orchards in western North America (fig. 53). Symptom expression appears to be erratic in affected trees, being strikingly expressed one year and obscure or entirely absent the next. No detrimental effects on affected trees have been seen. Symptoms have been observed on several varieties of sweet cherry in Utah.

All attempts to transmit the disorder by grafting scions from affected trees onto healthy trees have failed. In some cases growth from the scion taken from an affected tree failed to show symptoms. The evidence thus indicates that the disorder is not of
Fig. 53. Bing cherry leaves showing variously shaped, cream-colored patterns called "white mottle." This disorder is erratic in its expression and all attempts to transmit it by grafting have failed.

virus nature, but is caused by some genetic or nutritional factor even though the symptoms are strikingly suggestive of virus.

**Italian Prune Leaf Spot**

**Italian Prune** leaf spot is the most common disorder of Italian Prune wherever it is grown. In Utah, affected trees probably can be found in every Italian Prune orchard. In general, the percentage of affected trees in the orchards will be high, approximating 100 percent in many. At present, the disorder stands out as the one which limits production of the Italian Prune. Because of it, many Italian Prune orchards in Utah have been abandoned or pulled out. Others are rapidly deteriorating.

The actual nature or cause of leaf spot is not known. However, from the standpoint that it is noninfectious and bud-perpetuated, the disorder falls definitely into the genetic category with Italian Prune crinkle leaf, sweet cherry crinkle leaf, and sweet cherry deep suture.

**How to Recognize Italian Prune Leaf Spot**

Leaf symptoms of the disorder are primarily of two types, a diffuse chlorotic mottling and a distinctive tan or brown necrotic spotting. These commonly occur together (pl. 7, B), but the necrotic spotting often occurs alone (pl. 8).

The chlorotic mottling type appears first in late spring and early summer in newly matured leaves; the first spring growth is normal. Once chlo-
rotic symptoms have appeared, they tend to increase in expression and are most prominent in late summer and early fall. Finally in late season they give rise to diffused or ring-spot-like necrotic areas with dark brown centers which do not fall out (pl. 7, B). Young trees may show only a slight indication of the disorder, but when bearing their first heavy fruit crop may show pronounced symptoms.

The necrotic spots vary greatly in size, shape, and number. When numerous they coalesce, producing large irregular blotches of dead tissue, frequently involving much of the leaf surface (pl. 8). Sometimes the entire leaf is killed. Some spots show definite zones, parts of which may drop out producing shot-holes of varying sizes. Severe foliage spotting is usually accompanied by defoliation, some trees losing up to 50 percent of their leaves.

Symptom expression appears to be directly influenced by temperature and is more pronounced during hot dry seasons. Many observations indicate that low fertility, heavy fruiting, or other unfavorable environmental conditions enhances the symptoms. Trees near driveways, barnyards, and chicken runs, or on sandy areas and dry clay knolls are usually more severely affected than those in good soil.

**Distribution and Economic Importance**

Few specific data are available as to the exact economic significance of leaf spot in Utah. The disorder, however, is known to cause a decrease in quality and quantity of fruit on severely affected trees and appears to reduce quality even on lightly affected trees. Severely affected trees usually suffer heavy defoliation, which in turn results in a heavy fruit drop or in a yield of sunburned, poor quality fruits. Severely affected trees in the nursery are usually dwarfed and seldom make satisfactory orchard trees; even under favorable cultural conditions such trees may deteriorate early and finally die. The senior author has seen deterioration and removal of several plantings because of leaf spot in Davis County. Most of these plantings were pulled out within period of six years (1940 to 1945).

Rhoads, employed to make special plant disease surveys in 1944 and 1945, reported both types of leaf spot present in orchards in all parts of Utah where Italian Prunes are grown. He reported that crop and tree losses were so severe that some growers were on the verge of abandoning prune growing unless some solution could be found for leaf spot and mottle.

**Varieties Affected**

So far as is known, the Italian Prune is the only fruit variety in Utah on which leaf spot is important. A similar condition has been seen on other prunes of Italian type, on Jefferson, and other less important domestica varieties. A selection of the Italian Prune made in Washington and carried under the designation "Pr-H1" appears vigorous and, although under some conditions it shows some of the leaf-spot-type mottle, it appears to suffer little damage from the disorder. An early-maturing, Italian-type prune, introduced by the May Nursery of Yakima, Washington, under the name of "Richards' Early Italian," has also shown promise of being little damaged by the prune leaf spot disorder.
Perpetuation and Distribution

All attempts to transmit leaf spot from diseased to healthy trees have so far failed. The disease is shown to be bud-perpetuated by the fact that buds and scions from affected trees invariably produce leaf-spot-affected shoots or trees. Leaf spot, therefore, must be regarded as probably a genetic abnormality or varietal weakness that has been distributed in nursery trees. Part of the reason for its wide occurrence is the fact that no stock free from it has been available to Utah nurserymen.

Control

Nursery trees propagated from severely affected orchard trees usually show symptoms in the nursery. Such nursery trees become severely affected when grown in the orchard; thus they should not be used. Nursery stock propagated from mildly affected trees may appear normal in the nursery row but develop symptoms after they become older or when they are grown under unfavorable conditions. Trees propagated even from vigorous, healthy-appearing orchard trees growing under favorable conditions may develop symptoms when grown in orchards under unfavorable conditions. The best procedure appears to be to propagate only from trees that have been progeny-tested and are known to give good performance under conditions favorable for expression of the disorder. Superior selections have been made and are available for distribution through experiment stations.

The effect of poor culture, particularly insufficient water, fertilizer, pest control, and weed control, as factors which promote and increase development of leaf spot has been well demonstrated. Good orchard care must not be overlooked in the control of leaf spot.

Italian Prune Crinkle Leaf

A DISORDER of Italian Prune, resembling sweet cherry crinkle leaf, occurs in many orchards. Although there is no known relation of the disorder on prunes to sweet cherry crinkle leaf, the similarity of the two disorders has prompted the use of the name “crinkle leaf” for the prune disorder. Like sweet cherry crinkle leaf, prune crinkle leaf has not been transmitted by grafting; hence is not considered infectious. Prune crinkle leaf is perpetuated in nursery stock propagated from affected trees; no doubt the present distribution is directly related to dissemination in such affected nursery stock.

How to Recognize Prune Crinkle Leaf

Leaves on affected trees are reduced in size, variously misshapen, slightly thickened, and commonly show a diffuse mottle (fig. 54). Leaves may be so misshapen and reduced in size that, removed from the tree, they would not be recognized as prune leaves. Instead of having the normal ovoid shape, some leaves develop lobes with irregular dentations. In the narrow portions of the leaves the lateral veins are distorted and crowded together, giving an appearance of excessive venation. On some
trees, especially slow growing ones, reduction in number and size of leaves is an outstanding feature. On small leaves the distortion of shape and venation are less striking.

The amount of crinkle-leaf-affected leaves on trees is variable. On some trees a large portion of the leaves may be affected, but on others crinkled leaves may be restricted to single arms or to a few twigs. On some trees affected twigs may be scattered among those bearing normal foliage. Occasionally twigs may bear mostly affected leaves with a few normal leaves on the same spur, or affected arms may bear whole spurs with normal leaves.

Affected trees and affected portions of trees bear little fruit. The few fruits which do mature are normal in color, shape, and flavor.

**Economic Importance and Distribution**

In Utah, crinkle leaf can be found in 90 percent of the Italian Prune plantings. In some only occasional trees are affected, but in others many trees show from a trace to a large portion of their leaves affected. Loss from crinkle leaf not only represents the annual reduction in crops from non-productiveness, but also the cost of producing the tree up to the bearing age.
Varieties Affected

Crinkle leaf has been seen on several varieties of domestica-type prunes besides Italian Prune, but since the disorder is not known to be transmissible no relation of the disorders on the various varieties is recognized. Progenies of certain clones of Italian Prune under test are free of crinkle leaf.

Control

If buds from crinkle-leaf-affected trees are used to produce nursery stock, the resulting stock will be affected. If such affected trees are top-worked with scions from normal trees, the scions will grow into normal trees.

The first essential of control, then, is to avoid the disorder by using only progeny-performance-tested trees as a source of propagating material. If trees in young orchards show crinkle leaf, they should be top-worked with scions from healthy trees.

Peach False Wart

A disorder of peach, named "false wart" to distinguish it from the transmissible virus-induced wart, occurs sporadically in mid- and late-season-maturing varieties in orchards in western North America. False wart occurs particularly in the varieties J. H. Hale, Rio Oso Gem, Candoka, Halehaven, and some Elberta types. It is more common in some years, in some orchards, and in some areas than in others.

Unlike the virus disease "peach wart," which is not known to occur in Utah, false wart affects only a few fruits on trees and may not occur on the same tree the following year. Although symptoms of false wart occur only on fruits, like those of peach wart, buds from trees bearing fruits with false warts will not transmit the disorder to normal trees, whereas the virus causing peach wart is readily transmissible.

How to Recognize False Wart

False warts occur anywhere on fruits, usually as erumpent mounds of tissue topped by ridges and furrows (fig. 55). These mounds vary from
small outgrowths the size of a garden pea up to $\frac{1}{2}$ inch high and 1 inch wide. In some instances some of the larger wart-like growths appear to have been formed by union of two separate growths. Some of the false warts have hard, horny plates and needles of various shapes in the furrows, composed of the same type of tissue which occurs in the pit.

The cause of false wart is unknown. The most plausible suggestion is that it is a response by the peach fruit to the feeding punctures of some insect or mite when the fruit was young, at which time a toxic material was injected. Various types of outgrowths are known on plants as the result of feeding of specific insects and mites.

Fig. 56. Yellow spot on leaves of Lovell peach seedlings, a result of the feeding punctures of the peach silver-leaf mite, *Vasates cornutus* (Banks).
Peach Yellow Spot

A disorder of peach trees, particularly prevalent on nursery stock, termed “yellow spot” is now known to be the effect of feeding punctures of the peach silver-leaf mite, Vasates cornutus (Banks). Extensive feeding by the mite causes silvering of the upper surface of mature peach leaves. If the mite feeds on immature leaves, yellow spots form around the feeding points and no silvering develops. The silver-leaf mite is widely distributed in North America, but its prevalence appears to vary with seasons and in different areas. Also, the mite prefers certain varieties of peach, particularly those having no or small glands on their leaf petioles. Yellow spot is common in Utah nurseries, especially on Lovell peach seedlings, where it has been questioned as a possible virus disease.

The yellow spot disorder is characterized by patterns formed by various groupings of the yellow areas (fig. 56). Where the spots are numerous, they are usually smaller than where they are farther apart. When a group of spots occur along a leaf vein, they may produce chlorosis, simulating a virus pattern. When the mites feed on very young leaves, the spots are larger and appear to cause more leaf distortion.

Yellow spot has been produced experimentally. When mites are allowed to feed for 24 hours on partially mature leaves and are then killed, yellow spots develop 5 to 8 days later. New growth on such plants, if protected from mite feeding, is normal.

Yellow spot is not transmitted in buds from affected trees. It is necessary to remove any mites from the buds used in such transmission studies and to protect the experimental trees from natural infestation. In one instance, where mite-infested buds were used, the mites moved to young foliage and produced yellow spot, thus simulating virus transmission.
Disorders Caused by Improper Nutrition and Chemical Injuries Which May Be Confused With Those of Virus Diseases or Viruslike Disorders

Stone fruit trees when free of viruses and viruslike disorders produce growth characteristic of healthy trees when grown in good fertile soil and given proper cultural care. On the other hand, they will show characteristic symptoms of nutrient deficiency or injury if one or more of the essential chemical elements is absent from the soil, or present in excess. Injury may result if chemicals either essential or nonessential for growth are applied to soils or to leaves in amounts sufficient to be toxic. Chemicals toxic to plants are often used for pest control, may occur naturally in soils classed as unsuitable, may be applied to plants in unsuitable irrigation water, or may be taken up by plants from contaminated air.

In general, most of the elements needed by plants except carbon and oxygen are normally absorbed from the soil in a water solution through their roots. Research has shown, however, that a few of the elements normally obtained from the soil can be absorbed when applied to leaves. Expression of injury on or improper nutrition of plants is usually seen as abnormal growth of the tops, but careful examination of plants showing abnormal top growth will show correspondingly abnormal root growth. Symptoms on the tops vary from reduced growth to various types of chlorosis, malformation, and necrosis in leaves; reduction in yield and quality of fruit; and dieback of twigs or even death of trees. Some of these same symptoms may be easily confused with those caused by viruses or with those of viruslike disorders. Some of the nutritional disorders cause such characteristic symptoms that they can be readily recognized. Injuries resulting from chemicals are less characteristic and it is necessary to know the treatments that plants have received in order to determine the cause of the injury. It is the purpose of this section to outline briefly and illustrate the nutritional disorders and injuries most common on stone fruits in Utah.

Nitrogen Deficiency

Nitrogen deficiency is the most widespread nutritional disorder in Utah, occurring in orchards in every fruit-growing area in the state. Tree mortality resulting from the disorder is never high, because affected trees survive for many seasons even though they may show severe nitrogen-deficiency symptoms. Growth and yield, however, may be seriously reduced. All stone fruits require more nitrogen than occurs in most soils and are especially responsive to nitrogen application; however, peach is the fruit that shows the greatest effect of nitrogen deficiency and re-
sponds most promptly to its application.

**How to Recognize Nitrogen Deficiency**

Pale-green to yellowish color in leaves, a result of less chlorophyll than normal, is the one generally recognized symptom of nitrogen deficiency in any of the stone fruits. There is usually enough nitrogen present in the soil in the spring to produce normal green leaves, but when it becomes deficient the older leaves gradually give up their nitrogen to the new tissue and are the first to show the change in color. The pale-green or yellowish-green color finally becomes pronounced and generally distributed through the leaves and throughout the tree, even on the new growth (fig. 57). With more advanced stages of nitrogen deficiency in peach, the leaf blades, petioles, and veins become tinged with red and in some varieties this red coloring is pronounced. Also, in advanced stages of nitrogen deficiency, red or reddish-brown spots appear between the larger veins and become necrotic. Some leaf fall may result, beginning first with the older leaves. Where soil nitrogen is low, linear growth of twigs is short, stems remain slender, and fruit buds are numerous but only a small percentage of flowers set fruits. On trees critically short of nitrogen, there may be relatively few fruit buds. Fruits are smaller than normal and of a poor quality.

Nitrogen deficiency is fairly easily distinguished from other elemental
deficiencies and from virus diseases. The pale-green color, small leaves, and short shoot growth are general characteristics. The red spotting of leaves on severely affected peach trees is characteristic. If buds from virus-free but nitrogen-deficient trees are placed on trees growing in soils with sufficient nitrogen, the shoot growth from them will be normal.

Control

Nitrogen deficiency can be corrected by application of nitrogen in any form. The organic sources, such as barnyard manure, plant or animal residues, are slower to become available and contain much less nitrogen than inorganic sources, such as ammonium sulfate, sodium nitrate, or ammonium nitrate. If nitrogen is applied to the soil around trees in the advanced stages of deficiency during the growing season, there will be some greening of the leaves present when the nitrogen becomes available, but the recovery will be most pronounced in the new growth. For best results, nitrogen should be available to trees when growth is rapid in the spring. Organic nitrogen is usually applied in the fall and winter, but inorganic nitrogen may be applied in the spring 3 to 4 weeks before growth starts.

Zinc Deficiency

Zinc deficiency, also called "little leaf," is widely distributed in western North America, but is relatively rare in the Eastern States. Thorne and Wann reported that about 6 percent of the orchards surveyed in Utah contained trees showing zinc-deficiency symptoms. They found more zinc deficiency in Box Elder and Davis Counties than in Utah, Weber, and Salt Lake. Chandler reported that in California zinc deficiency occurred essentially on trees in sandy soils and in soils treated with large amounts of organic matter. In Utah the disorder has been found most severe on sandy and gravelly soils, around barnyards, abandoned corrals, or old stacking grounds. In general, zinc deficiency is associated with light soils, whereas iron deficiency occurs on heavy soils; therefore the two are seldom found together. Chandler reported sweet cherry to be the most susceptible of the stone fruits and listed the Japanese plum, domestica plum, peach, and apricot, ranging in descending order of sensitivity.

How to Recognize Zinc Deficiency

Insufficient zinc has a definite retarding effect on growth of fruit trees. The first noticeable effect is reduced leaf size in the tops of trees. On more severely affected trees, this reduction in leaf size in the tops, as contrasted with normal or more nearly normal foliage at the bases of trees, becomes more striking. Trees may be unequally affected, one side showing severe dwarfing and the other being nearly normal (fig. 58). Extreme deficiency may result in severe dieback and even death of trees.

Leaf symptoms are most characteristic of zinc deficiency. Affected trees generally show a progressive reduction in leaf size from normal or near
normal at the base to smaller leaves in the top, and from the base of twigs toward the growing point. On severely affected trees, leaves which develop during the first few weeks in the spring at the apex of shoots grown the preceding season are small, nearly sessile, and rigid, but not materially misshapen; hence the name “little leaf” (pl. 9, A-C). The small narrow leaves at the shoot apex are packed together in clumps, giving a rosetted appearance. Leaves on the lower branches of such severely affected trees, or on the upper branches of less severely affected trees, are larger but show chlorotic patterns and tend to have wavy or undulated edges. The chlorosis appears to begin as small, light green areas between the lateral veins. These enlarge and gradually coalesce until they involve most of the interveinal tissue and extend from near the midrib to the edge of the leaf. This extension of the chlorosis may continue until only the tissue along the larger veins remains green (fig. 59). At the end of the
summer newly formed leaves are so completely chlorotic that little green remains even along the veins. On severely affected peach, leaf fall is frequently severe and on some trees only terminal tufts of leaves remain in late summer (fig. 60).

Fruit production on zinc-deficient trees tends to be normal or nearly normal at the base, with a progressive decline in size toward the top, being especially small on the upper branches. Few fruits set on severely affected portions of trees, and those which do set are severely reduced in size, misshapen, and some are cracked and do not mature normally. Fruits of peach (fig. 61) and Japanese plum are not only smaller, but are flattened and constricted near the apex, some forming a terminal beak. Affected apricot fruits are more nearly round

Fig. 59. Italian Prune: Left twig showing small chlorotic leaves and rosetted growth characteristic of zinc deficiency; right, normal.

Fig. 60. Young peach tree showing severe defoliation with rosettes of leaves at end of branches, characteristic of zinc deficiency.
than normal and on severely affected branches may be no larger than a medium-sized sweet cherry. On mildly affected trees, reduced fruit size in the tops of trees may be a good indication of zinc deficiency. Such trees have in some cases given good response to zinc treatment.

Zinc-deficiency symptoms are sometimes confused with those of other deficiencies and with those of virus diseases. The symptoms of zinc deficiency most closely resemble those of nitrogen deficiency, but can be distinguished by the chlorotic patterns in leaves and the general difference in size of leaves at the base of trees as contrasted to those on the tops. In questionable cases, it may be necessary to treat some trees with zinc and others with nitrogen. Manganese and magnesium deficiencies are easily distinguished from zinc and nitrogen deficiencies because they do not reduce leaf size. Some comparisons of zinc-deficiency symptoms and those of western X decline on peach have been made. Symptoms of western X decline do not appear until mid- or late-summer, and are not associated with reduction in leaf size. In late summer severely defoliated trees may be somewhat confusing but usually those affected with western X decline will still bear some leaves with symptoms typical of that disease.

**Control**

Zinc deficiency can be easily cured on most stone fruits. For peaches, plums, and apricots the most satisfactory treatment is a dormant spray of zinc sulfate, 25 to 50 pounds per 100 gallons of water, depending on
the severity of symptoms. (Trees must be thoroughly dormant.) Sweet cherries do not respond to this treatment. Some success on sweet cherries has been obtained with foliage sprays of zinc oxide, 2 pounds per 100 gallons of water. The first application is made 2 weeks after leaves appear and repeated at 2-week intervals for three or four sprays, depending on the severity of symptoms. Growers having trouble with fruit sizing should determine whether zinc is a limiting factor.

**Iron Chlorosis**

Iron deficiency, commonly called iron chlorosis and lime-induced chlorosis, is found in plants growing in calcareous soils in arid regions in which iron, although it may be present, is relatively unavailable. It is common in western North America, but rare in eastern North America where rainfall is more plentiful and soils are generally acid in reaction. The surveys of Thorne and Wann showed iron chlorosis present in 23 percent of Utah orchards. It causes considerable losses in stone fruit orchards in Cache, Carbon, Emery, Utah, and Washington Counties.

Surveys show that peaches and pears are most susceptible, followed in approximate order of susceptibility by sweet cherries, plums, apricots, apples, and sour cherries.

**How to Recognize Iron Chlorosis**

The feature which most distinctively characterizes iron chlorosis is the striking deficiency in the amount of chlorophyll in leaves (pl. 10, E). Chlorosis appears first and is most evident in leaves at the tips of the terminal shoots. These terminal leaves are chlorotic when formed. Older leaves are successively less chlorotic and even in accentuated cases those at the base of shoots may be normally green. Chlorosis in the leaf blade may vary from slight yellowing between the veins at the tip of the leaf to complete loss of chlorophyll; severely affected leaves are frequently nearly white (fig. 62).

The leaf symptom most commonly seen consists of distinct green veins, which shade off to yellow or white in the interveinal areas between the veins. This amount of green may decrease until in advanced stages of chlorosis it may be scarcely perceptible (pl. 10, E). Iron chlorosis is distinguished from manganese deficiency by the fact that symptoms of iron deficiency initiate in the youngest leaves and develop progressively toward the bases of shoots, whereas in manganese deficiency the sequence is the reverse. Leaves on trees affected by iron deficiency are generally more chlorotic than those expressing other minor elemental deficiencies. Veins on iron-deficient leaves remain green until the entire blade has become yellow. Some individual leaves can be picked from trees suffering from zinc, manganese, magnesium, or iron deficiency which cannot be distinguished one from the other.

Trees which are only mildly affected, particularly which do not show symptoms until late in the season, appear to live indefinitely without serious crop reduction. On trees more severely affected, symptoms usually
develop earlier in the season. On such trees the leaves and terminals of shoots may become necrotic, and dieback may result. Trees suffering dieback usually decline and many eventually die. Fruit production is reduced in proportion to the severity of the chlorosis. If chlorosis becomes severe before the fruit is well formed the fruit will not mature normally, but if chlorosis does not develop until late in the season the crop may be nearly normal. The chief loss from iron chlorosis appears to be weakening and decline of trees. Trees weakened by iron deficiency are more subject to winter injury than normal ones.

**Control**

The cure for iron chlorosis is a complex process, and no measures entirely satisfactory for orchard use have been developed. Iron chelates have shown some promise in the cure of chlorosis in Utah. Several of these chelates have been used with considerable success in curing iron chlorosis in Florida and California, and one of these, an iron salt of diethylenetriaminepentacetic acid, DTPA-Fe, has given some control in field trials in Utah during the last two seasons. Application rates of DTPA-Fe range from 1 teaspoonful for a rose bush to $\frac{1}{2}$ to 1 pound for a full grown tree; this is scattered on the soil and watered in. The beneficial effects last for about one year. Iron chelates are expensive but with wider use the price may be reduced.

Other methods of control have been used with some success. Injection of iron salts into trunks will
green up the trees for a period of one year or more. In orchards only mildly affected, addition of sulfur in combination with barnyard manure has given some benefit. Keeping soils wet brings more alkali into solution, thereby fixing more of the iron and increasing iron chlorosis.

Iron chlorosis is not transmissible; buds from severely affected trees produce normal growth when transferred to unaffected trees. Certain viruses may so interfere with the metabolism of the available iron in trees that symptoms of iron chlorosis result.

Fig. 63. Babcock peach leaves showing marginal and interveinal chlorosis characteristic of manganese deficiency; normal leaf at right.

**Manganese Deficiency**

The disorder in stone fruit trees caused by a deficiency in manganese is distributed generally in Utah fruit-growing districts. Thorne and Wann found that of some 1,440 orchards surveyed 260 (18 percent) showed manganese-deficiency symptoms. Sweet cherry, sour cherry, and plum showed some symptoms, but apricot and peach were more severely affected. Manganese-deficiency symptoms were found in almost all the orchards in Wayne County, but only a small percent of the orchards in
Carbon, Davis, Emery, Utah, and Washington Counties showed the disorder.

How to Recognize Manganese Deficiency

Symptoms distinctive of manganese deficiency are similar on all stone fruits. Foliage on affected trees is normal during the early part of the growing season and leaves attain normal size. Symptoms develop in full-size leaves, beginning first on the oldest leaves and advancing progressively in leaves toward the growing point. The green color fades out and the leaves become chlorotic first along the margins at the apical portion. The pale green seems to fade out progressively from this margin in bands toward the midrib between the larger lateral veins (fig. 63). The result is a pattern in which the midrib and main lateral veins with bordering bands of tissue of varying width remain green, whereas the interveinal and peripheral areas of the leaf become chlorotic (pl. 10, A-D). In severe expression of the disorder, only a small portion of the leaf near the petiole may retain its normal green color. Color in affected tissue is a distinctive yellowish-green and seldom, if ever, approaches the extreme yellow or complete bleaching characteristic of iron chlorosis.

All leaves on severely affected trees may become chlorotic, while on other trees only part of the branches may be involved. Necrosis may result when deficiency is severe.

No specific symptoms occur on the twigs or fruits; however, Thorne and Wann report that in Utah the disease is so severe in certain areas that foliage has a general yellow color, growth is reduced, and in some cases trees have been killed. There are no data to show that mild symptoms of manganese deficiency reduce yield or produce any permanent damage to trees. More severe symptoms may cause reduction in size and yield of fruits in proportion to the amount of chlorosis.

Control

Manganese deficiency is easily corrected by foliage sprays of manganese sulfate in water in early summer. Concentrations of 2 to 10 pounds per 100 gallons of water, depending on the severity of symptoms, have been given complete recovery in 2 to 4 weeks.

Magnesium Deficiency

Magnesium deficiency is fairly common on stone fruits grown in alkaline soils in western North America. It is not recognized as the cause of serious losses. In Utah magnesium deficiency appears to be limited to local areas and often to spots in orchards.
of leaves at their widest part. With progressive severity, the yellowing extends toward the midrib, the tissue along the midrib being the last to become affected. Usually some leaves with a characteristic A-shaped green triangle remaining at the base of the leaf (pl. 10, F) can be found on affected trees. Ovate leaves show this green triangle more than linear leaves. When affected leaves become entirely yellowed, they usually fall. Severely defoliated trees are fairly rare in Utah.

**Control**

Magnesium deficiency symptoms can be cured by adding magnesium sulfate (epsom salts) to acid or slightly alkaline soils. For trees growing in alkaline soils, magnesium is supplied by spraying the foliage.

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**Copper Deficiency (Exanthema)**

Copper deficiency appears to be limited to certain isolated areas and soil types. Symptoms have been seen on Japanese and domestica-type plums in western North America. Frequently, copper deficiency is associated with zinc deficiency. Both occur on light sandy soils and are associated with old corral sites. Copper deficiency would probably be much more prevalent except for the wide usage of copper bordeaux as a fungicide.

**How to Recognize Copper Deficiency**

The initial stages of copper deficiency are expressed by veinal chlorosis, cessation of terminal growth, rosetting of leaves, and multiple-bud formation. Since these symptoms are somewhat common to other elemental deficiencies, trees with mild to moderate amounts of copper deficiency probably go undiagnosed. The later stages of copper deficiency are characterized by terminal dieback (pl. 9, D) and shoot growth of lateral buds below the dead terminal. In this stage, the injury resembles somewhat that caused by excess boron, but the corky areas do not appear on the leaf petioles. Gumming is common on the stems of some hosts below the killed areas.

**Control**

Copper deficiency is easily controlled by the application of a copper bordeaux spray to the young leaves. Peach foliage is sensitive to copper; therefore for peaches copper sulfate may be added to the soil.

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**Sodium Injury**

Although sodium is commonly present in many soils of western North America as one of the components of common salt, it is not recognized as an element essential to plant growth. In fact, sodium is known to be toxic to fruit trees if it reaches high enough concentrations. Sodium in concentrations below the toxic level may cause reduced growth.
but no other symptoms. Above this level, trees become strikingly affected and often die. Sodium salts are common to soils in Utah, especially those in low-lying areas with poor drainage.

**How to Recognize Sodium Injury**

Sodium injury is characterized by abrupt and striking marginal leaf scorch. Apricot (pl. 11, C) is particularly susceptible, and peach (pl. 11, D) is commonly affected. The scorch may initiate at the tip of the leaf and progressively involve the remainder. The type of scorch is not singularly diagnostic. Scorched leaves commonly show 0.5 percent of the dry matter as sodium, and may occur on soil with 250 parts per million of total soluble salt. Sodium injury is often brought on by conditions which cause fluctuations in the soil water table, bringing sodium from below up into the root zone. In some cases, symptoms have been produced by application of sodium as sodium nitrate.

**Control**

Trees showing sodium injury have already been damaged, but if symptoms are not too severe, they can be benefited by leaching with non-saline water. Addition of gypsum, sulfur, and like materials, followed by periodic leaching, depending on soil conditions, may be beneficial. Fertilizers containing sodium, such as sodium nitrate, should not be used on alkaline soils.

**Arsenic Injury**

Arsenic injury on peach and apricot was formerly of common occurrence in Utah. Trees were injured when acid lead arsenate used for controlling codling moth on apples drifted onto peaches planted near the apples, or indirectly when peaches planted on old apple land absorbed arsenic which had accumulated in the soil. With the advent of new insecticides, lead arsenate has almost disappeared as a fruit tree pesticide. Occasional cases of arsenic injury still occur where peaches are planted on old apple orchard sites.

**How to Recognize Arsenic Injury**

The first indication of arsenic injury is the appearance of brown to reddish-brown spots along the leaf margins on the oldest or basal leaves, occurring about midsummer. The terminal leaves usually remain normal. Spots also develop between the veins on leaves. The brown spots rapidly become necrotic and drop out, producing a shot-hole effect (pl. 11, E). On severely affected trees the leaves drop, sometimes leaving the trees almost bare; fruits remain on such trees but are small, lack flavor, and ripen prematurely (pl. 11, F).

**Control**

There is no practical way to remove arsenic from the soil. If young trees are planted in large holes filled with arsenic-free soil and given ample nitrogen they soon put out roots below the contaminated surface soil and outgrow the injury.
Fig. 64. Fluorine injury on Chinese apricot; leaves showing sharply delineated marginal necrosis. The necrotic tissue often breaks away and falls, leaving an irregularly-shaped leaf.
Fluorine occurs in a combined state in all forms of plant and animal life. Likewise, it also is present universally in the rocks and in the various soils making up the earth's crust. Even water often contains small quantities of fluorine. In most processes of heating or acidulating fluorine-containing materials, fluorine in some form is liberated either into the water or into the atmosphere. The burning of coal, a residue of plant life, is a common source of this most active element.

Under certain conditions, especially in metropolitan or urban industrialized areas, compounds of fluorine may accumulate in the atmosphere in such quantities as to injure fluorine-sensitive vegetation. Such atmospheric fluorine may be absorbed by leaves and stored within them. So far as known, these fluorine compounds are not moved or conducted from the leaf through the petiole to the stems, roots, flowers, or fruits; instead, the trapped fluorine in certain sensitive plants appears to move outward with the transpiration stream toward the leaf periphery where it may reach lethal levels, killing tissues at the margin. Here at the leaf margin the necrotic or dead tissue takes on structural or color patterns characteristic of the species of plants affected. On stone fruits in Utah, measurable amounts of leaf necrosis have been reported only on certain varieties of apricots and on Italian Prune.

How to Recognize Fluorine Injury

Fluorine injury in stone fruits, especially on apricot and Italian Prune, primarily involves the leaf margin. In mild expressions of the disorder, small light-colored necrotic areas appear along the leaf margin. These necrotic areas may occur at any one point in the periphery of the leaf or they may appear at the same time at several points along the margin. With certain concentrations and under cultural conditions favoring injury, the entire leaf margin may be necrotic. The bands of dead or necrotic tissue thus formed may vary greatly in width and in severe cases local necrotic areas may develop between the veins. At successive intervals, the necrotic areas once initiated may increase in size, often producing a definite zonal effect (fig. 64).

Necrotic or killed peripheral por-
tions of the leaf are sharply delimited from the green tissue without any visible gradation. This necrotic tissue frequently separates from the green portion of the leaf, which remains attached to the tree where it continues to function apparently unimpaired.

Both in apricot and Italian Prune (fig. 65) the marginal necrosis may appear on only occasional leaves scattered through the tree, or it may be restricted to leaves on a few branches or even to a single branch; or it may be generally distributed, involving a high percentage of the mature leaves on the tree. Factors responsible for such erratic distribution in the tree are not well understood. It is known, however, that fluorine-type necrosis can result from other causes such as wind, heat, drought, and injury to trunks or roots, making diagnosis extremely difficult. Reliable diagnosis cannot be made safely from detached injured leaves or from a single isolated tree in an orchard.

Control

Control of leaf necrosis induced by fluorine in stone fruits involves two things: Better cultural practices in orchards and effective reduction of fluorine in the air. Repeated observations have shown that both apricot and Italian Prune trees show far less necrosis under good cultural conditions than in neglected, run-down, and poorly irrigated orchards. Especially is this true where trees suffer severely from lack of water. In certain areas where fluorine results from a particular process, large sums of money are being spent in establishing control operations. The problem can be and is being solved in a number of such areas.

Sulfur Dioxide Injury

Injury to vegetation by sulfur dioxide gas (SO\textsubscript{2}) emitted from metal smelters and industrial plants (smelter injury) has been known for many years and now is generally controlled by removal of the SO\textsubscript{2} from the flue gases at the point of origin. Occasionally, however, injury does occur where removal equipment becomes faulty or for other reasons SO\textsubscript{2} is liberated. The existence of extensive smelting and refining industries in Utah has resulted in much SO\textsubscript{2} injury to crop plants and in the development of research laboratories where much of the fundamental work on effect and control has been done.

Agricultural plants differ in sensitivity to SO\textsubscript{2} injury. Alfalfa, roses, and certain forest trees, including ash, spruce, and other conifers, are the most sensitive. Cherry, apple, and peach are regarded as intermediate in sensitivity. Apricot is especially tolerant to SO\textsubscript{2}.

How to Recognize Sulfur Dioxide Injury

Diagnosis of sulfur dioxide injury is not simple, but injury patterns are so characteristic that they can usually be recognized. On fruit trees, the initial symptoms are yellowish-brown to reddish-brown killed areas in the interveinal areas of leaves, with part
of the tissue around the margins and along the main veins remaining green. Because of this localized tissue killing, affected leaves develop artistic and bizarre color patterns (fig. 66). The amount of red color bordering the killed areas varies with species and is more prevalent on species with red fruits. Often the dead tissues drop out, leaving a shot-holed or tattered condition. Where SO₂ injury is severe, all of the leaves may turn brown and trees may be killed.

If SO₂ injury on stone fruits is suspected, other plants in the vicinity should be examined. Alfalfa and many other plants are more sensitive; on alfalfa, affected leaves are bleached white, beginning at the margin, leaving an irregular green area along the midrib. Sugar beets develop intercostal reddish-brown discoloration and potatoes may show reddish-purple tints. These colorations are not in themselves proof of SO₂ injury, because similar effects, at least on some plants, may result from drought, frost, or other unfavorable environmental conditions.

**Control**

The control of sulfur dioxide injury (smelter injury) is entirely a matter of prevention by removal of the SO₂ from flue gases. Most of the large smelters, the chief emitters of SO₂, have installed equipment for prevention of SO₂ emission into the air.

**2,4-D Injury**

The use of 2,4-D for killing broadleaf weeds in and around stone fruit orchards has caused serious losses, particularly on peach. The injury symptoms on peach fruits strikingly resemble the symptoms of red suture, a virus disease affecting peach in Michigan and Maryland. The increasing use of 2,4-D for killing bindweed in orchards and weeds in fence rows and grain fields adjoining peach orchards in Utah is sure to result in drift of the 2,4-D to peach trees with resulting injury. Peaches sprayed with equipment which was previously used for application of 2,4-D may suffer injury unless the equipment is properly and thoroughly cleaned between uses. It seems worthwhile to include a brief description of the injury.

**How to Recognize 2,4-D Injury**

Unlike many other broadleaf plants, stone fruits are somewhat tolerant to 2,4-D. Direct application of 10 to 20 ppm causes little malformation of
Fig. 67. Halehaven peach fruits showing swollen and prematurely-ripened sutures typical of effects of 2,4-D.

the leaves of peach, apricot, and prune. Leaves of peach become somewhat thickened and roughened, and the edges roll downward. Apricot leaves are more misshapen, losing their petiolar sinus and developing typical crowded cleared veins with accentuated dentations.

Fruits, however, are more strikingly affected. Affected peach fruits ripen prematurely along the suture, and the suture becomes swollen from increase in size of the cells (fig. 67). Swelling of the suture is often accompanied by cracking. Premature ripening of the suture influences formation of abscission layers and causes fruit drop before the remainder of the fruit is ripe enough to harvest. Apricot fruits are slightly larger than normal in size, ripen prematurely, and often are severely cracked.

If 2,4-D injury is suspected on fruit trees, examination of more sensitive plants in the orchard will furnish evidence that it has been there. Grapes, boxelder, tomatoes, and cotton are very sensitive and serve as good indicator plants. Injury from 2,4-D sometimes occurs in locations far from where the material was applied, especially when volatile forms are used. Cotton has been injured 6 miles from where grain fields were treated. Peaches located in a canyon where wind currents came directly from treated grain fields several miles distant were injured.

Control

Control of 2,4-D injury is a matter of prevention. Serious injury to peach has occurred when 2,4-D has drifted
on the trees from adjoining areas where the material was applied as a weed killer on other crops. Injury has resulted on peaches several miles from the point of application when the material was applied from airplanes.
Selected References


Richards, B. L. The stone fruit virus disease situation in Utah and what we are doing about it. Utah State Hort. Soc. Proc. 1947: 34-38.


Glossary

ACQUISITION PERIOD—The period during which a vector is exposed to a virus source.

ACUTE SYMPTOMS—The stage when symptoms are most accentuated.

ASTEROID—Starlike.

BUD-PERPETUATED—A condition present in all the tissue resulting from the growth of a bud.

CANKER—A lesion in the bark of a tree caused by decay.

CHLOROPLAST—A small body within a plant cell bearing green color.

CHLOROSIS—A loss or lack of the normal green color.

CHRONIC STAGE—The stage when symptoms have become stabilized and remain consistent; opposed to acute.

CLONE—A progeny derived from a single individual by vegetative propagation.

COLOR BREAKING—A streaking or variation of the normal color found in the flower petals of some virus-infected plants.

COMPLEX—Two or more viruses or virus strains which together cause characteristic disease in a host, the symptoms of which are different from those produced by the individual components.

DECLINE—Reduced vigor, severe stages of which may result in death of plant.

DIAGNOSIS—The recognition of a disease from its symptoms.

DIAGNOSTIC HOST—A plant on which a virus produces the characteristic symptoms by which the disease it causes is recognized.
DIFFERENTIAL HOST—A plant that produced symptoms distinctive for a specific virus, sufficiently distinctive to differentiate it from other viruses.

DISEASE—A condition of a plant in which it is altered from the normal.

DISORDER—A synonym of disease, used most commonly for noninfectious types of disease.

ENATION—Abnormal outgrowth which varies from a gland-like protuberance to a leaf-like structure; most commonly found on leaf surfaces.

EXCRESCEENCE—An abnormal outgrowth such as a wart.

EXPERIMENTAL—An operation undertaken to investigate an unknown principle or effect.

FORM—A virus which incites reproducible symptoms distinguishable from a type but sufficiently similar to show a relationship. The symptoms of forms usually fit into a gradient which merges with those of the type; new isolates from nature cannot be specifically identified with those obtained previously.

GALL—A swelling of plant tissue, incited by a virus or other agent.

GENETIC DISORDER—A heritable disorder, one which passes through seeds or arises locally in tissue by mutation within the cells of growing points. Such disorders are perpetuated in growth from affected tissue but are not transmitted to plants on which affected tissue is budded or grafted.

HOST—A plant susceptible to infection.

IMMUNE PLANT—A plant which cannot be infected.

INCUBATION PERIOD—The period between infection and appearance of characteristic symptoms.

INDEXING—Determination of the virus or viruses present in a suspect plant by inoculation from it to an indicator host, by chemical or other means.

INDEX HOST—A plant used for indexing.

INDICATOR HOST—A plant that will manifest symptoms for viruses that are latent in other hosts.

INFECTION—The process in which the virus becomes established in the susceptible host and begins to multiply and produce symptoms.

INFECTIOUS—Capable of being transmitted by inoculation.

INFECTIVE—Able to carry a pathogenic virus to a susceptible plant resulting in infection.

INFECTIVE PERIOD (applied to vectors)—The period between acquisition and the time when the vector becomes infective.

INFEST—To become established for feeding purposes.

INOCULATE—The act of placing inoculum in a plant in a manner so that infection may result.

INOCULUM—Infective material.

INSIDIOUS (disease)—One which awaits the opportunity to become established and cause damage.

ISOLATE—A virus entity.

LACE LEAF—A leaf condition in which the interveinal tissue has fallen out, giving an appearance of lace work.

LATENT—Refers to a virus infection in a plant without symptom expression.

LATENT PERIOD—The period between infection and the appearance of symptoms; in vectors this term is used synonymously with infective period.

LINE PATTERN—A type of mosaic mottling in which the chlorotic areas of leaves are surrounded by irregular, narrow, more striking chlorotic bands.

MASKED—Refers to symptoms which are conditionally prevented from expression.

MIXTURE—Two or more viruses occurring together in a host plant, each causing its own characteristic symptoms.

MOSAIC—Irregular mottled patterns of chlorotic and greener tissue.

MOTTLED—Irregular spots and blotches of different colors.

MUTATION—A sudden change, differing from the parent, which becomes characteristic in subsequent growth.

NATURAL SPREAD—Spread which takes place by natural agents without the aid of man.

NECROSIS—Death and destruction of cells or tissue.

NONTOLERANT—Varieties or species which are seriously damaged.
OAK-LEAF PATTERN—A type of mosaic mottling in which the chlorotic area is centrally located on a leaf, with the general outline of an oak leaf.

PARASITE—A virus or other agent which lives on another organism to the detriment of that organism.

PARENCHYMA—The thin-walled cells of plants making up the bulk of fruits, growing points, and leaves, and also found widely distributed in other plant parts.

PERPETUATION—Characters which continue to be associated with new growth from tissues; contrasted with transmission.

PERSISTENT VIRUSES—Viruses which, once acquired by a vector, are retained for more than one day.

PROLIFERATION—Production of new abnormal tissue.

PURE CULTURE (virus)—A virus culture freed from all other viruses.

PURIFIED VIRUS—A virus freed from all plant and extraneous matter.

RASP LEAF—Leaves bearing abnormal leaf edge-like outgrowths from the surface, resembling the surface of a wood rasp.

RESISTANT—Less reactive either to infection or to symptom production after infection.

RING SPOT—Leaves bearing chlorotic or necrotic areas in the form of various sized spots or in rings surrounding islands of normal tissue. When islands of tissue become surrounded by necrotic areas they die and often drop out, forming a tattered and lace-leaf effect.

ROGUING—Removal of diseased plants from among unaffected ones.

ROOTSTOCK—The portion of a plant, composing the root system, usually grown from seeds or cuttings and differing from the variety top of horticultural trees produced by top-working the variety on the rootstock.

RUGOSE—Wrinkled; the veins sunken and the interveinal tissue of leaves raised and distorted.

SCION—A piece of a shoot bearing buds used in graft propagation.

SHOCK—The stage of a disease in which symptom expression is most severe; often in virus diseases immediately following the onset of symptoms.

SHOT HOLE—A condition of leaves resulting from dropping out of necrotic areas.

SPECIES—A botanical unit of classification including plants of a single kind, which when grown from seed are true to type.

STERILITY—Unfruitful.

STRAIN—A virus which incites reproducible symptoms distinguishable from a type but sufficiently similar to show a relationship, yet sufficiently constant to enable the entity to be recognized whenever encountered.

SYMPTOM—Any character representing a change from the normal which develops in roots, systemic in the woody cylinder, causal agent.

SYSTEMIC—Throughout the system; often used in a restricted sense, e.g., systemic in roots, systemic in the woody cylinder, etc.

TEST FEEDING PERIOD—That period in which viruliferous insects or mites are exposed to the test plant.

TEST PLANT—A plant used to indicate the presence of a virus, either from transmission by vectors, tissue grafts, or other means.

TOLERANT VARIETY—A variety which tolerates a virus infection without development of severe damaging symptoms.

TRANSMISSION—The transfer of an infectious agent from a diseased plant to a previously healthy plant, where the agent regenerates and causes disease. For viruses this is done in nature by vectors and experimentally through tissue grafts, infected juice transfer, and by other means.

VECTOR—An agent of dissemination. For viruses the natural vectors are mostly insects and mites.

VIRULENT—Having capability to produce severe or violent symptoms.

VIRULIFEROUS—Virus-bearing.

VIRUS—An extremely small infectious agent, which lives and reproduces in other living organisms.

VIRUS RESERVOIR—A host plant which maintains a virus and from which spread may take place to other plants.

YELLOWS—A general, uniform development of yellow color in plants.
Common and Scientific Names of Plants

Almond (*Prunus amygdalus* Batsch; syn. *P. communis* (L.) Arcang.)

Apple (*Malus sylvestris* Mill.)

Apricot (*Prunus armeniaca* L.)

Bushcherry, Chinese (*P. japonica* Thunb.)

Cherry, almond (*Prunus glandulosa* Thunb.)

Cherry, Bessey (*P. besseyi* Bailey)

Cherry, bitter (*P. emarginata* [Dougl.] Walp.)

Cherry, bitter (var. mollis) (*P. emarginata* var. *mollis* [Dougl.] Brewer & Wats.)

Cherry, black (*P. serotina* Ehrh.)

Cherry, duke (*P. avium* × *P. cerasus*)

Cherry, Hansen bush (*P. besseyi* Bailey)

Cherry, mahaleb (*P. mahaleb* L.)

Cherry, Manchu (*P. tomentosa* Thunb.)

Cherry, mazzard (*P. avium* L.)

Cherry, oriental flowering (*P. serrulata* Lindl.)

Cherry, pin (*P. pennsylvanica* L.)

Cherry, sand (*P. pumila* L.)

Cherry, sour (*P. cerasus* L.)

Cherry, sweet (*P. avium* L.)

Chokecherry, common, or eastern (*P. virginiana* L.)

Chokecherry, western (*P. virginiana* var. *demissa* [Nutt.] Torr.; syn. *P. demissa* [Nutt.])

Mazzard (*P. avium* L.)

Nectarine (*P. persica* var. *nectarina* Ait.) Maxim.; syn. *P. persica* var. *nucipersica* [Borkh.] Schneid.)

Peach (*Prunus persica* [L.] Batsch)

Peach, desert (*P. andersonii* Gray)

Peach, purple-leaved (*P. persica* var. *atropurpurea* Schneid.)

Peach, smoothpit (*P. mira* Koehne)

Pear (*Pyrus communis* L.)

Plum, American (*Prunus americana* Marsh.)

Plum, apricot (*P. simonii* Carr.)

Plum, Bokar (*P. bokhariensis* Schneid.)

Plum, beach (*P. maritima* Marsh.)

Plum, chickasaw (*P. angustifolia* Marsh.)

Plum, damson type, or bullace (*P. instititia* L.)

Plum, domestica, or garden (*P. domestica* L.)

Plum, flatwoods (*P. umbellata* Ell.)

Plum, hog (*P. reverchonii* Sarg.)

Plum, hortulan (*P. hortulana* Bailey)

Plum, Indian (*Osmaronia cerasiformis* [Torr. & Gray] Greene)

Plum, Japanese (*Prunus salicina* Lindl.)

Plum, klamath (*P. subcordata* Benth.)

Plum, Mexican (*P. mexicana* S. Wats.)

Plum, myrobalan (*P. cerasifera* Ehrh.)

Plum, pottawattomie, or wildgoose (*P. munsoniana* Wight & Hedr.)

Plum, purple, or ornamental (*P. cerasifera* var. *atropurpurea* Jaeger; syn. *P. cerasifera* var. *pisardi* [Carr.] Bailey)

Plum, wildgoose (*P. munsoniana* Wight & Hedr.)

Prune (*P. domestica* L.)
PLATE 1. Western X decline of peach: A, Elberta leaves showing early-season yellowing, necrosis, and separation of necrotic tissue from the more normal tissue; B, Orange Cling branch in advanced stage of disease, showing mid- to late-season yellow and red color and tattered condition of leaves (in hot dry seasons such features dominate the symptom pattern in Elberta peaches in Utah); C, Salwey tree showing affected branches and shoots interspersed with normal ones.
PLATE 2. A and B, Western X decline of peach: A, Comparable normal and affected Elberta fruits; B, Elberta tree in advanced stage of disease, having only one unaffected branch. C and D, Western X red leaf of chokecherry: C, Normal fruits at left compared with affected fruits at right, showing smaller size, varying color, and conical shape (the diseased fruits are from plants inoculated with the western X virus from cherry trees showing western X little cherry); D, affected chokecherries (right) showing reddish-green color and rosetting characteristic of second-year symptoms of the disease; normal at left.
PLATE 3. Peach mosaic: A, Florence peach flower showing broken color pattern; B and C, J. H. Hale peach, B affected fruits, showing reduced size, bumpy surface, and uneven color as compared with normal at lower right, and C leaves showing a gradient of mosaic mottling (veinlet clearing, right; brilliant mottle, center; and marginal striping, left); D, partially affected J. H. Hale peach tree, showing retardation on affected side.
PLATE 4. A, Bing cherry top-worked separately on arms of mahaleb cherry understock 2 to 3 inches above the crotch; outer branches showing wilt were subsequently inoculated with western X virus. B, Normal Montmorency cherries in comparison with western-X-little-cherry-affected fruits. C, Thirty-year-old Bing trees on mahaleb rootstock, left and right naturally affected with western X virus, showing wilt and decline (photographed 1947). D, Two young Napoleon cherry trees inoculated the previous growing season with western X virus, showing wilt and decline; normal uninoculated tree at right.
PLATE 5. A, Western-X-little-cherry-affected Napoleon fruits showing pale color, dull surface, conical shape, and decreased size, as compared with normal fruits from healthy limb on same tree, Utah; B, similar normal and affected Napoleon fruits from Washington; C, normal and affected Lambert fruits from Washington; D, Montmorency cherry tree showing advanced state of decline induced by western X virus; E, 5-year-old Bing tree on mahaleb rootstock third season after inoculation with western X virus, showing stunting, excess fruiting, yellowing, and reduced size of leaves; normal Bing tree of same age at right.
PLATE 6. A-C, Sour cherry yellows on Montmorency cherry: A, Leaf showing irregular pale-green or yellowish mottling; B, leaf showing more complete yellowing with the retention of green along the veins; C, leaf and fruit buds that have grown into shoots, producing willowy type of growth and leaving few buds for fruit production. D, Comparable normal fruiting branch.
PLATE 7. Top: Bing leaf and fruits affected with sweet cherry deep suture in contrast with one normal fruit at left. B, Italian Prune leaves arranged to show a gradient of Italian Prune leaf spot and mottle symptoms in contrast with normal leaf (upper left).
PLATE 8. Italian Prune leaves showing only the necrotic type of leaf spot: A, Leaves from trees at Emmett, Idaho; B, from Bountiful, Utah.
PLATE 9. A - C, Plant parts showing symptoms of zinc deficiency, in contrast with healthy ones at right: A, Peach showing chlorosis, upward rolling of leaves, and some defoliation and rosetting of leaves at tip of branch; B, apricot showing chlorosis, defoliation, cupping, and reduced leaf size; C, sweet cherry showing small rigid, wavy-margined, chlorotic leaves. Cherry fruit is smaller than normal, more pointed, and lighter in color. D, Prune shoots showing symptoms of copper deficiency.
PLATE 10. A, Peach leaves showing a gradient of manganese-deficiency symptoms; B, sour cherry leaf showing manganese-deficiency symptoms, Michigan; C and D, sour cherry leaves C from a manganese-deficient branch and D from a branch cured by injection of manganese sulfate, New York; E, prune leaves showing symptoms of iron deficiency, in contrast with normal leaves; F, peach leaves showing symptoms of magnesium deficiency.
PLATE 11. C and D, Leaves showing symptoms of sodium injury; C Apricot; D, peach. E and F, Peach leaves and shoots showing symptoms of arsenic injury.